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RISK ASSESSMENT WORK PLAN FOR KENO HILLS, YUKON

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September 2008

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1.0 INTRODUCTION

This document provides the work plan for the preliminary quantitative risk assessment that will be carried out for the Keno Hills Mine Site mine sites. The document outlines the procedure for the selection of constituents of potential concern (COPC), describes the ecological and human receptors that will be considered, indicates pathways of exposure that will be considered, discusses toxicity reference values (TRVs) that will be used in the assessment and provides a proposed Table of Contents. While this work plan provides the essential and fundamental guidance for the HHERA, the final aspects of the risk assessment (e.g., COPC, locations, etc.) will be determined when the results of the 2008 sampling program have been reviewed.

2.0 SELECTION OF CONSTITUENTS OF POTENTIAL CONCERN

The approach to identify constituents of potential concern (COPC) at the Keno Hills mine site is similar to approaches used at the Anvil Mine Range in the Yukon and other mine sites in the Northwest Territories. As metals are generally the COPC at mine sites, the largest amount of data is collected on metals levels in water, soils, and other media. Total petroleum hydrocarbons (PHCs) and polychlorinated biphenols (PCBs) are also present at some sites and if the data are available will be assessed to identify whether there are any organic COPC.

Metals that will be considered for this assessment include: aluminum (Al), antimony (Sb), arsenic (As), barium (Ba), beryllium (Be), bismuth (Bi), boron (B), cadmium (Cd), calcium (Ca), chromium (Cr), cobalt (Co), copper (Cu), iron (Fe), lead (Pb), lithium (Li), magnesium (Mg), manganese (Mn), mercury (Hg), molybdenum (Mo), nickel (Ni), phosphorus (P), potassium (K), selenium (Se), silicon (Si), silver (Ag), sodium (Na), strontium (Sr), thallium (Tl), thorium (Th), tellurium (Te), tin (Sn), titanium (Ti), tungsten (W), uranium (U), vanadium (V), zirconium (Zr) and zinc (Zn).

2.1 SELECTION PROCESS

The process that will be applied to identify constituents of potential concern in soil and water is presented in Figure 2.1-1. A slightly different screening procedure will be used to select constituents based on measured concentrations in sediment and vegetation samples; this is discussed in more detail in a later section.

In general, the COPC identification process for surface water and soil involves four steps:

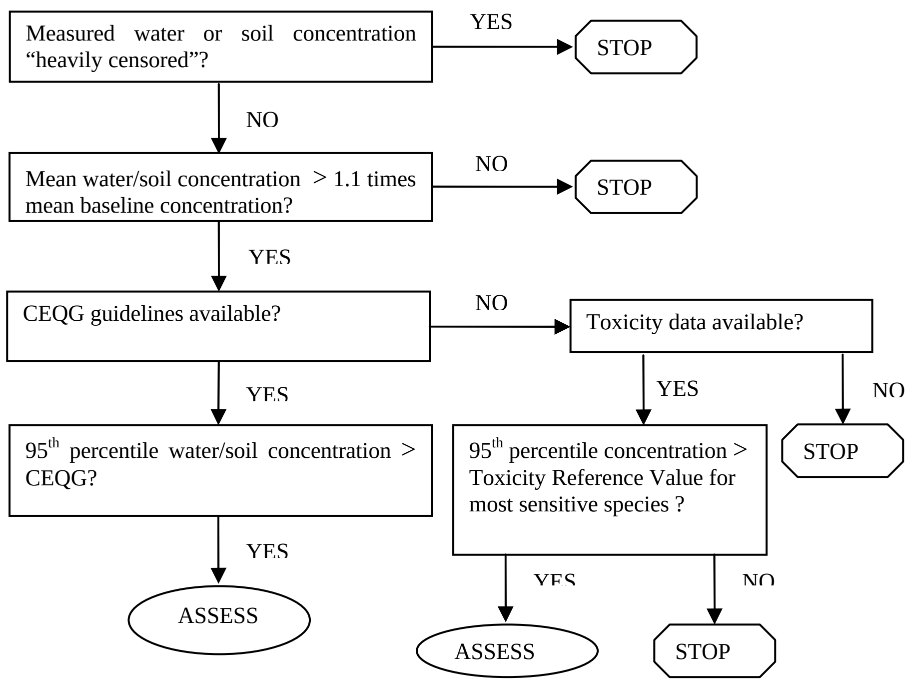
Step 1 – If more than 90% of measured concentrations on samples from an affected area are reported as below MDL, the data will be considered as heavily censored and will not be considered further. If sufficient data are available to calculate summary statistics on constituent concentrations on samples from affected and/or reference areas, measurements reported as below the laboratory method detection limit (MDL) will be set equal to one-half of the MDL.

Step 2 – Concentrations in the affected areas will be compared to concentrations obtained from reference sites. If the mean concentration from an affected area is higher than the reference mean, then a statistical test will be used to determine if the difference is statistically significant at a 0.10 level. Only constituents with concentrations in an affected area that are significantly higher than reference concentrations will undergo further assessment.

Step 3 – Constituents identified to have higher concentrations in affected areas than those at reference sites (i.e. from Step 2) will then be compared to the appropriate screening criteria (e.g., the CCME guidelines for protection of aquatic life in fresh water and soil (residential/parkland land use)). For constituents with at least 20 site measurements, the 95th percentile will be compared to the screening criteria. For constituents with less than 20 site measurements, the maximum will be used instead. Constituents with concentrations lower than the screening criteria will be dropped from further assessment.

Step 4 – Constituents identified in Step 3 will then be checked to see if corresponding toxicity data are available. Constituents without appropriate toxicity data would not be further assessed and will be addressed qualitatively in the assessment.

FIGURE 2.1-1
SELECTION PROCEDURE FOR CONSTITUENTS OF POTENTIAL CONCERN



Notes:
"heavily censored" – >90% of measurements used to calculate the mean were below the method detection limit
CEQG – Canadian Environmental Quality Guidelines for protection of freshwater aquatic life (water) and for residential/parkland use (soil) (CCME 2003).

2.2 SEDIMENT AND VEGETATION SCREENING

The screening process for sediment and vegetation samples will be carried out only to identify additional constituents that are possibly not selected in the general screening process described above. A slightly different methodology will be used in this case. Samples will still compared to background; however only constituents for which sediment quality criteria or phytotoxicty data are available will be considered further to select additional COPC. Phytotoxicty levels that will be used in the assessment are presented in Table 2.2-1 below. Sediment quality guidelines are presented in Section 4.0. If no sediment quality guidelines or phytotoxicty levels are available, then these constituents will not be considered further.

TABLE 2.2-1
PHYTOTOXIC LEVELS IN PLANTS

Constituent	Units	Phytotoxicty Level
Al	µg/g dry	50
As	µg/g dry	3
Ba	µg/g dry	400
Cd	µg/g dry	5
Co	µg/g dry	3
Cr	µg/g dry	5
Hg	µg/g dry	1
Mo	µg/g dry	10
Pb	µg/g dry	20
Sb	µg/g dry	150
Se	µg/g dry	5
Tl	µg/g dry	11
V	µg/g dry	5
Zn	µg/g dry	100

Overall List of COPC Considered

An overall summary table of the COPC for the Keno Hills Mine site will be provided as shown in Table 2.2-2. In summary, surface water and soil samples will be used to identify the main list of COPC and sediment and vegetation samples will be used to infill the list if necessary. Therefore from a long list of approximately 40 constituents, the list will be narrowed down to focus the risk assessment.

Based on our knowledge of the available data, there seems to be a lack of soil quality data for the area. Thus the risk assessment may only screen for contaminants of potential concern in the aquatic environment (i.e. water and sediments). It is anticipated that COPC may comprise the following list: antimony, arsenic, cadmium, copper, chromium, lead, manganese, zinc and petroleum hydrocarbons.

TABLE 2.2-2
EXAMPLE TABLE OF COPC SELECTED FOR MINE SITES

	COPC for the various environmental media?				**Mine Site**
Constituent	**Water**	**Soil**	**Sediment**	**Vegetation**	**List of COPC**
Ag	Y		Y		**Ag**
Al	Y				**Al**
As	Y	Y	Y	Y	**As**
B					
Ba					
Be					
Bi					
Ca					
Cd					
Co					
Cr	Y		Y		**Cr**
Cu	Y	Y	Y		**Cu**
Fe					
Hg					
K					
La					
Li					
Mg					
Mn	Y		Y		**Mn**
Mo					
Na					
Ni					
P					
Pb					
S					
Sb			Y		**Sb**
Se					
Si					
Sn					
Sr					
Te					
Th					
Ti					
Tl					
U					
V					
W					
Zn		Y	Y		**Zn**
Zr					

3.0 RECEPTOR CHARACTERIZATION

One of the key considerations, which defines the scope of a risk assessment, is the selection of ecological and human receptors. In selecting receptors it is important to identify plants, animals and people that are likely to be most exposed to COPC at the Keno Hills mine site as well as those that may be important for other ecological or social reasons. This section details the ecological (aquatic and terrestrial) species and people that will be selected for the assessment and the rationale behind their selection. Based on the available information, the initial risk assessment may only focus on the aquatic environment since there may not be available data to evaluate the terrestrial environment.

3.1 ECOLOGICAL RECEPTORS

The first step in an ecological risk assessment is the determination of which ecological species should be examined. It is not necessary to evaluate all species; ecological receptors are generally chosen to capture various types and magnitudes of exposure due to their different behavioural and dietary characteristics. They are also selected if they are considered important: (1) in the functioning of the ecosystem; (2) in the production of food for subsistence; or, (3) due to their cultural, legal or medicinal significance. In these assessments, exposure is primarily due to aquatic pathways, thus several ecological receptors will be selected to capture exposure from drinking water and consumption of aquatic plants, fish, invertebrates and sediments. Secondary exposure pathways also affected by the Keno Hills Mine site will be similarly included, and thus selected wildlife species that receive most of their exposure via atmospheric and terrestrial pathways will be also considered.

Ecological receptor characteristics will be assumed to represent a reasonable maximum exposure scenario, in that cautious assumptions will be made regarding the receptor's behaviour and home range. Ecological receptors will be assumed to spend considerable amounts of time near the areas affected by the mines, when in reality it is unlikely that wildlife would spend much time in those areas due to the lack of suitable habitat or sufficient food.

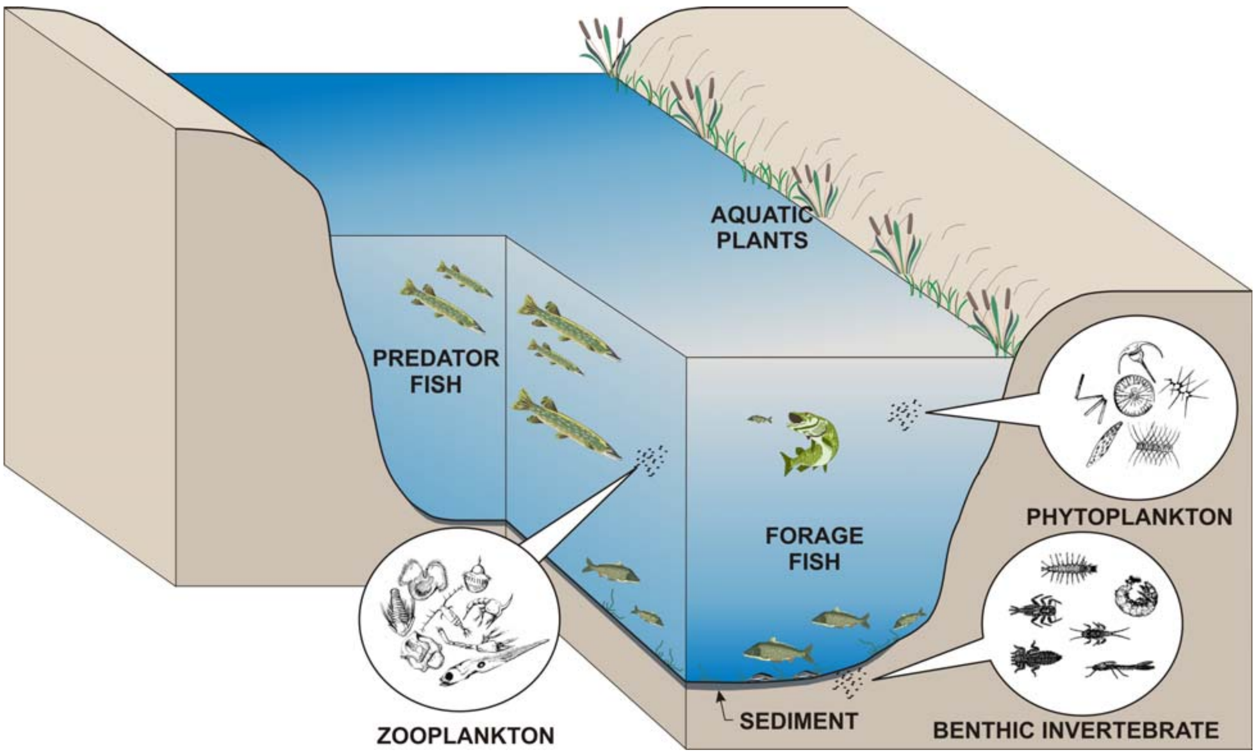
3.1.1 Aquatic Receptors

The aquatic species chosen for these assessments cover all food chain (trophic) levels in lake systems. Figure 3.1-1 portrays the species considered in the assessment. They include:

Primary Producers - Primary producers occupy the lowest level in the food chain. These organisms are generally plants that use the sun and inorganic molecules to produce food.

Aquatic plants in most lake ecosystems usually constitute the majority of the primary producer biomass. Aquatic plants are often consumed by moose, muskrat and other animals, thereby forming a link between aquatic and terrestrial ecosystems. Besides being an important food resource, aquatic plants also provide habitat to aquatic organisms.

FIGURE 3.1-1
AQUATIC RECEPTORS INCLUDED IN THE ASSESSMENT



Phytoplankton are also part of the first level in the aquatic food chain. Members of the division *Chlorophyta* have been studied extensively and are relatively common in most northern aquatic ecosystems. Even though the overall contribution of *Chlorophyta* to northern aquatic ecosystems is relatively small, they are a primary food resource for grazing zooplankton.

Primary Consumers - Primary consumers occupy the second level in the food chain. These organisms generally eat plant material such as phytoplankton. Zooplankton such as *Cladocerans* are found in most northern aquatic ecosystems. Although *Cladocerans* may be seasonally quite abundant, their overall contribution to northern aquatic ecosystems is relatively small.

Benthic invertebrates both live and feed within sediments and provide a link between aquatic and terrestrial ecosystems. For example, *Chironomidae* (midge) larvae are usually the most abundant benthic invertebrate taxa present in aquatic ecosystems in the northern climate. Many species feed on decaying organic matter and thereby form an important link between the decomposer and primary consumer levels. Furthermore, midge larvae are a main food source for small/juvenile fish and larger omnivorous fish. The adults are capable of flight and are frequently consumed by birds and bats. This life stage provides an important link between aquatic and terrestrial ecosystems in the region.

Secondary Consumers - Ecological receptors at the secondary consumer level include forage fish that feed primarily on benthic invertebrates and smaller individuals, and are an important food source for larger predatory fishes. Examples of forage fish are slimy sculpin.

Tertiary Consumers - Tertiary consumers are found at the top end of the aquatic food chain and consist of larger predatory fish species that consume other fish species. Examples include arctic grayling. Predatory fish are also an important component of the human food chain. Both forage and predatory fish are an important component of the diet of omnivores (e.g., merganser) and carnivores (e.g., eagle).

3.1.2 Terrestrial Receptors

The terrestrial receptors that will be chosen for these assessments are presented in Table 3.1-1. Since data are only available for the aquatic environment, only receptors with an aquatic based diet will be selected. If terrestrial data become available then other receptors presented below would be considered. These receptors are similar to the ones used at the Anvil Range risk assessment.

TABLE 3.1-1
POSSIBLE TERRESTRIAL RECEPTORS FOR
THE KENO HILLS MINE ASSESSMENT

Herbivores	Omnivores	Carnivores
Beaver	Black bear	Mink
Grouse	Waterfowl (mallard, scaup, merganser)	Wolf
Snowshoe hare	Red fox	
Hoary marmot	Grizzly Bear	
Moose		
Dall's sheep		
Woodland Caribou		

The receptors were selected to represent a wide range of exposures and include:

Herbivores - Herbivores convert vegetable matter to animal protein, and in turn are consumed by omnivores and carnivores. They are also trapped or hunted for fur and food. Beaver, hare, moose, muskrat, grouse and caribou are herbivores selected for this assessment.

Beaver – The beaver habitat is largely in the aquatic environment, although terrestrial vegetation comprises a significant part of their diet. Beaver will be included in this assessment because they have been identified as being consumed by Yukon First Nations people and because they are an indicator of both potential aquatic and terrestrial impacts.

Caribou – Caribou consume predominantly lichen, which are mostly impacted by contaminant deposition from the air. Caribou were chosen since they are in the area and represent a portion of the diet of the Yukon First Nations people. They will be included if sufficient terrestrial information is available.

Grouse – Grouse (and ptarmigan) are terrestrial birds common in the northern environment. The diets of both species are primarily terrestrial, comprising primarily berries and browse. Grouse will be selected to represent both species. They will be included if sufficient terrestrial information is available.

Snowshoe Hare – The snowshoe hare is chosen as it may be trapped in the area and used as a food source. Browse and forage comprise most of the diet of hare. They will be included if sufficient terrestrial information is available.

Hoary Marmot – The hoary marmot feeds on grass and other green plants. The only aquatic pathway of exposure is from consumption of water. Hoary marmots are protected from being hunted in the Yukon, except by First Nations people.

Moose – Moose consume aquatic macrophytes and browse and thus are potentially an exposed species. Moose may be exposed to COPC via terrestrial pathways and given that moose are an important source of food to people in the study area, they will be included in the assessment, if enough terrestrial information is available.

Dall Sheep – There are 19,000 to 23,000 Dall Sheep in the Yukon. Dall Sheep have been reported in the Keno Hills area. They are also a protected species but are hunted by First Nations people. Their diet consists of terrestrial vegetation (forage). They will be included if sufficient terrestrial information is available.

Omnivores - Omnivores consume both plant and animal matter. Vertebrate omnivores included are bear and waterfowl.

Bear – A bear’s diet is composed of terrestrial vegetation, berries, fish and carrion (moose and caribou) and therefore, the bear is an important indicator of potential effects of atmospheric deposition and constituent transfer from air and soil (terrestrial vegetation and berries), and effects on the aquatic environment (fish). Grizzly bears and black bears have similar diets.

Waterfowl (i.e. mallard, merganser, scaup) – Waterfowl are often the most exposed ecological receptors, since their diet is almost entirely obtained from the aquatic environment. The waterfowl diet includes aquatic vegetation, fish, and aquatic (benthic) invertebrates. Given their vulnerability to impacts from the aquatic environment, they will be included in the risk assessment. In addition, the different duck species are representative of other waterfowl that are present in the area. The three species were selected to take into account differences in the diets of mallard (consumes primarily aquatic plants), merganser (consumes mainly fish) and scaup (consumes mostly benthic invertebrates). Ducks are also part of the human food chain.

Red Fox - Foxes are predatory species and thus are exposed via food chain effects. They are omnivores and consume a varied diet including berries, ducks and hare.

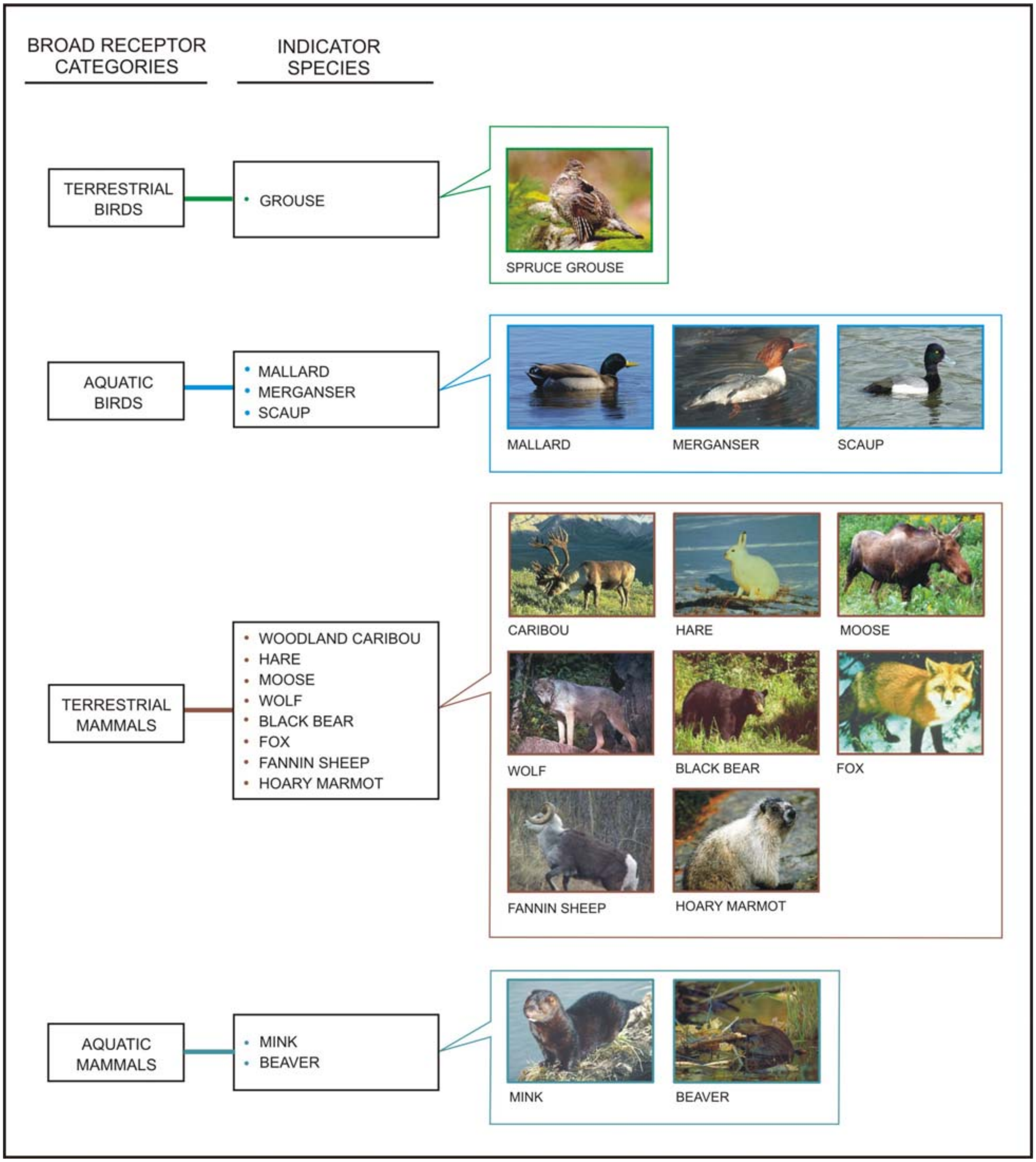
Carnivores - Predators represent the top level of the food chain. Predators interact with prey species (usually herbivores) and may influence population levels and distribution of prey. Terrestrial predators to be included in the assessment include the mink and wolf.

Mink – Mink are found in the Yukon and consume aquatic plants, benthic invertebrates, ducks and fish and are thus potentially exposed via the aquatic pathways.

Wolf - The wolf is a predatory species that consumes a number of other species including caribou, hare and moose. Food chain effects are assessed in this study through the inclusion of the wolf. They will be included if sufficient terrestrial information is available.

All of the terrestrial species mentioned above were assumed to consume soil or sediment, depending on where they obtained their food from, in addition to the food sources noted above.

FIGURE 3.1-2
TERRESTRIAL RECEPTORS CONSIDERED FOR THE ASSESSMENT



Figures 3.1-3a to 3.1-3d provides schematic representations of the potential pathways of exposure for each terrestrial receptor considered in this assessment. The figures also provide the ingestion rates for all the pathways considered for each of the terrestrial receptors. In these assessments, terrestrial species are either located primarily in the aquatic environment (i.e., beaver, ducks, and mink) or primarily in the terrestrial environment (i.e., bear, caribou, hare, moose, ptarmigan/grouse and wolf).

3.1.3 Location of Ecological Receptors

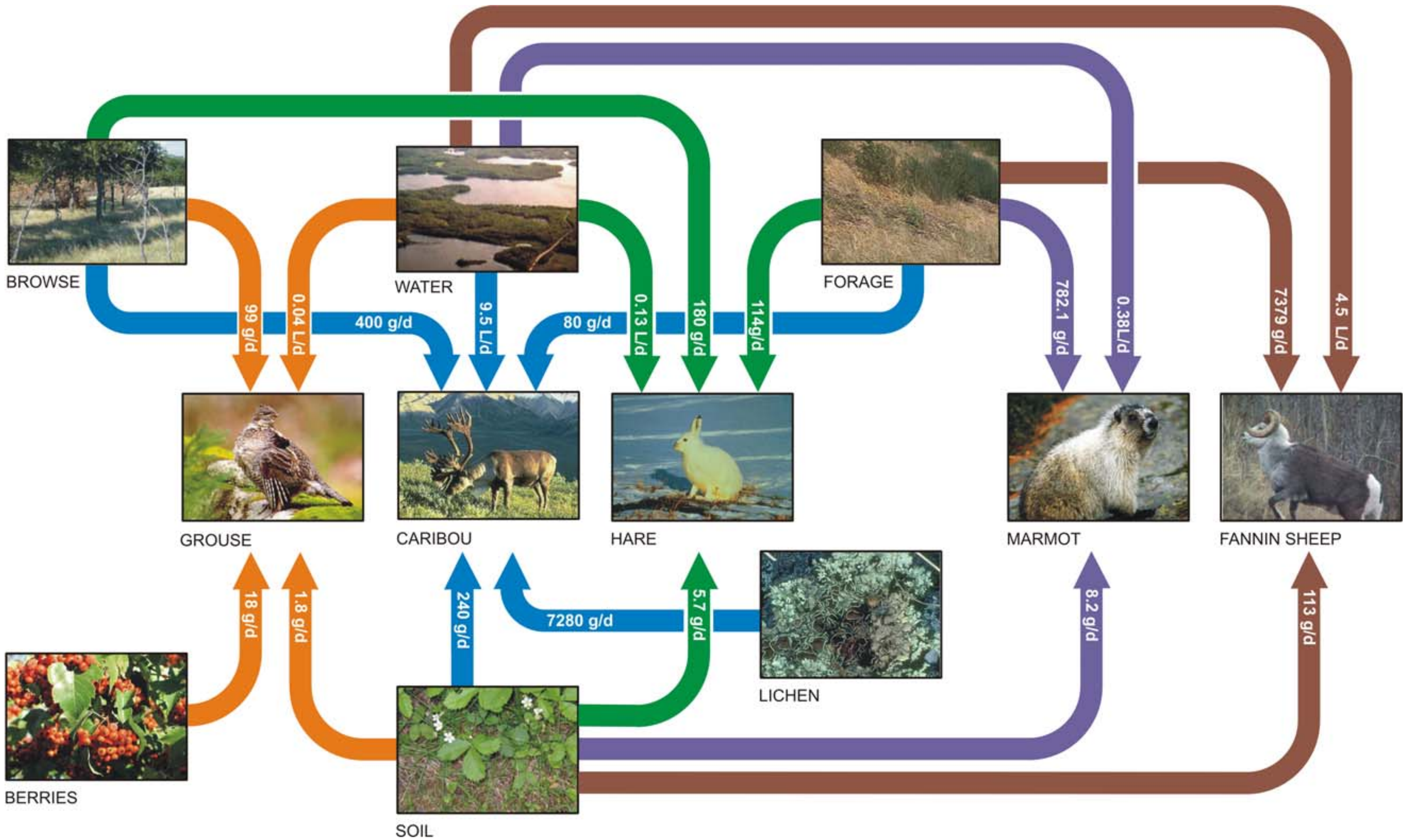
3.1.3.1 Aquatic Receptors

The aquatic receptors identified in Section 3.1.1 will be assumed to be present in all water bodies (see Table 3.1-2) potentially affected by the Keno Hills site such as Lighting Creek, Chrystal Creek, Flat Creek, and McQuesten River.

3.1.3.2 Terrestrial Receptors

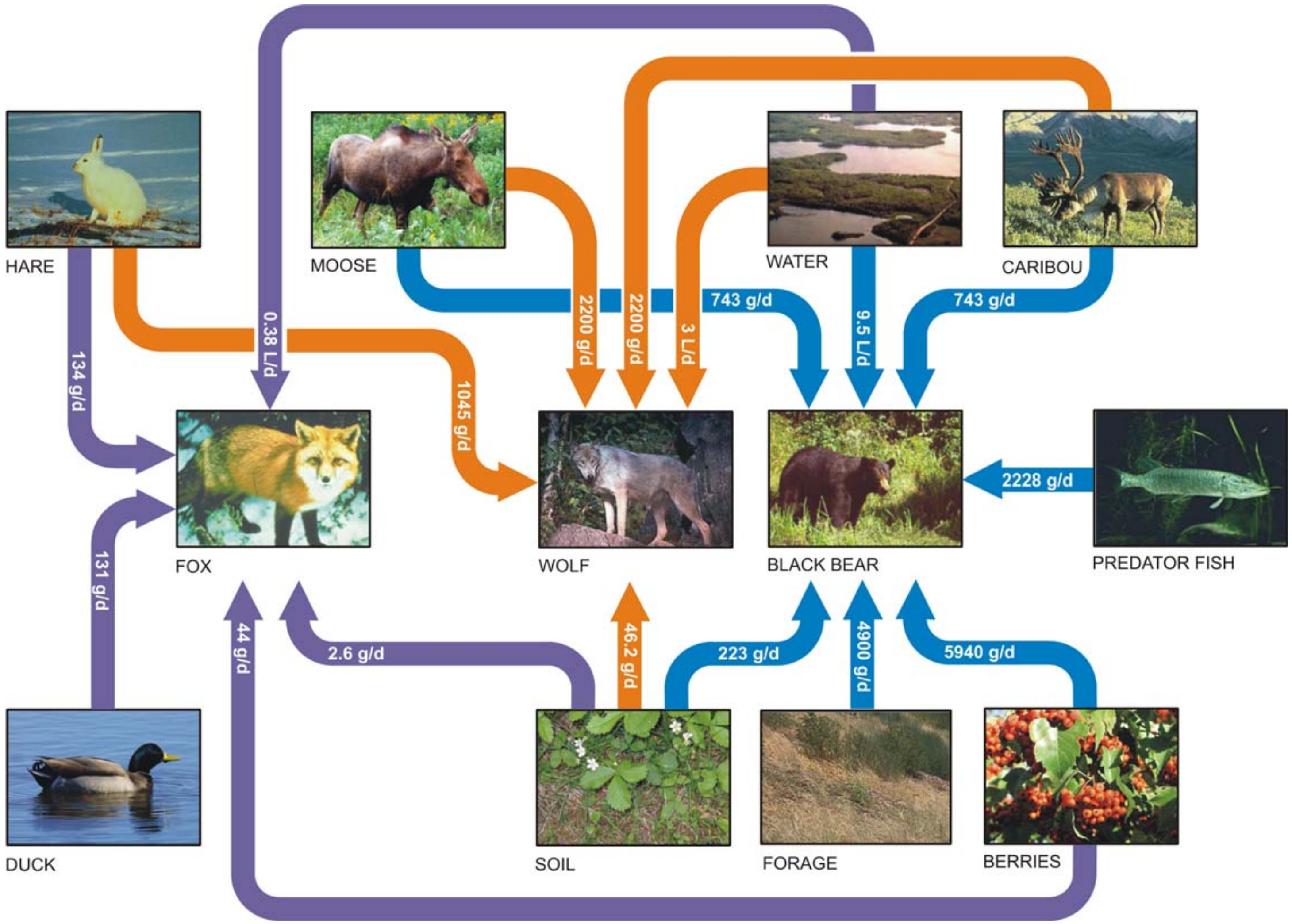
As noted in Section 3.1.2, terrestrial receptors will be selected to include species that obtain most or all of their diet from the aquatic environment and species that obtain all or most of their diet from the terrestrial environment. In selecting locations where terrestrial species will be assumed to be present in the vicinity of the Keno Hills site, factors that will be taken into consideration included not only dietary characteristics but also the home range of the species and locations where the species would likely receive a range of exposures to the COPC. The locations where the smaller terrestrial animals are placed at the site will be determined once appropriate data in the terrestrial environment are available. For example, depending on the locations and types of COPC, more than 1 species (e.g., hare) will be considered at different locations across the site. Table 3.1-3 summarizes the home range of the receptors and the proposed fraction of time that is assumed to be spent on-site.

FIGURE 3.1-3a
POTENTIAL PATHWAYS OF EXPOSURE FOR CARIBOU, GROUSE, HARE, MARMOT AND SHEEP



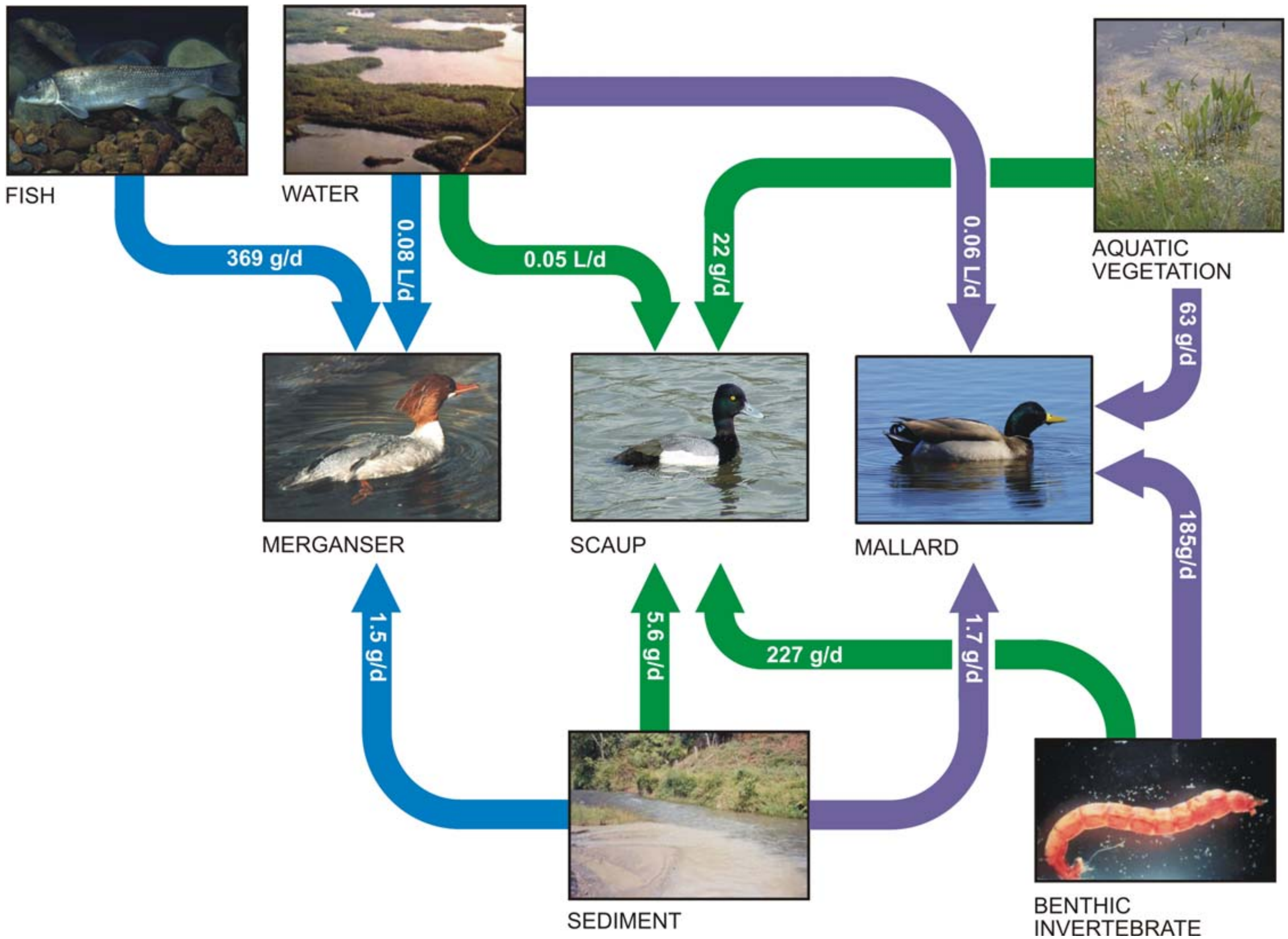
Note: All intakes are on a wet weight basis except for soil and sediment which is on a dry weight basis.

FIGURE 3.1-3b
POTENTIAL PATHWAYS OF EXPOSURE FOR BLACK BEAR, FOX AND WOLF



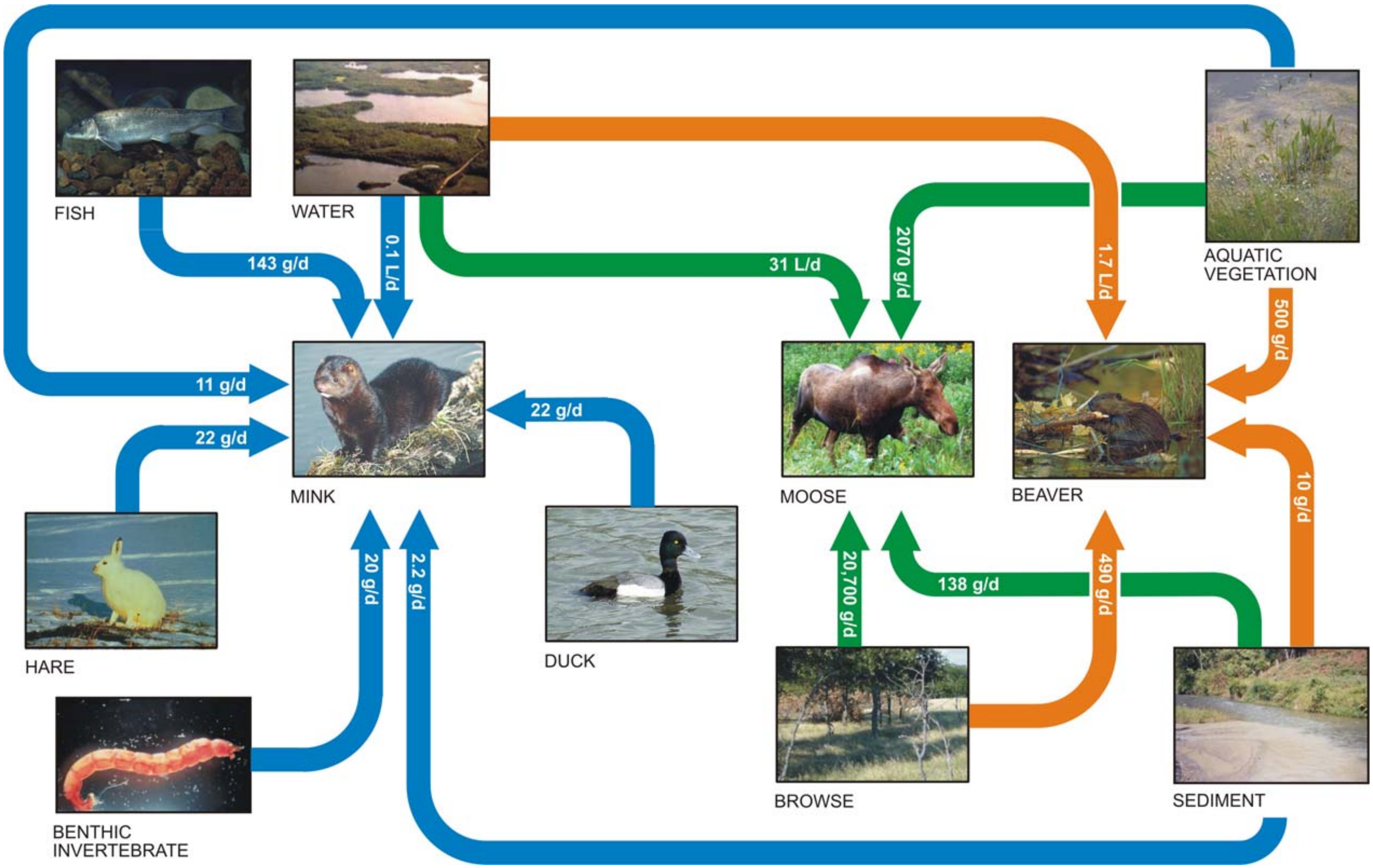
Note: All intakes are on a wet weight basis except for soil and sediment which is on a dry weight basis.

FIGURE 3.1-3c
POTENTIAL PATHWAYS OF EXPOSURE FOR MALLARD, MERGANSER AND SCAUP



Note: All intakes are on a wet weight basis except for soil and sediment which is on a dry weight basis.

FIGURE 3.1-3d
POTENTIAL PATHWAYS OF EXPOSURE FOR MINK, MOOSE AND BEAVER



Note: All intakes are on a wet weight basis except for soil and sediment which is on a dry weight basis.

TABLE 3.1-2
AQUATIC ECOLOGICAL RECEPTORS ASSUMED FOR ASSESSMENT

	Lighting Creek	**Chrystal Creek**	**Flat Creek**	**McQuesten River**
Aquatic plants	✓	✓	✓	✓
Phytoplankton	✓	✓	✓	✓
Zooplankton	✓	✓	✓	✓
Benthic invertebrates	✓	✓	✓	✓
Forage fish	✓	✓	✓	✓
Predator fish	✓	✓	✓	✓

Notes: ✓ species assumed to be present at the indicated location.

TABLE 3.1-3
EXPOSURE CHARACTERISTICS ASSUMED FOR TERRESTRIAL ECOLOGICAL RECEPTORS FOR RISK ASSESSMENT

Receptor	Pathways of Exposure	Fraction of time at site	Home Range
Bear	Water, soil, herbaceous vegetation, berries, fish, moose, caribou	1.0	20 km^2 (2.6 to 155 km^2) [a]
Beaver	Water, sediment, terrestrial vegetation, aquatic vegetation	1.0	0.04 km^2 (varies throughout the year from 0.25 ha to 10 ha; focussed on waters edge) [b]
Woodland Caribou	Water, soil, summer forage, browse, lichen	0.5	250 km^2 [c]
Fox	Water, soil, berries, duck, hare	1.0	6 km^2 (4 to 8 km^2 around den site) [d]
Grouse	Water, soil, browse, berries	1.0	0.2 km^2 (no migration - resident year round; 0.04 to 0.40 km^2) [e]
Hare	Water, soil, browse, herbaceous vegetation	1.0	0.08 km^2 (6 to 10 ha) [d]
Hoary Marmot	Water, soil, herbaceous vegetation	1.0	0.09 km^2
Mallard	Water, sediment, benthic invertebrates, aquatic vegetation	0.5	5.8 km^2 [f] (home range in spring - possibly in area from Mar /Apr/May to Sept/Oct/Nov)
Merganser	Water, sediment, fish	0.5	Possibly in area from April to Sept/Oct
Mink	Water, sediment, aquatic vegetation, benthic invertebrates, duck, fish, hare	1.0	0.14 km^2 [f] (7.8 to 20.4 ha depending on vegetation)
Moose	Water, sediment, browse, aquatic vegetation	1.0	60 km^2 [g] (15 to to 100 km^2)
Scaup	Water, sediment, benthic invertebrates, aquatic vegetation	0.5	0.89 km^2 [f] (possibly in area from Apr/May to Sept/Oct)
Sheep	Water, soil, herbaceous vegetation	1.0	Resident in the area year-round
Wolf	Water, soil, moose, caribou, hare	0.25	1000 km^2 (100 to 2500 km^2) [h]

Notes:

a Home range for a female bear can range between 2.6 km^2 to 40 km^2; the home range for a male bear can range from 21 km^2 to 155 km^2 (American Bear Association 2003).

b Wheatley 1994; Kent Wildlife Trust (2003).

c Rock (1992).

d Hinterland Who's Who (Canadian Wildlife Service 2005).

e Home range for a female grouse is from 0.16 km^2 to 0.40 km^2; home range for a male grouse is from 0.04 km^2 to 0.20 km^2 (North Carolina State University 1995).

f U.S. EPA (1993).

g In northern Saskatchewan, it is reported that moose may range over 25 to 100 km^2 (Cameco 2004). Home range studies based on radio-collared individuals were reported to average 59 km^2 for the Copper River Delta in south-central Alaska (MacCracken *et al.* 1997). In more southerly regions such as Idaho, the home range for female moose has been observed to range from 15.5 to 25.9 km^2, and for male from 31 to 51.8 km^2 (Pierce and Peck 1984).

h In Alaska, the home range may include some 200 to 600 square miles (520-1560 km^2) of habitat (Woodland 2005). Home range is 100 to 2500 km^2 (Resources Inventory Committee 1998).

3.2 HUMAN HEALTH RISK ASSESSMENT

The following section outlines the assessment of potential incremental exposures to humans that may utilize the Keno Hills site. For the purposes of the assessments, assumed human characteristics will be defined to calculate potential exposures under current site conditions. Many of the assumptions discussed here will have to be formalized once the information from the Traditional Knowledge study is available. In the mean time, the assumptions that were used at the Anvil Range Mine will assumed to apply.

3.2.1 Human Receptor Selection

This assessment will consider the potential for adverse effects on hypothetical individuals who would camp at the site while hunting and gathering. It will be assumed that the time that a hypothetical camper might spend on-site would encompass the time frame that trappers and other occasional users might be on the site. It will be assumed that a family would camp on the site and carry out their hunting and gathering activities; therefore, an adult, child and toddler will be considered in the assessment. It will be assumed that campers would be present at the site for approximately 1.5 months of the year. The location of the campers will be determined after the Traditional Knowledge Study has been completed as well as in discussions with the study teams working on the site.

3.2.2 Human Receptor Characteristics

The exposure to humans from COPC at the Keno Hills site depends on behavioural characteristics, such as time at the site and source of drinking water and food. Conservative assumptions are made in the characterization of human receptors for these assessments.

Dietary data from a regional survey of First Nations people in the Yukon were used to define the dietary characteristics for campers who would be present at the Anvil Range Mine Complex. Other exposure data, such as drinking water consumption and body weight, were obtained from a survey of the general Canadian population and are acceptable for use in the current assessment.

The dietary characteristics for the assessment were based on a study of Yukon First Nations communities in 1998. It is acknowledged that this information was not collected for the purposes of this assessment; however, it is the best information available at present for conducting this assessment. Assumptions regarding the intakes of the adult and child receptors are outlined below.

Food Consumption

Traditional and market food intake rates for the Yukon First Nations' group were derived from Receveur *et al.* (1998). The use of region-specific dietary intake rates was deemed more appropriate than the use of other data sources and methods to estimate traditional and market food intakes, and is associated with less uncertainty.

Receveur *et al.* (1998) collected dietary intake data using methods including food frequency questionnaires and 24-hour recall surveys administered by trained interviewers during the late winter and fall of 1995. Ten communities in the Yukon participated in the study, including Dawson City, Mayo, Carmacks, Ross River, Watson Lake, Lower Post, Beaver Creek, Burwash Landing, Carcross and Atlin. Only adults in the communities were sampled, and included both men and women divided into age categories including ages 20-40, ages 41-60 and 61+ years.

Traditional food intake rates were available for both consumers only (people who only eat traditional foods) and consumers and non-consumers (people who eat a mixture of store bought food and traditional food). Rates for consumers and non-consumers provide an average estimate for the community, while rates for consumers only provide an upper bound on the average estimate for the community. Intake rates for both groups were derived for this assessment.

To derive average traditional food intake rates for an adult at Keno Hills Mine, rates provided by Receveur *et al.* (1998) were first averaged for both sexes over the three adult age groups for which data were available. Traditional food items were subsequently grouped into categories including meat and poultry, fish, and berries in order to calculate intake rates for these based on a summation of the group items. The food groupings chosen were those typically used in Canadian total diet studies (Richardson 1997, Health Canada 2005).

Traditional food items in the meat and poultry group contained some items for which concentrations of COPC cannot be estimated in the risk assessment, such as moose bone marrow and heart. The amount consumed of each of these items was counted nonetheless so as not to under-estimate intake of traditional foods. However, it was assumed that the metals content of these items was the same as that in the flesh of the animal under consideration; this would provide an underestimate of the COPC intake as organs generally contain higher concentrations of COPC. A sensitivity analysis will be performed to determine the effect of organ consumption.

The intake for a child was estimated assuming that the ratio of child to adult of a particular category of food for the general population (Richardson 1997) could be applied to the information for the Yukon First Nations region.

Table 3.2-1 provides the average traditional food intake rates (people who eat traditional food and a mixture of store bought food) derived from Receveur *et al.* (1998) for a Yukon First Nation adult and child.

Total meat and fish intake rates were derived for risk assessment purposes, as well as fractions of the intake rate attributable to different traditional foods. This rate is presented in Table 3.2-1 and the fractions are presented in Table 3.2-2.

TABLE 3.2-1
INTAKE OF TRADITIONAL FOODS FOR YUKON FIRST NATION

Food Category	Mean Intake Rate (g/d)	
	Child	Adult
Meat and poultry	128	173
Fish and shellfish	16.0	21.6
Fruit	1.21	1.63
Total meat and fish	144	195

Source: Receveur *et al*. 1998

TABLE 3.2-2
COMPOSITION OF DIFFERENT FRACTIONS OF MEAT AND FISH INTAKE FOR YUKON FIRST NATION

Traditional Food Item	Dietary Fraction
Fraction that is caribou	0.06
Fraction that is moose	0.75
Fraction that is dall sheep	0.004
Fraction that is hare	0.03
Fraction that is hoary marmot	0.02
Fraction that is poultry	0.01
Fraction that is beaver	0.02
Fraction that is fish	0.11

Source: Receveur *et al*. 1998.

Table 3.2-3 provides the traditional food intake rates for consumers only derived from Receveur *et al*. (1998) for a Yukon First Nations adult and child.

Again, a total meat and fish intake rate was also derived for risk assessment purposes, as well as fractions of the intake rate attributable to different traditional foods. This rate is presented in Table 3.2-3 and the fractions are presented in Table 3.2-4.

TABLE 3.2-3
INTAKE OF TRADITIONAL FOODS FOR YUKON FIRST NATION (CONSUMERS OF TRADITIONAL FOOD ONLY)

Food Category	Mean Intake Rate (g/d)	
	Child	Adult
Meat and poultry	2804	3790
Fish and shellfish	1544	2086
Fruit	501	677
Total meat and fish	4348	5876

Source: Receveur *et al*. 1998

TABLE 3.2-4
COMPOSITION OF DIFFERENT FRACTIONS OF MEAT AND FISH INTAKE FOR YUKON FIRST NATION (CONSUMERS OF TRADITIONAL FOOD ONLY)

Traditional Food Item	Dietary Fraction
Fraction that is caribou	0.05
Fraction that is moose	0.36
Fraction that is dall sheep	0.03
Fraction that is hare	0.04
Fraction that is hoary marmot	0.02
Fraction that is poultry	0.05
Fraction that is beaver	0.11
Fraction that is fish	0.36

Source: Receveur *et al*. 1998

Intake rates for commercial market foods were also available in Receveur *et al*. (1998). Similar to the methods used for the intakes of traditional foods, adults of different age groups were interviewed regarding their consumption of market food using 24-hour dietary recall surveys conducted by trained interviewers during the late winter and fall of 1995. Intake rates for individual market food items for consuming and non-consuming adults of different age groups and sexes were subsequently calculated by Receveur *et al*. (1998).

To derive average market food intake rates for an adult at the Keno Hills Mine, rates by Receveur *et al.* (1998) for the Yukon First Nation were first averaged for both sexes over the three adult age groups for which data were available. Market food items were then grouped into their respective food categories, which included milk and dairy products, meat and poultry, fish and shellfish, soups, bakery goods and cereals, vegetables, fruits and fruit juices, fats and oils, sugar and candies, beverages and miscellaneous items based on a summation of individual items in the group. The food groupings chosen were those typically used in Canadian total diet studies (Richardson 1997, Health Canada 2005). Market foods intake rates were only available for consumers and non-consumers combined (zeros in).

Again, the intake for a child was estimated assuming that the ratio of child to adult of a particular category of food for the general population (Richardson 1997) could be applied to the information for the Yukon First Nation region. A similar ratio was used to determine toddler intakes.

Table 3.2-5 provides the market food intake rates derived from Receveur *et al.* (1998) for a Yukon First Nation adult and child.

TABLE 3.2-5
INTAKE OF MARKET FOODS BASED ON THE YUKON FIRST NATION

Food Category	Mean Intake Rate of Market Foods (g/d)	
	Child	**Adult**
Milk and dairy products	52	70
Meat and poultry	132	178
Fish and shellfish	2.6	3.5
Soups	104	141
Bakery goods and cereals	137	185
Vegetables	143	193
Fruit and fruit juices	46	63
Fats and oils	14	19
Sugar and candies	25	34
Beverages	1021	1379
Miscellaneous	56	76

Source: Receveur *et al.* 1998

As a significant amount of the beverage intake is coffee and tea, the beverage intake for the child is likely overstated.

In this assessment, the dietary intakes for people who eat a mixture of traditional foods and store bought foods will be used as this seems indicative of the community around the Keno Hills Mine.

Medicinal Tea Intake

First Nations People generally use Labrador tea and other teas made from natural plants for medicinal purposes. The Yukon dietary survey (Receveur *et al.* 1998), for the area only, indicated that in the winter 12% of the population consumes Labrador Tea 1.2 days/week and in the summer 23% of the population consumes Labrador Tea 1.4 days/week. This equates to an average Labrador Tea consumption of 1.3 days per week. The survey does not provide the amount of Labrador tea or other medicinal teas that are consumed.

In an attempt to determine the amount of medicinal tea consumed by members of the community, a web search was undertaken. The web search indicated that "*Labrador Tea contains small amounts of the toxin andromedotoxin which can cause headaches, cramps, paralysis and intestinal problems if too much is consumed. As a general rule, this tea should be consumed in moderation. One cup is often considered the safe amount.*" - *http://www.laurentiancenter.com/plantkey/plants/labradortea.html*

Therefore, it will be assumed in this assessment that an adult (70kg body weight) would consume 250 mL (1 cup) of medicinal teas for 1.3 days a week. This equates to a consumption rate of 0.17 cups/day. There was no information available on the amount of fresh Labrador tea used in the brewing process. In this assessment, it was assumed that approximately 3 g were used. This is equivalent to a bag of regular tea. If information becomes available from the Traditional Knowledge study with respect to the intake of Labrador Tea, that information will be used to derive an appropriate tea intake value.

Water Intake

The water intakes for an adult and child will be obtained from the *"Compendium of Canadian Human Exposure Factors for Risk Assessment"* (Richardson, 1997). The average water intakes for an adult, child (5 to 11 years of age) and toddler (0.5 to 4 years of age) is estimated to be 1.5 L/d, 0.8 L/d and 0.6 L/d respectively.

Soil Intake

Soil intake rates will be obtained from Health Canada (2003) and are based on the information obtained from CCME (1996) and the Massachusetts Department of Environmental Protection

(MADEP 2002). The mean daily soil intake provided for an adult was 20 mg/d, and, for a child was 20 mg/d. The mean soil intake for a toddler is reported to be 80 mg/d.

Body Weight

The body weight (bw) of a toddler, child and adult are also necessary in order to calculate a daily intake (mg/(kg (bw) d)). In this assessment, the body weights used for the toddler, child and adult receptors will be 16.5 kg, 32.9 kg and 70.7 kg, respectively (Richardson 1997).

3.2.3.1 Summary of Receptor Characteristics

The nominal amount of traditional foods and berries that will be assumed to be consumed by the adult, child and toddler receptors present at the Keno Hills Mine are summarized in Table 3.2-6.

TABLE 3.2-6
AVERAGE AIR, WATER AND LOCAL FOOD INTAKE RATES USED FOR ADULT, CHILD AND TODDLER IN THE PATHWAYS MODELLING

	Receptor		
	Adult	**Child**	**Toddler**
Air (m^3/d)	15.8	14.5	9.3
Water (L/d)	1.5	0.8	0.6
Soil Intake (mg dw/d)	20	20	80
Local Meat (g ww/d)			
Caribou	11.8	8.7	6.1
Moose	146	108	75.9
Sheep	0.7	0.5	0.4
Other small mammals	13.5	10	7
Local Poultry (g ww/d)			
Ground birds	0.7	0.5	0.4
Water birds	0.3	0.2	0.17
Local Fish (g ww/d)	21.6	17.5	10.8
Total local meat, fish and poultry (g ww/d)	195	145	101
Other (g ww/d)			
Berries	1.6	1.8	1.6

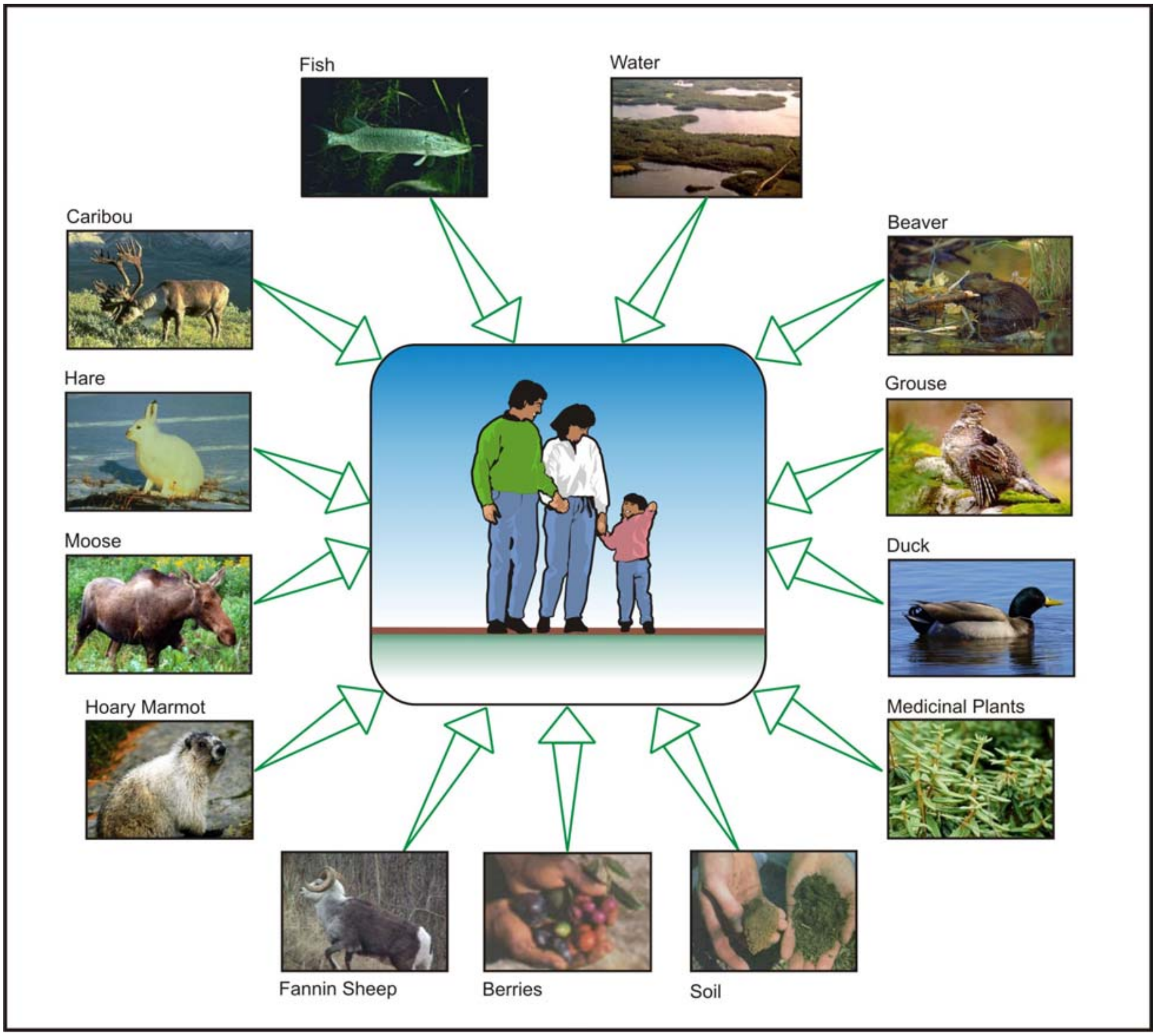
<u>Note:</u> Adult Weight = 70.7 kg; Child Weight = 32.9 kg; Toddler = 16.5 kg

3.2.3 Pathways of Exposure

The human exposure analysis focused on the pathways as shown on Figure 3.2-1. They include:

- consumption of drinking water containing COPC from waterbodies such as such as Lighting Creek, Chrystal Creek, Flat Creek, and McQuesten River by humans;
- uptake by caribou of COPC from water, soil and vegetation and consumption of caribou flesh by humans;
- uptake by moose of COPC from water, sediment and aquatic and terrestrial vegetation and consumption of moose flesh by humans;
- uptake by sheep of COPC from water, soil and vegetation and consumption of sheep flesh by the human receptors;
- uptake by snowshoe hare of COPC from water, soil and vegetation and consumption of hare flesh by the human receptors;
- uptake by hoary marmot of COPC from water, soil and vegetation and consumption of marmot flesh by the human receptors;
- uptake by grouse of COPC from water, soil and vegetation and consumption of grouse flesh by humans;
- uptake by beaver of COPC in water, sediments and vegetation and consumption of beaver flesh by humans;
- uptake by duck of COPC in water, sediment, benthic invertebrates and vegetation and consumption of duck flesh by humans;
- uptake by fish of COPC from the aquatic environment (such as Lighting Creek, Chrystal Creek, Flat Creek, and McQuesten River) and consumption of fish flesh by humans;
- uptake of COPC in soil by berries/medicinal plants and consumption of berries/medicinal plants by humans; and,
- inadvertent ingestion of soil containing COPC and dermal contact with soil by humans.

FIGURE 3.2-1
CONCEPTUAL MODEL FOR HUMANS



4.0 HAZARD ASSESSMENT

4.1 ECOLOGICAL RISK ASSESSMENT

Within the Ecological Risk Assessment (ERA) framework, assessment endpoints for valued ecosystem components (VECs) or ecological receptors are based on potential effects at population or community levels. At these levels of biological organization, VEC population and community characteristics can be defined over fairly extended temporal and spatial scales making the potential for the direct measurement of effects challenging (Environment Canada, 1997).

Due to the difficulty in measuring direct effects on population assessment endpoints, "*measurement endpoints*" are adopted to provide a framework for the evaluation of predicted effects. A measurement endpoint is defined as "…*a quantitative summary of the results of a toxicity test, a biological study, or other activity intended to reveal the effects of a substance*" (Suter, 1993). In lieu of direct assessment endpoint effects measures, the adoption of measurement endpoints provides a consistent basis for the evaluation of potential effects due to exposure to constituents.

Measurement endpoints are commonly selected at the individual level of biological organization, and are typically based on exposure responses that are meant to act as a proxy for population and community characteristics such as reproduction and abundance (Environment Canada, 1997). Such measurement endpoints are commonly based on literature-derived toxicity dose-response relationships, examined through laboratory experimentation (i.e., the response of a particular organism to a certain level of exposure). When derived from toxicity studies, such measurement endpoints are often referred to as toxicity benchmarks or toxicity reference values (TRVs).

These toxicity reference values are used in risk assessments to judge whether the predicted (estimated) exposures (or doses or intakes) may potentially have an adverse effect on ecological species. Site-specific information was incorporated into the selection process for TRVs where available. A discussion of selected literature and the associated toxicity reference values that will be used depending on the COPC selection is provided in the following sections.

4.1.1 Toxicity Reference Values

Toxicity reference values are based on exposure levels in the case of aquatic species and total intakes (or doses) for terrestrial species. Unless otherwise stipulated, it is assumed that the entire amount of each COPC taken into the stomach and/or lungs of a terrestrial species is transferred into the body stream of the species.

4.1.1.1 Aquatic Toxicity Reference Values

Table 4.1-1 summarizes the aquatic toxicity reference values for a selection of COPC. The final set of TRVs will be determined once the screening for the COPC has been completed. Table 4.1-1 outlines the references from which the toxicity reference values were obtained, the test species and the rationale for selecting the appropriate toxicity reference values. It is noted that TRV are provided for the COPC discussed in Section 1 (i.e., Al, Sb, As, Cd, Cr, Co, Cu, Pb, Mn, Mo, Ni, Ag, Se, and Zn).

It was not the intent of this assessment to extensively search the primary literature to obtain toxicity reference values; rather, these assessments will rely on values that have been collated by various agencies for use in risk assessments. The U.S. DOE database (Suter and Tsao, 1996) and the CCME Guidelines for the protection of aquatic life provide aquatic toxicity benchmarks and were the initial sources that were considered. This U.S. DOE database contains toxicity reference values for the protection of aquatic life from constituents in water. EC_{20} values provided in this database were selected as appropriate toxicity benchmarks. The toxicity reference values developed for use in risk assessments have been peer reviewed and are routinely used in risk assessments. This database provides documentation on the sources and derivations of the values and discusses the relative conservatisms in the toxicity reference values.

If data were not available from the DOE database or CCME then the U.S. EPA database AQUIRE was examined for infilling purposes. The data summarized in this database are from a variety of sources including peer-reviewed literature. Toxicity information provided in the Ontario water quality guidelines was also considered. It is acknowledged that these databases may not have the most recent data; however, the toxicity reference values selected are in our opinion appropriate.

Decision rules for the selection of test species were conservatively based on the lowest values from the available data. Such conservatism aims for the protection of the most sensitive ecological species present at the site, and also to protect any species at risk that may be present at the site. For example, choosing the lowest available values for aquatic plants attempts to protect the most sensitive aquatic plant species that may be present at the site.

The selection of reference values for the specific constituent was based on:

- Antimony – The aquatic toxicity reference values were obtained from Suter and Tsao (1996) for phytoplankton, zooplankton and forage fish. Benthic Invertebrates were obtained from Brooke *et al.* (1986). The aquatic toxicity reference values for predatory fish was derived based on Doe *et al.* (1987)

- Aluminium – References cited in CCME (1999) and U.S. EPA AQUIRE database were used to derive the aquatic toxicity reference values for Al.

- Arsenic – toxicity benchmarks were obtained from the Suter and Tsao (1996) and U.S. EPA AQUIRE (2003) databases as well as the CCME (1999) as outlined in Table 4.1-1.

- Cadmium – The aquatic toxicity benchmarks were obtained from US. EPA AQUIRE database and Suter and Tsao (1996). See Table 4.1-1 for details.

- Chromium – Reference cited in Suter and Tsao (1996) were used to obtain the aquatic toxicity reference values for chromium (VI).

- Cobalt – the toxicity benchmark for zooplankton was obtained from the U.S. EPA AQUIRE (2003) database while the other toxicity values were obtained from an Ontario Ministry of the Environment (1996) document which summarized aquatic toxicity values.

- Copper - the primary literature was used to obtain the toxicity values for all species with the exception of benthic invertebrates as outlined in Table 4.1-1. For aquatic plants, data for *Myrophillium* (Stanley 1974) were selected over data for *Lemna minor* (Jenner and Janssen-Mommen 1993) since *Lemna minor* are not present in the study area. Data from the U.S. EPA AQUIRE (2003) database were used for benthic invertebrates.

- Lead - the DOE database (Suter and Tsao 1996) was used to obtain toxicity benchmarks for phytoplankton, zooplankton and predator fish and the U.S. EPA AQUIRE (2003) database was used to obtain data for bottom feeder fish. For aquatic plants data for *Myriophyllum sp* were used over *Lemna minor* as discussed previously.

- Manganese - U.S. EPA AQUIRE (2003) was used for all TRVs.

- Molybdenum - the DOE database (Suter and Tsao 1996), U.S. EPA AQUIRE (2003) and CCME Water Quality document (1999) were used to derive the TRVs.

- Nickel – for daphnids, CVs of 0.0148 mg/L and 0.007 mg/L for *Daphnia magna* (Chapman *et al*. 1980, and Lazareva 1985) exist; however the DOE database (Suter and Tsao 1996; pages 9 and 26 under Ni) did not consider these values as appropriate and calculated a test EC_{20} of 0.045 mg/L from studies carried out by Münzinger (1990). From this EC_{20}, an EC_{25} was calculated using linear interpolation. For phytoplankton (*Microcystis aeruginosa*), an EC_{20} of 5 mg Ni/L is provided in the DOE database; however, this value is below the CCME aquatic life guideline of 0.025 mg/L. Chao and Chen (2000) developed an EC_{10} value of 0.016 mg/L for 10% growth reduction in *Selenastrum capricornutum* in batch culture. The value was extrapolated to an EC_{20} assuming a linear relationship and used in the assessment. There are several toxicity values for rainbow trout including a 28-d LC_{50} of 0.050 mg Ni/L for rainbow trout embryo & larva (Birge 1978). This value has been rejected by Suter and Tsao (1996) and replaced by an EC_{20} of 0.062 mg/L from Nebeker *et al*. (1985). Suter and Tsao (1996) reported that much of the data included in criteria documents issued prior to 1985 are no longer considered acceptable by EPA (Stephan *et al*. 1985). The above references (Birge 1978, Chapman *et al*. 1980, and Lazareva 1985) for instance were reviewed against criteria in Stephan *et al*. (1985) and were not considered as high quality standard data.

- Selenium – the DOE database (Suter and Tsao 1996) was used to obtain the toxicity benchmarks for zooplankton and predator fish. A 48 hr LC_{50} of 1.8 mg/L has been used for benthic invertebrates based on a study on *Chironomus sp* by Ingersoll *et al*. (1990). There exists a lower benchmark value of 0.07 mg/L based on a 336-h LC_{50} for *Hyallela azteca* (Halter *et al*. 1980). However, this lower value has not been used in the assessment since Halter *et al*. (1980) did not make a distinction between sodium selenite (Na_2SeO_3) and sodium selenate (Na_2SeO_4). This is particularly important because Se as selenate is generally (much) less toxic than Se as selenite. The U.S. EPA AQUIRE (2003) database was used to obtain the toxicity data for aquatic plants and bottom feeder fish. These values were adjusted to the EC_{25} toxicity benchmark as discussed below.

- Silver – no data were available for aquatic plants, phytoplankton or benthic invertebrates. Aquatic toxicity reference values for zooplankton, predator fish, and forage fish were obtained from Erickson *et al.* (1998).

- Zinc – The aquatic toxicity benchmarks were obtained from US. EPA AQUIRE (2003) database and Suter and Tsao (1996). See Table 4.1-1 for details.

Chronic effects concentration (EC_{25}) reference values were selected as recommended by Environment Canada (1997). The EC_{25} is the lowest concentration that results in 25% of the population being affected (i.e., population effect such as growth). Where possible, effects concentration (EC) data were used over mortality (LC) data. Different models exist for translating chemical exposure (or dose) to toxic responses. For EC_{50} toxicity values, in the absence of detailed dose-response functions, a linear approximation can be established with a single toxicity reference value assuming zero effect at zero exposure. This linearization is pessimistic since the predicted effect will be greater than that observed using the commonly encountered sigmoidal dose-response function for low dose exposures. For acute toxicity values (LC_{50} values for 96 hour tests), a factor of 10 was applied to derive a toxicity reference value that would approximate an EC_{20} (Environment Canada and Health Canada 2003). For LC_{50} data derived from chronic tests a factor of 4 was applied to derive a toxicity reference value that would approximate an EC_{20}. The factors of 4 and 10 are empirical factors based on the results of other toxicity tests.

It should be noted that the aquatic TRVs presented in Table 4.1-1 for benthic invertebrates and all other aquatic receptors are based on water exposure only; sediment exposure is discussed and assessed separately using toxicity data based on sediment toxicity studies. TRVs related to bioaccumulation of selenium in tissues are also discussed below.

TABLE 4.1-1
AQUATIC TOXICITY REFERENCE VALUES FOR ECOLOGICAL RISK ASSESSMENT

Aquatic Receptor	Aluminum (mg/L)				
	Test Species	**LC/EC_{50}**	**Toxicity Reference Value**	**Reference**	**Comments**
Aquatic Plants	*Myriophyllum*	45.7	22.8	Gostomoski (1990)	from Gensmer and Playle (1999); EC_{50} (growth); used EC_{25} – pH 7.6-8.2
Phytoplankton	*Selenastrum*	0.46	0.23	Call (1984)	from U.S. EPA (1988); 4-d EC_{50} (biomass); used an EC_{25} - pH 8.2
Benthic Invertebrates	Not provided	--	0.32	Borgmann *et al.* (1980)	from CCME (2003); 14-d EC_{20}
Zooplankton	*Daphnia* sp.	2.6	0.26	Wakabayashi *et al.* (1988)	from U.S. EPA AQUIRE; 24-h LC_{50}; derived benchmark using a factor of 10 based on an empirical relationship between an acute LC_{50} and an EC_{20}
Predatory Fish	Not provided	--	3.29	U.S. DOE (2005)	Lowest chronic value (LCV) Fish Surface Water Screening Benchmark
Forage Fish	Not provided	--	4.70	U.S. DOE (2005)	EC_{20} Fish Surface Water Screening Benchmark

Aquatic Receptor	Antimony (mg/L)				
	Test Species	**LC/EC_{50}**	**Toxicity Reference Value**	**Reference**	**Comments**
Aquatic Plants	--	--	--	--	no data available
Phytoplankton	*Selenastrum capricornutum*	0.61	0.15	Kimball (n.d.)	from Suter and Tsao (1996); 4-d EC_{50}; derived an EC_{25} by linear extrapolation
Benthic Invertebrates	*Gammarus pseudolimnaeus*	26	2.6	Brooke *et al*., 1986	4-day LC_{50} derived TRV using a factor of 10 based on an empirical relationship between an acute LC_{50} and EC_{50}
Zooplankton	*Daphnia magna*	5.4	1.35	Kimball (n.d.)	from Suter and Tsao (1996); 28-d LC_{50}; derived TRV using a factor of 4 based on an empirical relationship between a chronic LC_{50} and an EC_{20}.
Predatory Fish	Rainbow trout	16	4	Doe *et al*, 1987	30-day LC_{50}; derived TRV using a factor of 4 based on an empirical relationship between a chronic LC_{50} and an EC_{20}.
Forage Fish	*Pimephales promelas*	--	2.31	Kimball (n.d.)	from Suter and Tsao (1996); lowest chronic test EC_{20} – early life stage tests

TABLE 4.1-1 (Cont'd)
AQUATIC TOXICITY REFERENCE VALUES FOR ECOLOGICAL RISK ASSESSMENT

Aquatic Receptor	Arsenic (mg/L)				
	Test Species	**LC/EC_{50}**	**Toxicity Reference Value**	**Reference**	**Comments**
Aquatic Plants	*Myriophyllum* sp.	0.63	0.32	Jenner and Janssen-Mommen (1993)	from U.S. EPA AQUIRE (2003); 14-d EC_{50} (population); derived EC_{25} by linear extrapolation
Phytoplankton	*Scenedesmus* sp.	0.05	0.025	Vocke *et al.* (1980)	from CCME (1999); 14-d EC_{50} (growth); EC_{25} derived by linear extrapolation
Benthic Invertebrates	*Calanus* sp.	--	0.34	Borgmann *et al.* (1980)	From CCME (1999); 14-d EC_{20}; used as TRV
Zooplankton	*Daphnia* sp.	--	0.91	Call *et al.* (1983), Lima *et al.* (1984)	from Suter and Tsao (1996); lowest chronic test EC_{20} – life-cycle tests; used as TRV
Predator Fish	Rainbow Trout	0.55	0.14	Birge *et al.* (1979a)	from CCME (1999); 28-d LC_{50}; derived TRV using a factor of 4 based on empirical relationship between a chronic LC_{50} and an EC_{20}
Forage Fish	Goldfish	0.49	0.12	Birge *et al.* (1979b)	from U.S. EPA AQUIRE (2003); lowest value for fathead minnow and goldfish based on 7-d LC_{50} (mortality); derived TRV using a factor of 4 based on an empirical relationship between a chronic LC_{50} and an EC_{20}

Aquatic Receptor	Cadmium (mg/L)				
	Test Species	**LC/EC_{50}**	**Toxicity Reference Value**	**Reference**	**Comments**
Aquatic Plants	*Myriophyllum*	7.4	3.0	Stanley (1974)	from U.S. EPA AQUIRE; EC_{50} (population) 32-d; used an EC_{20} from linear extrapolation
Phytoplankton	*Scenedesmus*	0.008	0.003	Fargasova (1994)	from U.S. EPA AQUIRE; EC_{50} (population) 12-d; used an EC_{20} from linear extrapolation
Benthic Invertebrates	*Chironomus sp.*	1.2	0.12	Rehwoldt *et al.* (1973)	from U.S. EPA AQUIRE; LC_{50} (mortality) 96-hr; derived TRV using a factor of 10 based on an empirical relationship between an acute LC_{50} and EC_{50}
Zooplankton	*Daphnia sp.*	--	$7.5x10^{-4}$	Elnabarawy *et al.* (1986)	from Suter and Tsao (1996); lowest chronic test EC_{20} – life-cycle tests
Predator Fish	Rainbow Trout	--	0.002	Carlson *et al.* (1982)	from Suter and Tsao (1996); lowest chronic test EC_{20} – early life stage tests
Forage Fish	Fathead Minnow	0.09	0.009	Hall *et al.* (1986)	from IPCS; 96-hr LC_{50}; derived TRV using a factor of 10 based on an empirical relationship between an acute LC_{50} and EC_{50}

TABLE 4.1-1 (Cont'd)
AQUATIC TOXICITY REFERENCE VALUES FOR ECOLOGICAL RISK ASSESSMENT

Aquatic Receptor	Chromium – based on Cr (VI) (mg/L)				
	Test Species	LC/EC_{50}	Toxicity Reference Value	Reference	Comments
Aquatic Plants	*Microcystis aeruginosa*	--	0.002	U.S. EPA (1985)	from Suter and Tsao (1996); lowest chronic test EC20 – early life stage test
Phytoplankton	--	--	--	--	No data available
Benthic Invertebrates	--	--	--	--	No data available
Zooplankton	*Daphnia* sp.	--	0.006	Mount (1982)	from Suter and Tsao (1996); lowest chronic test EC20 – early life stage test
Predator Fish	Rainbow Trout	--	0.073	Sauter *et al*. (1976)	from Suter and Tsao (1996); lowest chronic test EC20 – early life stage test
Forage Fish	--	--	--	--	No data available

Aquatic Receptor	Cobalt (mg/L)				
	Test Species	LC/EC_{50}	Toxicity Reference Value	Reference	Comments
Aquatic Plants	--	--	--	--	no data available
Phytoplankton	*Chlorella*	0.55	0.27	Coleman *et al.* (1971)	from MOE (1996); EC_{50} 21-d; derived EC_{25} by linear extrapolation
Benthic Invertebrates	Cyclops	16	1.6	Baudouin and Scoppa (1974)	from MOE (1996); this value is the lowest value for all test invertebrate species and is the same as the value for a mayfly. LC_{50} 48-hr (acute); derived TRV using a factor of 10 based on an empirical relationship between an acute LC_{50} and an EC_{20}
Zooplankton	*Daphnia* sp.	--	0.005	Kimball (n.d.)	from Suter and Tsao (1996); lowest chronic test EC_{20} – 28-d life-cycle tests; used as TRV
Predator Fish	Rainbow Trout	0.47	0.12	Birge (1978)	from MOE (1996); LC_{50} embryos 28-d; derived TRV using a factor of 4 based on an empirical relationship between a chronic LC_{50} and an EC_{20}
Forage Fish	Goldfish	0.81	0.20	Birge (1978)	from MOE (1996); lowest value of fathead minnow, tilapia, stickleback and goldfish. LC_{50} 7-d; derived TRV using a factor of 4 based on an empirical relationship between a chronic LC_{50} and an EC_{20}

TABLE 4.1-1 (Cont'd)
AQUATIC TOXICITY REFERENCE VALUES FOR ECOLOGICAL RISK ASSESSMENT

Aquatic Receptor	Copper (mg/L)				
	Test Species	LC/EC_{50}	Toxicity Reference Value	Reference	Comments
Aquatic Plants	*Myriophyllum* sp.	0.30	0.15	Stanley (1974)	from U.S. EPA AQUIRE; EC_{50} (population) 32-d; derived EC_{25} by linear extrapolation
Phytoplankton	*Chlorella* sp.	--	0.0040	Franklin *et al.* (2000)	from EC/HC PSL2 (2003); EC_{20} for cell growth, cell division and cell size for *Chlorella* and *Selenastrum*
Benthic Invertebrates	*Calanus* sp.	0.8	0.080	Hooftman *et al.* (1989)	from U.S. EPA AQUIRE; LC_{50} (mortality) 72-hr; derived TRV using factor of 10 based on an empirical relationship between an acute LC_{50} and an EC_{20}
Zooplankton	*Daphnia* sp.	0.02	0.0041	Mastin and Rodgers (2000)	LC_{50} (mortality) 48-hr; derived TRV using a factor of 10 based on an empirical relationship between an acute LC_{50} and an EC_{20}; used CCME value since EC_{20} was less than CCME value
Predator Fish	Bass	--	0.0086	U.S. DOE RAIS (2005)	EC_{25} bass population surface water screening benchmark
Forage Fish	--	--	0.0041	--	used CCME value since calculated EC_{20} was less than CCME value

Aquatic Receptor	Lead (mg/L)				
	Test Species	LC/EC_{50}	Toxicity Reference Value	Reference	Comments
Aquatic Plants	*Myriophyllum* sp.	363	182	Stanley (1974)	from U.S. EPA AQUIRE; EC_{50} (population) 32-d; derived EC_{25} by linear extrapolation
Phytoplankton	*Chlorella* sp.	--	0.63	U.S. EPA (1985)	from Suter and Tsao (1996); EC_{20} (growth inhibition)
Benthic Invertebrates	*Hyallela azteca*	0.018	0.002	Mackie (1989)	96-hr LC_{50}; derived TRV using a factor of 10 based on an empirical relationship between an acute LC_{50} and EC_{50}
Zooplankton	*Daphnia* sp.	--	0.02	Chapman *et al.* (1980)	from Suter and Tsao (1996); lowest chronic EC_{20} 21-d tests
Predator Fish	Rainbow Trout	--	0.028	Sauter *et al.* (1976)	from Suter and Tsao (1996); lowest chronic test EC_{20}
Forage Fish	Goldfish	1.66	0.42	Birge *et al.* (1979b)	from U.S. EPA AQUIRE; lowest value of fathead minnow, snakehead catfish and goldfish LC_{50} (mortality) 7-d; derived TRV using a factor of 4 based on an empirical relationship between a chronic LC_{50} and an EC_{20}

TABLE 4.1-1 (Cont'd)
AQUATIC TOXICITY REFERENCE VALUES FOR ECOLOGICAL RISK ASSESSMENT

Aquatic Receptor	Manganese (mg/L)				
	Test Species	**LC/EC_{50}**	**Toxicity Reference Value**	**Reference**	**Comments**
Aquatic Plants	*Lemna minor*	31	15.5	Wang (1986)	from U.S. EPA AQUIRE; 4-d EC_{50} (growth); derived EC_{25} by linear extrapolation
Phytoplankton	*Spirostomum ambiguum*	92.8	46	Nalecz-Jawecki and Sawicki (1998)	from U.S. EPA AQUIRE; 24-hr EC_{50} (deformation); derived EC_{25} by linear extrapolation
Benthic Invertebrates	*Dugesia gonocephala*	46	46	Palladini *et al*. (1980)	from U.S. EPA AQUIRE; 8-d NOEC (locomotion)
Zooplankton	*Daphnia magna*	4.7	2.4	Baird *et al*. (1991)	lowest value from U.S. EPA AQUIRE; 48-hr EC_{50} (immobility); derived EC_{25} by linear extrapolation
Predatory Fish	Rainbow Trout	2.91	0.73	Birge (1978)	lowest value from U.S. EPA AQUIRE; 28-d LC_{50} (mortality); derived TRV using a factor of 4 based on an empirical relationship between a chronic LC_{50} and an EC_{20}
Forage Fish	Goldfish	8.22	2.1	Birge (1978)	from U.S. EPA AQUIRE; 7-d LC_{50} (mortality); derived TRV using a factor of 4 based on an empirical relationship between a chronic LC_{50} and an EC_{20}

Aquatic Receptor	Molybdenum (mg/L)				
	Test Species	**LC/EC_{50}**	**Toxicity Reference Values**	**Reference**	**Comments**
Aquatic Plants	--	--	--	--	no data available
Phytoplankton	*Chlorella* sp.	50	5.0	Sakaguchi *et al.* (1981)	from U.S. DOC NTIS (1989); assumed EC_{50} (growth) 96-hr; derived TRV using a factor of 10 based on an empirical relationship between an acute LC_{50} and an EC_{50}
Benthic Invertebrates	*Tubifex tubifex*	29	14.5	Khangarot (1991)	96-hr EC_{50} (immobilization); derived EC_{20} by linear extrapolation
Zooplankton	*Daphnia* sp.	--	0.45	Kimball (n.d.)	from Suter and Tsao (1996); lowest chronic test EC_{20}, 28-d life-cycle test
Predator Fish	Rainbow Trout	0.73	0.2	Birge *et al.* (1979b)	from U.S. EPA AQUIRE; LC_{50} (mortality) 28-d; derived TRV using a factor of 4 based on an empirical relationship between a chronic LC_{50} and an EC_{20}
Forage Fish	Goldfish	60	15	Birge (1978)	from CCME (1999); lowest toxicity value of fathead minnow, bluegill sunfish and goldfish. 7-d LC_{50}; study results had a large CI; derived TRV using a factor of 4 based on an empirical relationship between a chronic LC_{50} and an EC_{20}

TABLE 4.1-1 (Cont'd)
AQUATIC TOXICITY REFERENCE VALUES FOR ECOLOGICAL RISK ASSESSMENT

Aquatic Receptor	Nickel (mg/L)				
	Test Species	**LC/EC_{50}**	**Toxicity Reference Value**	**Reference**	**Comments**
Aquatic Plants	*Lemna* sp.	0.45	0.22	Wang (1986)	From U.S. EPA AQUIRE; EC_{50} (growth) 4-d; derived EC_{25} by linear extrapolation
Phytoplankton	*Selenastrum capricornutum*	--	0.03	Chao and Chen (2000)	Based on an EC_{10} of 0.016 mg/L for 10% growth reduction in batch tests; derived EC_{20} by linear extrapolation
Benthic Invertebrates	*Chironomus* sp.	8.6	0.86	Rehwoldt *et al.* (1973)	From U.S. EPA AQUIRE; LC_{50} (mortality) 96-hr; derived TRV using a factor of 10 based on an empirical relationship between an acute LC_{50} and an EC_{20}
Zooplankton	*Daphnia* sp.	--	0.06	Munzinger (1990)	From Suter and Tsao (1996); lowest chronic test EC_{20}
Predator Fish	Rainbow Trout	--	0.08	Nebeker *et al.* (1985)	From Suter and Tsao (1996); lowest chronic test EC_{20}, early life stage test
Forage Fish	Fathead Minnow	2.9	0.29	Lind *et al.* (1978)	From U.S. EPA AQUIRE; LC_{50} (mortality) 96-hr; derived TRV using a factor of 10 based on an empirical relationship between an acute LC_{50} and an EC_{20}

Aquatic Receptor	Silver (mg/L)				
	Test Species	**LC/EC_{50}**	**Toxicity Reference Value**	**Reference**	**Comments**
Aquatic Plants	--	--	--	--	no data available
Phytoplankton	--	--	--	--	no data available
Benthic Invertebrates	--	--	--	--	no data available
Zooplankton	*Daphnia* sp.	$5.8x10^{-3}$	$5.8x10^{-4}$	Erickson *et al*. (1998)	96-h LC_{50} (mortality); derived benchmark using a factor of 10 based on an empirical relationship between an acute LC_{50} and an EC_{20}.
Predator Fish	Rainbow Trout	$9.2x10^{-3}$	$9.2x10^{-4}$	Erickson *et al*. (1998)	96-h LC_{50} (mortality); derived benchmark using a factor of 10 based on an empirical relationship between an acute LC_{50} and an EC_{20}.
Forage Fish	Goldfish	$1.04x10^{-2}$	$1.04x10^{-3}$	Erickson *et al*. (1998)	96-h LC_{50} (mortality); derived benchmark using a factor of 10 based on an empirical relationship between an acute LC_{50} and an EC_{20}.

TABLE 4.1-1 (Cont'd)
AQUATIC TOXICITY REFERENCE VALUES FOR ECOLOGICAL RISK ASSESSMENT

Aquatic Receptor	Selenium (mg/L)				
	Test Species	**LC/EC_{50}**	**Toxicity Reference Value**	**Reference**	**Comments**
Aquatic Plants	*Lemna* sp.	1.7	0.9	Jenner *et al.* (1993)	from U.S. EPA AQUIRE; EC_{50} (population) 14-d; derived EC_{25} by linear extrapolation
Phytoplankton	*Scenedesmus* sp.	--	0.1	Vocke *et al.* (1980)	from Suter and Tsao (1996); chronic 14-d EC_{20} for reduced growth
Benthic Invertebrates	*Chironomus* sp.	1.8	0.2	Ingersoll *et al.* (1990)	from U.S. EPA AQUIRE; LC_{50} (ITX) 48-hr; derived TRV using a factor of 10 based on an empirical relationship between an acute LC_{50} and an EC_{20}
Zooplankton	Daphnia sp.	--	0.04	Johnston (1987)	from Suter and Tsao (1996); lowest chronic test EC_{20}, 28-d; derived an EC_{25} value from empirical equation provided in Suter and Tsao (1996)
Predator Fish	Rainbow Trout	--	0.05	Goettl and Davies (1976)	from Suter and Tsao (1996); lowest chronic test EC_{20}, early life stage tests
Forage Fish	Fathead Minnow	0.6	0.2	Halter *et al.* (1980)	from U.S. EPA AQUIRE; LC_{50} (mortality) 14-d; derived TRV using a factor of 4 based on an empirical relationship between a chronic LC_{50} and an EC_{20}

Aquatic Receptor	Zinc (mg/L)				
	Test Species	**LC/EC_{50}**	**Toxicity Benchmark**	**Reference**	**Comments**
Aquatic Plants	*Myriophyllum* sp.	21.6	10.8	Stanley (1974)	from U.S. EPA AQUIRE; EC_{50} (growth) 32-d; derived EC_{25} by linear extrapolation
Phytoplankton	*Selenastrum* sp.		0.04	Bartlett and Rabe (1974)	from Suter and Tsao (1996); 7-d test EC_{20} for growth inhibition
Benthic Invertebrates	*Chironomus* sp.	1.13	0.6	Phipps *et al.* (1995)	from U.S. EPA AQUIRE; LC_{50} (mortality) 10-d; used LC_{25}
Zooplankton	*Daphnia* sp.		0.04	Chapman *et al.* (1980)	from Suter and Tsao (1996); lowest chronic value, life-cycle tests
Predator Fish	Rainbow Trout		0.06	Spehar (1976)	from Suter and Tsao (1996); lowest chronic test EC_{20}
Forage Fish	Fathead Minnow	0.238	0.1	Norberg and Mount (1985)	from U.S. EPA AQUIRE; LC_{50} (mor) 7-d; derived an EC_{25} value based on empirical equation obtained from data provided in Suter (1993)

Note: LC_{50} – Lethal concentration that results in mortality to 50% of population in short-term acute exposure tests.
EC_{50} – Effects concentration that inhibits the growth rate on reproductive success of species in long-term chronic exposure tests.
Toxicity Reference Value – Inhibitory concentration (EC_{20} or EC_{25} value) that affects growth or reproductive success in 20 to 25% of species population.

As discussed above, the toxicity reference values for selenium presented in Table 4.1-1 are for exposure to water only. Exposures for bioaccumulative constituents such as selenium can occur through pathways other than water alone and may be related primarily to the diet. Because it is recognized that selenium has the ability to bioaccumulate through aquatic food webs, comparisons to other toxicity reference values are also made for selenium, some of them are based on fish tissue concentrations. In light of this, toxicity reference values that will also be used in this assessment for determining potential adverse effects from exposure to selenium are presented in Table 4.1-2.

TABLE 4.1-2
SELENIUM LEVELS OF CONCERN FOR FISH AND WILDLIFE

Source or Tissue Residue	**Effects Concentration**	**Affected Receptors**	**Toxic Effect**	**Source**
Water	> 2 to 5 µg/L [a]	Fish and Waterfowl	Reproductive failure or mortality due to food-chain bioconcentration	Lemly and Smith (1987)
Whole Body Tissue Residue	≥ 7.9 µg/g (dw) or 2.4 µg/g (ww)	Fish	Reduced survival following water and dietary exposure	Lemly (1993a) in U.S. EPA (2004b)

The U.S. EPA (2004) draft selenium aquatic life criterion provides a threshold effects concentrations which is equivalent to a No Observable Effect Concentration (NOEC) of 7.91 µg/g dw. Recent research studies completed by the University of Saskatchewan on northern fish species suggest that an effects criteria (EC_{20}) of 14 µg/g dw based on whole-fish tissue concentrations is a more appropriate benchmark. This value is supported by other research in Alberta and British Columbia that demonstrates NOEC or EC_{15} values at >14 µg/g dw for other northern fish species. A conversion is needed to convert the whole body tissue concentration into a muscle concentration since this is one of the tissues that are generally used in monitoring programs. It is generally found that the muscle concentrations are approximately 25 to 30% higher than whole body concentrations. The equation that converts whole body concentrations to muscle concentrations is based on a large number of fish samples and is as follows:

$$\text{Se conc (whole body)} = \exp\left(0.13 + 0.89 \ln\left(\text{Se conc (muscle)}\right)\right)$$

Therefore, a whole body tissue concentration of 7.9 µg/g (dw) converts into a muscle concentration of 8.8 µg/g (dw). Using a moisture content ranging between 75 to 80% results in a selenium muscle concentration of 2.2 to 1.8 µg/g (ww). Therefore, a value of 2 µg/g (ww) will be used in this assessment.

Additionally, studies performed by Lemly *et al*. (1993b) and Bryson *et al.* (1985) suggest that populations that have been historically exposed to selenium may not be as sensitive as the fish studied in the Lemly (1993a) study. Lemly (1998) indicates that selenium concentrations in muscle at levels of 1.8 µg/g (ww) (based on a moisture content of 85%) results in no effects levels in fish. This is similar to the value of 2 µg/g (ww) selected in this assessment.

4.1.1.2 Sediment Toxicity Evaluations

The potential ecological effects of sediment contamination at the Keno Hills mine site will be addressed in part through the examination of potential effects on benthic invertebrates. In contrast to the approach outlined above to assess the ecological risks to aquatic species from exposure to COPC in the water column, the sediment toxicity evaluation involved comparison of measured and/or predicted levels of COPC in sediments to sediment quality guidelines.

Table 4.1-3 outlines selected sediment toxicity benchmarks reported in the literature for those COPC for which guidelines have been established.

The Canadian Environmental Quality Guidelines for metals in sediments provide what are designated Interim Sediment Quality Guidelines (ISQGs) and Probable Effect Levels (PELs). In narrative description, an ISQG represents the concentration below which adverse biological effects are expected to occur rarely. A PEL defines the level above which adverse effects are expected to occur frequently (*i.e.*, more than 50% of adverse effects occur above the PEL, or above which adverse effects are usually or always observed). The CCME acknowledges the associative basis of the guidelines and maintains that the use of sediment quality guidelines in exclusion of other information (such as background concentrations of naturally occurring substances and biological tests) can lead to erroneous conclusions.

The theoretical toxicity benchmarks from Thompson *et al*. (2005) are specific to uranium-bearing regions of Canada (*e.g.*, northern Saskatchewan and northern Ontario) and are considered Canadian Nuclear Safety Commission (CNSC) working reference values. These values are considered applicable to sites in northern Canada.

TABLE 4.1-3
SEDIMENT QUALITY TOXICITY BENCHMARKS

Constituent	CCME 2003		Thompson *et al.* 2005	
	ISQG	PEL	LEL	SEL
Arsenic	5.9	17	9.8	346.4
Copper	3.7	197	22.2	268.8
Lead	35	91.3	36.7	412.4
Molybdenum	-	-	13.8	1238.5
Nickel	18[b]	35.9[b]	23.4	484
Selenium	-	-	1.9	16.1
Zinc	123	315	-	-

Note: "-" no data available
ISQG - interim sediment quality guideline PEL - probable effect level
LEL - lowest effect level SEL - severe effect level
b – guideline under review by CCME.

4.1.1.3 Terrestrial Wildlife Toxicity Reference Values

To determine possible effects on terrestrial ecological receptors, Lowest Observable Adverse Effect Level (LOAEL) toxicity reference values and No Observable Adverse Effect Level (NOAEL) toxicity reference values will be used. NOAELs are generally used for screening level type assessments whereas LOAELs are used to determine potential effects on ecological species since more specific assumptions have been made to obtain a more realistic estimate of COPC exposure (Sample *et al.*, 1996).

For terrestrial mammals, there is a lack of data available on the individual terrestrial mammals. In the absence of species specific toxicity data, data for laboratory animals (usually mice and rats) are generally used. For mammals, the data are generally available for mice and rats. For avian receptors, the test species are generally ducks or chicks. The general consensus in the risk assessment community is that scaling up from the body weight of the laboratory animal to the test animal species is no longer necessary. Thus, the TRVs from laboratory species are directly applicable to wildlife. In this assessment, it is proposed that there will be no scaling of laboratory TRVs.

The background information for the toxicity reference values developed for the test species are provided in Table 4.1-4 for mammals and Table 4.1-5 for birds. These tables include test species, study duration and toxicological endpoint for those COPC for which toxicity data are available.

TABLE 4.1-4
SUMMARY OF TOXICITY DATA FROM LABORATORY ANIMAL STUDIES

COPC	**Antimony**	**Arsenic**	**Cadmium**	**Chromium**	**Cobalt**	**Copper**	**Lead**	**Manganese**
Source of Reference values	Sample *et al*. (1996)	Sample *et al*. (1996)	Sample *et al.* (1996)	Sample *et al.* (1996)	ATSDR (2001)	Sample *et al*. (1996)	Sample *et al*. (1996)	Sample *et al*. (1996)
Original Reference	Schroeder *et al.* (1968)	Schroeder and Mitchener (1971)	Sutou *et al.* (1980)	MacKenzie *et al.* (1958)	Pedigo *et al*. (1988)	Auerlich *et al.* (1982)	Azar *et al*. (1973)	Laskey *et al*. (1982)
Chemical Species	Antimony Potassium Tartrate	Arsenite	Cadmium Chloride	Cr^{+6} as $K_2Cr_2O_4$	Cobalt Chloride	Copper Sulphate	Lead Acetate	Manganese Oxide
Test Species	Mouse	Mouse	Rat	Rat	Mouse	mink	rat	rat
Body Wt. (g)	30	30	303	350	35	1000	350	350
Study Duration	lifetime (>1 yr)	3 generations (>1 yr)	6 weeks	1 year	13 weeks	357 days	3 generations (>1 yr)	From gestation to 224 days
Endpoint	Lifespan, longevity	Reproduction	Reproduction	Body weight and food consumption	Reproduction	Reproduction	Reproduction	Reproduction
Comments	The study was carried out during a critical life stage and is considered to be chronic exposure.	Study carried out during critical life stage – taken as chronic exposure.	The study was carried out through mating and gestation and was considered to be chronic exposure.	Study carried out during critical life stage – taken as chronic exposure.	Study carried out during critical life stage – taken as chronic exposure.	Study carried out during critical life stage – taken as chronic exposure.	Study carried out during critical life stage – taken as chronic exposure.	The study was carried out during a critical life stage and is considered to be chronic exposure
Logic	Median lifespan was reduced among female mice exposed to the 5 ppm dose level and the study considered exposure throughout the entire lifespan, this dose was considered to be a chronic LOAEL. NOAEL derived by applying an uncertainty factor of 0.1 to the LOAEL.	Mice displayed declining litter sizes with each successive generation at a dose of 1.26 mg/kg-d and thus this dose was considered to be a chronic LOAEL.	No adverse effects were observed at the 1 mg/kg-d dose level. At the 10 mg/kg-d dose level, fetal implantations were reduced by 28%, fetal survivorship was reduced by 50% and fetal resorptions increased by 400%.	No significant differences were observed at any dose level studied and the study considered exposure over 1 year, the maximum dose was considered to be a chronic NOAEL.	Reversible testicular degeneration was observed at 20 mg/kg/d which was considered the LOAEL.	The survival of kits at 25 ppm was actually higher than the controls and this level was taken to be the NOAEL. At 50 ppm the percentage mortality in kits was increased and this was considered to be the LOAEL.	None of the Pb exposure levels affected the pregnancy rate, live birth rate or other reproductive indices. But, 1000 ppm exposure gave reduced offspring wt. and produced kidney damage in young - LOAEL. NOAEL100ppm	Decreased pregnancy percentage and fertility noted at the highest dose of 3550 ppm, while other reproductive parameters were not affected. This was taken as the LOAEL. No effects seen at 1100 ppm or lower and therefore NOAEL.
NOAEL (mg/kg/d)	**0.125**	**-**	**1**	**3.28**	**5**	**11.7**	**8**	**88**
LOAEL (mg/kg/d)	**1.25**	**1.26**	**10**	**-**	**20**	**15.14**	**80**	**284**

TABLE 4.1-4 (Cont'd)
SUMMARY OF TOXICITY DATA FROM LABORATORY ANIMAL STUDIES

COPC	Molybdenum	Nickel	Silver	Selenium	Zinc
Source of Reference values	Sample *et al.* (1996)	Sample *et al.* (1996)	ATSDR (1990)	Sample *et al.* (1996)	Sample *et al.* (1996)
Original Reference	Schroeder and Mitchener (1971)	Ambrose *et al.* (1976)	Walker (1971)	Rosenfeld and Beath (1954)	Schlicker & Cox (1968)
Chemical Species	Molybdate	Nickel Sulphate Hexahydrate	Silver Nitrate	Potassium Selenate	Zinc Oxide
Test Species	Mouse	Rat	Rat	Rat	Rat
Body Wt. (g)	30	350	350	350	350
Study Duration	3 generations (>1 yr)	3 generations (>1 yr)	14 days	1 year through 2 generations	Days 1 to 16 of gestation
Endpoint	Reproduction	Reproduction	Mortality	Reproduction	Reproduction
Comments	Study carried out during critical life stage – taken as chronic exposure.	Study carried out during critical life stage – taken as chronic exposure.	This study was based on short term exposure	Study carried out during critical life stage – taken as chronic exposure.	The study was carried out during a critical life stage and is considered to be chronic exposure
Logic	Mice displayed reduced reproductive success with a high incidence of runts.	No adverse effects observed over 3 generations at a concentration of 500 ppm. Therefore this value considered to be a NOAEL. At 1000 ppm reduced offspring body weights observed. Thus considered LOAEL	A 2-week study on rats exposed to silver nitrate in their drinking water resulted in the death of 3 out of 12 test animals. Test animals were observed to drastically decrease their drinking water intake and appeared listless and poorly groomed. Lethality was not observed in the lower dose group.	No adverse effects on reproduction at a concentration of 1.5 mg/L. This value is the NOAEL. At 2.5 mg/L the number of second-generation young was reduced by 50% among females. This value is the LOAEL.	No effects seen at 2000 ppm Zn therefore NOAEL. At 4000 ppm there was increased rates of fetal resorption and reduced fetal growth rates.
NOAEL (mg/kg/d)	**-**	**40**	**181.2**	**0.2**	**160**
LOAEL (mg/kg/d)	**2.6**	**80**	**362.4**	**0.33**	**320**

TABLE 4.1-5
SUMMARY OF TOXICITY DATA FROM LABORATORY BIRD STUDIES

COPC	Antimony	Arsenic	Cadmium	Chromium	Cobalt	Copper	Lead	Manganese
Source of Reference values	*No toxicity reference values available.*	Sample *et al*. (1996)	Sample *et al.* (1996)	Sample *et al.* (1996)	*No toxicity reference values available*	Sample *et al*. (1996)	Sample *et al*. (1996)	Sample *et al.* (1996)
Original Reference		USFWS (1964)	White and Finley (1978)	Haseltine *et al*. (1985)		Mehring *et al*. (1960)	Edens *et al.* (1976)	Laskey and Edens (1985)
Chemical Species		Arsenite	Cadmium chloride	Cr^{+3} as $CrK(SO_4)_2$		Copper Oxide	Lead Acetate	Manganese Oxide
Test Species		mallard duck	Mallard duck	Black duck		1 day old chicks	Japanese quail	Japanese Quail
Body Weight (g)		1000	1153	1250		534	150	150
Study Duration		128 d (>10 wks)	90d (>10wks)	10 months		10 wks	12 wks	75 d (>10 wks)
Endpoint		Mortality	Reproduction	Reproduction		Growth, mortality	Reproduction	Growth, aggressive behaviour
Comments		The study was carried out over a period greater than 10 weeks and is considered to be chronic exposure.	The study was carried out over a period greater than 10 weeks during reproduction and is considered to be chronic exposure.	The study was carried out over 10 weeks and is considered to be chronic exposure.		The 10-week study was considered to be chronic in duration.	The study was carried out over 10 weeks and is considered to be chronic exposure.	The study was carried out over longer than 10 weeks and was considered to be chronic.
Logic		Over 128 days, at a dose of 100 ppm sodium arsenite, the ducks experienced no mortality, and thus this value is considered to be the NOAEL. A dose of 250 ppm resulted in 12% mortality and is considered to be a chronic LOAEL.	At a dose of 15.2 ppm, no change in egg production was observed therefore this is considered to be a NOAEL. At 210 ppm, there was a significant decrease in egg production.	While duckling survival was reduced at the 50 ppm dose level, no significant differences were observed at the 10 ppm Cr^{+3} dose level. Because the study considered exposure throughout a critical lifestage (reproduction), the dose 50 ppm dose was considered to be a chronic LOAEL and the 10 ppm dose was considered to be a chronic NOAEL.		Consumption of copper up to 570 ppm had no effect on the growth of chicks and was considered to be the NOAEL. At 749 ppm there was a 30% reduction in growth and a 15% mortality. This level is considered to be the LOAEL.	Reproduction not impaired by the 10 ppm dose, but egg hatching success reduced at the 100 ppm dose. Therefore 10 and 100 ppm are considered the NOAEL and LOAEL, respectively.	The study noted no effect on growth in birds fed 5056 ppm Mn in the diet. Aggressive behaviour was noted to be reduced by 25 to 50% relative to controls but this was not considered an adverse effect. NOAEL was taken as 977 mg/kg-d.
NOAEL (mg/kg/d)		**5.135**	**1.45**	**1**		**47**	**1.13**	**977**
LOAEL (mg/kg/d)		**12.84**	**20**	**5**		**61.7**	**11.3**	**-**

TABLE 4.1-5 (Cont'd)
SUMMARY OF TOXICITY DATA FROM LABORATORY BIRD STUDIES

COPC	Molybdenum	Nickel	Silver	Selenium	Zinc
Source of Reference values	Sample *et al*. (1996)	Sample *et al.* (1996)	*No toxicity reference values available.*	Sample *et al.* (1996)	Sample *et al.* (1996)
Original Reference	Lepore and Miller (1965)	Cain and Pafford (1981)		Heinz *et al*. (1987)	Stahl *et al.* (1990)
Chemical Species	Sodium Molybdate	Nickel Sulphate		Sodium Selenite	Zinc sulphate
Test Species	chicken	mallard duckling		mallard duck	White leghorn Hens
Body Weight (g)	1500	782		1000	1935
Study Duration	21 d	90 d (>10 wks)		78 d (>10 wks)	44 weeks (>10 wks)
Endpoint	Reproduction	Mortality, growth, behaviour		Reproduction	Reproduction
Comments	The study was carried out through the reproductive cycle and is considered to be chronic exposure.	The study was carried out over 10 weeks and was considered to be chronic.		The study was carried out through the reproductive cycle and is considered to be chronic exposure.	The study was carried out over 10 weeks and was considered to be chronic
Logic	Embryonic viability was reduced to zero at the lowest dose of 500 ppm and this was considered to be a LOAEL.	Consumption of up to 77.4 ppm Ni in diet did not increase mortality or decrease growth and is thus the NOAEL. At 1069 ppm there was a 70% mortality which is considered to be a LOAEL.		Consumption of 1, 5 and 10 ppm had no effect on weight and survival of adults but 10 and 25 ppm resulted in significantly larger frequency of lethally deformed embryos as compared to 1 or 5 ppm. Therefore 10 ppm is the LOAEL and 5 ppm is the NOAEL.	No adverse effects seen at 228 ppm Zn therefore NOAEL. At 2028 ppm Zn a 20% decrease in egg hatchability over controls was observed. Therefore LOAEL.
NOAEL (mg/kg/d)	**-**	**77.4**		**0.5**	**14.5**
LOAEL (mg/kg/d)	**35.3**	**107**		**1**	**130.9**

4.2 HUMAN HEALTH

The hazard assessment identifies what potential adverse effects are associated with the identified constituents, and what is the relationship between the magnitude of exposure and the probability of occurrence of adverse effects. In general, the hazard assessment uses results from animal (and when available human) studies to determine the likelihood of an adverse health effect occurring as a result of a given exposure. However, it should be noted that exposure above a TRV does not mean that an effect will occur, but instead means that there is an increased risk of an adverse effect occurring.

4.2.1 Toxicity Reference Values

Exposure to COPC is conventionally assessed against toxicity reference values (TRVs). Toxicity is the potential of a chemical to cause some type of damage, either permanent or temporary, to the structure or functioning of any part of the body. The toxicity depends on the amount of the chemical taken into the body (generally termed the intake or dose) and the length of time a person is exposed. Every chemical has a specific dose and duration of exposure that is necessary to produce a toxic effect in humans. Toxicity assessments generally involve the evaluation of scientific studies, based either on laboratory animal tests or on workplace exposure investigations, by a number of experienced scientists in a wide range of scientific disciplines in order to determine the maximum dose that a human can be exposed to without having an adverse health effect. Levels that are likely to result in no appreciable risks or no measurable adverse effects are known as exposure limits.

For many non-carcinogenic effects, protective biological mechanisms must be overcome before an adverse effect from exposure to the chemical is manifested. For this reason, scientists generally agree that there is a level (threshold) below which no adverse effects would be measurable or expected to occur. These toxicity reference values are generally called reference doses (RfDs), tolerable daily intakes (TDIs) or acceptable daily intakes (ADIs) and are generally derived by regulatory agencies such as Health Canada and the United States Environmental Protection Agency (U.S. EPA). These TRVs are usually expressed as the quantity of a chemical per unit body weight per unit time (mg/(kg-day)) and have generally been derived for sensitive individuals in the public using the most sensitive endpoint available. Additionally, these factors involve the incorporation of “safety factors” by regulatory agencies to provide additional protection for members of the public.

As discussed above, there are several regulatory sources that report TRVs. Some of the most used sources are Health Canada, U.S. EPA Integrated Risk Information System (IRIS) database, U.S. EPA health assessment reports (HEAST), U.S. EPA National Center for Environmental Assessment (NCEA), the World Health Organization (WHO) and the Agency for Toxic Substances and Disease Registry (ATSDR). Given that this assessment is within a Canadian jurisdiction, TRVs provided by Health Canada were preferentially selected for evaluation of the health impacts on people. The Toxicology Evaluation Section of the Health Products and Food Branch of Health Canada has published Tolerable Daily Intakes for a number of trace elements found in foodstuff. These values were also considered for use in these assessments. In addition, the U.S. EPA IRIS database is another major source for TRVs and was used to infill data gaps in the Health Canada database.

Carcinogenic TRVs

Carcinogenesis is generally assumed to be a "non-threshold" type phenomenon whereby it is assumed that any level of exposure to a carcinogen poses a finite probability of generating a carcinogenic response. Carcinogenic TRVs or slope factors are used to estimate an upper-bound lifetime probability of an individual developing cancer as a result of exposure to a particular level of a potential carcinogen. The carcinogenic TRV is, therefore, the lifetime cancer risk per unit of dose.

Arsenic exposure via the oral route is considered to be carcinogenic based on the incidence of skin cancers in epidemiological studies examining human exposure through drinking water (Tseng *et al.* 1968; Tseng 1977). A slope factor of 2.8 $(mg/(kg\ d))^{-1}$ was derived from these studies by Health Canada (2003). New data have become available that suggest that the risk of internal cancers due to ingestion of drinking water is greater than previously believed (Health Canada 2004). An evaluation was completed of the cancer potency indices for liver, lung, bladder and kidney cancers. The lifetime risks of cancer in the general population in Canada associated with ingestion of 1 µg/L of arsenic in drinking water were estimated to be 4.5×10^{-6} (based on kidney cancer in men) to 2.2×10^{-5} (based on lung cancer in women) (Health Canada 2004). Based on the 95% upper bound level, an oral slope factor of 1.2 $(mg/(kg\ d))^{-1}$ can be determined. Both of these values will be used in the assessments.

Cadmium and chromium are the only other COPC that exert carcinogenic effects. For these COPC, the effects are related to the inhalation pathway only.

Non Carcinogenic TRVs

The remaining COPC are considered to be non-classifiable with respect to human carcinogenicity, indicating that there are no human or animal data to indicate that they are carcinogens. For many non-carcinogenic effects, protective biological mechanisms must be overcome before an adverse effect is manifested from chronic exposure to a toxicant. This is known as a "threshold" concept. Non-carcinogens are often referred to as "systemic toxicants" because of their effects on the function of various organ systems.

Table 4.2-1 provides a summary of the some selected TRVs that will be used in the assessment depending on the identification of COPC. The value, toxicological endpoint and reference for each TRV are provided in the table.

TABLE 4.2-1
SELECTED TOXICITY REFERENCE VALUES FOR HUMAN RECEPTORS

	Pathway of Exposure	Carc. vs Non-Carc.[a]	Value	Units	Health Effect	Ref[b]
Antimony	oral	non-carc.	0.0004	mg/(kg-d)	Longevity, blood glucose and cholesterol	IRIS (U.S. EPA, 2008)
Arsenic	oral	carc.	2.8	$(mg/(kg\text{-}d))^{-1}$	Internal cancers (liver, lung, bladder, kidney)	Health Canada (2004)
	inhal.	carc.	28	$(mg/(kg\text{-}d))^{-1}$	Lung cancer (inhalation, human, occupational exposure)	Health Canada (2004)
Cadmium	oral	non-carc.	0.0008	mg/(kg-d)	Not provided	Health Canada (2004)
	inhal.	carc.	42.9	$(mg/(kg\text{-}d))^{-1}$	Not provided	Health Canada (2004)
Chromium (Total)	oral	non-carc.	0.001	mg/(kg-d)	Not provided	Health Canada (2004)
	inhal.	carc.	47.6	$(mg/(kg\text{-}d))^{-1}$	Not provided	Health Canada (2004)
Cobalt	oral	non-carc	0.02	mg/(kg-d)	Not provided	U.S. EPA National Centre for Environmental Assessment (NCEA 2002)
Copper	oral	non-carc.	0.03	mg/(kg-d)	Not provided	Health Canada (2004)
Lead	oral	non-carc.	0.0036	mg/(kg-d)	Not provided	Health Canada (2004)
Manganese	oral	non-carc.	0.14	mg/(kg-d)	Neurological effects	IRIS (U.S. EPA, 2008)
Molybdenum	oral	non-carc.	0.005	mg/(kg-d)	Increased uric acid levels	IRIS (U.S. EPA, 2008)
Nickel, metallic	oral	non-carc.	0.02	mg/(kg-d)	Decreased body and organ weights (oral exposure, rats)	IRIS (U.S. EPA, 2008)
Silver	oral	non-carc.	0.005	mg/(kg-d)	Argyria	IRIS (U.S. EPA, 2008)
Selenium	oral	non-carc.	0.0007	mg/(kg-d)	Not provided	Health Canada (2002)
Zinc	oral	non-carc.	0.3	mg/(kg-d)	Nutritional effects	IRIS (U.S. EPA, 2008)

TABLE 4.2-1 (Cont'd)
SELECTED TOXICITY REFERENCE VALUES FOR HUMAN RECEPTORS

	Pathway of Exposure	Carc. vs Non-Carc.[a]	Value	Units	Health Effect	Ref[b]
PHC F1 Aliphatic	oral	non-carc.	5.0	mg/(kg-d)	Neurotoxicity	TPHCWG (1997); CCME (2000)
	inhalation	non-carc.	18.4	mg/m^3	Neurotoxicity	TPHCWG (1997); CCME (2000)
PHC F1 Aromatic	oral	non-carc.	0.04	mg/(kg-d)	Decreased body weight	TPHCWG (1997); CCME (2000)
	inhalation	non-carc.	0.20	mg/m^3	Decreased body weight	TPHCWG (1997); CCME (2000)
PHC F2 Aliphatic	oral	non-carc.	0.1	mg/(kg-d)	Hepatic and haematological changes	TPHCWG (1997); CCME (2000)
	inhalation	non-carc.	1.0	mg/m^3	Hepatic and haematological changes	TPHCWG (1997); CCME (2000)
PHC F2 Aromatic	oral	non-carc.	0.04	mg/(kg-d)	Decreased body weight	TPHCWG (1997); CCME (2000)
	inhalation	non-carc.	0.20	mg/m^3	Decreased body weight	TPHCWG (1997); CCME (2000)
PHC F3 Aliphatic	oral	non-carc.	2.0	mg/(kg-d)	Hepatic (foreign body) Granuloma	TPHCWG (1997); CCME (2000)
	inhalation	non-carc.	na	--	--	--
PHC F3 Aromatic	oral	non-carc.	0.03	mg/(kg-d)	Nephrotoxicity	TPHCWG (1997); CCME (2000)
	inhalation	non-carc.	na	--	--	--

Notes:
na - not available
[a] Carcinogenic (non-threshold) vs. non-carcinogenic (threshold) effect.
[b] IRIS Integrated Risk Information System on-line database (U.S. EPA, 2005).

5.0 CONCEPTUAL TABLE OF CONTENTS

The conceptual Table of Contents for the report will follow the outline given below. If there is not enough available information on the terrestrial environment then the risk assessment focus will be on the aquatic environment; however, a terrestrial assessment will also be done for animals with an aquatic-based diet.

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REFERENCES

Agency for Toxic Substances and Disease Registry (ATSDR) 2001. *Toxicological Profile for Cobalt.*

Ambrose, A.M., P.S. Larson, J.F. Borzelleca, and G.R. Hennigar, Jr. 1976. *Long-term Toxicologic Assessment of Nickel in Rats and Dogs*. J. Food. Sci. Tech. 13: 181-187.

American Bear Association (ABA) 2003. *The American Bear Association, Vince Shute Wildlife Sanctuary*. Internet: http://www.americanbear.org/Habitat%20-%20Home%20Range.htm.

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

Auerlich, R.J., R.K. Ringer, M.R. Bleavins, *et al.* 1982. *Effects of Supplemental Dietary Copper on Growth, Reproductive Performance and Kit Survival of Standard Dark Mink and the Acute Toxicity of Copper to Mink.* J. Animal Sci. 55: 337-343. {cited in Sample *et al.* 1996}.

Azar, A., H.J. Trochimowicz and M.E. Maxwell 1973. *Review of Lead Studies in Animals Carried out at Haskell Laboratory: Two-Year Feeding Study and Response to Hemorrhage Study.* In: Environmental Health Aspects of Lead: Proceedings International Symposium. Barth et al., eds. Commission of European Communities. pp. 199-210. {cited in Sample *et al.* 1996}.

Baird, D.J., I. Barber, M. Bradley, A.M.V.M Soares and P. Calow 1991. *A Comparison Study of Genotype Sensitivity to Acute Toxic Stress Using Clones of Daphnia magna Straus*. Ecotoxicol. Environ. Saf. 21(3): 257-265. {cited in U.S. EPA AQUIRE 2003}.

Baudouin, M.F. and P. Scoppa 1974. *Acute Toxicity of Various Metals to Freshwater Zooplankton.* Bull. Environ. Contam. Toxicol. 12: 745-751. {cited in MOE 1996}

Birge, W.J., J.E. Hudson, J.A. Black and A.G. Wasterman 1979a. *Embryo-larval Bioassays on Inorganic Coal Elements and In Situ Biomonitoring of Coal-waste Effluents*. In: Surface mining and fish/wildlife needs in the eastern United States, D.E. Samuel, J.R. Stauffer, C.H. Hocutt, and W.T. Mason Jr., eds. pp. 97-104. Fish and Wildlife Service, Office of Biological Services. FWS/OBS-78/81. U.S. Government Printing Office, Washington, D.C. {cited in CCME 1999}.

Birge, W.J., J.A. Black, and A.G. Westerman 1979b. *Evaluation of Aquatic Pollutants Using Fish and Amphibian Eggs as Bioassay Organisms*. In: S.W. Nielsen, G. Migaki, and D.G. Scarpelli (Eds.), Symp. Animals Monitors Environ. Pollut., 1977, Storrs, CT 12: 108-118. {cited in U.S. EPA AQUIRE }.

Birge, W.J. 1978. *Aquatic Toxicology of Trace Elements of Coal and Fly Ash*. In: Energy and environmental stress in aquatic systems, J.H. Thorp and J.W. Gibbons, eds. Department of Energy Symposium Series CONF 771114. {cited in U.S. EPA AQUIRE, CCME 1999, MOE 1996, MOE 1991}.

Borgmann, U., R. Cove and C. Loveridge 1980. *Effects of Metal on the Biomass Production Kinetics of Freshwater Copepods*. Can. J. Fish. Aquat. Sci. 37: 567-575. {cited in CCME 1999}.

Brooke, L.T., D.J. Call, S.H. Poirier, C.A. Lindberg, and T.P. Markee 1986. *Acute Toxicity of Antimony III to Several Species of Freshwater Organisms*. Report to Battelle Memorial Research Institute. Center for Lake Superior Environmental Studies. University of Wisconsin-Superior. Superior, Wisconsin. {Cited In: MOEE 1996}.

Bryson, W.T., W.R.Garrett, M.A. Mallin, K.A. MacPherson, W.E. Partin, and S.E. Woock 1985. *Roxboro Steam Electric Plant Hyco Reservoir 1983 Bioassay Report*. Environmental Services Section. Carolina Power & Light Company. September 1985.

Cain, B.W. and E.A. Pafford 1981. *Effects Of Dietary Nickel On Survival And Growth Of Mallard Ducklings*. Arch. Environm. Contam. Toxicol 10: 737-745.

Call, D.J., L.T. Brooke, N. Ahmed and J.E. Richter 1983. *Toxicity and Metabolism Studies with EPA Priority Pollutants and Related Chemicals in Freshwater Organisms*. EPA-600/3-83-095. U.S. Environmental Protection Agency. Duluth, Minn. {cited in Suter and Tsao 1996}.

Call, D.J., L.T. Brooke, N. Ahmed and J.E. Richter 1983. *Toxicity and Metabolism Studies with EPA Priority Pollutants and Related Chemicals in Freshwater Organisms*. EPA-600/3-83-095. U.S. Environmental Protection Agency. Duluth, MN {cited in Suter and Tsao 1996}.

Cameco Corporation 2004. *Construction, Operation and Decommissioning Environmental Assessment Study Report*. January.

Canadian Council of Ministers of the Environment (CCME) 2003. *Canadian Environmental Quality Guidelines.* Includes updates to the Original 1999 Version.

Canadian Council of Ministers of the Environment (CCME) 2000. *Canada-Wide Standards for Petroleum Hydrocarbons (PHCs) in Soil: Scientific Rationale*. Supporting Technical Document. December.

Canadian Council of Ministers of the Environment (CCME) 1999. *Canadian Water Quality Guidelines. Prepared by the Task Force on Water Quality Guidelines of the Canadian Council of Ministers of the Environment*. December

Canadian Council of Ministers of the Environment (CCME) 1996. *A Protocol for the Derivation of Environmental and Human Health Soil Quality Guidelines.* Report CCME EPC-101E, CCME. March.

Canadian Wildlife Service 2005. *Hinterland Who's Who.* www.hww.ca/index_e.asp

Carlson, A.R. *et al.* 1982. *Cadmium and Endrin Toxicity to Fish in Water Containing Mineral Fibers.* EPA-600/3-82-053. U.S. Environmental Protection Agency, Duluth, Minn. {cited in Suter and Tsao 1996}.

Chao, M-R and C. Chung-Yuan 2000. *No-Observed-Effect Concentrations in Batch and Continuous Algal Toxicity Tests.* Environ. Toxicol. Chem., 19(6), 1589-1596.

Chapman, G.A., S. Ota and F. Recht 1980. *Effects of Water Hardness on the Toxicity of Metals to Daphnia magna*. U.S. Environmental Protection Agency, Corvallis, Oregon. {cited in Suter and Tsao 1996}.

Coleman, R.D., R.L. Coleman and E.L. Rice 1971. *Zinc and Cobalt Bioconcentration and Toxicity in Selected Algal Species*. Bot. Gaz. 132: 102-109. {cited in MOE 1996}.

Doe, K.G., Parker, W.R., Ponsford, S.J. and Vaughan, J.D.A 1987. The acute and chronic toxicity of antimony to *Daphnia magna* and rainbow trout. Canadian Technical Report of Fisheries and Aquatic Sciences 1575:40-43.

Edens, F., W.E. Benton, S.J. Bursian, and G.W. Morgan 1976. *Effect of Dietary Lead on Reproductive Performance in Japanese Quail*, Coturnix coturnix japonica. Toxicol. Appl. Pharmacol. 38: 307-314. {cited in Sample *et al*. 1996}

Elnabarawy, M.T., A.N. Welter, and R.R. Robideau 1986. *Relative Sensitivity of Three Daphnid Species to Selected Organic and Inorganic Chemicals*. Environ. Toxicol. Chem. 5: 393-398. {cited in Suter and Tsao 1996}.

Environment Canada 1997. *Environmental Assessments of Priority Substances Under the Canadian Environmental Protection Act*. Guidance Manual Version 1.0, March.

Environment Canada/Health Canada (EC/HC PSL2) 2003. *Priority Substances List Assessment Report: Releases of Radionuclides from Nuclear Facilities (Impact on Non-Human Biota).*

Erickson, R.J., L.T. Brooke, M.D. Kahl, F.V. Venter, S.L. Harting, T.P. Markee, and R.L. Spehar 1998. *Effects of Laboratory Test Conditions on the Toxicity of Silver to Aquatic Organisms*. Environ.Toxicol.Chem. 17(4):572-578. {cited in U.S. EPA AQUIRE 2003}

Fargasova, A. 1994. *Comparative Toxicity of Five Metals on Various Biological Subjects*. Bull. Environ. Contam. Toxicol. 53(2): 317-324. {cited in U.S. EPA AQUIRE 2003}.

Franklin, N.M., J.L. Stauber, S.J. Markich and R.P. Lim 2000. *pH-dependent Toxicity of Copper and Uranium to a Tropical Freshwater Algae (*Chlorella *sp.)*. Aquatic Toxicology 48: 275-289. {cited in EC/HC PSL2 2003}.

Gensmer, R.W. and R.C. Playle 1999. *The Bioavailability and Toxicity of Aluminum in Aquatic Environments*. Critical Reviews in Environmental Science and Technology, 29(4): 315-450.

Goettl, J.P. Jr. and P.H. Davies 1976. *Water Pollution Studies*. Federal Aid Project F-33-R-11. Department of Natural Resources, Colorado Division of Wildlife. {cited in Suter and Tsao 1996}.

Gostomoski, F. 1990. *The Toxicity of Aluminum to Aquatic Species in the U.S. Environ.* Geochem. Health, 12: 51-54. {cited in Gensemer and Playle 1999}.

Hall, W.S., R.L. Paulson, L.W. Hall, and D.T. Burton 1986. *Acute Toxicity of Cadmium and Sodium Pentachlorophenate to Daphnids and Fish*. Bull. Environ. Contam. Toxicol. 37: 308-316. {cited in IPCS 135 1992}.

Halter, M.T., W.J. Adams, and H.E. Johnson 1980. *Selenium Toxicity to Daphnia magna, Hyallela azteca, and the Fathead Minnow in Hard Water*. Bull. Environ. Contam. Toxicol. 24(1): 102-107. {cited in U.S. EPA AQUIRE}.

Haseltine, S.D., L. Sileo, D.J. Hoffman, and B.D. Mulhern 1985. *Effects Of Chromium On Reproduction And Growth In Black Ducks.* {cited in Sample *et al.* 1996}.

Health Canada 2005. *Uranium – Classification and Assessment. Environmental and Workplace Health*. Last updated May 14, 2004. Accessed August 22, 2005. http://www.hc-sc.gc.ca/ewh-semt/pubs/water-eau/doc_sup-appui/uranium/index_e.html.

Health Canada 2004. *Arsenic in Drinking Water. Document for Public Comment.* Prepared by the Federal-Provincial-Territorial Committee on Drinking Water. Comment period ended May 17, 2005. November.

Health Canada 2003. *Federal Contaminated Site Risk Assessment in Canada Part II: Health Canada Toxicological Reference Values.* Version 1.0. Environmental Health Assessment Services. Safe Environments Program. Ottawa, Ontario. October 3.

Health Canada 2002. *Toxicological Reference Doses for Trace Elements. Food Directorate*. Last updated August 20, 2002.

Heinz, G.H., D.J. Hoffman, A.J. Krynitsky, and D.M.G. Weller 1987. *Reproduction in Mallards Fed Selenium*. Environ. Toxicol. Chem. 6: 423-433. {cited in Sample *et al*. 1996}.

Hooftman, R.N., D.M.M. Adema, and J. Kauffman-Van Bommel 1989. *Developing a Set of Test Methods for the Toxicological Analysis of the Pollution Degree of Waterbottoms*. Rep.No.16105, Netherlands Organization for Applied Scientific Research: 68 p.(DUT). {cited in U.S. EPA AQUIRE}.

Ingersoll, C.G., F.J. Dwyer, and T.W. May 1990. *Toxicity of Inorganic and Organic Selenium to* Daphnia magna *(Cladocera) and* Chironomus riparius *(Diptera).* Environ. Toxicol. Chem. 9(9): 1171-1181. {cited in U.S. EPA AQUIRE}.

Jenner, H. A., and J. P.M. Janssen-Mommen 1993. Duckweed Lemna minor *as a Tool for Testing Toxicity of Coal Residues and Polluted Sediments*. Arch. Environ. Contam. Toxicol. 25(1): 3-11. {cited in U.S. EPA AQUIRE}.

Johnston, P.A. 1987. *Acute Toxicity of Inorganic Selenium to Daphnia magna (Straus) and the Effect of Sub-Acute Exposure upon Growth and Reproduction*. Aquat. Toxicol. 10: 335-352. {cited in Suter and Tsao 1996}.

Kent Wildlife Trust (KWT) 2003. *About Beavers.* Internet: http://www.kentwildlife.org.uk/beaver/beavers.htm.

Khangarot, B.S. 1991. *Toxicity of Metals to a Freshwater Tubificid Worm, Tubifex tubifex (Muller)*. Bull. Environ. Contam. Toxicol. 46:906-912.

Kimball, G. n.d. *The Effects of Lesser Known Metals and One Organic to Fathead Minnows (Pimephales promelas) and Daphnia magna.* U.S. Environmental Protection Agency, Duluth, MN. {cited in Suter and Tsao 1996 and CCME 1999}.

Laskey, J.W., G.L. Rehnberg, J.F. Hein and S.D. Carter 1982. *Effects of Chronic Manganese (Mn_3O_4) Exposure on Selected Reproductive Parameters in Rats*. J. Toxicol. Environ. Health. 9: 677-687.

Laskey, J.W. and F.W. Edens 1985. *Effects Of Chronic High-Level Manganese Exposure on Male Behaviour in the Japanese Quail (Cotirnix coturnix japonica)*. Poult. Sci. 64: 579-584.

Lazareva, L.P. 1985. *Changes in Biological Characteristics of* Daphnia magna *from Chronic Action of Copper and Nickel at Low Concentrations.* Hydrobiol. J. 21: 69-52.

Lemly, A.D. 1998. *A Position Paper on Selenium in Ecotoxicology: A Procedure for Deriving Site-Specific Water Quality Criteria - Review*. Ecotoxicology and Environmental Safety 39, 1 – 9.

Lemly, A.D. 1993a. *Metabolic Stress During Winter Increases the Toxicity of Selenium to Fish*. Aquat.Toxicol. (Amsterdam) 27(1-2):133-158.

Lemly, A.D. 1993b. *Teratogenic Effects of Selenium In Natural Populations of Freshwater Fish*. Ecotoxicol. Environ. Safety 26(2):181-204.

Lemly, A.D. and G.J. Smith 1987. *Aquatic Cycling of Selenium: Implications for Fish and Wildlife.* United States Department of the Interior, Fish and Wildlife Service, Fish and Wildlife Leaflet 12.

Lepore, P.D. and R.F. Miller 1965. *Embryonic Viability as Influenced by Excess Molybdenum in Chicken Breeder Diets*. Proc. Soc. Exp. Biol. Med. 118: 155-157.

Lima, A.R. C. Curtis, D.E. Hammermeister, T.P. Markes, C.E. Northcott, L.T. Brooke 1984. *Acute and Chronic Toxicities of Arsenic (III) to Fathead Minnows, Flagfish, Daphnids, and an Amphipod*. Arch. Environ. Contam. Toxicol. 13: 595-601. {cited in Suter and Tsao 1996}.

Lind, D., K. Alto, and S. Chatterton 1978. *Regional Copper-Nickel Study*. Draft Report, Minnesota Environmental Quality Board St. Paul, M N:54. {cited in U.S. EPA AQUIRE}.

MacCracken, J.G., *et al.* 1997. *Habitat Relationships of Moose on the Copper River Delta in Coastal South-Central Alaska*. Wildlife Monographs 136: 5-52.

Mackie, G.L. 1989. *Tolerances of Five Benthic Invertebrates to Hydrogen Ions and Metals (Cd, Pb, Al)*. Archives of Environmental Toxicology. 18: 215-223.

Mackenzie, R.D., R.U. Byerrum, C.F. Decker, C.A. Hoppert, and R.F. Langham 1958. *Chronic Toxicity Studies, II. Hexavalent and Trivalent Chromium Administered in Drinking Water to Rats.* Am. Med. Assoc. Arch. Ind. Health 18: 232-234. {cited in Sample *et al*. 1996}.

Massachusetts Department of Environmental Protection (MADEP) 2002. *Technical Update: Calculation of Enhanced Soil Ingestion Rate.* Office of Research and Standards, MADEP, Boston, MA. http://www.state.ma.us/dep/ors/files/soiling.doc

Mastin, B.J. and J.H. Rodgers Jr. 2000. *Toxicity and Bioavailability of Copper Herbicides (Clerigate, Cutrine-Plus, and Copper Sulphate) to Freshwater Animals*. Arch. Environ. Contam. Toxicol. 39: 445-451.

Mehring, A.L. Jr., J.H. Brumbaugh, A.J. Sutherland, and H.W. Titus 1960. *The Tolerance of Growing Chickens for Dietary Copper*. Poult. Sci. 39: 713-719.

Mount, D.I. 1982. *Memorandum to Charles E. Spehar.* U.S. Environmental Protection Agency, Duluth, Minn. June 7.

Munzinger, A. 1990. *Effects of Nickel on Daphnia magna During Chronic Exposure and Alterations in the Toxicity to Generations Pre-exposed to Nickel*. Wat. Res. 24: 845-852. {cited in Suter and Tsao 1996}.

Nalecz-Jawecki, G.and J. Sawicki 1998. *Toxicity of Inorganic Compounds in the Spirotox Test: A Miniaturized Version of the Spirostomum ambiguum Test*. Arch. Environ. Contam. Toxicol. 34(1):1-5. {cited in U.S. EPA AQUIRE}.

Nebeker, A.V., C. Savonen and D.G. Stevens 1985. *Sensitivity of Rainbow Trout Early Life Stages to Nickel Chloride*. Environ. Toxicol. Chem. 4: 233-239. {cited in Suter and Tsao 1996}.

North Carolina State University (NCSU) 1995. *Ruffed Grouse*. Forest Stewardship. Internet: http://www.ces.ncsu.edu/nreos/forest/steward/www9.html.

Ontario Ministry of the Environment (MOE) 1996. *Scientific Criteria Document for the Development of a Provincial Water Quality Objective for Cobalt (Stable Isotope)*. October.

Palladini, G., V. Margotta, A. Carolei and M.C. Hernandez 1980. D*opamine Agonist Performance in Plenaria After Manganese Treatment*. Experientia (Basel) 36(4): 449-450. (Author Communication Used). {cited in U.S. EPA AQUIRE}.

Pedigo, N.G., W.J. George, M.B. Anderson 1988. *Effects of Acute and Chronic Exposure to Cobalt in Male Reproduction in Mice*. Reprod. Toxicol. 2: 45-53. {cited in ATSDR 2001}.

Phipps, G.L., V.R. Mattson, and G.T. Ankley 1995. *Relative Sensitivity of Three Freshwater Benthic Macroinvertebrates to Ten Contaminants. Arch. Environ. Contam. Toxicol*. 28(3): 281-286. {cited in U.S. EPA AQUIRE}.

Pierce, J.D., and J.M. Peck 1984. *Moose Habitat Use and Selection Patterns in North-Central Idaho*. Journal of Wildlife Management 48: 1335-1343.

Receveur, O., N. Kassi, H.M. Chan, P.B. Berti, H.V. Kuhnlein 1998. *Yukon First Nations' Assessment of Dietary Benefit/Risk*. Centre for Indigenous Peoples' Nutrition and Environment (CINE). McGill University. Ste-Anne-de-Bellevue, Quebec. May.

Rehwoldt, R., L. Lasko, C. Shaw, and E. Wirhowski 1973. *The Acute Toxicity of Some Heavy Metal Ions Toward Benthic Organisms*. Environ. Pollut. 62(1): 73-85. {cited in U.S. EPA AQUIRE}.

Resources Inventory Committee 1998. *Inventory Methods for Wolf and Cougar: Standards for Components of British Columbia's Biodiversity No. 34.* Prepared by the Ministry of the Environment, Lands and Parks Resources Inventory Branch for the Terrestrial Ecosystems Task Force Resources Inventory Committee. Published by the Province of British Columbia. Internet: http://srmwww.gov.bc.ca/risc/pubs/tebiodiv/wolfsandcougars/.

Richardson, G.M. 1997. *Compendium of Canadian Human Exposure Factors for Risk Assessment.* O'Connor Associates Environmental Inc.

Rock, T.W. 1992. *A Proposal for the Management of Woodland Caribou in Saskatchewan.* Wildlife Technical Report 92-3. Saskatchewan Natural Resources, Wildlife Branch. May.

Rosenfeld, I. and O.A. Beath 1954. *Effect of Selenium on Reproduction in Rats*. Proc. Soc. Exp. Biol. Med. 87: 295-297.

Sakaguchi, T., A. Nakajima, and T. Horikoshi 1981. *Studies on the Accumulation of Heavy Metal Elements in Biological Systems. XVIII. Accumulation of molybdenum by green microalgae*. European J. Appl. Microbiol. Biotechnol. 12: 84-89. {cited in U.S. DOC NTIS 1989}.

Sample, B.E., D.M. Opresko and G.W. Suter II 1996. *Toxicological Benchmarks for Wildlife: 1996 Revision*. U.S. Department of Energy. June.

Sauter, S., K.S. Buxton, K.J. Macek, and S.R. Petrocelli 1976. *Effects Exposure to Heavy Metals on Selected Freshwater Fish.* EPA-600/3-76-105. U.S. Environmental Protection Agency, Duluth, Minn. {cited in Suter and Tsao 1996}.

Schlicker, S.A. and D.H. Cox 1968. *Maternal Dietary Zinc, and Development and Zinc, Iron and Copper Content of the Rat Fetus*. J. Nutr. 95: 287-294.

Schroeder, H.A. and M. Mitchener 1971. *Toxic Effects of Trace Elements on the Reproduction of Mice and Rats*. Arch. Environ. Health. 23: 102-106. {cited in Sample *et al*. 1996}.

Schroeder, H.A., M. Mitchener, J.J. Balassa, M. Kanisawa and A.P. Nason 1968. *Zirconium, Niobium, Antimony, and Fluorine in Mine: Effects on Growth, Survival and Tissue Levels.* J. Nutr. 95: 95-101. {Cited in Sample *et al.* 1996}.

Spehar, R. L. 1976. *Cadmium and Zinc Toxicity to Flagfish, Jordanella floridae*. *J. Fish. Res. Board Can*. 33:1939–1945.

Stahl, J. L., J. L. Greger, and M. E. Cook 1990. *Breeding-Hen And Progeny Performance When Hens Are Fed Excessive Dietary Zinc.* Poult. Sci. 69: 259-263.

Stanley, R.A. 1974. *Toxicity of Heavy Metals and Salts to Eurasian Watermilfoil* (Myriophyllum spicatum L.). Arch. Environ. Contam. Toxicol. 2(4): 331-341. {cited in U.S. EPA AQUIRE}.

Stephan, C.E., D.I. Mount, D.J. Hansen, J.H. Gentile, G.A. Chapman, and W.A. Brungs 1985. *Guidelines for Deriving Numerical National Water Quality Criteria for the Protection of Aquatic Organisms and their Uses.* National Technical Information Service, EPA-440/5-86-001. U.S. Environmental Protection Agency, Washington, DC.

Suter and Tsao 1996. *Toxicological Benchmarks for Screening Potential Contaminants of Concern for Effects on Aquatic Biota: 1996 Revision*. U.S. Department of Energy. June.

Suter, G.W. II 1993. *Ecological Risk Assessment*. Lewis Publishers: Chelsea, Michigan, USA, pp. 1-538.

Sutou, S., K. Yamamoto, H. Sendota, K. Tomomatsu, Y. Shimizu and M. Sugiyama 1980. *Toxicity, Fertility, Teratogenicity, and Dominant Lethal Tests in Rats Administered.*

Thompson, P.A., J.A. Kurias and S.S. Mihok 2005. *Dervation and Use of Sediment Quality Guidelines fro Ecological Risk Assessement of Metals and Radionuclides Released to the Environment from Uranium Mining and Milling Activities in Canada.* Environmental Monitoring and Assessment 110 (1-3): 71-85. November.

Total Petroleum Hydrocarbon Criteria Working Group (TPHCWG) 1997. *Volume 4 – Development of Fraction Specific Reference Doses (RfDs) and Reference Concentrations (RfCs) for Total Petroleum Hydrocarbons (TPH).* Amherst Scientific Publishers, Amherst, Mass.

Tseng, W.-P. 1977. *Effects and Dose-response Relationships of Skin Cancer and Blackfoot Disease with Arsenic.* Environ. Health Perspect. 19: 109-119.

Tseng, W.-P., H.M. Chu, S.W. How, J.M. Fong, C.S. Lin and S. Yeh 1968. *Prevalence of Skin Cancer in an Endemic Area of Chronic Arsenicism in Taiwan.* J. Natl. Cancer. Inst. 40(3): 453-463.

United States Department of Commerce (U.S. DOC) National Technical Information Service (NTIS) 1989. *Molybdenum Hazards to Fish, Wildlife, and Invertebrates: a Synoptic Review*. U.S. Patuxent Wildlife Research Center, Laurel, M.D. August 1989.

United States Department of Energy (U.S. DOE) Risk Assessment Information System (RAIS) 2005. *Ecological Benchmark*. On-line database.

United States Environmental Protection Agency (U.S. EPA) 2008. *Integrated Risk Information System (IRIS) – On-line database*. http://www.epa.gov/iris/.

United States Environmental Protection Agency (U.S. EPA) 2004. *Draft Aquatic Life Water Quality Criteria for Selenium – 2004*. EPA-822-D-04-001. U.S. EPA, Washington D.C. Office of Water. November 12. http://www.epa.gov/waterscience/criteria/selenium/pdfs/complete.pdf

United States Environmental Protection Agency (U.S. EPA) 2002. *National Center for Environmental Assessment – NCEA – TRV for Cobalt.*

United States Environmental Protection Agency (U.S. EPA) 1985. *Ambient Water Quality Criteria for Lead – 1984*. EPA 440/5-84-027. U.S. EPA, Washington, D.C. {cited in Suter and Tsao 1996}.

United States Environmental Protection Agency (U.S. EPA) Aquatic Toxicity Information Retrieval Database (AQUIRE). On-line database. http://www.epa.gov/ecotox/.

United States Environmental Protection Agency (U.S. EPA) 1988. *Human Health Risk Assessment Protocol for Hazardous Waste Combustion Facilities*. Peer Review Draft. EPA 530-D-98-001.

United States Environmental Protection Agency (U.S. EPA) 1993. *Wildlife Exposure Factors Handbook.* EPA/600/R-93/187).

United States Fish and Wildlife Service (USFWS) 1964. *Pesticide-wildlife Studies, 1963: a Review of Fish and Wildlife Service Investigations During the Calendar Year*. FWS Circular 199.

Vocke, R.W., K.L. Sears, J.J. O'Toole, and R.B. Wildman 1980. *Growth Responses of Selected Freshwater Algae to Trace Elements and Scrubber Ash Slurry Generated by Coal-fired Power Plants*. Water. Res. 14: 141-150. {cited in CCME 1999 and Suter and Tsao 1996}.

Wakabayashi, M., R. Konno, and T. Nishiido 1988. *Relative Lethal Sensitivity of Two Daphnia Species to Chemicals*. Tokyo-to Kankyo Kagaku Kenkyusho Nenpo:126-128 (JPN). {cited in U.S. EPA AQURE}.

Walker F. 1971. *Experimental Argyria: A Model for Basement Membrane Studies*. Br J Exp Pathol 52:589-593.

Wang, W. 1986. *Toxicity Tests of Aquatic Pollutants by Using Common Duckweed*. Environ. Pollut. Ser. B Chem. Phys. 11(1): 1-14. {cited in U.S. EPA AQUIRE}.

White, D.H. and M.T. Finley 1978. *Uptake and Retention of Dietary Cadmium in Mallard Ducks*. Environ. Res. 17: 53-59.

Woodland 2005. *Gray Wolf Fact Sheet. Woodland Park Zoo.* http://www.zoo.org/educate/fact_sheets/wolf/wolf.htm