REPORT

Assessment of the Potential Lifetime Cancer Risks Associated with Exposure to Inorganic Arsenic among Indigenous People living in the Wood Buffalo Region of Alberta

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EXECUTIVE SUMMARY

At the request of Alberta Health and Wellness (AHW), the lifetime cancer risks that might be presented to indigenous people living in the Wood-Buffalo region from exposure to inorganic arsenic were examined. The request was prompted, in part, by the findings of a recent human health risk assessment (HHRA) completed by Golder Associates (2006) in support of a proposed industrial oil sands development project (*i.e.*, the Suncor Voyageur project), which suggested these people may be at increased risk of developing cancer during their lifetime as a result of arsenic exposure. In this regard, Golder Associates reported the "incremental lifetime cancer risk" (ILCR) attributable to arsenic exposure to be approximately 450 (*i.e.*, equivalent to 450 extra cases of cancer in a population of 100,000 people).[1] The reported ILCR was well above the "benchmark" value of 1 x 10^{-5} (*i.e.*, one extra cancer case in a population of 100,000 people) deemed to be acceptable by most leading regulatory authorities for the protection of public health. The reported ILCR also was much higher than the lifetime cancer risks from arsenic exposure reported historically as part of EIAs completed in support of oil sands projects. To test the veracity of the original risk estimates reported by Golder Associates, AHW retained CANTOX ENVIRONMENTAL INC. (CEI) to re-assess the potential lifetime cancer risks involved.

The Terms of Reference for the work were assigned by AHW, and were organized around four separate, but related, tasks. These tasks were:

- To complete a review of the recently published literature describing the health effects associated with arsenic exposure, with particular attention given to the cancer-causing potential of arsenic. The review was to include identification of the various "Exposure Limits" (*i.e.*, acceptable levels of exposure) for arsenic developed by leading scientific and/or regulatory authorities.
- To review the HHRA performed by Golder Associates (2006), with special attention given to the methodology used to predict the lifetime cancer risk(s) to people living in the Wood-Buffalo region from arsenic exposure. The review was to include consideration of the various assumptions used to calculate the original ILCR (*i.e.*, ≈ 450).
- To re-evaluate the potential cancer risk(s) that could result from arsenic exposure in the region applying methodology routinely used by CEI. The re-assessment was to include re-calculation of the lifetime cancer risks, with documentation of the assumptions used as well as discussion of the departures from the original calculations performed by Golder Associates (2006).
- To assess the potential lifetime cancer risks that could be presented to people living in the Wood-Buffalo region from arsenic exposure associated with the consumption of moose meat, venison, and cattail root based on arsenic levels measured in representative samples of these traditional food items gathered from the area as part of a recent sampling program commissioned by Alberta Health and Wellness (AHW 2006).

[1] The original HHRA completed by Golder Associates (2006) examined three development scenarios (designated "baseline", "application", and "planned development case" (PDC)). An incremental lifetime cancer risk (ILCR) of 452-453 was reported for the development scenarios.

Some direction was received from AHW in terms of the overall approach to be followed, namely:

- The re-assessment was to closely mimic the original assessment performed by Golder Associates in terms of overall design, with allowance for some level of refinement in order to better match the approach typically followed by CEI. To further ease comparison between the two assessments, the work also was to rely on the same information concerning arsenic levels in the environment used by Golder Associates as part of the original HHRA, with the option to: i) substitute information deemed to be more relevant; and/or, ii) supplement the information with additional data. In either case, the aim was to increase the robustness of the cancer risk estimate(s).
- Unlike conventional cancer risk assessments, the work was to consider not only the incremental lifetime cancer risk that could result from arsenic exposure contributed by future anthropogenic sources (including the proposed Suncor Voyageur project), but also the potential lifetime cancer risk presented by existing levels of arsenic in the environment, both naturally-occurring arsenic as well as arsenic contributed by already existing anthropogenic sources in the region.
- The work was to focus specifically on the potential lifetime cancer risks that could be presented to indigenous people living in the Wood-Buffalo region, with consideration given to the lifestyle and dietary habits of these individuals.
- The work was to focus on the cancer risks associated with exposure to arsenic *via* the oral route (*i.e.*, ingestion), with consideration given to the potential arsenic exposures that might result from the incidental ingestion of soil, the consumption of drinking water, traditional plant-foodstuffs (*e.g.*, berries, roots), game meat, and sport fish.
- The potential lifetime cancer risks were to be calculated on the basis of the cancer potency factors and corresponding Exposure Limits for arsenic (*i.e.*, acceptable level of exposure) developed by both the Contaminated Sites Division *and* the Water Quality and Health Bureau of Health Canada. These limits corresponded to Risk-Specific Doses (RsD) of 0.003 µg/kg BW/day and 0.006 µg/kg BW/day, respectively.

The work followed a conventional risk assessment paradigm, with the lifetime cancer risks expressed as Exposure Ratios (ERs) based on comparison of the estimated exposures to inorganic arsenic that might be received by an indigenous receptor across an 80-year lifetime against the set of Exposure Limits for arsenic recommended by Health Canada (2004; 2006). The ERs corresponded to the number of potential cancer cases attributable to the arsenic exposure that might occur among a population of 100,000 people. To assist in the interpretation of the significance of the findings, the predicted lifetime cancer risks also were expressed in terms of the actual contingent of indigenous people living in the Wood-Buffalo region based on recent census data compiled for communities in the area.

In keeping with the overall approach followed for the original HHRA performed by Golder Associates, the re-assessment examined the potential lifetime cancer risks that could be presented under three different development scenarios:

- A “baseline” scenario in which the potential lifetime cancer risk associated with existing sources of arsenic exposure in the Wood-Buffalo region was calculated. The baseline exposure represented a combination of exposure from naturally-occurring sources of arsenic in the environment and exposure from already existing anthropogenic sources in the region
- A “future” scenario in which the incremental lifetime cancer risk presented by arsenic exposure contributed by projected future development activities in the region was calculated. The scenario was based on an 80-year projection. Reliance was placed on the Suncor Voyageur application (Suncor 2005a,b) for information relating to the future development activities and the amounts of arsenic that might potentially be released into the environment.
- A “combined” scenario in which the lifetime cancer risk calculated for the baseline scenario and the incremental lifetime cancer risk contributed by the future scenario were added. The combined cancer risk was based on the sum total of arsenic exposures contributed by naturally-occurring sources, existing anthropogenic sources, and prospective future industrial sources in the region.

It is important to note that the above scenarios differed from those examined as part of the original assessment performed by Golder Associates. Specifically, whereas the original HHRA was structured following a conventional EIA “protocol” in terms of both the definition of the development scenarios examined as well as the terminology used to describe each scenario (*i.e.,* baseline, application and PDC), the re-assessment departed from convention and proceeded on a more generic basis, with less emphasis given to a specific proponent and/or application and different terminology assigned to each scenario (*i.e*., baseline, future and combined). In other words, both the nature and the nomenclature of the development scenarios differed between the two assessments. In addition, whereas, the original HHRA included a development scenario specific to the project under consideration (*i.e*., the so-called “application” scenario), the re-assessment did not focus on the incremental lifetime cancer risk associated with the Suncor Voyageur project *per se*, but rather was concerned with estimating the incremental lifetime cancer risk that could result from *all* projected future development activities in the region, including the Suncor Voyageur project (*i.e*., the “future” scenario). In other words, unlike the original HHRA, the re-assessment did not consider the Suncor Voyageur project on a stand-alone basis, but instead was deliberately designed to examine the potential incremental lifetime cancer risk associated with future development activities on a broader temporal and regional scale.

The principal findings that emerged from the re-assessment were:

- The ER values predicted for the baseline development scenario ranged from ≈ 17 to 33 (depending on the Exposure Limit used in the calculations), signifying that lifetime exposure to inorganic arsenic among indigenous people living in the Wood-Buffalo region *via* the exposure pathways examined potentially could contribute to ≈ 17 to 33 cases of cancer when calculated on a 100,000-person population basis. The “acceptability” of this potential lifetime cancer risk from a public health perspective cannot be determined following a conventional approach since an acceptable “benchmark” cancer risk level for exposure to background levels of carcinogens is not available for comparison.

When re-expressed in terms of the actual contingent of indigenous people living in the Wood-Buffalo region (*i.e.*, ≈ 8,000 people of indigenous descent), the number of cancer cases attributable to exposure to inorganic arsenic predicted to occur among the population under the baseline development scenario ranged from one to three (1 to 3), depending on the Exposure Limit used in the calculation. The adjusted numbers are shown in the table below.

Both sets of predicted numbers very likely represent over-estimates as a result of the conservatism incorporated into the re-assessment. When interpreted in the context of this conservatism, the significance of the numbers becomes questionable, with the findings suggesting that the current indigenous population is at little, if any, risk of developing cancer as a result of exposure to inorganic arsenic contributed by both naturally-occurring sources and existing anthropogenic sources in the region *via* the exposure pathways examined as part of the work.

- The ER values predicted for the future development scenario ranged from ≈ 1 to 2, signifying that lifetime exposure to inorganic arsenic contributed by future anthropogenic activities in the Wood-Buffalo region might potentially contribute to 1 to 2 extra cancer cases among indigenous people living in the area when calculated on a 100,000-person population basis. The predicted incremental lifetime cancer risks "bridged" the benchmark ILCR of 1×10^{-5} (*i.e.*, one extra cancer case in a population of 100,000 people) deemed to be acceptable by most leading scientific and regulatory authorities. More specifically, the ILCR calculated using the Exposure Limit recommended by the Water Quality and Health Bureau of Health Canada (*i.e.*, 0.006 µg/kg BW/day) was slightly below the comparison benchmark (*i.e.*, ER = 0.9); whereas, the ILCR determined using the limit recommended by the Contaminated Sites Division of Health Canada (*i.e.*, 0.003 µg/kg BW/day) was slightly above the comparison benchmark (*i.e.*, ER = 1.8).

  When re-expressed in terms of the actual contingent of indigenous people living in the Wood-Buffalo region (*i.e.*, ≈ 8,000 people of indigenous descent), the number of cancer cases attributable to exposure to inorganic arsenic predicted to occur among the population under the future development scenario was less than one (<1), regardless of the Exposure Limit used in the calculation. The adjusted numbers are provided in the table below.

  Both sets of numbers signify that the current indigenous population is at essentially no risk of developing cancer as a result of exposure to any inorganic arsenic that might be contributed by projected future anthropogenic activity in the region *via* the exposure pathways examined. Added confidence is provided by the conservatism incorporated into the re-assessment.

- The ER values predicted for the combined development scenario (calculated on the basis of the combined exposures determined for both the baseline *and* future scenarios) ranged from ≈ 18 to 35, closely mimicking those predicted for the baseline scenario itself (*i.e.*, ER = ≈ 17 to 33). The same pattern emerged following adjustment of the predicted cancer risks to reflect the actual contingent of indigenous people living in the Wood-Buffalo region, as shown in the table below. Both sets of findings signify that the lifetime cancer risks that could potentially result from exposure to inorganic arsenic among indigenous people living in the Wood-Buffalo region are dominated by the exposures contributed by already existing naturally-occurring and anthropogenic sources of arsenic in the region, with very little incremental risk presented by projected future anthropogenic activities.

Estimated Number of Cancer Cases Predicted to Occur Among the Indigenous Population of the Wood-Buffalo Region From Exposure to Inorganic Arsenic[(1)]

Estimate based on:	Estimated Number of Cancer Cases		
	Baseline Development Scenario	Future Development Scenario[(2)]	Combined Development Scenario
Exposure Limit recommended by Water Quality and Health Bureau of Health Canada (2006) (RsD = 0.006 μ/kg BW/day)	1 (1.36)	<1 (0.07)	1 (1.44)
Exposure Limit recommended by Contaminated Sites Division of Health Canada (2004) (RsD = 0.003 μ/kg BW/day)	3 (2.71)	<1 (0.15)	3 (2.86)

(1) Based on an estimated total indigenous population of 8,122 individuals (RMWB 2006). Values refer to the number of cancer cases that might potentially occur from exposure to inorganic arsenic *via* the complete set of exposure pathways examined (*i.e.*, all pathways combined). Values in parentheses correspond to the actual numerical ER values calculated after adjustment for the specific population number indicated above.

(2) Values refer specifically to the number of *extra* number of cancer cases that might potentially occur among the indigenous population as a result of projected future anthropogenic activities in the Wood-Buffalo region.

- Regardless of the development scenario examined, the predicted exposures to inorganic arsenic that might be received by indigenous people living in the Wood-Buffalo region were dominated by certain exposure pathways, notably the consumption of drinking water and the consumption of sport fish, which contributed up to 27% and up to31% of the total combined predicted exposure, respectively. Lesser, but still significant, contributions were revealed for the consumption of roots and other below-ground plants (as part of the consumption of traditional plant foodstuffs) and the consumption of game meat, depending on the development scenario assessed. The contribution from the remaining exposure pathways was negligible.

- Despite the general guidance received from AHW to design the re-assessment to match that of the original assessment completed by Golder Associates (2006) as much as possible, significant differences existed between the two assessments in the particulars surrounding the work. Many of the differences stemmed from the fact that each assessment was performed under different terms and for different reasons. Whereas the original assessment completed by Golder Associates was commissioned by Suncor Energy and formed part of the EIA performed in support of the Suncor Voyageur project (Suncor 2005b), the re-assessment completed by CEI was commissioned by AHW and was meant to serve as a “second opinion” of the lifetime cancer risks that might be presented to the indigenous people living in the Wood-Buffalo region from exposure to inorganic arsenic. On this basis alone, the scope and nature of the two assessments necessarily differed. The differences extended to the definition of the development scenarios examined, the manner in which the lifetime cancer risks were differentiated, the Exposure Limits used in the calculations, and the assumptions that were applied to accommodate the uncertainties surrounding the work. The net result of the differences was that the lifetime cancer risks predicted by CEI were significantly lower than those originally reported by Golder Associates across all development scenarios, especially in terms of the incremental lifetime cancer risks predicted for the future development case.

- It is important to note that the predicted lifetime cancer risk estimates (both unadjusted and adjusted) are population-based. They refer to the potential cancer risks from exposure to inorganic arsenic presented to the indigenous population of the Wood-Buffalo region as a whole. They do not refer to the potential lifetime cancer risks faced by an individual person. The circumstances governing the exposures to inorganic arsenic that might be received by an individual person are many and varied and were not captured as part of either the original assessment completed by Golder Associates nor the present re-assessment. Depending on circumstances, the individual lifetime cancer risks could be greater or lower than those predicted for the population as a whole. However, that said, the conservatism incorporated into the re-assessment was deliberately meant to ensure that the cancer risks would not be understated, thereby reducing the first possibility. In addition, the predicted lifetime cancer risks presented herein only relate to the exposures to inorganic arsenic received through the exposure pathways examined. Other sources of arsenic exposure exist (*e.g.* store-bought foods and beverages, tobacco smoke *etc.*) that may be relevant to the indigenous population within the Wood Buffalo Region, but these additional sources were not evaluated as part of this re-assessment. Exposure to inorganic arsenic from these sources could theoretically add to the estimated lifetime cancer risks.

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1.0 INTRODUCTION AND BACKGROUND

The toxicity of arsenic (As) continues to be the subject of considerable research, as well as some controversy, particularly in relation to its potential to cause cancer. Exposure to arsenic is widespread due to its presence in the earth's crust, with most human exposure arising from naturally-occurring arsenic in drinking water. The effects of arsenic exposure depend on many factors, including its chemical and physical form, the amount and duration of exposure, the nutritional status of those exposed, and the extent of co-exposure to other chemicals, notably carcinogens (*e.g.*, cigarette smoke, UV light). Arsenic does not commonly exist in its elemental form, but rather combines with other elements to form a number of inorganic and organic derivatives. The inorganic forms of arsenic (*e.g.*, arsenates, arsenites) are recognized to be more toxic than either elemental arsenic or its organic derivatives, both in terms of cancer-related and non-cancer-related health effects. Inorganic arsenic occurs in natural waters and drinking water as well as in foods (WHO 2001; Health Canada 2006a).

A recent human health risk assessment (HHRA) completed by Golder Associates (2006) in support of a proposed industrial oil sands development project (Suncor 2005b) suggested that people exposed to arsenic in the Wood-Buffalo region of Alberta may be at increased risk of developing cancer during their lifetime. An incremental lifetime cancer risk (ILCR) attributable to arsenic exposure was calculated by Golder Associates to be approximately 450 (*i.e.*, equivalent to 450 extra cases of cancer in a population of 100,000 people).[2]

In order to better understand and assess the significance of the elevated cancer risk from arsenic exposure predicted by Golder Associates (2006), Alberta Health and Wellness (AHW) retained CANTOX ENVIRONMENTAL INC. (CEI) to review the approach used by Golder to calculate the ILCR as well as to re-assess the potential cancer risk using refined methodology consistent with that typically used by CEI. The work was meant to serve as a "second opinion" of the lifetime cancer risk that could be presented to people living in the Wood-Buffalo region from arsenic exposure. The Terms of Reference for the work are outlined below.

[2] The original HHRA completed by Golder Associates (2006) examined three development scenarios (designated "baseline", "application", and "planned development case" (PDC)). An incremental lifetime cancer risk (ILCR) of 452-453 was reported, regardless of scenario (*i.e.*, the reported ILCR was the same for all three of the development scenarios examined).

2.0 TERMS OF REFERENCE

The Terms of Reference for the work were organized around four separate, but related, tasks. These tasks were:

- To complete a review of the recently published literature describing the health effects associated with arsenic exposure, with particular attention given to the cancer-causing potential of arsenic. The review was to include identification of the various "Exposure Limits" (*i.e.*, acceptable levels of exposure) for arsenic developed by leading scientific and/or regulatory authorities, including limits developed on the basis of the potential cancer risks presented from arsenic exposure.
- To review the HHRA performed by Golder Associates (2006), with special attention given to the methodology used to predict the lifetime cancer risk(s) to people living in the Wood-Buffalo region from arsenic exposure. The review was to include consideration of the various assumptions used to calculate the original ILCR (*i.e.,* ≈ 450).
- To re-evaluate the potential cancer risk(s) that could result from arsenic exposure in the region applying methodology routinely used by CEI. The re-assessment was to include re-calculation of the lifetime cancer risks, with documentation of the assumptions used as well as discussion of the departures from the original calculations performed by Golder Associates (2006).
- To assess the potential lifetime cancer risks that could be presented to people living in the Wood-Buffalo region from arsenic exposure associated with the consumption of moose meat, venison, and cattail root based on arsenic levels measured in representative samples of these traditional food items gathered from the area as part of a recent sampling program commissioned by Alberta Health and Wellness (AHW 2006).

In terms of the third task (*i.e.*, re-evaluation of the potential lifetime cancer risks(s) using CEI methodology), some direction concerning the overall approach to be followed was provided by AHW. The direction was as follows:

- Unlike conventional cancer risk assessments, the work was to consider not only the incremental lifetime cancer risk that could result from arsenic exposure contributed by future anthropogenic sources (including the proposed Suncor Voyageur project), but also the potential lifetime cancer risk presented by existing levels of arsenic in the environment, both naturally-occurring arsenic as well as arsenic contributed by already existing anthropogenic sources in the region.
- The work was to rely on the same information concerning arsenic levels in the environment used by Golder Associates (2006) as part of the original HHRA, with the option to: i) substitute information deemed to be more relevant; and/or, ii) supplement the information with additional data. In either case, the aim was to increase the robustness of the cancer risk estimate(s).

- The work was to focus on the potential lifetime cancer risks that could be presented to indigenous people living in the Wood-Buffalo region, with consideration given to the lifestyle and dietary habits of these individuals.
- The work was to focus on the cancer risks associated with exposure to arsenic *via* the oral route (*i.e.*, ingestion), with consideration given to the potential arsenic exposures that might result from the consumption of drinking water, traditional plant-foodstuffs (*e.g.*, berries, roots), game meat, and sport fish.
- The potential lifetime cancer risks were to be calculated on the basis of the cancer potency factors and corresponding Exposure Limits for arsenic developed by both the Contaminated Sites Division *and* the Water Quality and Health Bureau of Health Canada.

Specific details surrounding each of the tasks, including the principal findings and conclusions reached, are presented below. Additional information respecting the work can be found in the accompanying appendices.

3.0 SUMMARY OF CEI REVIEW OF GOLDER ASSOCIATES (2006)

As described in the Terms of Reference, CEI reviewed the arsenic HHRA calculations in Golder Associates (2006). It should be noted that one of the key differences between the Golder Associates (2006) and Suncor (2005a; 2005b) assessments and the CEI assessment is that the Golder and Suncor documents related to an Environmental Impact Assessment (EIA) for a specific application (Suncor Voyageur), while this report by CEI is intended to be a broader assessment of the potential cancer risks associated with oral arsenic exposure in the Wood Buffalo Region. The review presented below is not intended to be a comprehensive review of Suncor (2005a; 2005b) or the Golder Associates (2006) assessment. Rather, the review was a means of gaining understanding regarding the approach used by Golder Associates to determine the lifetime cancer risk of 450 in 100,000. Further, the intent of the review was to assist in CEI's evaluation of the potential cancer risks in the Region, and to help evaluate the various assumptions and input values that are associated with the Golder Associates (2006) cancer risk estimate. Details of the Golder Associates (2006) approach include:

- Lifetime cancer risks were labelled as "incremental" and were reported to be numerically similar (*i.e.*, ILCR = 452-453). This approach did not permit clear identification of the lifetime cancer risks associated with the Suncor Project alone, or the impact of all planned future projects in the area. As some reliance was placed upon measured data from the Wood Buffalo Region (*e.g.* moose meat, plants), the scenarios evaluated by Golder Associates included natural background sources of arsenic, and contributions from anthropogenic sources of arsenic. Thus, despite the labelling of the cancer risks as being representative of *incremental* lifetime cancer risks (ILCR), the lifetime cancer risks reported in Golder Associates (2006) were not truly incremental due to the inclusion of background sources in the exposure calculations. The cancer risks predicted by Golder Associates (2006) represent lifetime cancer risks, for which there is no 'acceptable' benchmark for comparison.
- The lifetime cancer risks calculated by Golder Associates (2006) were based on comparison of the predicted exposures to inorganic arsenic against a single Exposure Limit (*i.e.*, 0.003 μ/kg BW/day, the origin of which was not stated, but presumably was based on the slope factor of 2.8 $(mg/kg\text{-}d)^{-1}$ used by the Contaminated Sites Division of Health Canada).
- The cancer risk assessment of arsenic by Golder Associates (2006) was likely affected by the use of various assumptions related to the estimation of the potential exposures to inorganic arsenic. Some examples include:
  - For some exposure pathways (game meat, berries, traditional plants), reliance was placed upon empirical data sets where arsenic concentrations were determined to be below analytical detection limits. In such cases, Golder Associates (2006) assumed that arsenic was present in those samples at a level of one-half the detection limit, which is an acceptable approach. However, in some cases, the analytical detection limits appeared rather high. For example, the limit of detection for arsenic for the historical dataset (used by Golder Associates) was reported to be as high as 0.5 mg/kg for moose meat (weight basis unknown). The reliance upon non-detectable data for several food ingestion pathways may have significantly over-estimated arsenic exposures.
  - With the exception of fish, Golder Associates (2006) assumed that 100% of the arsenic content of the foods (game meat, traditional plants, berries) consisted of inorganic arsenic

as no adjustment appears to have been made. Fish was assumed to contain about 10% inorganic arsenic, however, the basis for this adjustment was not clear from the materials reviewed. The U.S. Agency for Toxic Substances and Disease Registry (2005) predicts that the proportion of inorganic arsenic in most foods is about 37%.

- It was assumed that exposure to inorganic arsenic *via* the incidental ingestion of soil could occur for 365 days per year, and consideration was not given to winter weather conditions where snow or ice may cover surface soils and thereby limit incidental ingestion.
- Raw, untreated surface water was assumed to be ingested for only six months of the year, however, no drinking water source for the remaining 6-months/year was included.
- Based upon the information available to CEI, a combination of wet weights and dry weights appear to have been used to express the arsenic content of foods.

Additional details regarding the assumptions relied upon by Golder Associates (2006), and the key differences between the CEI and the Golder Associates (2006) evaluation of arsenic cancer risks are presented in Section 7.0 of this report.

4.0 SUMMARY OF ARSENIC TOXICITY

A review of the recently published literature detailing the toxicity of arsenic was completed. Emphasis was given to identifying and summarizing information respecting the cancer-causing potential of arsenic, consistent with the principal objective of the work (*i.e.*, to estimate the lifetime cancer risks presented to people living in the Wood-Buffalo region from arsenic exposure). The review was meant to: i) provide an overview of the health effects associated with arsenic exposure, including cancer; ii) summarize the weight-of-evidence surrounding the carcinogenic potential of arsenic; iii) introduce the various Exposure Limits developed for arsenic by different scientific and regulatory authorities; iv) discuss the basis of the Exposure Limits, with emphasis on those limits intended to protect the general public against the cancer risks presented by arsenic exposure; and, v) provide an indication of the amounts and sources of arsenic in the environment to which humans might be exposed. The review relied primarily on secondary literature sources (*i.e.*, review articles and toxicological monographs prepared by a number of leading regulatory agencies). Key journal articles describing original scientific research also were retrieved and examined in order to provide more in-depth coverage, particularly in relation to the carcinogenic potential of arsenic. A detailed summary of the health effects and available Exposure Limits for arsenic can be found in Appendix A. Highlights are provided below.

The health effects that can result from arsenic exposure are very much dose- (*i.e.*, amount), time- and species-dependent. Unfortunately, there are no definitive animal models that can be used to adequately represent the toxicity of arsenic to humans because of differences between humans and animals in the metabolism of arsenic as well as in the nature of the toxic responses observed following exposure. Fortunately, arsenic has been well studied in human populations, even at environmentally-relevant concentrations, allowing for good understanding of its toxic potential, both in terms of cancer-related and non-cancer-related effects. However, conflicting findings from human studies have contributed to the challenge of assessing the health risks associated with arsenic exposure, particularly the cancer risks associated with low-dose exposures. Adding to the challenge is the fact that the nature and severity of the health outcomes attributed to arsenic exposure also are influenced by the type of arsenic involved (*i.e.*, elemental *vs*. organic *vs*. inorganic), the nutritional status of those exposed, and co-exposure to other chemicals, notably carcinogens (see above). As already indicated, inorganic arsenic is known to possess a higher order of toxicity than elemental or organic arsenic. The majority of adverse health outcomes reported in the literature refer to effects observed following exposure of people to inorganic arsenic.

Interestingly, there is no known biological function for arsenic; however, evidence suggests that it may act as an essential element, at least in some animal species. Depressed growth, reduced fertility and neonatal mortality have been demonstrated among animals fed arsenic-deficient diets, with the effects disappearing once the diets were replenished. Similarly, low serum arsenic levels among haemodialysis patients have been associated with an increased incidence of central nervous system (CNS) and vascular disease as well as elevated cancer rates. Some authorities have indicated that, despite the adverse health effects associated with over-exposure to inorganic arsenic, there is some evidence that the small amounts of arsenic typically found in the normal diet may confer some benefit(s) to health.

Acute Toxicity

Short-term or "acute" exposure to high levels of arsenic can lead to a classic set of symptoms characterized by gastrointestinal distress (*i.e.*, abdominal pain, vomiting, diarrhea), muscular numbness, weakness, cramping or pain, flushing of the skin, skin rash, and thickening of the skin on the palms of the hands or soles of the feet. These symptoms have been reported to follow acute oral exposure to as little as 1 milligram of arsenic per kilogram body weight per day (mg/kg BW/day). The minimum lethal oral dose for humans has been reported to range from 1 to 3 mg As/kg BW.

Chronic Toxicity

Long-term or "chronic" exposure to arsenic can lead to a number of different health outcomes, both cancer- and non-cancer-related. The most commonly observed response following chronic oral exposure is a pattern of skin changes presenting as hyper-pigmentation (*i.e.*, dark or light spots on the skin) and hyperkeratosis (*i.e.*, thickening of the skin, with small corns or warts), particularly affecting the palms of the hands and soles of the feet. In some cases, these changes can reportedly progress to skin cancer. Other non-cancer-related health outcomes attributed to chronic exposure include effects on the cardiovascular system (*e.g.*, abnormal heart rhythm), digestive system (*e.g.*, nausea and diarrhea), haematopoietic system (*e.g.*, decreased production of blood cells), respiratory system, and the musculature and connective tissues (*e.g.*, weakness or numbness in the hands and feet). The relationship between some of these latter effects and arsenic exposure is not well established. The effects have most commonly been reported to occur following long-term consumption of drinking water containing relatively high concentrations of arsenic (*i.e.*, at levels often above 100 microgram per litre (μg/L) and as high as 2,000 μg/L). Isolated reports of peripheral vascular disease associated with chronic exposure to arsenic also have appeared in the literature (*i.e.,* so-called "Blackfoot Disease"). In addition, a number of different cancers affecting the internal organs have been attributed to long-term exposure to arsenic (see below).

Reproductive Toxicity

The reproductive toxicity of arsenic is not well understood. Although studies conducted in Bangladesh, India, Taiwan, Chile and Hungary have variably reported positive associations between arsenic exposure *via* the drinking water and elevated rates of spontaneous abortions, stillbirths, preterm births, neonatal mortality and low birth weights, the evidence for arsenic-induced adverse reproductive outcomes is considered to be preliminary or inconclusive at this time. Not only is much of the evidence conflicting, but the positive associations noted in the studies were invariably confounded by co-exposure to other chemicals and/or lack of adjustment for maternal age, socioeconomic status, previous reproductive history, nutritional status, and other factors known to influence reproductive function and performance.

Carcinogenicity

It is generally accepted that arsenic can act as a human carcinogen, with positive associations between exposure to inorganic arsenic and a number of different types of cancer, including cancer of the skin, lung, bladder and kidneys, commonly reported in the literature. However, considerable debate surrounds the carcinogenic potency of arsenic, especially at low-dose levels, with different authorities offering differing opinions. Fuelling the debate are: i) conflicting findings from different investigations; ii) conflicting results from the re-analyses of the same investigations; iii) conflicting depictions of the shape of the dose-response curve; and, iv) conflicting evidence of the genotoxicity of arsenic. Uncertainty also surrounds the mechanism by which arsenic causes cancer. Finally, the limitations inherent to epidemiological studies in establishing cause-and-effect vis-à-vis chemical exposures and adverse health outcomes apply to much of the evidence surrounding the carcinogenicity of arsenic. This evidence is summarized in Appendix A. Evidence considered to be especially relevant to the present assessment includes:

a) Much of the evidence supporting an association between arsenic exposure and cancer is based on the findings of epidemiological investigations of cancer incidence among populations exposed to inorganic arsenic in the drinking water. A number of such investigations have been performed in different parts of the world, including South America, South-East Asia, Europe, and the United States. Conflicting results have emerged, with some studies showing strong positive associations between the consumption of drinking water containing inorganic arsenic and the occurrence of various cancers, whereas other investigations have found either no association or weak negative associations, possibly signalling a protective effect of arsenic exposure. The differences in findings might be attributable to: i) differences in the arsenic content of the drinking water, with some studies following populations exposed to very high arsenic levels (*i.e.*, >500 µg/L), whereas other investigations followed people exposed to relatively low amounts of arsenic (*i.e.*, <50 µg/L); ii) differences in exposure classification and stratification, with some studies relying on more refined estimates of exposure than other investigations (*i.e.*, segregation of exposures into "high" or "low" categories *vs*. no sub-grouping of exposures); iii) differences in the lifestyles of the populations followed, with variations in diet, prevalence of smoking, alcohol consumption, *etc.*, possibly influencing the study outcomes; iv) differences in the statistical methods used to analyze the findings; and, v) differences in environmental factors affecting the study populations, which may have contributed directly to the cancers observed.

b) Data originating from villages in south-eastern Taiwan are commonly cited as proof of an association between arsenic exposure and the occurrence of various types of cancer. These data form the basis of the Exposure Limits established by a number of regulatory authorities, including Health Canada (see below). However, consensus has not been reached within the general scientific community over the strength of the association as well as the relevance of the data to populations living outside of Taiwan. The original dataset purportedly showed significant associations between exposure to inorganic arsenic in drinking water and increased risk of skin cancer as well as cancers of the internal organs, namely the bladder, lungs and kidneys.

However, re-analyses of the data have led to conflicting opinions. Some investigators maintain that the original cancer risk estimates may have over-stated the carcinogenic potency of arsenic, especially at low-dose levels. Other investigators claim that the Taiwanese data are not relevant to North American populations because of differences in lifestyle and nutritional status, with the suggestion that the increased incidence of cancer observed in the Taiwanese villages was likely confounded by the villagers' poor diet and limited access to health care. Some authorities have refuted this claim. Still other investigators are convinced that some unknown environmental factor is largely responsible for the observed cancers because of the unique prevalence of "Blackfoot Disease" among the Taiwanese populations studied (*i.e.*, the occurrence of the disease is confined to the Taiwanese villages, suggesting that the adverse health outcomes affecting the population, including cancer, may have a unique etiology). Details surrounding the original Taiwanese dataset as well as the various re-analyses can be found in Appendix A.

c) In an attempt to address the uncertainty surrounding the carcinogenic potency of inorganic arsenic at low-dose levels in drinking water, a meta-analysis of grouped data from several independent epidemiological investigations performed in different areas of the world was recently completed, with an emphasis on discerning the relationship, if any, between low-level exposure to arsenic and the occurrence of bladder cancer (Exponent 2005). The analysis was meant, in part, to increase understanding of the cancer risk presented by inorganic arsenic in drinking water to North American populations since the arsenic content of most North American drinking water supplies is relatively low. The analysis revealed no significant association between low-level exposure to inorganic arsenic in drinking water and bladder cancer.

d) The mechanism(s) by which arsenic induces cancer is not well established, nor is the shape of the dose-response curve well understood, especially at lower doses. Mixed results have emerged from studies aimed at assessing the genotoxic potential of arsenic. The findings from some studies indicate that inorganic arsenic does not act as a bacterial mutagen, does not cause point mutations, and does not directly damage DNA. Other evidence suggests that inorganic arsenic is clastogenic (*i.e.*, exposure can result in damage or breakage of the chromosomes); however, the mechanism involved is not known. The clastogenicity may result from direct action of inorganic arsenic and/or its metabolites on the chromosomes, or alternatively, it may occur secondary to cytotoxicity, diminished DNA repair and/or oxidative stress. The former mechanism supports the position taken by some authorities that the carcinogenicity of inorganic arsenic is genetically-mediated and that arsenic qualifies as a "non-threshold" carcinogen, whereas the latter mechanisms suggest that the carcinogenicity may represent a high-dose phenomenon and that arsenic acts as a "threshold" carcinogen. The mixed findings have contributed to different approaches being taken by different authorities in assessing the carcinogenic potency of arsenic as well as in regulating exposures to arsenic (see below).

Exposure Limits

Exposure Limits refer to “safe” or acceptable levels of exposure to chemicals that are established by scientific and/or regulatory authorities to protect people from the adverse health effects that can follow over-exposure to the substances. The limits are based on an understanding of the toxicity of the chemical of interest, with careful consideration given to the concentration-time-response characteristics of the substance. Distinction is often made between cancer-related and non-cancer-related health outcomes when assigning an Exposure Limit to a chemical, with further distinction made between “threshold” and “non-threshold” carcinogens. Arsenic presents several challenges in this regard, which explains, in part, the diversity of limits that have been developed for this chemical. Certain exposure limits assigned to arsenic are meant to protect against the occurrence of health effects other than cancer, whereas other limits are strictly cancer-based. Since both the carcinogenic potency of arsenic and its mechanism of carcinogenic action are not fully understood, even the cancer-based limits show considerable diversity, not only in terms of the manner in which they were derived, but also in the level of protection afforded. A listing of the Exposure Limits developed for arsenic by a number of different authorities, including Health Canada, the World Health Organization (WHO), and the U.S. Environmental Protection Agency (U.S. EPA) is presented in Table 1. The basis of each limit is discussed in Appendix A.

Table 1. Summary of Oral Exposure Limits for Arsenic

Agency/Organization	Limit Type	Limit Value	Reference
Health Canada – Food Directorate	Tolerable Daily Intake (TDI)	1 μg/kg BW/day	Lo 2006
Health Canada – Water Quality and Health Bureau	“Negligible Risk Level”	0.3 μg/L[(1)]	Health Canada 2006b
Health Canada – Contaminated Sites Division	Slope Factor (SF)	2.8 $(mg/kg\ BW/day)^{-1}$ [(2)]	Health Canada 2004
World Health Organization (WHO)	Provisional Maximum Tolerable Daily Intake (PMTDI)	2 μg/kg BW/day	WHO 1983; JECFA/WHO 1989; WHO 2003
Health Council of the Netherlands	Tolerable Daily Intake (TDI)	1 μg/kg BW/day	HCON 2000
Agency for Toxic Substances and Disease Registry (ATSDR)	Minimal Risk Level (MRL)	MRL(acute): 5 μg/kg BW/day MRL (chronic): 0.3 μg/kg BW/day	ATSDR 2005
United States Environmental Protection Agency (U.S. EPA)	Reference Dose (RfD), Slope Factor (SF)	0.3 μg/kg BW/day 1.5 $(mg/kg\ BW/day)^{-1}$	U.S. EPA IRIS 1998

(1) Equivalent to a Risk-Specific Dose (RsD) of 0.006 μ/kg BW/day.

(2) Equivalent to a Risk-Specific Dose (RsD) of 0.003 μ/kg BW/day.

Based on direction received from Alberta Health and Wellness (see 2.0 TERMS OF REFERENCE), emphasis was given to the following Exposure Limits for the purpose of calculating the potential lifetime cancer risks associated with arsenic exposures in the Wood-Buffalo region:

- The "negligible risk level" of 0.3 µ/L developed by the Water Quality and Health Bureau of Health Canada (*i.e.*, equivalent to a Risk-Specific Dose of 0.006 µ/kg BW/day); and,
- The "slope factor" of 2.8 (mg/kg BW/day)$^{-1}$ determined by the Contaminated Sites Division of Health Canada (*i.e.*, equivalent to a Risk-Specific Dose of 0.003 µ/kg BW/day).

Both limits assume that inorganic arsenic acts as a non-threshold carcinogen. The choice of limits was based, in part, on: i) their jurisdictional relevance (*i.e.*, "made-in-Canada" limits); ii) the solid reputation of the responsible authority (*i.e.*, Health Canada); and, iii) the high degree of protection afforded. In addition, being cancer-based, the limits were properly suited to the issue at hand (*i.e.*, understanding the potential cancer risks that might be presented to people living in the Wood-Buffalo region from arsenic exposures). Further details concerning each of these limits are provided in Appendix A.

Summary

A number of key items having a direct bearing on the present assessment emerged from the above review of the toxicity of arsenic. These items not only influenced the design of the work (see 6.0 CANCER RISK ASSESSMENT (CEI METHODOLOGY)), but also the interpretation of the findings (see 8.0 DISCUSSION). They are:

1. Although it is generally recognized that arsenic can act as a human carcinogen, its carcinogenic potency is not fully understood, especially at low dose levels. These low dose levels are most relevant to much of the North American population.

2. Much of the evidence supporting an association between exposure to inorganic arsenic in drinking water and elevated cancer rates originates from epidemiological studies of people living in south-east Asia and South America. The relevance of these studies to North American populations has been questioned by some authorities. Most studies completed in North America have shown no association between arsenic levels in drinking water and the occurrence of cancer.

3. The mechanism by which arsenic causes cancer is not well established. Debate surrounds whether arsenic acts directly *via* a genetically-mediated mechanism, thereby qualifying arsenic as a "non-threshold" carcinogen … or through one or more non-genotoxic mechanisms, thereby qualifying arsenic as a "threshold" carcinogen. The difference in classification can have a considerable bearing on cancer risk estimates.

4. Mounting evidence suggests that the carcinogenicity of arsenic may represent a high-dose phenomenon only (*i.e.*, consistent with a threshold-type response). Some evidence even suggests that low doses of arsenic may confer a protective effect against the occurrence of cancer.

5. Exposure Limits derived on the basis of the premise that arsenic acts as a “non-threshold” carcinogen may overstate the carcinogenic potency of arsenic, especially if the limit assumes a linear dose-response at low dose levels. Cancer risks estimates calculated on the basis of these Exposure Limits may be exaggerated and should be interpreted with caution, and with full understanding of the conservatism incorporated into the limit. Both of the Exposure Limits chosen for use in the present re-assessment assume that arsenic acts as a “non-threshold” carcinogen.

5.0 ARSENIC CONTENT OF TRADITIONAL FOODSTUFFS – SUMMARY OF ALBERTA HEALTH AND WELLNESS (2006) SAMPLING PROGRAM

Among the tasks specified in the Terms of Reference developed for the work was the need to assess the potential cancer risks that could be presented to people living in the Wood-Buffalo region from arsenic exposures associated with the consumption of moose meat, venison, and cattail root based on arsenic levels measured in representative samples of these traditional food items gathered from the area as part of a recent sampling program commissioned by Alberta Health and Wellness (see 2.0 TERMS OF REFERENCE). The purpose of the sampling program was to add to the current understanding of the arsenic content of these traditional food items using improved analytical methodologies capable of detecting lower levels of arsenic than methods employed as part of earlier sampling exercises, including programs commissioned as part of EIAs completed in support of existing and/or proposed oil sands projects.

Samples were collected from the Wood-Buffalo region as well as from "control" (*i.e.*, reference) locations found both within and outside of the province. The latter locations were well removed from the Wood-Buffalo area and outside the zone of influence of any current oil sands activity. Samples were gathered during the Fall of 2006, and submitted to the Department of Laboratory Medicine and Pathology, Division of Analytical and Environmental Toxicology, University of Alberta for analysis. The results of the analyses were subsequently forwarded to CEI for use in the re-assessment of the potential lifetime cancer risks that could be presented to indigenous people consuming the food items as part of a traditional diet (see 6.0 CANCER RISK ASSESSMENT – CEI METHODOLOGY). The type and number of food items collected as part of the sampling program are listed in Table 2. The results of the analyses (expressed as the total arsenic content of the food items on a wet-weight basis) are summarized in Table 3. A more detailed listing of the total arsenic content of the cattail root samples, segregated according to the individual parts of the plant that were analyzed, can be found in Table 4. The variability surrounding the data is illustrated in Figure 1. More detailed information concerning the sampling locations and the analytical findings may be found in Appendix C. Highlights surrounding the findings are listed below, including an indication of the challenges presented by the data in terms of their usefulness for predicting the exposures to inorganic arsenic and the corresponding cancer risks that could result from the consumption of these traditional foodstuffs:

- All findings referred to the total arsenic content of the food items, with no indication of the inorganic arsenic content. As a result, for the purposes of the present work, the reported data necessarily had to be converted to the corresponding inorganic arsenic concentrations based on generic conversion factors available in the published literature for foods in general. The use of the generic conversion factors necessarily introduced uncertainty with respect to the estimation of the potential exposures to inorganic arsenic that might result from the consumption of the food items.

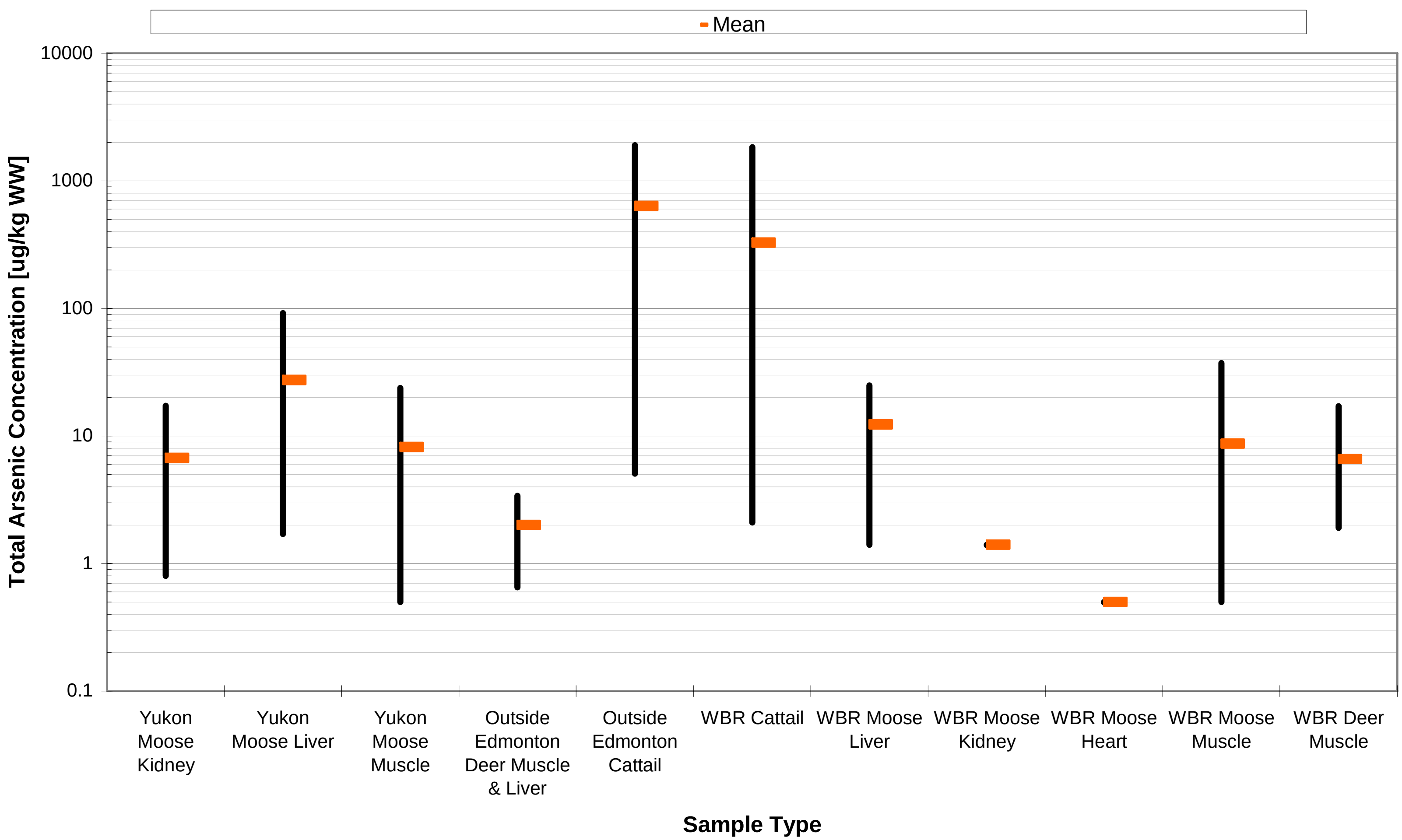

Figure 1. **Distribution of Measured Total Arsenic Concentrations (Range from the 5th Percentile to the 95th Percentile of the Data Distribution) for Various Cattail and Game Meat Samples (AHW 2006)**

- For certain of the food items, the sample size was limited and the results could not necessarily be considered representative. This limitation applied especially to the samples of vital organs (*i.e.*, heart, liver, kidney) obtained from the deer and/or moose, particularly among the animals harvested from locations other than the Yukon. In some cases, the sample size was restricted to one organ from a single animal only (see Table 2). As a result, for the purposes of the present work, estimates of the exposures to inorganic arsenic that might result from the consumption of game meat relied only on the measured arsenic concentrations in the muscle tissue obtained from deer and moose, for which reasonably comprehensive datasets were available.
- A good deal of variability was evident in the measured arsenic content of the food items from all locations (see Figure 1). This variability was especially obvious for the cattail root samples, with the average arsenic concentration measured in the individual plant tissues varying by up to 200-fold (see Table 4). In light of the variability, the total arsenic content of the food items was deemed to be best represented by the 95th percentile of the upper confidence limit of the mean measured value (95% UCLM)[3].
- Owing, in part, to the considerable variability surrounding the arsenic concentrations measured in the different parts of the cattail root samples, combined with a lack of understanding of the actual part(s) of the plant that are consumed as part of the traditional diet of the indigenous people living in the Wood-Buffalo region, for the purposes of the present work, the arsenic content of the cattail root was deemed to be best represented by the 95% UCLM calculated for *all* of the plant parts combined.
- Comparison of the arsenic content of the game meat samples across the different locations revealed no obvious pattern, with no clear distinction between the samples gathered from the Wood-Buffalo region and those collected from the reference locations in terms of the measured total arsenic concentrations. However, the comparison is necessarily limited because of the small sample sizes involved, especially for the Outside Edmonton[4] and Peace River/Grande Prairie locations (see Table 2). It is of some interest that the arsenic concentrations measured in the samples of moose muscle tissue obtained from the Wood-Buffalo region closely matched the measured arsenic content of the moose muscle samples gathered from the Yukon territory, both in terms of the average measured concentrations (*i.e.*, 0.0087 mg/kg wet-weight *vs*. 0.0082 mg/kg wet-weight) and the 95% UCLM values (*i.e.*, 0.020 mg/kg wet-weight *vs*. 0.0134 mg/kg wet-weight) (see Table 3).
- The cattail root samples from Wood-Buffalo region presented lower mean and maximum detected total arsenic concentrations than the Outside Edmonton samples. However, the 95%UCLM for the Wood-Buffalo samples was slightly higher than the 95% UCLM for the Outside Edmonton[4] samples. Comparison of the measured total arsenic concentration in the cattail root samples (*all* plant parts combined) gathered from the Wood-Buffalo region against the total arsenic concentrations measured in cattail root and rat root samples collected

[3] The 95% UCLM represents the upper estimate of the mean (average) in a dataset, with 95% coverage (Pagano and Gauvreau 1993).

[4] The 'Outside Edmonton' area from which the samples were collected included the Strathcona Wilderness Centre and locations near Westaskiwin Alberta.

from the same region and reported as part of previous EIAs performed in support of oil sands projects (Albian Sands Energy Inc. 2005; CNRL 2003; Golder Associates 2000; Imperial Oil 2006; Petro-Canada 2001; Shell Canada 2002; Suncor 2005) revealed the average concentration measured in the former samples (*i.e.*, 0.33 mg/kg wet-weight) to be well within the range of concentrations reported for the latter samples (*i.e.*, < 0.3 to 1.96 mg/kg wet-weight – see Table 15). However, the comparison is necessarily hindered by lack of information concerning the exact manner in which the latter samples were harvested and processed for analysis (*i.e.*, whole plant basis *vs.* individual or selected plant parts).

Complete details concerning the manner in which the findings from the above sampling program were used to estimate the arsenic exposures that might be received through the consumption of traditional foodstuffs are provided in the section that follows (see 6.0 CANCER RISK ASSESSMENT – CEI METHODOLOGY)

Table 2. Summary of Cattail Root and Game Meat Samples Collected as Part of the AHW (2006) Sampling Program

Sampling Location	Designation	Species	Type and Number of Samples (n)
Yukon[1]	Reference location	Moose	Kidney (5)
		Moose	Liver (10)
		Moose	Muscle (22)
Outside Edmonton[2]	Reference location	White-tailed deer	Muscle (1)
		White-tailed deer	Liver (1)
		Cattail	Mixture of root, root hair, root skin, whole root, washed root, and stem (20)
Peace River/Grand Prairie	Reference location	White-tailed deer	Muscle (2)
Wood Buffalo Region (WBR)	Primary location of interest	Cattail	Mixture of root, root hair, root skin, whole root, washed root, and stem (45)
		Moose[3]	Kidney (1)
		Moose[3]	Liver (3)
		Moose[3]	Heart (1)
		Moose[3]	Muscle (23)
		White-tailed deer[4]	Muscle (10)

(1) Moose meat samples from the Yukon Territory were provided for comparative analysis by Dr. Mary Gamberg of Gamberg Consulting, Whitehorse, Yukon (Gamberg *et al.* 2005)

(2) The 'Outside Edmonton' area from which the samples were collected included the Strathcona Wilderness Centre and locations near Westaskiwin Alberta.

(3) Moose muscle samples from the WBR comprised of tissues collected from the back, hind leg, thigh, neck, or shoulder.

(4) Deer muscle samples from the WBR were comprised of tissues collected from the neck, hind leg, or front leg.

Table 3. Summary of Total Arsenic Concentrations Measured in Traditional Food Items Collected as Part of the AHW (2006) Sampling Program

Region	Type	Total Arsenic Concentration (mg/kg wet-weight)		
		Mean	95%UCLM	Range
Yukon	Moose - Kidney	0.0067	0.014	< 0.001 to 0.02
	Moose - Liver	0.027	0.072	0.0014 to .1314
	Moose - Muscle	0.0082	0.0134	0.0013 to 0.043
Outside Edmonton[1]	Deer – Muscle and Liver	0.002	--	< 0.001 to 0.0036
	Cattail (all parts)	0.635	1.33	0.003 – 4.7
Peace River/ Grande Prairie	Deer - Muscle	0.0005	--	< 0.001
Wood-Buffalo Region (WBR)	Cattail (all parts)	0.327	1.98	< 0.001 to 2.7
	Moose - Kidney	0.0015	-- (1)	0.0015
	Moose – Heart	0.0005	-- (1)	<0.001
	Moose – Liver	0.012	-- (1)	<0.001 to 0.027
	Moose - Muscle	0.0087	0.020	< 0.001 to 0.068
	Deer - Muscle	0.0066	0.011	0.005 to 0.020

"--" indicates that too few data points were generated to calculate a 95% UCLM.

(1) The 'Outside Edmonton' area from which the samples were collected included the Strathcona Wilderness Centre and locations near Westaskiwin Alberta.

Table 4. Summary of Total Arsenic Concentrations Measured in Cattail Roots Collected as Part of the AHW (2006) Sampling Program (Wood Buffalo Region Only)

Cattail Root Part	Total Arsenic Concentration (mg/kg wet-weight)		
	Mean	95%UCLM	Range
Root hairs	1.12	1.9	0.068 to 2.6
Root skin	0.575	0.89	0.016 to 1.4
Root starch	0.026	0.042	0.002 to 0.082
Whole root	0.20	0.49	0.037 to 0.60
Root stem	0.006	0.0077	0.0005 to 0.012
Meal (all root data)	0.33	- -	- -
Overall 95% UCLM	- -	1.9	- -

"- -" indicates that either a mean arsenic concentration of all cattail root parts or a 95% UCLM for all cattail root parts was not applicable.

6.0 CANCER RISK ASSESSMENT (CEI METHODOLOGY)

6.1 Overview

As indicated above, the incremental lifetime cancer risks (ILCRs) presented to people living in the Wood-Buffalo region from arsenic exposures under different development scenarios were originally predicted by Golder Associates (2006). To test the veracity of the original risk estimates, the cancer risks were re-evaluated by CEI, with direction on the overall approach to be followed provided by AHW (see 2.0 TERMS OF REFERENCE). A general description of the approach is provided below. Details concerning the specific methods that were followed can be found in the next section (Section 6.2 SPECIFIC METHODS).

The re-assessment followed a conventional risk assessment paradigm (see Figure 2), with a specific focus on the potential lifetime cancer risks that could be presented to indigenous people living in the Wood-Buffalo region from exposure to inorganic arsenic *via* the oral route.[5] Three development "scenarios" were examined:

- A "baseline" scenario in which the potential lifetime cancer risk associated with existing sources of arsenic exposure in the region was calculated. The baseline exposure represented a combination of exposure from naturally-occurring sources of arsenic in the environment *and* exposure from already existing anthropogenic sources in the region
- A "future" scenario in which the incremental lifetime cancer risk presented by arsenic exposure contributed by projected future development activities in the region was calculated. The scenario was based on a 80-year projection. Reliance was placed on the Suncor Voyageur application (Suncor 2005a; 2005b) for information relating to the future development activities and the amounts of arsenic that might potentially be released into the environment. The calculation of the incremental lifetime cancer risk proceeded on the basis of the predicted arsenic deposition that might occur from future anthropogenic activities independent of baseline conditions.
- A "combined" scenario in which the lifetime cancer risk calculated for the baseline scenario and the incremental lifetime cancer risk contributed by the future scenario were added. The combined cancer risk was based on the sum total of arsenic exposures contributed by naturally-occurring sources, existing anthropogenic sources, and prospective future industrial sources in the region.

It is important to note that the above scenarios differed from those assessed by Golder Associates as part of the original HHRA performed in support of the Suncor Voyageur project. Key differences were as follows:

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[2] Emphasis was given to assessing the cancer risks associated with exposure to arsenic *via* the oral route since the original assessment performed by Golder Associates (2006) showed very little contribution from other routes of exposure, including inhalation.

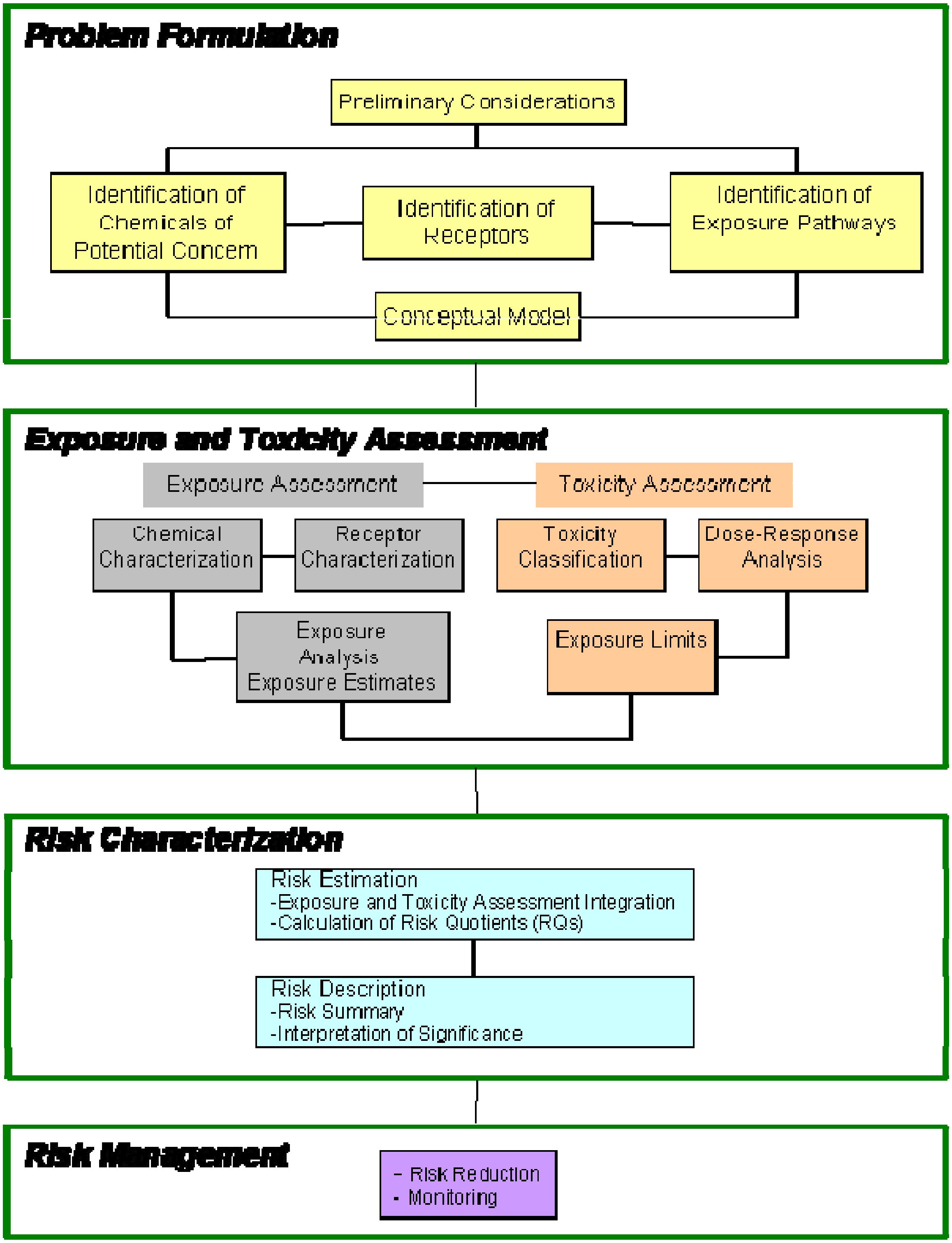

Figure 2. The Risk Assessment Paradigm

- Whereas the original HHRA was structured following a conventional EIA "protocol" in terms of both the definition of the development scenarios examined as well as the terminology used to describe each scenario (*i.e.,* baseline, application and PDC), the re-assessment departed from convention and proceeded on a more generic basis, with less emphasis given to a specific proponent and/or application and different terminology assigned to each scenario (*i.e*., baseline, future and combined). In other words, both the nature and the nomenclature of the development scenarios differed between the two assessments.
- Whereas, the original HHRA included a development scenario specific to the project under consideration (*i.e.*, the so-called "application" scenario), the re-assessment did not focus on the incremental lifetime cancer risk associated with the Suncor Voyageur project *per se*, but rather was concerned with estimating the incremental lifetime cancer risk that could result from *all* projected future development activities in the region, including the Suncor Voyageur project (*i.e.*, the "future" scenario). In other words, unlike the original HHRA, the re-assessment did not consider the Suncor Voyageur project on a stand-alone basis, but instead was deliberately designed to examine the potential incremental lifetime cancer risk associated with future development activities on a broader temporal and regional scale.
- Whereas, following convention, the so-called "baseline" scenario examined as part of the original HHRA included consideration of the arsenic exposures that might be contributed by approved but not yet operating oil sands projects in the Wood-Buffalo region, the baseline scenario included as part of the re-assessment was confined to the examination of the arsenic exposures and corresponding cancer risks associated with existing sources only. (Note that the future development scenario examined as part of the re-assessment considered both approved *and* prospective future oil sands projects to be constructed in the Wood-Buffalo region across an 80-year timeline).

Other features that distinguish the re-assessment are listed below:

- Whereas, by convention, cancer risks are calculated on an incremental basis only and refer to the added risks contributed by individual projects without consideration of the risks presented by existing and/or "background" sources, the re-assessment extended to all three of the above development scenarios (*i.e*., baseline, future *and* combined), consistent with the Terms of Reference assigned to the work.[6]
- For the purposes of the future scenario, emphasis was given to the incremental arsenic exposures that could result from the air emissions originating from the projected development activities. Consideration was *not* given to exposures that might occur from arsenic contained in effluents since surface water discharges from the types of facilities associated with the future development were deemed to be negligible.
- Per the instructions received from AHW, the re-assessment relied on the original datasets used by Golder Associates to estimate the potential exposures to inorganic arsenic that might

[6] Both the original assessment performed by Golder Associates (2006) and the re-assessment completed by CEI were somewhat unique in that a "background" scenario *per se* was not evaluated, but rather a "baseline" scenario was examined which represented a combination of background <u>*and*</u> already existing anthropogenic sources of arsenic in the region.

be received by people living in the area, supplemented with information available in-house as well as with additional data sourced from the published literature and elsewhere.

- In accordance with the instructions received from AHW, two sets of cancer risk estimates were derived for each of the development scenarios examined, with one set of calculations based on the use of the Exposure Limit for inorganic arsenic recommended by the Water Quality and Health Bureau of Health Canada (*i.e.*, RsD = 0.006 μ/kg BW/day) and the other set of calculations based on the use of the limit developed by the Contaminated Sites Division of Health Canada (*i.e.*, RsD = 0.003 μ/kg BW/day) (see Table 1).

6.2 Specific Methods

6.2.1 Problem Formulation

By convention, the Problem Formulation step of the risk assessment paradigm is concerned with identifying: i) the chemicals of potential concern (COPCs); ii) the receptors that might be exposed to the chemicals; and, iii) the routes and pathways by which the receptors might be exposed. For the present assessment, the choice of chemicals, receptors and exposure pathways to be examined was dictated largely by the Terms of Reference assigned to the work (see 2.0 TERMS OF REFERENCE). Details are given in Table 5. The specific exposure pathways selected for assessment are illustrated in Figure 3.

Table 5. Problem Formulation Specific to the Re-assessment of the Lifetime Cancer Risks Associated with Arsenic Exposures among People Living in the Wood-Buffalo Region

Parameter	Selection	Comments
COPC(s)	Inorganic Arsenic	Consistent with the Terms of Reference assigned to the work, and dictated, in part, by the need to further understand the potential lifetime cancer risks associated with exposure to arsenic among people living in the Wood-Buffalo region. The inorganic form of arsenic was chosen by default owing to its higher order of toxicity compared to elemental arsenic and its organic derivatives as well as on the basis of its known carcinogenic potential.
Receptor(s)	Indigenous People (*i.e.*, First Nations)	Consistent with the Terms of Reference assigned to the work, and based, in part, on consideration of lifestyle and dietary factors that might contribute to potentially higher exposures to arsenic among indigenous people compared to other populations living in the area.
Exposure Route	Oral (*i.e.*, ingestion)	Consistent with the Terms of Reference assigned to the work, and based largely on the findings from the original assessment performed by Golder Associates (2006) which showed the oral route to be the greatest contributor to arsenic exposure.
Exposure Pathways	Incidental ingestion of soil Consumption of surface waters(1) Consumption of traditional plant foodstuffs Consumption of game meat Consumption of sport fish	Most of the listed pathways were dictated by the Terms of Reference assigned to the work, and were included as part of the original assessment performed by Golder Associates (2006).

(1) Surface waters were conservatively assumed to act as a major source of drinking water.

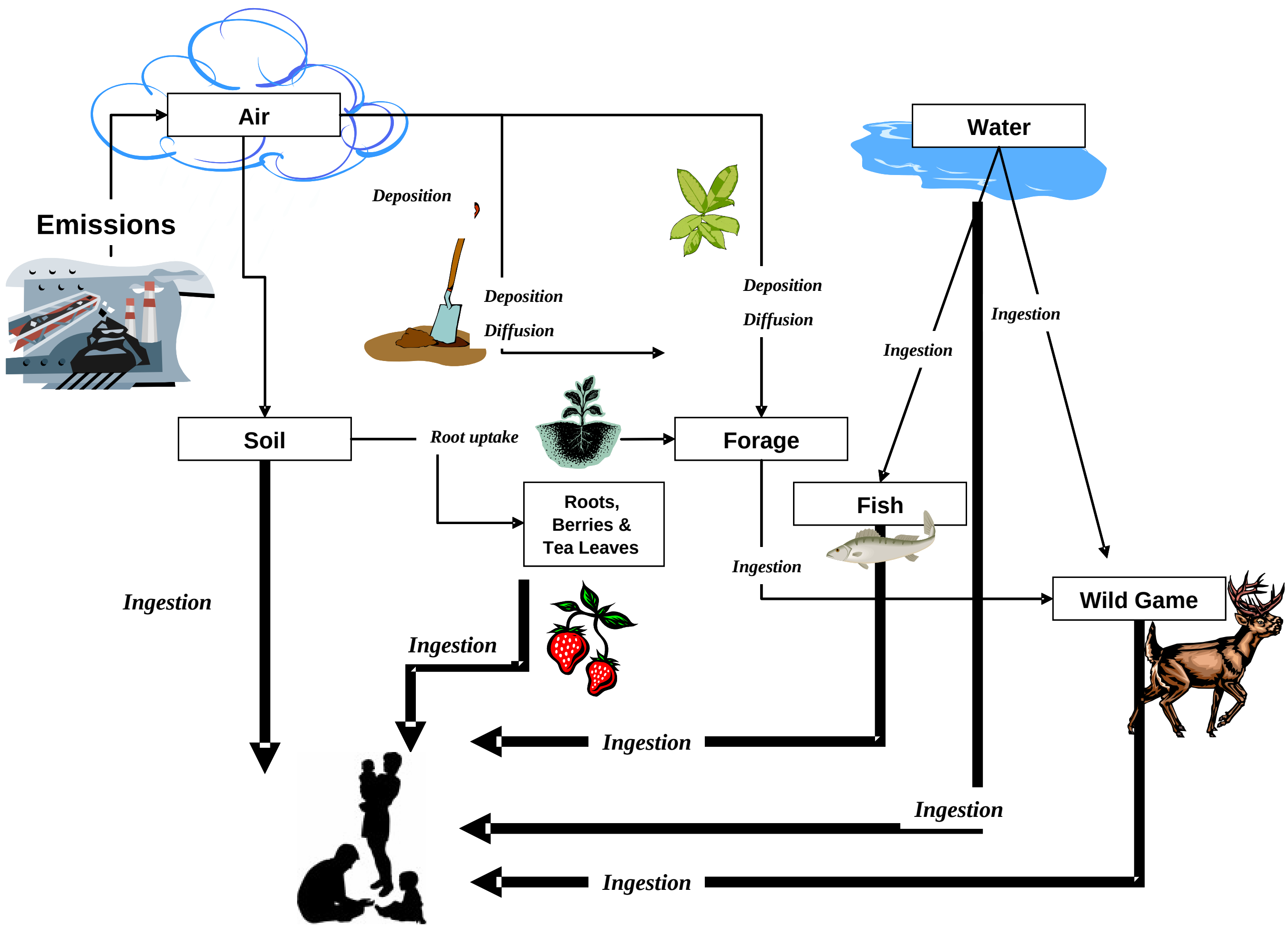

Figure 3. Exposure Pathways Considered as Part of the Re-assessment

Items emerging from the Problem Formulation step of the CEI re-assessment that require some clarification are:

- First, the choice of indigenous people as the receptor of interest required that the lifestyle and dietary habits of these individuals be considered as part of the work, especially any unique characteristics that could directly bear on the arsenic exposures that might be received. Accordingly, allowance was made for the receptor to live in both a community-based setting as well as a more traditional setting dedicated to hunting, fishing, trapping and other outdoor pursuits. It was assumed that the receptor would spend six months of the year in each setting, with much of the winter spent in the community. Allowance also was made for the consumption of traditional foods as well as untreated surface waters by the receptor, especially while in the field.

- Second, following convention, different age categories were assigned to the receptor (*i.e.*, infant, toddler, child, adolescent and adult), largely for the purposes of apportioning the exposures to arsenic that might be received at different ages throughout the course of a lifetime. For the purposes of estimating the lifetime cancer risks, emphasis was necessarily assigned to a "composite" receptor representing all age categories combined. The cancer risks were calculated on the basis of the predicted "lifetime average daily dose" (LADD) of inorganic arsenic that might be received by the composite receptor.
- Third, uncertainty surrounded the source(s) of drinking water in the region, especially in relation to the lifestyle assumed for the receptor. It was anticipated that, while in the field, the receptor could very likely drink raw surface water from local creeks, streams, rivers and/or lakes, at least while the waters were open (*i.e.*, outside of the winter months); whereas, while residing in the community, drinking water would likely be sourced from the treated water supply. Since uncertainty also surrounded the status of the water treatment facility servicing the community, especially in relation to the ability of the treatment system to successfully remove arsenic from the water supply, it was conservatively assumed that the arsenic content of the treated water would be equivalent to that found in the raw surface water(s) drawn by the facility. Use of groundwater drawn from local aquifers as a source of drinking water was deemed to be unlikely based on information compiled by Suncor (2005b) indicating that artesian water in the region is both difficult to access and of poor quality.
- Finally, the exposure pathways selected for assessment were deliberately chosen and configured to permit estimation of the arsenic exposures and corresponding cancer risks unique to indigenous people living in the Wood-Buffalo region. For example, emphasis was given to the arsenic exposures that might be received through the consumption of traditional plant foodstuffs and game meat as well as the consumption of raw surface waters while in the field in recognition of the unique lifestyles and dietary habits of these people. It should be noted that the arsenic exposures that could potentially be received through other pathways and/or from other sources (*e.g.*, tobacco smoke, store-bought foods and beverages) were not accounted for in the assessment.

6.2.2 Toxicity Assessment

The Toxicity Assessment step of the risk assessment paradigm is concerned with identifying and understanding the potential health effects that can result from exposure to the COPC(s), with particular interest assigned to the concentration-time-response characteristics of the chemical(s) in relation to each of the health endpoints affected. This step also is concerned with identifying Exposure Limits for the COPCs, with the limits signifying exposures deemed to be "safe" or acceptable vis-à-vis the protection of health.

For the present re-assessment, reliance was placed on the information contained in Appendix A summarizing the health effects associated with exposure to arsenic. The choice of the Exposure Limit(s) to be used in the re-assessment was made in collaboration with Alberta Health and Wellness (Hopkins 2007), and was based largely on consideration of the following criteria:

- The need to accommodate long-term exposure to arsenic, consistent with the 80-year projection captured in the future and combined development scenarios (see above).
- The need to focus on the health effects known to be associated with exposure to arsenic *via* the oral route (*i.e.*, ingestion), consistent with the Terms of Reference assigned to the work.
- The need to recognize the carcinogenic potential of arsenic.
- The need for sufficient conservatism to be embraced by the limit such that the lifetime cancer risks would not be understated.
- Other conventional criteria relating to the adequacy and reliability of the limit, including the reputability of the source, the availability of supporting documentation, and the level of protection afforded.

Based on direction received from Alberta Health and Wellness, the choice of limits was narrowed to the following:

- The "negligible risk level" (NRL) of 0.3 μ/L recommended by the Water Quality and Health Bureau of Health Canada, and described in the *Canadian Drinking Water Guideline* for inorganic arsenic (Health Canada 2006). According to Health Canada, the "negligible risk level" corresponds to an incremental lifetime cancer risk of 1.9×10^{-6} to 14×10^{-6} (*i.e.*, 2 to 14 extra cases of cancer in a population of one million people <u>*or*</u> 0.2 to 1.4 extra cancer cases in a population of 100,000 people). The NRL is equivalent to a Risk-Specific Dose (RsD) of 0.006 micrograms of inorganic arsenic per kilogram of body weight per day (μg/kg BW/day), based on the daily consumption of 1.5 L of water by a 70-kg adult.
- The slope factor (SF) of 2.8 $(mg/kg\text{-}d)^{-1}$ recommended by the Contaminated Sites Division of Health Canada. The SF corresponds to an incremental lifetime cancer risk of 1×10^{-5} (*i.e.* one extra cancer case in a population of 100,000), and is equivalent to an RsD of ≈ 0.003 micrograms of inorganic arsenic per kilogram of body weight per day (μg/kg BW/day).

Additional details concerning the above limits can be found in Appendix A. Use of the limits necessarily required that, as part of the interpretation of the cancer risk estimates, consideration be given to the following items since they had a direct and significant bearing on the level of conservatism incorporated into the re-assessment:

- The limits are based on the associations between exposure to inorganic arsenic in drinking water and elevated cancer rates witnessed in Taiwanese villages (Chen *et al*. 1992; Morales *et al*. 2000; Wu *et al*. 1989 – see Appendix A). As indicated above, some authorities have questioned the relevance of these finding to North American populations because of: i) the relatively high arsenic content of the village drinking water; ii) the poor nutritional status of the villagers; and, iii) the possible presence of an environmental "factor" apart from arsenic contributing to the increased cancer rates.
- The limits are based on linear extrapolation of the observed cancer incidence data among villagers exposed to relatively high levels of arsenic to low-dose levels (*i.e.*, a conservative approach). As outlined earlier, debate surrounds the shape of the dose-response curve for

arsenic, especially at lower dose levels. In fact, some authorities have argued that exposure to arsenic at low dose levels may confer a protective effect against the occurrence of cancer, suggesting that a non-linear curve (*i.e.*, biphasic or concave) may better represent the carcinogenic potency of arsenic.

- The limits assume that arsenic acts as a non-threshold carcinogen (*i.e.*, any amount of exposure carries some level of risk). As discussed above, the mechanism by which arsenic causes cancer is not fully understood. Debate surrounds whether arsenic qualifies as a threshold or non-threshold carcinogen, with some authorities claiming that the carcinogenicity of arsenic represents a high-dose phenomenon only, mediated by cytotoxicity, suppression of DNA repair mechanisms and/or oxidative stress, for which thresholds exist.

6.2.3 Exposure Assessment

The Exposure Assessment of the risk assessment paradigm was concerned with estimating the exposures to inorganic arsenic that might be received by indigenous people living in the Wood-Buffalo region under each of the development scenarios examined (*i.e.*, baseline, future, and combined – see above). The assessment relied on both: i) empirical data collected from different environmental "compartments" in the area (*i.e.*, soils, surface waters, fish tissues, game animal tissues, traditional plant foodstuffs, *etc*.) showing measured arsenic levels; *and*, ii) modelled predictions based on mathematical algorithms describing the behaviour and transport of arsenic in the environment. The former data were used largely to estimate the arsenic exposures that might be received under the baseline development scenario, whereas the predictive modelling was used primarily to forecast the incremental exposures that could be encountered under the future development scenario. Predictive modelling also was applied in cases in which the empirical data were judged to be incomplete or lacking. *In all cases, the potential exposures were expressed in terms of inorganic arsenic.*

Much of the empirical data was obtained from the following sources, supplemented with information contained in the published literature as well as information available in-house:

- The original HHRA performed by Golder Associates (2006).
- The Application submitted by Suncor Energy in support of the Voyageur Project (Suncor 2005a), including the Environmental Impact Assessment and Environmental Setting reports.
- Supplemental information provided by Suncor Energy in support of the Voyageur Project (Suncor 2005b).
- The sampling and analysis program aimed at determining the arsenic content of traditional foodstuffs in the Wood-Buffalo region recently commissioned by Alberta Health and Wellness (see above).

The forecast data were derived using predictive models developed by CEI either as part of earlier modelling exercises or *de novo* for specific use in the current re-assessment.

Unfortunately, in terms of the empirical dataset, it was not always obvious from the above reports as to which form of arsenic was represented, with no clear indication provided in some cases as to whether the measured levels referred to total arsenic, organic arsenic, or inorganic arsenic. Whenever doubt existed, conservative assumptions were applied by default in order to ensure that inorganic arsenic was not under-represented (*i.e.*, unless indicated otherwise, in the absence of information, it was assumed that the empirical data referred to inorganic arsenic). *The exposure estimates and corresponding lifetime cancer risks were always expressed in terms of inorganic arsenic.*

Details concerning the Exposure Assessment are given below.

6.2.3.1 Receptor Age Categories

As indicated earlier, the re-assessment was concerned with estimating the *lifetime* cancer risk that could result from exposure to inorganic arsenic under each development scenario. As such, the cancer risks were calculated for a "composite" receptor who was assumed to spend his/her entire life in the region, and who was assumed to be exposed to inorganic arsenic daily throughout his/her entire lifetime.[7] In order to differentiate the arsenic exposures that might be received at different ages and to refine the estimates of the daily exposures that might be received across a lifetime, different life stages were examined. Details concerning these life stages, including an indication of the averaging times used to apportion the exposures across an 80-year lifetime are provided in Table 6. The lifetime cancer risks were calculated on the basis of the lifetime average daily dose (LADD) of arsenic received by the composite receptor.

Table 6. Age Categories Used to Represent the "Composite" Receptor[(1)]

Age Category	Exposure Frequency [days/year]	Exposure Duration [years]	Body Weight [kg]	Averaging Time [days]
Infant (0 to 6 mo)	365	0.5	8.2	182.5
Toddler (7 mo to 4yr)	365	4.5	16.5	1,277.5
Child (5 to 11 yr)	365	7	32.9	2,920
Adolescent (12 to19 yr)	365	8	59.7	2,555
Adult (>20)	365	61	70.7	22,265

(1) Information sourced from Health Canada (2006).

6.2.3.2 Exposure Pathways

Consistent with the Terms of Reference assigned to the work, the Exposure Assessment was directed at estimating the inorganic arsenic exposures that might be received *via* the oral route (*i.e.*, by ingestion), with the following exposure pathways examined:

[7] The term "composite" was used to denote the combination of all age categories (*i.e.*, infant to adult). An 80-year lifetime was assumed, consistent with the 80-year projection used to define the "future" development scenario. Exposure to arsenic was assumed to occur daily, 365 days per year (except for direct soil ingestion which was assumed to occur for six months of each year).

Incidental ingestion of soil

- Consumption of surface waters (as sources of drinking water);
- Consumption of traditional plant foodstuffs (*e.g.*, berries, roots, leaves);
- Consumption of game meat; and,
- Consumption of sport fish.

For the purposes of the assessment, it was assumed that the entire drinking water supply of the receptor and the entire complement of traditional/natural food items examined were sourced from the Wood-Buffalo region. Consideration was *not* given to the other types of food that typically comprise the diet (*e.g*., grains and cereals, milk and other beverages, snacks and convenience foods) as they would likely originate from sources outside of the region.[8] Similarly, other sources of arsenic exposure (*e.g.*, tobacco smoke) were *not* considered since again they were not unique to the region. Finally, as already indicated, other exposure pathways (*e.g.*, inhalation) were not included since their contribution previously has been shown to be insignificant (Golder Associates 2006).

Descriptions of each of the exposure pathways examined follow. Details surrounding the calculations performed can be found in the attached Excel® spreadsheet (Appendix B).

6.2.3.2.1 Incidental Soil Ingestion Pathway

Potential exposures to inorganic arsenic that could result from incidental ingestion of soil were estimated by one of two means depending on the development scenario under consideration. For the baseline scenario, the estimates were based on measured levels of arsenic in soils collected from the Wood-Buffalo region; whereas, for the future scenario, the estimates relied on predictive modelling of the amounts of arsenic that might be deposited on soil as a result of air emissions from future anthropogenic sources. Details are given below for each scenario.

Baseline Development Scenario

A summary of arsenic concentrations measured in soils collected from the Wood-Buffalo region and reported as part of EIAs performed in support of earlier oil sands projects is presented in Table 7. These data served as the basis of the exposure estimates calculated for the baseline development scenario. The use of the data presented several challenges:

[8] The exclusion of other dietary constituents from the assessment was based on two considerations: First, the primary aim of the assessment was to further understanding of the lifetime cancer risks presented to people living in the Wood-Buffalo region from arsenic exposure associated with existing (*i.e.*, baseline) and on-going (*i.e.*, future) development activities in the area. It was not intended to specifically address the "background" risks associated with naturally-occurring arsenic, including the potential risks that might be associated with the consumption of foods originating from other areas (*i.e.*, store-bought foods). Second, per the instructions received from AHW, the re-assessment was to mimic the original assessment performed by Golder Associates (2006). The original assessment considered only traditional foods.

- First, it was not obvious from the data as to what form of arsenic was measured. For the purposes of the re-assessment, it was conservatively assumed that the measurements referred to inorganic arsenic.
- Second, it was unclear from the data as to whether the soil concentrations were reported on a wet-weight or dry-weight basis. By default, it was assumed the measurements were expressed on a dry-weight basis.
- Third, many of the soil samples (*i.e.*, 18 of 62) reportedly showed non-detectable amounts of arsenic, signifying that, if any arsenic was present, it was at a level below the analytical detection limit. In order to calculate the average soil concentration, it became necessary to assign a value to these samples, with several choices available, as follows: i) assume the arsenic concentration was zero; ii) assume the arsenic concentration was equivalent to one-half of the analytical detection limit; or, iii) assume the arsenic concentration was equal to the detection limit. Each of these choices was tested, with the outcomes shown in Table 8. Based on the findings, it was decided that, for the purposes of the re-assessment, the 95% upper confidence limit of the mean measured concentration (95% UCLM) should be used to represent the arsenic level in soil. Use of the 95% UCLM was based on the fact that it remained unchanged regardless of which assumption surrounding the detection limit was applied, combined with the fact that it represented the upper end of the average concentration range (*i.e.*, a reasonably conservative choice).

Table 7. Summary of Arsenic Concentrations Measured in Soil Samples Collected From the Wood-Buffalo Region

Designation	Source	Number of Samples	Arsenic Concentration (mg/kg)[1]
North Steepbank	Suncor (2005a)	6	0.5 to 1.6
Voyageur	Suncor (2005a)	7	<0.1 to 2.1
Fort McMurray	Suncor (2005a)	8	0.2 to 3.9
Kearl	Imperial Oil (2006)	7	0.2 to 2.3
Muskeg River Mine Expansion	Albian Sands Energy Inc. (2005)	7	0.4 to 1.2
Horizon	CNRL (2003)	10	<0.5 to 1.2
Fort McMurray	Shell Canada (2002)	3	0.8 to 2.7
Jackpine Mine	Shell Canada (2002)	14	<0.5 to 0.7

(1) Note that it was not possible from the data supplied to determine the form of arsenic that was measured (*i.e.,* total *vs*. organic *vs*. inorganic) nor the manner in which the soil concentrations were expressed (*i.e.*, dry-weight vs. wet- weight basis. For the purposes of the re-assessment, it was conservatively assumed that the data referred to inorganic arsenic, and that the soil concentrations represented dry-weight values.

Table 8. Calculated Mean and 95% UCLM Concentrations of Inorganic Arsenic in Soil[(1)]

Detection Limit Assumption	Arsenic Concentration (mg/kg dry-weight)	
	Mean	95% UCLM
Arsenic present at analytical detection limit	0.92	1.3
Arsenic present at ½ detection limit	0.85	1.3
Arsenic concentration is zero or blank if less than detection limit	1.1	1.3

(1) Measured values assumed to refer to inorganic arsenic (see text above for explanation).

Based on the above, the concentration of inorganic arsenic in soil was set at 1.3 mg/kg (dry-weight) for the purpose of estimating the exposures that might be received through the incidental ingestion of soil under the baseline development scenario. This concentration was translated to the corresponding exposure estimate (*i.e.,* µg/kg BW/day) for each of the receptor age categories based on soil ingestion rates reported by Health Canada (2004). The estimates are shown in Table 9. As part of the translation, consideration was given to the fact that soils in the Wood-Buffalo region would likely be frozen and/or snow-covered for much of the year (Environment Canada 2006), thereby precluding any reasonable opportunity for exposure *via* this pathway at these times. As a result, for the purposes of the re-assessment, exposure to inorganic arsenic from the incidental ingestion of soil was assumed to occur for only six months of the year.

Future Development Scenario

Estimates of the potential incremental exposures to inorganic arsenic that might be received *via* the incidental consumption of soil under the future development scenario were based on the premise that arsenic contained in the air emissions originating from any added facilities would deposit on the local soils and would be available for ingestion. Reliance was placed on an already-predicted annual deposition rate of 0.00001 kg arsenic/hectare (Suncor 2005a).[9] Estimates were calculated using the equation shown below (*i.e.*, Equation #1). No losses from soil (*e.g., via* erosion) were assumed to occur over the projected 80-year future development period.

$$C_{is} = \frac{DR \times CF}{D \times BD} \times T \qquad \textbf{\textit{Equation \#1}}$$

Where:

C_{is}	=	incremental soil concentration contributed by future development (calculated to be 0.0053 mg/kg dry-weight)
DR	=	deposition rate (0.00001 kg/ha/yr)
CF	=	conversion factor from ha to m^2 and kg to mg (1,000,000 mg/kg / 10,000 m^2/ha)
D	=	soil depth (0.01 meters)
BD	=	bulk density of soil (1,500 kg/m^3)
T	=	period of deposition (80 years)

[9] In the absence of information, the deposition rate was assumed to apply to inorganic arsenic.

The concentration of inorganic arsenic predicted to occur in soil from the future development activities in the Wood-Buffalo region was calculated to be 0.005 mg/kg dry-weight. As described above, this soil concentration was translated to the corresponding exposure estimate (µg/kg BW/day) for each age category, with adjustment for the six-month period per year that the soil would be frozen and/or snow-covered and not readily accessible. The estimates are shown in Table 9.

Summary – Incidental Soil Ingestion Pathway

Estimates of the exposures to inorganic arsenic that might be received *via* the incidental ingestion of soil under each of the development scenarios are summarized in Table 10. The estimates shown for the combined scenario represent the sum of the exposures predicted for the baseline *and* future scenarios.

Table 9. Estimated Exposures to Inorganic Arsenic Received *via* the Soil Ingestion Pathway (µg/kg BW/day)

Age Category	Soil Ingestion Rate [g/day][1]	Development Scenario		
		Baseline	Future	Combined
Infant	0.02	0.002	0.000007	0.002
Toddler	0.08	0.0032	0.000013	0.0032
Child	0.02	0.00040	0.0000016	0.00040
Adolescent	0.02	0.00022	0.00000089	0.00022
Adult	0.02	0.00018	0.00000075	0.00018
***Composite*[2] (*LADD*)**		0.00038	0.0000016	0.00038

(1) Source: Health Canada (2004).

(2) The composite receptor exposure value or "Lifetime Average Daily Dose" (LADD) was calculated by adding the predicted exposure for each age category and averaging the exposure over an 80-year lifetime. This approach is consistent with guidance provided in Health Canada (2006), and was used throughout the re-assessment.

Examination of the estimates reveals that the incremental exposures that might be received from the incidental ingestion of soil as a result of the future development activities are negligible across all age categories. The combined exposures are dominated by those contributed by the baseline scenario, with essentially no change in exposures expected from future development.

6.2.3.2.2 Consumption of Drinking Water Pathway

Estimates of the potential exposures to inorganic arsenic that could result from the consumption of drinking water again were calculated by one of two means depending on the development scenario under consideration. For the baseline scenario, exposures were predicted on the basis of the measured arsenic content of samples of surface waters collected from the Wood-Buffalo region, specifically samples drawn from the Athabasca River and Ells River. For the future scenario, reliance was placed on modelled predictions of the incremental increase in arsenic concentrations in local surface waters that might result from future development activities in the region (Suncor 2005a). The approach followed for each scenario is outlined below. Note that the assessment distinguished between the arsenic exposures that might be received by the indigenous receptor through the drinking water pathway while outdoors in the field versus while in a community setting.

Baseline Development Scenario

Surface water quality data gathered as part of the Regional Aquatic Monitoring Program (RAMP) for the Municipality of Wood-Buffalo for the period 2002 to 2004 were obtained in an electronic format for sorting and review (RAMP 2002; 2003; 2004). Data specific to the arsenic levels measured in the Athabasca River and Ells River were extracted for use as follows:

- Measured levels of arsenic in the Athabasca River were used to predict the arsenic exposures that might be received by an indigenous person from drinking raw, untreated surface waters while in the field for the purpose of hunting, fishing, trapping, or as part of other outdoor pursuits. As discussed earlier (see 6.2 PROBLEM FORMULATION), it was assumed that the receptor would spend six months of the year in such a field setting, and would drink freely from local creeks, streams, rivers and/or lakes. The six-month period was chosen, in part, to allow for the fact that the surface waters would likely be frozen and largely inaccessible during the winter months.
- Measured levels of arsenic in the Ells River were used to estimate the arsenic exposures that might be received by the same indigenous person from drinking treated water in a community setting for the remaining six months of the year. (Note that the Ells River serves as the source of drinking water for the Community of Fort MacKay). Since details concerning the water treatment technology used by the community were not known (*i.e.*, information respecting the ability of the technology to successfully remove arsenic from the water was unavailable), it was conservatively assumed that the arsenic content of the treated drinking water would be the same as that measured in the Ells River.

The arsenic content of samples of raw water taken from both rivers is summarized in Table 10. It was assumed that the measurements referred to the inorganic form of arsenic (*i.e.*, a reasonable, but still conservative assumption). These measured concentrations were used to predict the exposures to inorganic arsenic that might be received through the consumption of drinking water under the baseline development scenario. For the purposes of the re-assessment, the 95% UCLM was used to represent the arsenic content of the drinking water.[10] This concentration was translated to the corresponding exposure estimate (µg/kg BW/day) for each setting (*i.e.*, community *vs*. field) and for each age category using drinking water ingestion rates reported by Health Canada (2004). The exposure estimates are shown in Table 11.

[10] Note that, unlike the soil measurements (see above), no correction was required to accommodate samples that registered “non-detect” since virtually all of the water samples collected showed arsenic present at levels above the analytical detection limit.

Table 10. **Summary of Arsenic Concentrations Measured in Samples of Untreated Surface Waters Collected From the Wood-Buffalo Region** [1]

	Inorganic Arsenic Content (mg/L)	
	Athabasca River (n=42)	**Ells River (n=15)**
Number of non-detect results	1	1
Mean	0.001	0.00094
Minimum	0.00048	0.0003
Maximum	0.0045	0.0022
95% UCLM	***0.0012***	***0.0012***

(1) Source: Regional Aquatic Monitoring Program for the Municipality of Wood Buffalo (RAMP) for the period 2002 to 2004. Measured values were assumed to refer to inorganic arsenic. Non-detect values were assumed equal to one-half the detection limit for the purposes of calculating the mean and 95% UCLM. The values shown in parentheses refer to the number of samples collected and analyzed.

Future Development Scenario

Estimates of the incremental arsenic exposures that might be received through the consumption of drinking water under the future development scenario were calculated on the basis of earlier forecasts of the incremental increase in the arsenic content of local surface waters that might result from the added emissions from future anthropogenic activities (Suncor 2005a). It was assumed that these earlier predictions referred to inorganic arsenic. For the purposes of the re-assessment, the *highest* predicted incremental increase in arsenic levels (*i.e.*, 0.0001 mg/L) was used in the calculations. This concentration was translated to the corresponding exposure estimate (µg/kg BW/day) for each setting and age category using the drinking water ingestion rates reported by Health Canada (2004). The results are listed in Table 11.

Summary – Consumption of Drinking Water Pathway

The estimated exposures to arsenic that might occur from the ingestion of drinking water are summarized in Table 11 for each of the development scenarios considered, with the potential exposures received from drinking raw surface waters while in the field distinguished from the exposures that might occur from drinking from the community water supply. The sum of these exposures represents the estimated daily dose *per year* of inorganic arsenic that an individual might receive from consuming drinking water from the field for six months/year and from the community for six months/year. The estimates shown for the combined development scenario represent the sum of the exposures predicted for the baseline and future scenarios. All values refer to inorganic arsenic. Additional details regarding the input values for these calculations are provided in Appendix B.

Table 11. Estimated Exposures to Inorganic Arsenic Received *via* the Consumption of Drinking Water (μg/kg BW/day)

Age Category	Water Ingestion Rate [L/day][1]	Development Scenario		
		Baseline	Future	Combined
Field Setting (6-months/year)[2]				
Infant	0.3	0.022	0.0018	0.024
Toddler	0.6	0.021	0.0018	0.023
Child	0.8	0.014	0.0012	0.015
Adolescent	1	0.0099	0.00084	0.011
Adult	1.5	0.013	0.0011	0.014
Composite (LADD)		*0.013*	*0.0011*	*0.014*
Community Setting (6-months/year)[3]				
Infant	0.3	0.021	0.0018	0.023
Toddler	0.6	0.021	0.0018	0.023
Child	0.8	0.014	0.0012	0.015
Adolescent	1	0.010	0.00084	0.011
Adult	1.5	0.012	0.0011	0.013
Composite(LADD)		*0.013*	*0.0011*	*0.014*
Total Estimated Annual Intake				
Composite Receptor (LADD)		*0.026*	*0.0022*	*0.028*

(1) Source: Health Canada (2004).

(2) Based on the consumption of untreated surface waters from creeks, streams, rivers, and/or lakes while in the field for the purpose of hunting, fishing, trapping, *etc.*

(3) Based on consumption of water from the community water supply.

Examination of the estimates reveals that the exposures predicted for the future development scenario are approximately one-tenth of those calculated for the baseline scenario, signalling that only a modest increase in exposures to inorganic arsenic *via* the consumption of drinking water would be expected from future anthropogenic activities in the region.

6.2.3.2.3 Consumption of Traditional Plant Foodstuffs Pathway

Predictions were made of the potential exposures to inorganic arsenic that might be received by indigenous people living in the Wood-Buffalo region through the consumption of traditional plant foodstuffs. For the purposes of the re-assessment, the foodstuffs were segregated into the following categories:

- Berries or fruits (*e.g.*, blueberries, cranberries, choke cherries)
- Tea leaves and other similar traditional above-ground plants (*e.g.*, Labrador tea, mint tea)
- Roots and other traditional below-ground plants (*e.g.,* cattail root)

The methodology used to estimate the potential exposures that could be received through the consumption of former two plant food categories (*i.e.*, berries and above-ground plant foodstuffs) was similar, and is described in the section that immediately follows (*i.e.*, Section 6.2.3.2.3.1). A different approach was used to evaluate the potential exposures that could result from the consumption of roots and other below-ground plants. The latter approach is outlined in Section 6.2.3.2.3.2.

6.2.3.2.3.1 Berries and Above-Ground Plant Foodstuffs

Baseline Development Scenario

A two-step process was followed in order to arrive at estimates of the potential exposures to inorganic arsenic that might result from the consumption of berries, leaves and other above-ground plant foodstuffs under the baseline development scenario. The first step involved retrieving, summarizing and interpreting the available empirical data on the measured levels of arsenic in representative above-ground plant foodstuffs gathered from the Wood-Buffalo region, and reported as part of recent EIAs completed in support of oil sands projects. These data are summarized in Tables 12 and 13. The measurements were assumed to refer to total arsenic since, for most plant tissue analyses, the arsenic content is reported as total arsenic (Wymer 2006).

Table 12. Summary of Arsenic Concentrations Measured in Samples of Berries or Fruits Collected From the Wood-Buffalo Region and Reported as Part of Recent EIAs

Designation	Source	Arsenic Content[1] (mg/kg dry-weight)		
		Blueberries	**Choke Cherries**	**Cranberries**
Kearl	Imperial Oil (2006)	<0.2 (5)		
Muskeg Mine	Albian Sands Energy Inc. (2005)	<0.2 (8)		
North Steepbank	Suncor (2005a)	<0.2 (4)		
Voyageur	Suncor (2005a)	<0.2 (5)		
Fort McMurray	Suncor (2005a)	<0.2 (5)		
Horizon	CNRL (2003)	<0.2 (8)		
Fort McMurray	CNRL (2003)	<0.2 (2)		
Jackpine	Shell Canada (2002)	<0.2 (9)		
Meadow Creek	Petro-Canada (2001)	<0.2 (8)		
Fort MacKay, Fort Chipewyan; Lease 13	Golder Associates (2000)	<0.5 (27)	<0.5 (2)	<0.5 (9)[2]
Average [3]		- -	- -	- -
95% UCLM		- -	- -	- -

(1) Assumed to represent total arsenic. Values in parentheses refer to number of samples collected and analyzed.

(2) Analysis of samples (n = 2) of wild rhubarb and yarrow flowers also revealed non-detectable levels of arsenic (< 0.5 mg/kg)

(3) Summary statistics could not be calculated since all measured concentrations were reportedly below the limit of analytical detection.

Table 13. Summary of Arsenic Concentrations Measured in Samples of Leaves Collected From the Wood-Buffalo Region and Reported as Part of Recent EIAs

Designation	Source	Arsenic Content[1] (mg/kg – dry weight)			
		Blueberry Leaves	Cranberry Leaves	Mint tea	Labrador tea
Kearl	Imperial Oil (2006)				<0.2 to 0.2 (6)
Muskeg Mine	Albian Sands Energy Inc. (2005)				<0.2 to 0.9 (11)
North Steepbank	Suncor (2005a)				<0.2 (6)
Voyageur	Suncor (2005a)				<0.2 (5)
Fort McMurray	Suncor (2005a)				<0.2 (6)
Horizon	CNRL (2003)				<0.2 to 1.6 (8)
Jackpine	Shell Canada (2002)				<0.2 to <0.05 (22)
Meadow Creek	Petro-Canada (2001)				<0.2 (7)
For MacKay, Fort Chipewyan; Lease 13	(Golder Associates 2000)	<0.5 (19)	<0.5 (9)	<0.5 to 1.1 (35)	<0.5 (53)
Average		- -[2]	- -	- -	- -
95% UCLM		- -	- -	- -	- -

(1) Assumed to represent total arsenic. Values in parentheses refer to number of samples collected and analyzed.

(2) Signifies (“- -“) that insufficient data were available to permit calculation of an average value and/or 95% UCLM. Calculation was precluded by the large numbers of samples for which the arsenic concentration was reported to be below the limit of analytical detection.

Examination of the empirical data reveals that the large majority of measured values were below the limit of analytical detection (*i.e., all* of the samples of berries and 6% of 25 mint samples and 2.5% of 118 Labrador samples were reported to contain no detectable amounts of arsenic), thereby limiting the usefulness of the data for the purpose of estimating the arsenic content of either category of plant foodstuffs as well as the corresponding exposure estimates.[11] As the second step, attention was directed toward estimating the arsenic content of the berries and above-ground plant foodstuffs through predictive modelling developed *de novo* by CEI. The modeling was configured to account for: i) uptake of arsenic from the soil *via* the plant root system; and, ii) deposition of arsenic from the air onto the leaves and other exposed plant surfaces. Details follow:

[11] The preponderance of “non-detect” measurements precluded that calculation of meaningful summary statistics (*e.g.*, mean, 95% UCLM) pertaining to the arsenic content of the berries, leaves, and other above-ground traditional plant foodstuffs.

Root Uptake

The contribution from root uptake was modeled based on the arsenic concentrations measured in soil samples collected from the region (as reported above – see 6.2.3.2.1 Incidental Soil Ingestion Pathway), with specific reliance on the 95% UCLM value as an indicator of the arsenic content of the soils (*i.e.*, 1.3 mg/kg as total arsenic – see Table 3). Two predictive models were employed, both of which were designed to provide estimates of the above-ground plant tissue concentrations of arsenic based on the uptake of arsenic from the soil *via* the roots. The first model (BCF model) was originally described by Baes *et al.* (1984), and relied on use of a soil-to-plant bioconcentration factor. The second model (Regression model) was based on regression analysis of soil and plant tissue concentrations (BLC 1998). Both models were developed, by design, to provide estimates of the total arsenic content of the above-ground plant tissues in recognition of the fact that the plant metabolic systems would likely operate to convert some of the inorganic arsenic obtained from the soil to organic derivatives. Thus, for the purposes of the re-assessment, the total arsenic concentrations in the plant tissues predicted by the models ultimately required adjustment to determine the amount of the inorganic form present in the plant. In this regard, it was conservatively assumed that 37% of the total arsenic content of the plant tissues was comprised of inorganic arsenic. In addition, the predictive models generated results in units of mg/kg dry-weight. Adjustment of the measured concentrations from a dry-weight to wet-weight basis was completed. The latter predictions relied on the use of predictive models that generated values in dry weight, which where then converted to wet weight. . The adjustment was based on the moisture content of plants (85%) reported by Suter *et al*. (2000).

The principal algorithms that were used to calculate the above-ground plant tissue concentrations of arsenic contributed through root uptake are shown below. The outcomes of the calculations for both models are listed in Table 14. Since the Regression model yielded a higher arsenic concentration than the BCF model (*i.e.*, 0.0088 mg/kg wet-weight *vs.* 0.003 mg/kg wet-weight, as inorganic arsenic), reliance was placed on the outcome of the former model as a conservative measure.

i) **BCF Model**

$$C_p = C_s \times BCF_s$$ ***Equation #2***

Where,

C_p = Concentration in plant;

C_s = Concentration in soil (1.3 mg/kg, as inorganic arsenic);

BCF_s = Soil-to-plant bioconcentration factor (0.04)

ii) **Regression Model**

$$\ln(C_p) = m\ln(C_s) + C$$ ***Equation #3***

Where

C_p = Concentration in plant

m = Slope of regression model (0.564 unitless);

C_s = Concentration in soil (1.3 mg/kg);

C = Y-Intercept (-1.992 mg/kg)

Table 14. Summary of the Predicted Inorganic Arsenic Content of Berries and Other Above-Ground Traditional Plant Foodstuffs Contributed Through Root Uptake)[(1)]

Plant Category	Arsenic Content (mg/k wet-weight)[(2)]	
	BCF Model	**Regression Model**
Above ground-plants (*e.g.*, berries, leaves)	0.003	0.0088

(1) The predictive models estimated arsenic concentrations for total arsenic. Based upon ATSDR (2005), it was assumed that 37% of arsenic in plants was in the inorganic form. This conversion has been applied to this table.

(2) The predictive models generated results in units of mg/kg dry-weight. Adjustment of the measured concentrations from a dry-weight to wet-weight basis was performed in order to maintain consistency with the predictions made for the other types of plant foodstuffs. The latter predictions relied on the use of predictive models that were configured on a wet-weight basis. The adjustment was based on the moisture content of plants (85%) reported by Suter *et al*. (2000).

Surficial Deposition

The contribution from surficial deposition (*i.e.,* deposition of arsenic from the air onto the leaves and other exposed plant surfaces) was calculated using algorithms developed by the US Environmental Protection Agency (U.S. EPA 2005c) to describe the deposition of air-borne particulate matter. The calculation was based on a deposition rate of 0.00001 kg arsenic/ha/yr, which was deemed to represent the arsenic loading rate from existing facilities in the region (Suncor 2005a). The calculation proceeded using the following equation:

$$Pd = \frac{CF \times DR \times R_p \times \left[1.0 - \exp\left(-kp \times Tp\right)\right]}{Yp \times kp}$$ ***Equation #4***

Where:

Pd = plant tissue concentration due to deposition (calculated to be 0.0000091 mg/kg dry-weight *or* 0.0000014 mg/kg wet-weight, as inorganic arsenic)

DR = deposition rate (0.00001 kg/ha/year *or* 0.000000001 kg/m^2/year)

CF = conversion factor (1,000,000 mg/kg)

R_p = intercept fraction of edible portions of plant (0.39 unitless)

kp = plant surface loss coefficient (18 year^{-1})

Tp = length of plant exposure to deposition per harvest of the edible portion of the i^{th} plant group (0.16 unitless)

Yp = crop yield or productivity (2.24 kg dry-weight/m^2)

Both the root uptake and surficial contributions were then combined (*i.e.*, 0.0088 mg/kg wet-weight from root uptake *and* 0.0000014 mg/kg wet-weight from surficial deposition) to arrive at the concentration of inorganic arsenic in the above-ground plant tissues (*i.e.*, 0.0088 mg/kg wet-weight). This predicted concentration was then used to estimate the potential exposures to inorganic arsenic that might received through the consumption of berries and traditional above-ground plants under the "baseline" scenario. The calculation involved translating the plant tissue concentration to the corresponding exposure estimates (µg/kg BW/day) using traditional plant ingestion rates reported in the literature. The results are shown in Table 18.

The most conservative estimate of above-ground plant inorganic arsenic concentrations appears to be associated with the use of the Regression model (*i.e.* 0.009 mg/kg wet weight). This predicted concentration was then used to estimate the intake of inorganic arsenic to humans *via* the ingestion of above-ground plants. The calculation involved translating the plant tissue concentration to the corresponding exposure estimates (µg/kg BW/day) using traditional plant ingestion rates reported in the literature. The results are shown in Table 18.

Future Development Scenario

Reliance was placed on predictive modeling performed *de novo* by CEI to predict potential arsenic levels in the plant tissues that might result from the uptake of arsenic from soils *via* the roots relied on a step-wise series of calculations using the equations already described (*i.e.*, Equations 1, 2 and 3). The first calculation involved estimation of the concentration of inorganic arsenic that might occur in soil from the deposition of emissions from future anthropogenic sources. The calculation was described earlier (*Incidental Soil Ingestion Pathway*). It was based on use of Equation #1, assuming a loading rate of 0.00001 kg As/ha/yr (Suncor 2005a), and yielded a soil concentration of 0.005 mg/kg (as inorganic arsenic). This soil concentration was subsequently entered into both the BCF model (Equation #2) and Regression model (Equation #3) in order to estimate the *total* arsenic content of the plant tissues, as outlined previously (see immediately preceding discussion).

Prediction of the potential arsenic levels in the plant tissues that might result from the surficial deposition of arsenic from the air onto the leaves and other exposed surfaces was calculated as outlined earlier using Equation #4. A deposition rate of 0.00001 kg arsenic/ha/yr was assumed based on information reported by Suncor (2005a). The calculation revealed that the contribution from surficial deposition would amount to 0.00000014 mg/kg wet-weight, as inorganic arsenic. These predicted values are presented in Table 15 below.

Table 15. Summary of Predicted Inorganic[(1)] Arsenic Concentrations Using Two Modelling Approaches (mg/kg wet weight)[(2)]

Plant Category	Future	
	BCF Model	Regression Model
Above-ground plants	0.000012	0.00040

(1) The predictive models estimated arsenic concentrations for total arsenic. Based upon ATSDR (2005), it was assumed that 37% of arsenic in plants was in the inorganic form. This conversion has been applied to this table.

(2) The predictive models generated results in units of mg/kg dry weight. Adjustment of the measured concentrations from a dry-weight to wet-weight basis was performed in order to maintain consistency with the predictions made for the other types of plant foodstuffs. The latter predictions relied on the use of predictive models that were configured on a wet-weight basis. The adjustment was based on the moisture content of plants (85%) reported by Suter *et al*. (2000).

The highest plant tissue concentration appears to be predicted by the Regression model - 0.00040 mg/kg wet-weight.

The inorganic arsenic content of the plant tissues revealed by the predictive modelling is shown in Table 16 (in Section 6.4.3.3 below) for both of the above-ground plant categories. The predicted concentrations were then used to estimate the potential exposures to inorganic arsenic that might be received through the ingestion of these traditional plant foods under the "future" development scenario by applying traditional plant ingestion rates reported in the literature.

6.2.3.2.3.2 Roots and Other Below-Ground Plant Foodstuffs

As mentioned previously, a different methodology was used to evaluate the contribution of arsenic intake from the consumption of below-ground roots, such as cattails. The approaches used for the baseline and future scenarios are presented below.

Baseline Development Scenario

Several EIAs have attempted to measure arsenic in cattail and rat root samples. This information, as well as the 95% UCLMs calculated by CEI for this historical data are presented in Table 16.

Table 16. Summary of Arsenic Concentrations Measured in Root Samples Collected From the Wood-Buffalo Region (from recent environmental impact assessments)

Designation	Source	As Content(1) (2) (mg/kg – dry weight)		
		Cattail root	Rat root	Cattail & rat root combined
Kearl	Imperial Oil (2006)	< 0.03 (5)		<0.2 (5)
Muskeg Mine	Albian Sands Energy Inc. (2005)	< 0.03 (5)		<0.2 (5)
North Steepbank	Suncor (2005a)	< 0.03 (5)		(0.2 (5)
Voyageur	Suncor (2005a)	<0.03 to 0.39 (5)		<0.2 to 2.6 (5)
Fort McMurray	Albian Sands Energy Inc. (2005)	0.045 to 1.02 (5)		0.3 to 6.8 (5)
Horizon	CNRL (2003)	<0.03 to 0.66 (8)		<0.2 to 4.4 (8)
Jackpine	Shell Canada (2002)	0.03 to 0.135 (5)		0.2 to 0.9 (5)
Meadow Creek	Petro-Canada (2001)	<0.03 to 1.14 (10)		<0.2 to 7.6 (10)
Fort McKay, Fort Chipewyan; Lease 13	(Golder Associates 2000)	<0.075 to 1.96 (21)	<0.075 to 1.2 (11)	<0.075 to 1.96 (32)
Mean (EIA Data, wet weight)*	-	***0.21***	***0.48***	***0.25***
95% UCLM (EIA Data, wet weight)*	-	***0.48***	***0.69***	***0.51***
Mean (AHW 2006 Data)	-	***0.33***	-	-
95% UCLM (AHW 2006 Data)	-	***1.9***	-	-

(1) Assumed to represent total arsenic. Values in parentheses refer to number of samples collected and analyzed.
(2) Calculated assuming that the arsenic content of samples that were "non-detect" was equivalent to one-half of the detection limit.

The 95% UCLM from the historical EIA data set ranges from 0.48 – 0.69 mg/kg wet weight for roots, with the maximum detected level being of 1.9 mg/kg wet weight). In comparison, the

AHW 2006 data set for cattails [12] (presented in Section 5 of this report) presents a 95% UCLM for total arsenic concentrations in cattail roots of approximately 1.9 mg/kg (wet weight). As detectable levels of arsenic were recorded for the cattail and rat root samples, the calculation of the arsenic exposures that might be received through the consumption of roots and other below-ground plants relied on the empirical data alone, without the need for predictive modelling. The root total arsenic concentration of 1.9 mg/kg wet weight, which is representative of *both* the 95% UCLM from the AHW 2006 study and the maximum detected level in the historical EIA data was used to predict the potential baseline inorganic arsenic exposures *via* the consumption of roots. Using the 37% conversion from ATSDR (2005), this value is approximately equivalent to an inorganic arsenic concentration of 0.7 mg/kg wet weight.

Future Development Scenario

For roots and other below-ground plants, a different approach was used to forecast the arsenic content of the plant tissues. As roots are found below ground, estimation of the amount of arsenic that might occur in the edible plant tissues from surficial deposition was deemed to be unnecessary. In addition, unlike the above-ground plants, cattail roots typically grow in an aquatic environment as opposed to a terrestrial environment, with the roots being in sediment rather than soil. Thus, the accumulation and sediment concentrations in the root environment for these plants are different than for above-ground plants growing in soil. Instead of calculating the root uptake from soil as described above for the above-ground plants, the incremental change in the total arsenic content of the roots was based on the earlier estimate provided by Golder Associates (2006) (*i.e.*, 0.00002 mg total arsenic/kg wet-weight). This value was then converted to the equivalent inorganic arsenic content, as outlined earlier. The result is shown in Table 16. It was then used to forecast the potential exposures to inorganic arsenic that might be received through the ingestion of roots under the "future" development scenario by applying traditional plant ingestion rates reported in the literature.

Summary – Consumption of Traditional Plant Foodstuffs Pathway

The estimated inorganic arsenic concentrations in traditional plant foods for the baseline, future, and combined scenarios that were used in the CEI risk assessment are presented in Table 17.

Table 17. Inorganic Arsenic Concentrations in Plant Tissues (mg/kg wet- weight) Used in CEI Exposure Model

Plant Category	Development Scenario		
	Baseline	Future	Combined
Berries	0.009	0.0004	0.0094
Aboveground plants	0.009	0.0004	0.0094
Below ground roots	0.703	0.0000074	0.19

[12] Adjustment of the measured concentrations from a dry-weight to wet-weight basis was performed in order to maintain consistency with the predictions made for the other types of plant foodstuffs. The latter predictions relied on the use of predictive models that were configured on a wet-weight basis. The adjustment was based on the moisture content of plants (85%) reported by Suter *et al*. (2000).

The potential exposures to inorganic arsenic that might be received by the indigenous receptor living in the Wood-Buffalo region through the consumption of traditional plant foodstuffs under each development scenario are listed in Table 18. The potential exposures are segregated by plant category and receptor age group. Examination of the estimates reveals that the exposures predicted for the future development scenario are considerably less than those calculated for the baseline scenario, signalling that only a very marginal increase in exposure to inorganic arsenic *via* the consumption of traditional plant foodstuffs would be expected from future anthropogenic activities in the region.

Table 18. Estimated Exposures to Inorganic Arsenic Received *via* Consumption of Traditional Plant Foodstuffs (μg/kg BW/day)

Age Category	Ingestion Rate[1,2] [g/day]	Development Scenario		
		Baseline	Future	Combined
Berries				
Infant	3	0.0029	0.0001288	0.003
Toddler	5	0.0029	0.0001288	0.003
Child	11	0.0029	0.0001288	0.003
Adolescent	19	0.0029	0.0001288	0.003
Adult	23	0.0029	0.0001288	0.003
Composite (LADD)		***0.0029***	***0.0001288***	***0.003***
Traditional Above-ground Plants[3]				
Infant	0	0.00037	0.0000168	0.00039
Toddler	1	0.00037	0.0000168	0.00039
Child	1	0.00037	0.0000168	0.00039
Adolescent	3	0.00037	0.0000168	0.00039
Adult	3	0.00037	0.0000168	0.00039
Composite (LADD)		0.00037	0.0000168	0.00039
Roots and Traditional Below-ground Plants[4]				
Infant	0.35	0.030	0.00000031	0.03
Toddler	1	0.030	0.00000031	0.03
Child	1	0.030	0.00000031	0.03
Adolescent	3	0.030	0.00000031	0.03
Adult	3	0.030	0.00000031	0.03
Composite(LADD)		***0.030***	***0.00000031***	***0.03***

(1) The consumption rate of 23 g/day for the adult receptor was obtained from a survey of food consumption in the Wood Buffalo National Park area by Wein (1989). The consumption rates for the other age groups were adjusted relative to body weight to the adult consumption rate. Due to this adjustment, the exposure estimates for the various age groups and the composite receptor appear to be the same.

(2) For all plants and roots, CEI assumed that individuals were exposed 365 days/year

(3) The ingestion rate for traditional plants was derived by CEI based upon the combined consumption rates for traditional mint and Labrador teas outlined in Wein (1989).

(4) The ingestion rate for roots (*e.g.,* cattail root) was assumed to be the combined ingestion rate of traditional mint and Labrador teas from Wein (1989). CEI has assumed that plant root infusions would be consumed for traditional medicinal purposes, due to a lack of information regarding how root preparations are used.

6.2.3.2.4 Game Meat Ingestion Pathway

Game animals are commonly used as a food source by indigenous people living in the Wood-Buffalo region, with large and small mammals, upland game birds, and waterfowl harvested. Estimates of the potential exposures to inorganic arsenic that could result from the consumption of game meat were calculated using one of two approaches depending on the development scenario under consideration. The approaches are outlined below:

Baseline Development Scenario

Two different approaches (A and B) were employed in the baseline assessment of game meat, with only one being used in the CEI risk assessment (Approach A). These are described below.

A Baseline Approach Using Analytical Data

Concentrations of arsenic in game have been presented in previous studies in the oil sands region, and are presented in Table 19. These studies have consistently reported non-detect values.

Table 19. Reported Arsenic Concentrations in Game Meat Samples prior to 2006 (mg/kg dry-weight)[(1)]

Source	Snowshoe Hare	Grouse	Beaver	Moose
Fort MacKay (1999 to 2000)[(2)]	<0.2 (14)	<0.2 (9)	<0.2 (7)	<0.2 (5)
Athabasca Oil Sands Report [(3)]	<0.2	<0.2 (2)	<0.2	<0.2

(1) Values in parentheses refer to the number of samples of each type of game meat that were collected and analyzed.

(2) Data obtained from a traditional food study commissioned by the Terrestrial Environmental Effects Monitoring subcommittee of the Wood Buffalo Environmental Association, referenced from the Shell Canada Jackpine Mine – Phase 1 project (Shell Canada 2002).

(3) Data obtained from the Athabasca Oil Sands Traditional Foods study (Golder Associates 2003).

In an effort to provide a better understanding of the baseline concentrations of arsenic in game meats, samples were recently collected (*i.e.*, AHW 2006 and reference samples) and analyzed with an analytical technique that permits a lower level of detection. A summary of the game meat tissue results from AHW is presented in Table 20.

The extent to which liver and kidney meat is consumed in the Wood Buffalo Region is unknown. In addition, very few liver and kidney samples from Wood Buffalo moose and deer were obtained, and as a result, the data that has been collected for these tissues may not be representative. It is likely that muscle meat is most frequently consumed, and as several muscle tissue samples were obtained, muscle was selected as the game meat sample type for inclusion in the risk assessment. Although both moose and deer muscle samples were collected for the Region, the moose presented higher mean levels of total arsenic than deer. Therefore, the 95%

UCLM for moose muscle of 0.0205 mg/kg wet weight was utilized to predict arsenic exposures that may be received as a result of game meat ingestion.

Table 20. Summary of Total Arsenic Concentrations (mg/kg wet weight)

Region	Type	Average	95%UCLM
Yukon (Reference)	Moose - Kidney	0.0067	0.014
	Moose - Liver	0.027	0.072
	Moose - Muscle	0.0082	0.0134
Outside Edmonton (Reference)(1)	Deer – Muscle & Liver	0.002	-- (2)
Peace River & Grande Prairie (Reference)	White-tailed Deer	0.0005	-- (2)
Wood Buffalo Region (Regional)	Moose - Kidney	0.0015	-- (1)
	Moose – Heart	0.0005	-- (1)
	Moose – Liver	0.012	-- (1)
	Moose - Muscle	0.008	0.020
	White-tailed deer	0.0066	0.011

(1) The 'Outside Edmonton' area from which the samples were collected included the Strathcona Wilderness Centre and locations near Westaskiwin Alberta.

(2) Too few observations to calculate value

This concentration was translated to the equivalent exposures (*i.e.*, µg/kg BW/day, assuming 37% of the arsenic in the meat consisted of inorganic arsenic) that might be received by an indigenous person through the consumption of various types of game meat using ingestion rates reported in the literature for large mammals, small mammals, waterfowl, and upland game birds (Wein 1989)[13].

B Baseline Approach Using Predictive Modelling

In order to validate the predictive models used to determine future game meat concentrations, the baseline assessment used measured concentrations in soil and surface water, and predicted concentrations in forage to estimate baseline moose meat concentrations for comparison. The values calculated using this model for baseline were **not** employed in the CEI risk assessment. Rather, this exercise is intended to validate the model developed by CEI with the measured analytical data to ensure that this model is sufficiently accurate in predicting tissue concentrations in game meat for the Future Scenario. Detail regarding the models, input values and modelling results for game meat may be found in Appendix D of this report.

Future Development Scenario

For the "future" development scenario, reliance was placed on predictions of the incremental increase in arsenic concentrations in game meats that might result from future development

[13] Game animals considered within these four categories included: moose, caribou, bison, bear, hare, muskrat, beaver, lynx, duck, good, swan, spruce hen, ruffed grouse, sharp-tail, ptarmigan (Wein 1989).

activities in the region. Exposures were predicted using a biotransfer model that provided estimates of the amount of inorganic arsenic that could be found in game meat as a result of the game animals being exposed to arsenic in the natural environment.

Moose and other wildlife consume above-ground vegetation (forage) as a food source, and these plants may take-up arsenic from soil *via* roots and surficial deposition. Root uptake of forage plants was calculated using the BCF and Regression Models (Equations 2 and 3) described previously in the Traditional Above-Ground Plants section. Using the Regression Model (Equation 3), the highest predicted amount of total arsenic in forage resulting from root uptake was determined to be 0.0071 mg/kg dry weight.

Arsenic accumulation due to surficial deposition in forage is calculated using a different algorithm from the U.S. EPA (2005c), as presented in Equation 5 below. The calculation was based on a deposition rate of 0.00001 kg As/ha/yr, which was deemed to represent the arsenic loading rate from existing facilities in the region (Suncor 2005a).

$$Pd = \frac{CF \times DR \times R_p \times [1.0 - \exp(-kp \times Tp)]}{Yp \times kp}$$ ***Equation #5***

Where:

P_d	=	plant tissue concentration due to deposition
DR	=	deposition rate (0.000000001 kg/m^2/year)
CF	=	conversion factor (1,000,000 mg/kg)
R_p	=	intercept fraction of edible portions of plant (0.5 unitless)
kp	=	plant surface loss coefficient (18 year^{-1})
Tp	=	length of plant exposure to deposition per harvest of the edible portion of the i^{th} plant group (0.12 unitless)
Yp	=	crop yield or productivity (0.24 kg DW/m^2)

The predicted total arsenic concentration in forage resulting from root uptake (0.0071 mg/kg dry weight) and surficial deposition (0.0001 mg/kg dry weight) is approximately 0.0072 mg/kg dry weight. This value is not converted to inorganic arsenic or units of dry weight as completed previously in order to be consistent with the calculation for game meat presented below. Equation 6 outlines the model (validated in Appendix D) used to predict game meat tissue concentrations.

$$C_m = BTF \times MF \times \left[(C_p \times FIR) + (C_s \times SIR \times BA_s) + (Cw \times WIR \times \mathrm{BA}_s(\mathrm{w}))\right]$$ ***Equation #6***

Where:

C_m	=	concentration of total arsenic in moose muscle (calculated to be 0.14 mg/kg wet-weight)
BTF	=	bio-transfer factor (0.002 day/kg wet-weight) (U.S. EPA 2005c)
MF	=	metabolism factor (Assumed to be 1.0; unitless)
C_p	=	concentration of total arsenic in plants (0.0072 mg/kg dry-weight)
FIR	=	food ingestion rate for moose (9.0 kg dry weight / day; U.S. EPA 1993)
C_s	=	0.005 mg/kg dry-weight (future soil concentration based upon deposition rate).
SIR	=	soil ingestion rate (0.132 kg/day = 2%×FIR; Suter *et al*. 2000)
BA_s	=	bioavailability of arsenic in soil (Assumed to be 1.0; unitless)

C_w = 0.0001 mg/L (*i.e.*, estimated future arsenic content of surface waters)
WIR = water ingestion rate (24 L/day)
$BA_{s(w)}$ = bioavailability of arsenic in drinking water (Assumed to be 1.0; unitless)

When converted using the assumption that 37% of the arsenic content is inorganic (ATSDR 2005), an estimated future inorganic arsenic concentration of 0.000051 mg/kg wet weight is calculated for moose muscle.

The corresponding estimates of the exposures (µg/kg BW/day) that might be received by an indigenous person through the consumption of all types of game meat are shown in Table 21.

Table 21. Inorganic Arsenic Content of Moose Meat (mg/kg wet-weight)

Game Meat	Development Scenario	
	Baseline	Future[1]
Measured (95% UCLM from AHW study)	0.0074	0.000051
Predicted (CEI Model)	0.0013	0.000051

[1] All moose meat arsenic concentrations within the Future scenario are modelled, and are added to the baseline (either measured or predicted) to obtain the estimated concentrations in the Combined scenario.

Summary – Game Meat Ingestion Pathway

The potential exposures to inorganic arsenic that might be received by the indigenous receptor living in the Wood-Buffalo region through the consumption of game meat under each development scenario are listed in Table 22. The potential exposures are segregated by age category. Examination of the estimates reveals that the exposures predicted for the future development scenario are well below those calculated for the baseline scenario. The findings indicate that any increase in exposure to inorganic arsenic *via* the consumption of game meat resulting from future anthropogenic activities in the region is likely to be negligible.

Table 22. Estimated Exposures to Inorganic Arsenic Received *via* Consumption of Game Meat (µg/kg BW/day)

Age Category	Game Meat Ingestion Rate [g/day] [(1)]	Development Scenario		
		Baseline	Future	Combined
Infant[(2)]	0	0.0	0.0	0.0
Toddler	25	0.011	0.000076	0.011
Child	49	0.011	0.000076	0.011
Adolescent	90	0.011	0.000076	0.011
Adult	106	0.011	0.000076	0.011
Composite (LADD)[(3)]		***0.011***	***0.000076***	***0.011***

(1) The game consumption rate for an adult was calculated based on information compiled by Wein (1989) as part of the Wood Buffalo National Park study. This rate is based upon a game meat consumption of 207 g/day, at a consumption frequency that is the sum of the consumption rates for large and small mammals, waterfowl, and upland birds. The game ingestion rates for the other age categories were adjusted relative to the adult rate with respect to body weight. Thus, the exposure concentrations for the different age groups appear to be the same.

(2) The game consumption rate for infants in Health Canada (2004) is reported to be zero. The extent of the transfer of inorganic arsenic from maternal blood to human breast milk is anticipated to be minimal (Concha *et al.* 1998a; 1998b).

(3) The exposure levels for the composite receptor appear to be the same as for each receptor category, due to the adjustment for consumption rate scaled with respect to body weight relative to the adult consumption rate.

6.2.3.2.5 Fish Consumption Pathway

Estimates of the potential exposures to inorganic arsenic that could result from the consumption of sport fish were calculated by one of two means depending on the development scenario under consideration. For the "baseline" scenario, exposures were predicted on the basis of the measured arsenic content of samples of fish fillets collected from the Wood-Buffalo region. For the "future" scenario, reliance was placed on predictions of the incremental increase in the arsenic content of fish tissues that might occur as a result of increases in the arsenic levels in local surface waters as a consequence of future development activities in the region (Suncor 2005a). The approach followed for each scenario is outlined below.

Baseline Development Scenario

The potential exposures that might be received under the "baseline" development scenario were estimated on the basis of measured concentrations of arsenic in fish fillets or muscle collected as part of the Regional Aquatic Monitoring Program (RAMP) for the Municipality of Wood-Buffalo for the period 1998 to 2004 (RAMP 1998; 2001; 2002; 2003; 2004). A variety of species of sport fish were sampled, including gold eye, walleye, lake white fish, and Northern pike. The fish were collected from a number of local water bodies, including the Muskeg River, Athabasca River, Clearwater River, and Gregoire Lake. A total of 34 samples of fish tissues were submitted for analysis of total arsenic content. Most measurements revealed non-detectable levels of arsenic in the fish tissues (*i.e.,* < 0.02 to 0.2 mg/kg). The results are summarized in Table 23.

Table 23. Summary of the Total Arsenic Content of Fish Fillets Collected From the Wood-Buffalo Region (mg/kg wet-weight)[1]

Detection Limit Assumption	Total As Concentration (mg/kg wet-weight)	
	Mean	95th UCLM
Arsenic assumed to be present at the analytical detection limit	0.16	0.21
Arsenic assumed to be present at one-half of the detection limit	0.081	0.10
Arsenic concentration assumed to be zero when non-detectable	0.02	NA[2]

(1) Source: RAMP (1998; 2001; 2002; 2003; 2004).

(2) Not available, as value could not be calculated due to data distribution.

Based on the above, the concentration of total arsenic in fish was set at 0.10 mg/kg (wet-weight) for the purpose of estimating the exposures that might be received from consuming local sport fish under the 'baseline' development scenario. This concentration was converted to the equivalent inorganic arsenic content (*i.e.*, 0.037 mg/kg wet-weight), assuming that 37% of the total arsenic found in fish tissue is present as the inorganic form (ATSDR 2005). The inorganic arsenic content of the fish tissues was then translated to the corresponding exposure estimates (µg/kg BW/day) for the various receptor age categories using fish consumption rates compiled by Health Canada (2004), after adjustment to accommodate the higher frequency of fish meals consumed by indigenous people (FMES 1997). The resulting exposure estimates are shown in Table 23.

Future Development Scenario

Estimates of the potential incremental exposures to inorganic arsenic that might be received *via* the consumption of sport fish under the "future" development scenario were calculated on the basis of the premise that arsenic contained in the emissions originating from any added facilities would deposit on the local surface waters and would then accumulate within the fish tissues. The calculation relied on an already-predicted annual deposition rate of 0.00001 kg As/hectare (Suncor 2005a).[14] The calculation proceeded using the equation shown below:

$$C_f = C_w \times BCF_f \qquad \textbf{\textit{Equation \#6}}$$

Where:

C_f = Concentration in fish (calculated to be 0.01 mg/kg wet-weight, as total arsenic)

C_w = Predicted change in median arsenic concentration in surface water (0.0001 mg/L) (Suncor 2005a)

BCF = Bioconcentration Factor for fish (110 L/kg) (Suncor 2005a)

The predicted total arsenic content of the fish tissues (*i.e.*, 0.01 mg/kg wet-weight) was converted to the equivalent inorganic arsenic content (*i.e.*, 0.0037 mg/kg wet-weight), as described previously. The inorganic arsenic content of the fish tissues was then translated to the

[14] In the absence of information, the deposition rate was assumed to apply to inorganic arsenic.

corresponding exposure estimates (μg/kg BW/day), as outlined above. The results are presented in Table 23.

Summary – Fish Consumption Pathway

The potential exposures to inorganic arsenic that might be received by the indigenous receptor living in the Wood-Buffalo region through the consumption of sport fish under each development scenario are shown in Table 23. The potential exposures are segregated by age category. Examination of the estimates reveals that the exposures predicted for the future development scenario are 10-fold lower than those calculated for the baseline scenario, signalling that any increase in exposure to inorganic arsenic *via* the consumption of sport fish as a result of future anthropogenic activities is expected to be marginal.

Table 24. Estimated Exposures to Inorganic Arsenic Received *via* the Consumption of Sport Fish (μg/kg BW/day)

Age Category	Fish Ingestion Rate [g/day] (1)	Development Scenario		
		Baseline	Future	Combined
Infant(2)	0	0.0	0.0	0.0
Toddler	22	0.049	0.0049	0.054
Child	40	0.045	0.0045	0.050
Adolescent	47	0.029	0.0029	0.032
Adult	51	0.027	0.0027	0.030
Composite (LADD)		***0.030***	***0.0030***	***0.033***

(1) Adjusted from fish consumption rates reported by Health Canada (2004). These rates were not considered to be representative of the amounts of fish that might be consumed by an indigenous receptor in the Wood-Buffalo region. Accordingly, the rates were adjusted on the basis of data collected as part of a survey of traditional food use among First Nations people living in the community of Fort MacKay (FMES 1997). The survey revealed a relatively high frequency of consumption of fish meals (*i.e.*, 85 per year) by these people. It was assumed that the amounts of fish reported to be consumed by Health Canada (2004) were eaten as part of each fish meal. Note that the calculated adult fish consumption rate of 51 grams/day is comparable to the median consumption rate of 46 grams/day previously reported by AHW (1997).

(2) The fish consumption rate in Health Canada (2004) is zero. The extent of the transfer of inorganic arsenic from maternal blood to human breast milk is anticipated to be minimal (Concha *et al*. 1998a; 1998b).

6.2.3.2.6 Summary of Exposure Estimates (All Pathways)

Estimates of the total exposures to inorganic arsenic that might potentially be received *via* the oral route (*i.e.*, *via* ingestion) by an indigenous person living in the Wood-Buffalo region under each development scenario are listed in Table 25. The estimates are segregated by age category. The total exposures represent the combined exposures received through all of the various exposures pathways examined (*i.e.*, incidental ingestion of soil, consumption of drinking water, consumption of traditional plant foodstuffs, consumption of game meat, and consumption of sport fish). As described above, the exposure estimates were calculated with consideration given to the lifestyle and dietary habits of the indigenous people living in the area. It is evident from the findings that the combined exposures are dominated by the contributions from the "baseline" development scenario, with more than 90% of the combined exposures being directly attributable to already existing sources of arsenic in the region. The incremental change in exposure that potentially could result from future anthropogenic activities in the area was judged to be marginal (*i.e.,* < 10% of the combined exposures).

Table 25. Summary of Potential Exposures to Inorganic Arsenic From All Exposure Pathways Examined (µg/kg BW/day)[(1)]

Age Category	Development Scenario		
	Baseline	Future	Combined
Infant	0.078	0.0038	0.082
Toddler	0.140	0.009	0.15
Child	0.118	0.0072	0.12
Teen	0.093	0.0048	0.1
Adult	0.096	0.0050	0.1
Composite(LADD)	***0.100***	***0.0054***	***0.1***

(1) The exposure pathways examined included: i) incidental ingestion of soil; ii) consumption of drinking water; iii) consumption of traditional plant foodstuffs; iv) ingestion of game meat; and, v) consumption of sport fish. The values shown represent the potential exposures that might be received from all pathways combined.

6.2.4 Risk Characterization

The Risk Characterization step of the risk assessment paradigm is concerned with quantifying the potential health risks that could result from exposure to the chemical(s) of potential concern (COPCs). The risks are expressed as Exposure Ratios (ERs), and are calculated by comparing the predicted exposures (determined as part of the Exposure Assessment step – see Section 6.2.3) against the corresponding Exposure Limits (determined as part of the Toxicity Assessment step – see Section 6.2.2) using the following equation:

$$ER = \frac{\text{Estimated Daily Exposure } (\mu g/kg \text{ BW})}{\text{Exposure Limit } (\mu g/kg \text{ BW/day})} \qquad \textbf{\textit{Equation 7}}$$

Consistent with the Terms of Reference developed for the present work (see 2.0 TERMS OF REFERENCE), the risk characterization step of the re-assessment was directed at estimating the lifetime cancer risks that could be presented to indigenous people living in the Wood-Buffalo region from exposure to inorganic arsenic. Two sets of cancer risk estimates were derived *per* the instructions received from Alberta Health and Wellness that that the risks be calculated on the basis of both:

- The Risk-Specific Dose (RsD) of 0.006 µ/kg BW/day determined from the "negligible risk level" for inorganic arsenic of 0.3 µ/L recommended by the Water Quality and Health Bureau of Health Canada (2006); and,
- The RsD of 0.003 µ/kg BW/day determined from the Slope Factor of 2.8 $(mg/kg\text{-}d)^{-1}$ recommended by the Contaminated Sites Division of Health Canada (2005).

As indicated earlier, the RsD values correspond to a *de minimus* risk of one extra cancer case in a population of 100,000 people (1×10^{-5}) (see Section 4 TOXICITY ASSESSMENT). The lifetime cancer risks were expressed as Exposure Ratios (ERs), calculated as follows:

$$ER = \frac{\text{Estimated Daily Exposure } (\mu g/kg \text{ BW/day})}{\text{RsD } (\mu g/kg \text{ BW/day})} \qquad \textbf{\textit{Equation 8}}$$

Interpretation of the ER values proceeded as follows:

- For the baseline *and* combined development scenarios, the ERs simply refer to the number of cancer cases that could potentially result from the estimated exposures to inorganic arsenic among a population of 100,000 people. Since an acceptable cancer incidence rate has *not* been recommended for exposure to background or baseline levels of carcinogens by any leading scientific or regulatory authority, interpretation of the significance of the ER values determined for these development scenarios was *not* based on comparison against a numerical "benchmark" *per se*, but instead was simply based on consideration of the number of predicted cancer cases adjusted to reflect the population base of indigenous people living in the Wood-Buffalo region (see Section 6.4 INTERPRETATION OF FINDINGS).
- For the future development scenario, the ERs refer to the extra cancer cases that could potentially result from the incremental exposures to inorganic arsenic contributed by future anthropogenic activities in the Wood-Buffalo region. Interpretation of these incremental lifetime cancer risks was based on comparison of the ER values against the "benchmark" ILCR of 1×10^{-5} (*i.e.,* one extra cancer case in a population of 100,000 people). This benchmark corresponds to a *de minimus* risk level that is considered acceptable by most leading authorities for the protection of public health. Interpretation of the ER values in this instance proceeded as follows:
  - $ER \leq 1.0$ Signifies an incremental lifetime cancer risk that is below the benchmark ILCR of 1×10^{-5} (*i.e.*, within the generally accepted limit deemed to be protective of public health).
  - $ER > 1.0$ Signifies an incremental lifetime cancer risk that is greater than the *de minimus* risk level of 1×10^{-5}, the interpretation of which must consider the conservatism incorporated into the assessment.
- Regardless of the development scenario examined, the calculation of the lifetime cancer risks applied to the "composite" receptor (*i.e.*, all age categories combined), and reflected the potential risks associated with exposure to inorganic arsenic over the course of an 80-year lifetime.
- Regardless of the development scenario examined, the interpretation of the significance of the potential lifetime cancer risks necessarily had to weigh both the uncertainties surrounding the assessment as well as the conservatism embraced by the work.

Further details concerning the interpretation of the cancer risk estimates can be found in Section 6.4 INTERPRETATION OF FINDINGS.

6.3 Results

The predicted lifetime cancer risks (expressed as ER values) associated with exposure to inorganic arsenic among indigenous people living in the Wood-Buffalo region are summarized in Table 26. The results are segregated according to:

- The Exposure Limit used in the calculations (*i.e.*, the RsD of 0.006 μ/kg BW/day recommended by the Water Quality and Health Bureau of Health Canada (2006) *vs*. the RsD of 0.003 μ/kg BW/day developed by the Contaminated Sites Division of Health Canada (2004));
- The development scenario examined (*i.e.*, baseline *vs*. future *vs*. combined); and,
- The exposure pathway involved (*i.e.*, incidental ingestion of soil *vs*. consumption of drinking water *vs*. consumption of traditional plant foodstuffs *vs*. consumption of game meat *vs*. consumption of sport fish *vs*. all pathways combined).

The percentage contribution of each exposure pathway to the lifetime cancer risks is shown in Table 27. For the Consumption of Traditional Plant Foodstuffs pathway, distinction is made between the contributions from each of the food categories examined (*i.e.*, berries *vs*. above-ground plants *vs*. roots and other below-ground plants).

Table 26. Estimated Lifetime Cancer Risks Presented to the Composite Receptor from Exposure to Inorganic Arsenic

Exposure Pathway	ER Value (unitless)		
	Development Scenario		
	Baseline	Future	Combined
Calculation based on RsD of 0.006 μ/kg BW/day (1)			
Incidental ingestion of soil	0.063	0.00026	0.063
Consumption of drinking water	4.3	0.36	4.7
Consumption of traditional plant foodstuffs	5.6	0.024	5.6
Consumption of game meat	1.9	0.013	1.9
Consumption of sport fish	4.9	0.49	5.4
All Pathways Combined	*16.7*	*0.90*	*17.7*
Calculation based on RsD of 0.003 μ/kg BW/day (2)			
Incidental ingestion of soil	0.13	0.00052	0.13
Consumption of drinking water	8.6	0.74	9.3
Consumption of traditional plant foodstuffs	11.0	0.049	11.1
Consumption of game meat	3.8	0.025	3.8
Consumption of sport fish	9.9	0.99	10.9
All Pathways Combined	*33.4*	*1.8*	*35.2*

(1) Recommended by the Water Quality and Health Bureau of Health Canada (2006).
(2) Recommended by the Contaminated Sites Division of Health Canada (2004).

Table 27. Contribution of Individual Exposure Pathways to Potential Lifetime Cancer Risks

Exposure Pathway	Pathway Contribution (%) Development Scenario		
	Baseline	Future	Combined
Incidental ingestion of soil	0.4%	0.03%	0.4%
Consumption of drinking water	26%	41%	27%
Berries	3%	2%	3%
Above-ground plants	0.4%	0.3%	0.4%
Roots and other below-ground plants	30%	0.01%	28%
Consumption of game meat	11%	1%	11%
Consumption of sport fish	30%	55%	31%
All Pathways Combined	*100%*	*100%*	*100%*

The principal findings that emerged from the re-assessment were:

- The ER values predicted for the baseline development scenario ranged from ≈ 17 to 33 (depending on the Exposure Limit used in the calculations), signifying that lifetime exposure to inorganic arsenic among indigenous people living in the Wood-Buffalo region *via* the exposure pathways examined potentially could contribute to ≈ 17 to 30 cases of cancer when calculated on a 100,000-person population basis. The "acceptability" of this potential lifetime cancer risk from a public health perspective cannot be determined following a conventional approach since an acceptable "benchmark" cancer risk level for exposure to background levels of carcinogens is not available for comparison. (Note that the predicted exposures determined for the baseline development scenario represented a combination of exposures from naturally-occurring or background sources of inorganic arsenic as well as exposures from existing anthropogenic sources of arsenic in the Wood-Buffalo region).
- The ER values predicted for the future development scenario ranged from ≈ 1 to 2, signifying that lifetime exposure to inorganic arsenic contributed by future anthropogenic activities in the Wood-Buffalo region might potentially contribute to 1 to 2 extra cancer cases among indigenous people living in the area when calculated on a 100,000-person population basis. The predicted incremental lifetime cancer risks "bridged" the benchmark ILCR of 1×10^{-5} (*i.e.*, one extra cancer case in a population of 100,000 people) deemed to be acceptable by most leading scientific and regulatory authorities. More specifically, the ILCR calculated using the Exposure Limit recommended by the Water Quality and Health Bureau of Health Canada (*i.e.*, 0.006 μ/kg BW/day) was slightly below the comparison benchmark (*i.e.*, ER = 0.9); whereas, the ILCR determined using the limit recommended by the Contaminated Sites Division of Health Canada (*i.e.*, 0.003 μ/kg BW/day) was slightly above the comparison benchmark (*i.e.*, ER = 1.8).
- The ER values predicted for the combined development scenario (calculated on the basis of the combined exposures determined for both the baseline *and* future scenarios) ranged from ≈ 18 to 35, closely mimicking those predicted for the baseline scenario itself (*i.e.*, ER = ≈ 17

to 33). The finding signifies that the lifetime cancer risks that could potentially result from exposure to inorganic arsenic among indigenous people living in the Wood-Buffalo region are dominated by the exposures contributed by already existing naturally-occurring and anthropogenic sources of arsenic in the region, with very little incremental risk presented by projected future anthropogenic activities.

- Regardless of the development scenario examined, the contribution to the total predicted exposure to inorganic arsenic that might be received by indigenous people living in the Wood-Buffalo region was dominated by certain exposure pathways, notably the Consumption of Drinking Water pathway (*i.e.*, up to 41%) and the Consumption of Sport Fish pathway (*i.e.*, up to 55%). Lesser, but still significant, contribution was revealed for the consumption of roots and other below-ground plants (as part of the Consumption of Traditional Plant Foodstuffs pathway) and the Consumption of Game Meat pathway, depending on the development scenario. The contribution from the remaining exposure pathways was negligible.

6.4 Interpretation of Findings

The interpretation of the significance of the lifetime cancer risks predicted for each of the development scenarios must carefully consider the following:

- The margins-of-safety incorporated into the risk estimates by virtue of the conservative assumptions that formed the basis of the work.
- The practical meaning of the risk estimates vis-à-vis the actual number of cancer cases that might occur among the population of indigenous people living in the Wood-Buffalo region.
- The uncertainties surrounding the cancer risk estimates … including the uncertainties intrinsic to the predictive modeling as well as the empirical datasets upon which the risk estimates were based … which were accommodated, in part, through the use of conservative assumptions.

It is important to recognize that the re-assessment was deliberately constructed to ensure that the lifetime cancer risks that might result from exposures to inorganic arsenic *via* the pathways examined would not be understated. It is also important that the conditions assumed as part of the re-assessment (and their influence on the outcomes of the work) be understood in relation to actual conditions, both in terms of the toxicity of arsenic *and* the exposures that might occur among the indigenous people living in the region. In addition, it is important to note that the predicted lifetime cancer risks were based on a 100,000-person population basis, and must be re-configured to reflect the actual numbers of indigenous people living in the Wood-Buffalo region for proper and meaningful interpretation. Finally, it is important to note that the predicted cancer risk estimates are population-based and refer to the potential risks presented to the indigenous population living in the Wood-Buffalo region as a whole. They do not refer to the risks faced by an individual person *per se* since individual circumstances affecting exposures to inorganic arsenic can be many and varied and were not addressed as part of the re-assessment. Each of these items is discussed more fully below.

6.4.1 Conservatism

A number of conservative assumptions were applied as part of the re-assessment, in part, to accommodate the uncertainties surrounding the various datasets that were used to generate the exposure estimates as well as to ensure that the potential cancer risks would not be under-estimated. A listing of the various conservative assumptions that applied can be found in Table 28. Some of the key assumptions are highlighted below.

- For the purposes of the re-assessment, it was conservatively assumed that inorganic arsenic acts as a "non-threshold" human carcinogen and exhibits a linear dose-response pattern, even at low-dose levels. As indicated previously (see 2.0 SUMMARY OF ARSENIC TOXICITY), debate surrounds both the carcinogenic potency of arsenic (… especially at low-dose-levels) as well as its mechanism of carcinogenic action. In other words, it is *not* clearly established that the carcinogenic potential of inorganic arsenic is best described by a linear non-threshold model. Some investigators claim that the carcinogenic activity of arsenic represents a high-dose phenomenon only, based, in part, on the fact that elevated rates of cancer have typically been observed only among non-North American populations chronically exposed to relatively high levels of arsenic in the drinking water (*i.e.*, >100 µg/L). By comparison, the inorganic arsenic content of the drinking water consumed by the receptor living in the Wood-Buffalo region was conservatively assumed to be 0.0012 µg/L (*i.e.,* 95% UCLM based on measured concentrations of arsenic in local rivers – see Table 5), a level more than 80,000-times lower than that reported to cause cancer.
- For the purposes of the re-assessment, the calculation of the predicted exposures to inorganic arsenic from the various exposure pathways was deliberately designed to yield the highest reasonable exposure estimates. For instance, in the absence of information, the measured amounts of arsenic in the various environmental media (*i.e.*, soil, drinking water, plant foodstuffs and fish) were assumed, by default, to represent the form (*i.e.*, total *vs*. organic *vs*. inorganic) that would correspond to the highest exposures to inorganic arsenic. Similarly, the measured levels of arsenic used to calculate the potential exposures often were described by the 95th percentile of the upper confidence limit of the mean (UCLM)[15] (*i.e.*, a reasonably conservative approach). In addition, for the purpose of calculating the incremental exposures to inorganic arsenic that might result from future anthropogenic activities, reliance was placed on conservative estimates of loading rates into the various environmental compartments (*e.g.,* the estimates of the exposures that might occur through the consumption of drinking water under the future development scenario were based on the highest predicted incremental increase in the arsenic content of local surface waters). Finally, the predictive modelling was deliberately fashioned through the choice of model input values to provide conservative forecasts of the arsenic exposures that might be received. Collectively, these measures very likely contributed to exposure estimates that over-stated that actual exposures to inorganic arsenic that might be received by indigenous people living in the region *via* the exposure pathways examined. Further examples of the conservative assumptions used as part of the Exposure Assessment can be found in Table 28.

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15 The 95% UCLM represents is the upper estimate of the mean (average) in a data set with 95% coverage (Pagano and Gauvreau 1993).

Table 28. Listing of Conservative Assumptions Applied as Part of Exposure Assessment Step of the Re-assessment

Exposure Pathway	Development Scenario	Assumption/Measure	Comments
Incidental ingestion of soil	Baseline	The arsenic content of the soils found in the Wood-Buffalo region was set at 1.3 mg/kg	The 95th upper confidence limit of the mean concentration of arsenic measured in soils gathered from the Wood-Buffalo region (*i.e.*, 1.3 mg/kg) was used to represent the arsenic content of soils for the purpose of calculating the exposures that might be received through the incidental ingestion of soil. It was conservatively assumed that people would be exposed to this soil concentration every day over a 80-year period, whereas it is much more likely that an individual would be exposed to concentrations at or near the average measured level of 0.8 mg/kg (*i.e.*, a level approximately 40% lower than that used in the calculations).
	Baseline	The arsenic content of the local soils was assumed to be comprised entirely of inorganic arsenic.	In the absence of information, it was conservatively assumed that the measured arsenic content of the soils collected from the Wood-Buffalo region was represented entirely by inorganic arsenic. It is possible that some or all of the soil analyses were conducted for total arsenic, and that some inorganic arsenic was transformed to the organic form(s) in the natural soil environment.
	Baseline	The measured arsenic content of the local soils was assumed to be expressed on a dry-weight basis.	It was not clear whether the measured soil arsenic levels were reported on a dry-weight or wet-weight basis as the original analytical data were not available. For the purposes of the re-assessment, the measured soil arsenic levels were assumed to be expressed on a dry-weight basis in order to maximize the soil arsenic content.
	All	The ingestion rate for soil was assumed to be that reported by Health Canada (2004).	For the purposes of the re-assessment, it was assumed that the indigenous receptor would consistently consume soil, every day over a 80-year lifetime, at rates reported by Health Canada (2004; 2006). Realistically, the amount of soil ingested daily over a lifetime would very likely be lower than the reported level(s) as a result of food washing, hand washing, and other hygienic practices.
	All	The arsenic found in soil was assumed to be 100% bio-available.	It was conservatively assumed that 100% of the arsenic consumed *via* the incidental ingestion of soil was bio-available (*i.e.*, fully absorbed). Oral arsenic bioavailability from soil in animal studies has been observed to be highly variable, ranging from 8 – 98% (ATSDR 2005).

Exposure Pathway	Development Scenario	Assumption/Measure	Comments
Consumption of drinking water	Baseline	The inorganic arsenic content of the drinking water was set at 0.0012 µg/L.	The 95th upper confidence limit of the mean concentration of arsenic measured in local surface waters gathered from the Wood-Buffalo region (*i.e.*, 0.0012 µg/L) served as the basis for estimating the exposures to inorganic arsenic that might received through the consumption of drinking water under the "baseline" development scenario. It was conservatively assumed that people would drink water having this arsenic content every day over an 80-year period.
	Baseline	The arsenic content of the local surface waters (… which served as the basis of estimating the exposures received through the consumption of drinking water) was assumed to be comprised entirely of inorganic arsenic	In the absence of information, it was conservatively assumed that the measured arsenic content of the local surface waters collected from the Wood-Buffalo region was represented entirely by inorganic arsenic.
	Future	The inorganic arsenic content of the drinking water was set on the basis of the highest incremental increase in the arsenic levels in local surface waters predicted to occur from future anthropogenic sources in the region.	Use of the highest predicted incremental increase in the arsenic concentration of the local surface waters likely resulted in over-statement of the exposures to inorganic arsenic received through the consumption of drinking water under the "future" development scenario.
	All	Whether in the field or community setting, it was assumed that the inorganic arsenic content of the drinking water consumed by the indigenous receptor was equivalent to the concentrations measured in the raw, untreated water.	It was conservatively assumed that no inorganic arsenic would be removed by the community water treatment system.

Exposure Pathway	Development Scenario	Assumption/Measure	Comments
Consumption of traditional plant foodstuffs	Baseline	Soil arsenic level of 1.3 mg/kg	The 95th upper confidence limit of the mean concentration of arsenic measured in soils gathered from the Wood-Buffalo region (*i.e.*, 1.3 mg/kg) was used to represent the arsenic content of soils. Over a 80-year period, it is likely that the plants consumed by residents in the Wood Buffalo area grew in soils with lower arsenic levels, and thus, actual risks are likely lower than reported.
	Baseline	People consume a mixture of parts from cattail roots (hairs, skin, stem, starch, whole root)	It was not clear which part(s) of roots are commonly used by individuals in the Region. Thus, to be protective, it was assumed that a mixture of the plant parts would be consumed, and the entire baseline analytical data set for cattails was considered (*i.e.* not limited to just one part of the plant). This approach is likely over-conservative, as only part(s) of the roots may be consumed.
	Baseline	Cattail roots contained the 95%th UCLM of measured arsenic concentrations	The 95th upper confidence limit of the mean concentration of arsenic measured in cattail roots gathered from the Wood-Buffalo region was used to represent the arsenic content of roots. Given the variability of this data set in relation to root part, it is likely that the use of the 95%UCLM is overly estimating exposure. Over an 80-year period, it is likely that the plants consumed by residents in the Wood Buffalo are grew in soils with lower arsenic levels, and would consume roots with variable arsenic concentrations, and thus, actual risks are likely lower than reported.
	All	Arsenic in soil and deposits on leaves did not vary with environmental factors	It was assumed that no weathering or leaching of soils and plants occurred. It is possible that natural environmental processes over time decrease the amount of arsenic in the soil. Similarly, precipitation may decrease the amount of soil dusts on the surface of plants, thus reducing the amount of arsenic taken up by the plant. Risks associated with plants may be over-stated.
	All	Plant leaves, roots and fruits all contained 37% inorganic arsenic.	It was conservatively assumed that foods contain approximately 37% inorganic arsenic, based on ATSDR (2005). As the laboratory was not able to measure inorganic arsenic in root samples, the analytical data were provided for total arsenic only. It is likely that the inorganic arsenic levels in plant foods varies, and is lower than 37%.
	All	Oral bioavailability of 100%	It was assumed that 100% of the inorganic arsenic from plant foods was absorbed in the gastrointestinal tract following absorption. Absorption and uptake of inorganic arsenic is likely to vary with the type of plant consumed, and as such, risks may be lower than what was predicted.

Exposure Pathway	Development Scenario	Assumption/Measure	Comments
Game meat ingestion	All	Game meat contains 37% inorganic arsenic	As the laboratory was not able to measure inorganic arsenic, the analytical data were provided for total arsenic only. Thus, it was conservatively assumed that foods contained only 37% inorganic arsenic, based upon ATSDR (2005). It is likely that the inorganic arsenic levels in roots vary, and is lower than 37%.
	Future	All relevant game animals (including birds) accumulate arsenic to the same degree as moose.	It was assumed that all game animals would be exposed to and accumulate arsenic to the same degree as a large, vegetation consuming animal (*i.e.*, moose or deer). The total daily intake of arsenic from plants (in association with metabolic demand) and the consumption rate for water were both based upon a large animal. While the predicted concentrations of inorganic arsenic in game meats may be appropriate for large animals, when applied to smaller animals with lower food and water ingestion rates, they may be over-stated.
	All	Game meats contain 37% inorganic arsenic.	It was conservatively assumed that foods contain approximately 37% inorganic arsenic, based on ATSDR (2005). It is likely that the inorganic arsenic level in plant foods varies, and is lower than 37%.
	All	Oral bioavailability of 100%	It was conservatively assumed that 100% of the inorganic arsenic from game meats was absorbed in the gastrointestinal tract following absorption. Absorption and uptake of inorganic arsenic is likely to vary with the type of food consumed.
Consumption of sport fish	Baseline	All fish in the area contain the 95% UCLM of measured arsenic concentrations (0.1 mg/kg ww)	The 95% UCLM of measured total arsenic concentrations from fish collected in the vicinity of the oil sands. As several of the samples evaluated presented un-detectable levels, it was assumed that the arsenic was present at ½ the detection limit, which is conservative.
	Future	The inorganic arsenic that may accumulate in fish over time was based upon the highest incremental increase in the arsenic levels in local surface waters predicted to occur from future anthropogenic sources in the region.	Use of the highest predicted incremental increase in the arsenic concentration of the local surface waters likely resulted in over-statement of fish arsenic levels.
	All	37% of arsenic in fish is inorganic.	The assumption that fish contain 37% inorganic arsenic was obtained from ATSDR (2005), is anticipated to be highly conservative as it represents an estimate of total inorganic arsenic in all foods (*i.e.*, not restricted to fish and seafood). Actual risk associated with ingestion of fish from water bodies in the vicinity of the oil sands is likely less.

6.4.2 *Significance of Risk Estimates*

To assist in the interpretation of the significance of the predicted lifetime cancer risks and to assign some practical meaning to the number of cancer cases that might potentially result from exposure to inorganic arsenic among the indigenous people living in the Wood-Buffalo region, the risk estimates were re-expressed in terms of the specific population numbers for the region. Recent census data compiled for the Regional Municipality of Wood-Buffalo (including the communities of Fort McMurray, Fort MacKay, and Fort Chipewyan) revealed a total population of 79,810 individuals living in the area, of which approximately 10% is comprised of people of indigenous descent (RMWB 2006). The census data are summarized in Table 29. A listing of the number of cancer cases that might potentially occur among the Wood-Buffalo population contingent *per se* from exposure to inorganic arsenic based on the lifetime cancer risk estimates revealed by the present assessment is provided in Table 30. The values shown simply reflect an adjustment of the number of cancer cases that were predicted on the basis of a 100,000-person population contingent (see 6.0 RESULTS) to reflect the actual population numbers for the Wood-Buffalo region. The listing captures each of the development scenarios examined. Two sets of numbers are provided to account for the use of both the Exposure Limit recommended by the Water Quality and Health Bureau of Health Canada (2006) *and* the limit developed by the Contaminated Sites Division of Health Canada (2004) in the calculations, as described earlier (see Section 6.2.4 Risk Characterization). Note that the values refer specifically to the number of cancer cases that might potentially occur among the total indigenous population of the Wood-Buffalo region from exposure to inorganic arsenic *via* the pathways examined as part of the present re-assessment.

Table 29. Summary of 2006 Census Data for the Regional Municipality of Wood Buffalo

Area	Total Population	Indigenous Population[(1)]
Fort McMurray	64,441	5,987
Anzac	711	210
Conklin	338	291
Draper	118	6
Fort Chipewyan	915	822
Fort MacKay	536	525
Gregoire Lake	285	32
Janvier	218	190
Saprae Creek	728	59
Total Regional Population	***79,810***	***8,122***

(1) Based upon an estimate that approximately 10% of the Wood-Buffalo population is of indigenous descent (RMWB 2006)

Table 30. Estimated Number of Cancer Cases Predicted to Occur Among the Indigenous Population of the Wood-Buffalo Region From Exposure to Inorganic Arsenic[1]

Estimate based on:	Estimated Number of Cancer Cases		
	Baseline Development Scenario	Future Development Scenario[2]	Combined Development Scenario
Exposure Limit recommended by Water Quality and Health Bureau of Health Canada (2006) (RsD = 0.006 μ/kg BW/day)	1 (1.36)	<1 (0.07)	1 (1.44)
Exposure Limit recommended by Contaminated Sites Division of Health Canada (2004) (RsD = 0.003 μ/kg BW/day)	3 (2.71)	<1 (0.15)	3 (2.86)

[1] Based on an estimated total indigenous population of 8,122 individuals (RMWB 2006). Values refer to the number of cancer cases that might potentially occur from exposure to inorganic arsenic *via* the complete set of exposure pathways examined as part of the present re-assessment (*i.e.*, all pathways combined). Values in parentheses correspond to the actual numerical ER values calculated after adjustment for the specific population number indicated above.

[2] Values refer specifically to the number of *extra* number of cancer cases that might potentially occur among the indigenous population as a result of projected future anthropogenic activities in the Wood-Buffalo region.

Examination of the values listed in Table 30 reveals the following:

- The number of cancer cases predicted to occur among the indigenous population of the Wood-Buffalo region from exposure to inorganic arsenic under the baseline development scenario ranges from one to three (1 to 3), depending on the Exposure Limit used in the calculation. These predicted numbers very likely represent over-estimates as a result of the conservatism incorporated into the re-assessment (see Section 6.4.1 Conservatism). When interpreted in the context of this conservatism, the significance of the numbers becomes questionable, with the findings suggesting that the current indigenous population is at little, if any, risk of developing cancer as a result of exposure to inorganic arsenic contributed by both naturally-occurring sources and existing anthropogenic sources in the region *via* the exposure pathways examined as part of the re-assessment.
- The number of extra cancer cases predicted to occur among the indigenous population of the Wood-Buffalo region from exposure to inorganic arsenic under the future development scenario was less than one (<1), regardless of the Exposure Limit used in the calculation. The findings signify that the current indigenous population is at essentially no risk of developing cancer as a result of exposure to any inorganic arsenic that might be contributed by projected future anthropogenic activity in the region *via* the exposure pathways examined. Added confidence is provided by the conservatism incorporated into the re-assessment.
- It is important to note that the above predicted lifetime cancer risk estimates (both unadjusted and adjusted) are population-based. They refer to the potential cancer risks from exposure to inorganic arsenic presented to the indigenous population of the Wood-Buffalo region as a whole. They do not refer to the potential lifetime cancer risks faced by an individual person.

The circumstances governing the exposures to inorganic arsenic that might be received by an individual person are many and varied and were not captured as part of either the original assessment completed by Golder Associates (2006) nor the present re-assessment. Depending on circumstances, the individual lifetime cancer risks could be greater or lower than those predicted for the population as a whole. However, that said, the conservatism incorporated into the re-assessment was deliberately meant to ensure that the cancer risks would not be understated, thereby reducing the first possibility.

6.4.3 Uncertainties

There are also some limitations and uncertainties associated with the re-assessment that must be acknowledged and considered in the interpretation of the findings. They include:

- The contribution of store-bought foods and beverages to the potential arsenic exposures that might be received by the indigenous people living in the region was *not* accounted for in the calculation of the lifetime cancer risks. In this regard, the Terms of Reference assigned to the work restricted consideration of food- and beverage-borne sources of inorganic arsenic exposure to those addressed as part of the original assessment performed by Golder Associates (2006), namely traditional plant foodstuffs, game meat, sport fish and local sources of drinking water. Since these items may constitute only a portion of a person's total food and beverage intake, and since food and water can be significant contributors to arsenic exposure, it may be that the lifetime cancer risks calculated as part of the re-assessment do not fully account for total cancer risks involved. However, that said, the calculations did embrace a high degree of conservatism, and were meant to specifically address the cancer risks that could be afforded by the traditional lifestyle of the indigenous people living in the region. Similarly, the re-assessment did not consider other sources of inorganic arsenic exposure apart from dietary items, such as tobacco smoke, metallic pigments, wood preservatives, *etc.* (ATSDR 2005), nor did the work consider exposure to inorganic arsenic *via* pathways other than ingestion as both of these items were outside the Terms of Reference assigned to the work.
- According to Suncor (2005b), groundwater is not used as a source of drinking water in the vicinity of the oil sands projects due to the poor quality of the artesian water, the depth to the aquifer(s), and the lack of demand for groundwater. As a result, the re-assessment did not consider the consumption of groundwater as an exposure pathway for inorganic arsenic. In the event that groundwater is in fact consumed by the indigenous people living in the region, the cancer risks estimates may need to be re-visited. However, that said, the re-assessment did allow for exposure to inorganic arsenic through the consumption of raw, untreated local surface waters using drinking water ingestion rates reported by Health Canada (2004).
- The extent to which cattail roots and other traditional roots are consumed is not clear. Details concerning the nature and extent to which roots are consumed by the indigenous people living in the region were not specifically addressed as part of the dietary surveys performed by Wein (1989). For the purposes of the re-assessment, it was assumed that the ingestion rate for roots was equivalent to that reported for traditional above-ground plants. This assumption necessarily introduced some uncertainty in the exposure estimates and corresponding lifetime cancer risks that were calculated. Additional area-specific information regarding this

exposure pathway (*i.e.*, local dietary surveys) may assist in more accurate predictions in future assessments.

- As already indicated, the re-assessment did not consider the exposures to inorganic arsenic that might be received *via* inhalation. Rather, the re-assessment concentrated on the oral route of exposure, with a specific focus on those pathways appropriate to the traditional way-of-life. That said, the original HHRA completed by Golder Associates (2006) revealed very little exposure to inorganic arsenic *via* the inhalation pathway.
- The consumption rates for game meat and fish for infants were assumed to be zero, based upon Health Canada (2004). Theoretically, it is possible that infants could be exposed to arsenic *via* lactation if arsenic residues were present in the mother's milk from recent and/or historical maternal exposures to arsenic through the consumption of game meat, fish, traditional plant foodstuffs and other sources. However, that said, ATSDR (2005) and Concha *et al*. (1998a; 1998b) have determined that arsenic (organic or inorganic) is not transferred to a significant extent into human milk based upon epidemiological studies. Moreover, any exposure to arsenic that might theoretically be received by the infant *via* lactation would contribute very little to the lifetime average daily dose (LADD) of arsenic received by the composite receptor given that the infant age category occupies only a very small proportion of the total 80-year lifetime under investigation.

7.0 COMPARISON OF CANCER RISK ASSESSMENT METHODOLOGIES – CEI *VS*. GOLDER ASSOCIATES

Among the tasks specified in the Terms of Reference developed for the work was the need to perform a comparison of the methods used in the original HHRA completed by Golder Associates (2006) to derive the lifetime cancer risk estimates against those used by CEI, highlighting the differences in approach. The key differences in the methodologies are outlined below. A more detailed listing of the differences can be found in Table 31. These differences account, in part, for the different lifetime cancer risks reported by Golder Associates versus those predicted herein. It should be noted that many of the differences originate from the fact that each assessment was performed under different terms and for different reasons. Specifically, whereas the original assessment completed by Golder Associates was commissioned by Suncor Energy and formed part of the EIA performed in support of the Suncor Voyageur project (Suncor 2005b), the re-assessment completed by CEI was commissioned by Alberta Health and Wellness (AHW) and was meant to serve as a "second opinion" of the lifetime cancer risks that might be presented to the indigenous people living in the Wood-Buffalo region from exposure to inorganic arsenic. On this basis alone, the scope and nature of the two assessments necessarily differed. More specifically, whereas the original assessment was governed by the Terms of Reference surrounding the EIA for the Suncor Voyageur project and was necessarily broad in scope, the re-assessment was based on the Terms of Reference prescribed by AHW and was more focused in nature. Apart from the above, many of the differences between the two assessments related to the following:

- Use of different datasets describing the levels of arsenic in the local environment. (Although, for the purposes of the re-assessment, an attempt was made to rely on the same datasets used in the original assessment, by virtue of the differences in the methods followed, reliance was ultimately placed on additional datasets as part of the present work).
- Use of different predictive models to describe the fate and transport of arsenic in the environment.
- Use of different Exposure Limits describing the "acceptable" level of arsenic exposure.
- Use of different assumptions to accommodate the uncertainty surrounding a number of elements intrinsic to each assessment.

A description of the major differences follows:

- As discussed earlier, the definition of the development scenarios examined differed between the two assessments. Whereas, the definitions adopted by Golder Associates were consistent with those typically used for EIAs both in terms of the nature of the scenarios and the terminology that applied (*i.e.*, baseline, application and planned development case), the scenarios assessed by CEI were somewhat more generic in nature and were labelled differently (*i.e.*, baseline, future and combined). Greater emphasis was given to the Suncor Voyageur project *per se* in the original assessment, with the "application" scenario devoted exclusively to the calculation of the incremental lifetime cancer risks associated with the

Project on a stand-alone basis. In contrast, the re-assessment did not focus specifically on the Suncor Voyageur project, but rather was concerned with assessing the incremental lifetime cancer risks that could result from *all* projected future anthropogenic activities over an 80-year time period.

- Unlike the original assessment in which the lifetime cancer risks were labelled as "incremental" and were reported to be numerically similar (*i.e.*, ILCR = 452-453) regardless of the development scenario examined, use of the term "incremental" was reserved for the lifetime cancer risks calculated for the future development scenario only in the re-assessment. Using the latter approach, distinction was made between the number of cancer cases predicted to occur as a result of exposure to inorganic arsenic originating from naturally-occurring and/or existing anthropogenic sources in the Wood-Buffalo region (*i.e.*, the baseline development scenario), and the *extra* cancer cases that might potentially result from the added exposures contributed by future anthropogenic activity (*i.e.*, the future development scenario). This approach followed convention and provided a much clearer representation of the truly "incremental" lifetime cancer risks involved. The significance of these incremental risks could then be appropriately judged by comparison against the acceptable benchmark cancer incidence rate of 1×10^{-5} (*i.e.*, one *extra* cancer case in a population of 100,000 people). Despite the labelling, the lifetime cancer risks reported as part of the original assessment were not truly incremental, and comparison of these risks against the benchmark value would represent a departure from convention.
- Unlike the original assessment in which the so-called incremental lifetime cancer risks were simply reported as a common number (*i.e.*, ILCR = 452-453) regardless of development scenario and without context, the lifetime cancer risks reported as part of the re-assessment were not only better differentiated (*i.e.*, distinction was made between cancer risks associated with baseline conditions and the true incremental risks associated with projected future anthropogenic activities), but were also expressed in terms of the number of potential cancer cases that might be expected to occur among the indigenous population contingent of the Regional Municipality of Wood-Buffalo *per se* in order to permit more meaningful interpretation of the significance of the risk estimates.
- Whereas the so-called incremental lifetime cancer risks calculated as part of the original assessment were based on comparison of the predicted exposures to inorganic arsenic against a *single* Exposure Limit (*i.e.*, 0.003 µ/kg BW/day – origin not stated, but presumably based on the slope factor of 2.8 $(mg/kg\text{-}d)^{-1}$ determined by the Contaminated Sites Division of Health Canada), two sets of calculations involving the use of two different Exposure Limits were included as part of the re-assessment (*i.e.*, 0.006 µ/kg BW/day, based on the negligible risk level reported by the Water Quality and Health Bureau of Health Canada, *and* 0.003 µ/kg BW/day, based on the slope factor determined by the Contaminated Sites Division of Health Canada). Use of the two limits provided added perspective.
- A number of differences between the two assessments originated from the use of different assumptions related to the estimation of the potential exposures to inorganic arsenic that might be received by the indigenous receptor living in the Wood-Buffalo region. Some examples of these differences are provided below. A more complete listing can be found in Table 31.

- Whereas, as part of the original assessment, Golder Associates assumed that exposure to inorganic arsenic *via* the incidental ingestion of soil could occur for 365 days per year, CEI assumed that exposure through this pathway would be limited to six months of the year because winter conditions and snow cover would likely preclude routine ingestion of soil during the winter months. The latter assumption was supported by data sourced from Environment Canada (2006) for the Fort McMurray area.
- Whereas, Golder Associates assumed that the indigenous receptor would consume raw, untreated surface waters for six months of the year, CEI assumed that the receptor would consume untreated water (or equivalent) during the entire year. More specifically, as part of the re-assessment, it was assumed that the receptor would spend six months in the field and drink directly from local creeks, rivers and/or lakes, and the remaining six months in a community setting drinking from a treated water supply. However, since the effectiveness of the water treatment system in removing arsenic was unknown, it was assumed that the arsenic content of the treated water was equivalent to that of the local untreated surface waters.
- Whereas Golder Associates assumed that the arsenic content of most foods was comprised entirely of inorganic arsenic[16], CEI assumed that 37% of the total arsenic content of the various food items examined (*i.e.*, game meat, sport fish, traditional plant foodstuffs) consisted of inorganic arsenic. The latter assumption was based on information sourced from the Agency for Toxic Substances and Disease Registry (ATSDR 2005).
- Whereas Golder Associates used a combination of wet weights and dry weights to express the arsenic content of foods, CEI adjusted and expressed the arsenic content of all food items of a wet-weight basis. This allowed for a more consistent approach and permitted more reliable translation of the arsenic content of the food items into the corresponding estimated daily intakes since the food ingestion rates used in the calculations (Health Canada 2004; Wein 1989) referred to the wet weight of the foodstuffs.
- Whereas Golder Associates relied on historical data detailing the arsenic content of traditional foodstuffs, including game meat, to predict the exposures that might be received under the baseline development scenario described in the original HHRA, CEI relied on the empirical data gathered as part of the recent sampling program commissioned by Alberta Health and Wellness (2006) to estimate the arsenic content of game meat and the corresponding exposures that might occur from consumption of this traditional food item under the baseline scenario described in the re-assessment. These two datasets differed significantly in terms of the limit of analytical detection that was achieved by the analytical testing facility. Whereas the limit of detection for arsenic for the historical dataset (used by Golder Associates) was reported to be as high as 0.5 parts-per-million (ppm) for moose meat (weight basis unknown), a much lower detection limit (*i.e.*, 0.001 ppm, wet-weight) was achieved for the more recent dataset. This difference is especially significant since the analysis of many of the samples of moose meat comprising the historical dataset showed no detectable levels of arsenic. By default,

[16] An exception was fish, for which Golder Associates assumed 10% of the total arsenic content consisted of inorganic arsenic.

Golder Associates assumed the arsenic content of these samples was equivalent to one-half of the maximum detection limit (*i.e.*, 0.25 ppm), possibly leading to over-estimation of the arsenic exposure that might result from the consumption of game meat, and certainly leading to much higher estimates of arsenic exposure than those predicted by CEI on the basis of the more recent dataset.

It is evident from the above discussion that, despite the general guidance offered by the Terms of Reference developed for the present work and the instruction received from Alberta Health and Wellness to structure the re-assessment to match the design of the original assessment completed by Golder Associates as much as possible, significant differences existed between the two assessments in the particulars surrounding the work. These differences extended to the definition of the development scenarios examined, the manner in which the lifetime cancer risks were differentiated, the Exposure Limits used in the calculations, and the assumptions that were applied to accommodate the uncertainties surrounding the work. As is evident from examination of Table 31, in some instances the assumptions used by CEI were more conservative than those used by Golder Associates; whereas, in other cases, the assumptions used in the re-assessment were less conservative than those applied in the original assessment. However, the net result was that the lifetime cancer risks predicted by CEI were significantly lower than those originally reported by Golder Associates across all development scenarios, especially in terms of the incremental lifetime cancer risks predicted for the future development case. More specifically, whereas Golder Associates reported an ILCR of 453 for the "application" scenario (*i.e.*, for the Suncor Voyageur project itself), CEI reported the ILCR for the "future" development scenario (*i.e.*, all projected future projects combined) to range from <1 to ≈ 2.

Table 31. Comparison of Key Assumptions Used in the Original Assessment (Golder Associates) and the Re-assessment (CEI)

Assumption	Original Assessment (Golder Associates 2006)	Re-assessment (CEI)	Comments
Soil concentration (mg/kg)	1.0	1.3	The CEI assumption is slightly more conservative.
Soil ingestion exposure frequency (days/year)	365	182.5	The Golder estimate is more conservative, however, does not consider that snow and ice will cover the surface soil during winter months
Surface water concentration (μg/L)	0.7	1.18	The CEI assumption is more conservative
Surface water exposure frequency (days/year)	182.5	365	The CEI assumption is more conservative (about 2-times)
Game meat concentration (mg/kg) [1]	0.25 mg/kg dw (*i.e.*, 0.08 mg/kg ww)	0.008 mg/kg ww	The Golder assumption is more conservative, but is based upon ½ a detection limit, while the CEI value represents the 95% UCLM on a measured data set.
Game meat consumption	270	106	The Golder assumption is

Assumption	Original Assessment (Golder Associates 2006)	Re-assessment (CEI)	Comments
rate for adult (g/day)			more conservative.
Berry concentration (mg/kg) [2]	0.1 mg/kg dw (*i.e.*, 0.015 mg/kg ww)	0.009 mg/kg ww	The Golder assumption is more conservative (about 11-times), but does not consider only the inorganic form.
Berry consumption rate for adult (g/day)	15	23	The CEI assumption is more conservative.
Plant concentration (mg/kg) [2]	0.1 mg/kg dw (*i.e.*, 0.015 mg/kg ww)	0.009 mg/kg ww	The Golder assumption is more conservative (about 11-times), but does not consider only the inorganic form.
Plant ingestion exposure frequency (days/year)	365	365	Assumptions are the same.
Plant ingestion rate for adult (g/day) [2]	13.7 g/day dw (2 g/day ww)	3 g/day ww	The CEI assumption is more conservative.
Fish inorganic arsenic concentration (mg/kg ww)[3]	0.0044	0.0037	The Golder assumption is slightly more conservative, although the two are similar.
Fish ingestion exposure frequency (days/year)	365	365	Assumptions are the same.
Fish ingestion rate for adult (g/day)	220 g/day	51	Golder assumption is more conservative (about 4-times).
Portion inorganic arsenic in fish	10%	37%	The CEI assumption is more conservative than Golder.
Root concentration (mg/kg) [2]	1.3 mg/kg dw (*i.e.*, 0.2 mg/kg ww)	0.70 mg/kg ww	The CEI value is more conservative, and is based upon measured data.
Root ingestion rate for adult (g/day) [2]	28 g/day dw (4.2 g/day ww)	3 g/day ww	The Golder assumption is more conservative.
Slope factor [$(mg/kg/day)^{-1}$]	2.8	1.7 and 2.8	The CEI assumption using the slope factor of 1.7 is less conservative than Golder. The use of the slope factor of 2.8 is the same

(1) Dry weight (dw) converted to wet weight (ww) assuming moisture content of 68% (Suter *et al*. 2000)

(2) Dry weight (dw) converted to wet weight (ww) assuming moisture content of 85% (Suter *et al*. 2000)

(3) Concentration values for total arsenic reported

8.0 SUMMARY AND CONCLUSIONS

At the request of Alberta Health and Wellness (AHW), the lifetime cancer risks that might be presented to indigenous people living in the Wood-Buffalo region from exposure to inorganic arsenic were examined. The request was prompted, in part, by the findings of a recent human health risk assessment (HHRA) completed by Golder Associates (2006) in support of a proposed industrial oil sands development project (*i.e.*, the Suncor Voyageur project), which suggested these people may be at increased risk of developing cancer during their lifetime as a result of arsenic exposure. A so-called "incremental lifetime cancer risk" (ILCR) attributable to arsenic exposure was reported by Golder Associates to be approximately 450 (*i.e.*, equivalent to 450 extra cases of cancer in a population of 100,000 people).[17] The reported ILCR was well above the "benchmark" value of 1 x 10^{-5} (*i.e.*, one extra cancer case in a population of 100,000 people) deemed to be acceptable by most leading regulatory authorities for the protection of public health. The reported ILCR also was much higher than the lifetime cancer risks from arsenic exposure reported historically as part of EIAs completed in support of oil sands projects. To test the veracity of the original risk estimates reported by Golder Associates, AHW retained CANTOX ENVIRONMENTAL INC. (CEI) to re-assess potential the lifetime cancer risks involved.

The re-assessment was performed in accordance with terms and conditions assigned by AHW (*i.e.*, Terms of Reference), with the understanding that the overall approach to be taken for the work was to closely mimic that used earlier by Golder Associates, but with allowance for some level of refinement in order to better match the approach typically followed by CEI. The work followed a conventional risk assessment paradigm, with the lifetime cancer risks expressed as Exposure Ratios (ERs) based on comparison of the estimated exposures to inorganic arsenic that might be received by an indigenous receptor across an 80-year lifetime against a set of Exposure Limits for arsenic (*i.e.*, acceptable level of exposure) recommended by Health Canada (2004 2006). The ERs corresponded to the number of potential cancer cases attributable to the arsenic exposure that might occur among a population of 100,000 people. The cancer risks were assessed for three different development scenarios, designated "baseline", "future" and "combined". Emphasis was given to the lifetime cancer risks associated with exposure to inorganic arsenic through the oral route (*i.e.*, *via* ingestion). Several different exposure pathways were examined, namely incidental ingestion of soil, ingestion of drinking water, consumption of traditional plant foodstuffs, ingestion of game meat, and consumption of sport fish. A number of conservative assumptions were applied to the work in order to ensure that the cancer risks would not be understated. To assist in the interpretation of the significance of the findings, the predicted lifetime cancer risks were re-expressed in terms of the actual contingent of indigenous people living in the Wood-Buffalo region based on recent census data compiled for communities in the area.

The principal findings that emerged from the re-assessment were:

- The ER values predicted for the baseline development scenario ranged from ≈ 17 to 33 (depending on the Exposure Limit used in the calculations), signifying that lifetime exposure

[17] The original HHRA completed by Golder Associates (2006) examined three development scenarios (designated "baseline", "application", and "planned development case" (PDC)). An incremental lifetime cancer risk (ILCR) of 452-453 was reported for the development scenarios.

to inorganic arsenic among indigenous people living in the Wood-Buffalo region *via* the exposure pathways examined potentially could contribute to ≈ 17 to 30 cases of cancer when calculated on a 100,000-person population basis. The "acceptability" of this potential lifetime cancer risk from a public health perspective cannot be determined following a conventional approach since an acceptable "benchmark" cancer risk level for exposure to background levels of carcinogens is not available for comparison.

When re-expressed in terms of the actual contingent of indigenous people living in the Wood-Buffalo region (*i.e.*, ≈ 8,000 people of indigenous descent), the number of cancer cases attributable to exposure to inorganic arsenic predicted to occur among the population under the baseline development scenario ranged from one to three (1 to 3), depending on the Exposure Limit used in the calculation.

Both sets of predicted numbers very likely represent over-estimates as a result of the conservatism incorporated into the re-assessment. When interpreted in the context of this conservatism, the significance of the numbers becomes questionable, with the findings suggesting that the current indigenous population is at little, if any, risk of developing cancer as a result of exposure to inorganic arsenic contributed by both naturally-occurring sources and existing anthropogenic sources in the region *via* the exposure pathways examined as part of the work.

The ER values predicted for the future development scenario ranged from ≈ 1 to 2, signifying that lifetime exposure to inorganic arsenic contributed by future anthropogenic activities in the Wood-Buffalo region might potentially contribute to 1 to 2 extra cancer cases among indigenous people living in the area when calculated on a 100,000-person population basis. The predicted incremental lifetime cancer risks "bridged" the benchmark ILCR of 1×10^{-5} (*i.e.*, one extra cancer case in a population of 100,000 people) deemed to be acceptable by most leading scientific and regulatory authorities. More specifically, the ILCR calculated using the Exposure Limit recommended by the Water Quality and Health Bureau of Health Canada (*i.e.*, 0.006 µ/kg BW/day) was slightly below the comparison benchmark (*i.e.*, ER = 0.9); whereas, the ILCR determined using the limit recommended by the Contaminated Sites Division of Health Canada (*i.e.*, 0.003 µ/kg BW/day) was slightly above the comparison benchmark (*i.e.*, ER = 1.8).

When re-expressed in terms of the actual contingent of indigenous people living in the Wood-Buffalo region (*i.e.*, ≈ 8,000 people of indigenous descent), the number of cancer cases attributable to exposure to inorganic arsenic predicted to occur among the population under the future development scenario was less than one (<1), regardless of the Exposure Limit used in the calculation.

Both sets of numbers signify that the current indigenous population is at essentially no risk of developing cancer as a result of exposure to any inorganic arsenic that might be contributed by projected future anthropogenic activity in the region *via* the exposure pathways examined. Added confidence is provided by the conservatism incorporated into the re-assessment.

- The ER values predicted for the combined development scenario (calculated on the basis of the combined exposures determined for both the baseline *and* future scenarios) ranged from

≈ 18 to 35, closely mimicking those predicted for the baseline scenario itself (*i.e*., ER = ≈ 17 to 33). The finding signifies that the lifetime cancer risks that could potentially result from exposure to inorganic arsenic among indigenous people living in the Wood-Buffalo region are dominated by the exposures contributed by already existing naturally-occurring and anthropogenic sources of arsenic in the region, with very little incremental risk presented by projected future anthropogenic activities.

- Regardless of the development scenario examined, the predicted exposures to inorganic arsenic that might be received by indigenous people living in the Wood-Buffalo region were dominated by certain exposure pathways, notably the consumption of drinking water and the consumption of sport fish, which contributed up to 41% and up to 55% of the total predicted exposure, respectively. Lesser, but still significant, contributions were revealed for the consumption of roots and other below-ground plants (as part of the consumption of traditional plant foodstuffs) and the consumption of game meat, depending on the development scenario assessed. The contribution from the remaining exposure pathways was negligible.

- Despite the general guidance received from AHW to design the re-assessment to match that of the original assessment completed by Golder Associates as much as possible, significant differences existed between the two assessments in the particulars surrounding the work. Many of the differences stemmed from the fact that each assessment was performed under different terms and for different reasons. Whereas the original assessment completed by Golder Associates was commissioned by Suncor Energy and formed part of the EIA performed in support of the Suncor Voyageur project (Suncor 2005b), the re-assessment completed by CEI was commissioned by AHW and was meant to serve as a "second opinion" of the lifetime cancer risks that might be presented to the indigenous people living in the Wood-Buffalo region from exposure to inorganic arsenic. On this basis alone, the scope and nature of the two assessments necessarily differed. The differences extended to the definition of the development scenarios examined, the manner in which the lifetime cancer risks were differentiated, the Exposure Limits used in the calculations, and the assumptions that were applied to accommodate the uncertainties surrounding the work. The net result of the differences was that the lifetime cancer risks predicted by CEI were significantly lower than those originally reported by Golder Associates across all development scenarios, especially in terms of the incremental lifetime cancer risks predicted for the future development case.

9.0 REFERENCES

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APPENDIX A
TOXICITY PROFILE

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APPENDIX A – TOXICITY PROFILE

A1.0 INTRODUCTION

The toxicity of arsenic has been and continues to be the subject of considerable research, as well as some controversy. Exposure is widespread due to its presence in the earth's soil and rock, with most human exposure globally arising from naturally occurring arsenic in drinking water. The effects of arsenic exposure depend on many factors, including its chemical and physical form, dose and duration of exposure, nutritional status of those exposed and extent of co-exposure to other chemicals or carcinogens (*e.g.*, cigarette smoking, UV light). Arsenic does not commonly exist in elemental form, but it combines with other elements to form a number of inorganic and organic compounds. Inorganic arsenics such as arsenates (pentavalent arsenic) or arsenites (trivalent arsenic) occur in natural waters or drinking water and at low levels in food. Organic arsenite species such as arsenobetaine and arsenocholine are abundant in seafood but they are generally considered to be much less harmful to health and are rapidly eliminated from the body in the urine (WHO 2001; Health Canada 2006a).

In Canada, the primary source of exposure to arsenic is food, followed by drinking water, soil and air (Dabeka *et al.* 1993; Health Canada 2006a). However, a substantial amount of arsenic in food, predominantly from fish, is in the less-toxic organic form. In estimating total daily intakes for a typical Canadian, Health Canada has assumed that 37% of arsenic in food is inorganic (Health Canada 1995) but others have proposed estimates of 2 to 10% (WHO 1989; GESAMP 1986). A recent study by Yost *et al.* 1998 suggests that inorganic arsenic may account for 21 to 40% of the total arsenic present in a mixed diet and that population exposure to inorganic arsenic through the ingestion of food may be greater than previously believed. Table A-1 illustrates estimated average *daily* arsenic exposure of a typical Canadian, with arsenic concentrations in food further categorized in Table A-2. Arsenic levels in Canadian drinking water are generally less than 5 µg/L (Health Canada 2006b). Due to the differences in toxic potential, it is important to measure the chemical form of arsenic, where feasible, in order to more accurately predict potential health risks.

Several regulatory bodies have comprehensively reviewed the toxicological literature on arsenic to determine its potential to cause cancer as well as its potential non-cancer hazards (*e.g.*, reproductive or developmental toxicity) (JECFA/WHO 1989; WHO 1993; 2003; U.S. EPA 2001; 2005b; U.S. NRC 1999; 2001; IARC 2004; ATSDR 2005; Health Canada 2006b). In some cases, they also made recommendations on "safe" intake levels through food, drinking water and other environmental sources or alternatively, cancer potency factors by which cancer risks can be predicted for a given exposure level. Inorganic arsenic is considered an established human carcinogen but the shape of the dose-response curve (*i.e.*, no-threshold versus threshold) and its cancer hazard at low doses is a subject of much debate. In general, a threshold model assumes that a carcinogen does not increase cancer risk below a certain minimum level, while a hormetic model assumes there are in fact benefits to low dose exposure.

The purpose of this review is to first summarize the general health effects associated with inorganic arsenic in human populations. Secondly, the health-based exposure limits (*i.e.*, safe intake levels) recommended by various regulatory agencies are described and contrasted. Some key areas of uncertainty that have bearing on those limits are also highlighted throughout.

Table A-1. Estimated average daily inorganic arsenic exposure of a typical Canadian adult male[1] (µg/d) (Health Canada 1995)

Media	Estimated Daily Exposure
Air	0.021
Dust	na
Food	15.6
Incidental Soil Ingestion	0.21
Drinking Water	7
TOTAL (no smoking)	22.8
Smoking	1.4
TOTAL (with smoking)	24.2

(1) Assumed to be an adult male weighing 70 kg, breathing 23 m^3 of air per day, drinking 1.5 L of water per day and with a soil intake of 20 mg per day

na = not available

Table A-2. Estimated Average Daily Inorganic Arsenic Exposure of a Typical Canadian Adult Male in Various Food Categories as Compared to Total Daily Intake (µg/d)[1]

Media	Concentration
Meat and Poultry	1.51
Fish[2]	10.13
Vegetables	0.65
Grains	2.24
Fruits	0.31
Milk [3]	0.4
Fats and Oils	0.18
Beverages	0.28
Total	15.7

(1) Based on Canadian market basket Total Diet studies (Dabeka *et al.* 1987; Dabeka *et al.* 1993; Health Canada 1995).

(2) Arsenic concentrations are based on shellfish, freshwater fish, marine fish and canned fish; Arsenic concentrations reported in total food studies were based on total arsenic concentrations, which were then converted to inorganic arsenic by assuming that inorganic arsenic comprises 37% of total arsenic (Health Canada 1995).

(3) Arsenic concentrations are based on shellfish, freshwater fish, marine fish and canned fish; Arsenic concentrations reported in total food studies were based on total arsenic concentrations, which were then converted to inorganic arsenic by assuming that inorganic arsenic comprises 37% of total arsenic (Health Canada 1995).

(4) Includes other dairy products (*i.e.*, cottage cheese, yoghurt, *etc.* and eggs).

A2.0 ARSENIC HEALTH EFFECTS

Arsenic is an unusual chemical in that a) it has been well studied in human populations, even at environmentally relevant concentrations and b) there are no definitive animal models to adequately represent the effects caused by arsenic in humans, due to differences in metabolism and toxicity among humans *versus* animals (Hopenhayn 2006). Risk assessments for many chemicals rely on extrapolation from high dose animal studies or very high occupational exposures. .

Most of what is known regarding the effects of oral arsenic ingestion in humans comes from studies of populations exposed to varying levels of arsenic in their drinking water. Hopenhayn (2006) have summarized some landmark observations of the association between arsenic ingestion *via* drinking water and health effects. These include:

- In 1917, the reporting of frequent skin disorders among residents of a town in Argentina was attributed to arsenic exposure from deep-water wells. Since then, several areas in the province of Cordoba, Argentina have been found to be affected by natural arsenic water contamination, with levels often above 100 µg/L and as high as 2,000 µg/L.

- In the 1920s, the appearance of a peripheral vascular disease called "Blackfoot Disease" (BFD) in south western Taiwan, subsequent to a switch in drinking water source from surface water to groundwater or artesian wells (in order to improve the microbiological quality of drinking water). BFD is a condition leading to gangrene of the toes and feet. Since that time, BFD prevalence has increased in SW Taiwan and been strongly associated with arsenic in artesian well water. Since BFD is rarely seen in other arsenic-exposed populations, some believe there are other factors that make the population of this area susceptible to BFD.

- In 1958, signs of arsenic toxicity became evident in the city of Antofagasta, Chile after they changed their water source to the Toconce River. Unknown to the population, it had very elevated arsenic concentrations (averaging 800 µg/L) and within a few years clinical reports of acute toxicity, skin disorders and systemic damage were apparent. By 1970 an arsenic removal plant was installed but health effects with long latencies such as cancer outlasted the exposure peak by decades; for example, elevated rates of bladder and lung cancer.

- In the 1980s, several epidemiological studies conducted in the arsenic endemic areas of Taiwan were published, indicating elevated risks for skin, bladder, lung and kidney cancer mortality. These opened the door for studies of exposed populations in several other countries, such as Chile, Argentina, Finland, China, England and Nicaragua.

- In Bangladesh and West Bengal, India, a well-intentioned change of water supply in the 1970s resulted in a very large arsenic epidemic. Microbial contamination of rivers and streams had resulted in numerous cases of disease and death, so thousands of shallow "tube" wells were dug to provide cleaner water that unfortunately was contaminated with arsenic. It has been estimated that in just Bangladesh, where 80 million people are thought to have used the contaminated tube well water, approximately 300,000 people have arsenic induced skin lesions and cancer (Chowdury *et al.* 2000).

A2.1 Acute Toxicity

Inorganic arsenic has a long history as a human poison, with milligram doses able to cause death following ingestion. Minimum lethal oral doses for humans have been reported to be in the range of 1 to 3 mg As/kg (Vallee *et al.* 1960; Armstrong *et al.* 1984). Short-term exposure to high levels of arsenic can lead to abdominal pain, vomiting and diarrhea, muscular cramping or pain, weakness and flushing of skin, skin rash, numbness, burning or tingling sensation in hand and feet, thickening of the skin on the palms of the hands and soles of the feet or loss of movement and sensory responses (Health Canada 2006a). The acute oral LOAEL in humans was reported to be 1 mg As/kg body weight per day (bw/d) for these effects (Armstrong *et al.* 1984).

Although there are many studies of the effects of arsenic inhalation in humans, there were no cases of lethality or severe impacts from short-term exposure in the scientific literature. This was interpreted by ATSDR (2005) as an indication that mortality is not likely to be of concern, even at the very high exposure levels (1 to 100 mg As/m^3) that were once associated with workplace exposures.

A2.2 Subchronic/chronic Toxicity

According to Health Canada (2006a), long-term exposure to high levels of arsenic in drinking water over a lifetime may increase the risk of cancer in internal organs such as the bladder, liver and lungs. Other health effects attributed to chronic exposure include: thickening and discoloration of the skin, nausea and diarrhea, decreased production of blood cells, abnormal heart rhythm and blood vessel damage, and numbness in the hands and feet (Health Canada 2006a).

The most commonly observed symptom following oral exposure *via* drinking water in humans is a pattern of skin changes called hyper-pigmentation (dark or light spots on the skin) and hyperkeratosis (thickening of the skin with small corns or warts), particularly on the palms and soles (Health Canada 2006b). This is observed typically after 5 to 15 years of exposure equivalent to 700 µg/d, or 6 months to 3 years of exposure equivalent to 2,800 µg/d (U.S. EPA 2001; Health Canada 2006b). These skin changes are not necessarily a health concern, but in some cases they may progress to skin cancer (OMOE 2001). As noted above, arsenic ingestion *via* drinking water has also been reported to increase the risk of internal cancer, especially in the liver, bladder, kidney and lungs. This is discussed further in Section 2.4. Studies in several countries have demonstrated that arsenic may cause mild forms of peripheral vascular disease, but the most severe form called “Blackfoot Disease” has been observed only in Taiwan (WHO 2001). Haematopoietic effects have been observed following occupational exposure to unspecified concentrations of arsenic but these effects appeared to be reversible (U.S. EPA 1984; Yoshida *et al.* 1987; CEPA 1993).

The relationship between arsenic exposure and other health effects is not considered to be conclusive, although some studies have suggested associations between arsenic exposure and cardiovascular disease, diabetes and cancers of other organs (WHO 2001; Hopenhayn 2006). During the period of high arsenic exposure in Antofagasta, Chile (mean arsenic concentration of 600 to 800 µg/L), effects on the skin, respiratory system, cardiovascular system and digestive system were observed in children under 16 years of age (Zaldivar 1980; Zaldivar and Ghai 1980; Health Canada 2006b). A study in West Bengal, India indicated that chronic consumption of arsenic contaminated water was associated with respiratory symptoms and reduced lung function in men, particularly among those with arsenic-related skin lesions (von Ehrenstein *et al.* 2006)

Navas-Acien (2005) recently conducted a systematic review of the epidemiological evidence for a relationship between arsenic exposure and cardiovascular disease. Thirteen studies were identified that were conducted in general populations and 16 studies in occupational populations with exposure assessed ecologically in most studies. Among the general population studies, 8 were conducted in Taiwan and indicated moderate to strong associations between high levels of arsenic in drinking water and cardiovascular outcomes. The reviewers noted, however, that methodological issues limit the interpretation of these associations (Navas-Acien 2005). In other general populations and in the occupational studies, the evidence was inconclusive with relative risks ranging from 0.40 to 2.14 (*i.e.*, both increased and decreased relative risks were observed). No cardiovascular outcome was consistently observed to be elevated among the populations studied.

A2.3 Reproductive and Developmental Toxicity

Several studies have investigated the potential effects of arsenic exposure on pregnancy and developmental outcomes in humans (Kwok *et al.* 2006; von Ehrenstein *et al.* 2006; Milton *et al.* 2005; Yang *et al.* 2003;Hopenhayn *et al.* 2003; Hopenhayn-Rich *et al.* 2000). Studies conducted in Bangladesh, India, Taiwan, Chile and Hungary have variably reported positive associations with spontaneous abortion, stillbirth, preterm births, neonatal mortality and birth weight but not consistently among studies. For example, an ecological study in Bangladesh reported statistically significant elevated risks for spontaneous abortion and stillbirth when comparing exposure to arsenic concentrations greater than 50 µg/L with concentrations of 50 µg/L or less (Milton *et al.* 2005). In contrast, a larger study in Bangladesh that used individual-level exposure and health data for 2006 pregnant women found no relationship between arsenic exposure and stillbirth, low birth-weight, impaired growth and weight gain in childhood (Kwok *et al.* 2006). They did, however, find a very small but statistically significant association between arsenic exposure and birth-defects (odds ratio = 1.005; 95% confidence interval (CI) 1.001 to 1.010). Another study in India reported that exposure to high concentrations of arsenic (> 200 µg/L) during pregnancy was associated with a 6-fold increase in stillbirth but no associations were found between spontaneous abortion or infant mortality (von Ehrenstein *et al.* 2006).

Health Canada (2006b) describes a case-control study of 270 children with congenital heart disease and 665 healthy children that reported an association between maternal consumption of arsenic in drinking water and a 3-fold increase in the occurrence of coarction of the aorta (Zierler *et al.* 1988). They note, however, that there was no adjustment for maternal age, socioeconomic status or previous reproductive history and since many of the mothers were served by multiple water supplies it was necessary to average contaminant concentrations.

In general, the evidence for arsenic-induced adverse reproductive outcomes is considered preliminary or inconclusive at this time (WHO 2001; Health Canada 2006b; Hopenhayn 2006). Many studies were of an ecological design and thus, they did not adequately control for potential confounders (Kwok *et al.* 2006). There is evidence from animal studies that high oral doses of arsenic during pregnancy can cause low birth weight, fetal malformations and fetal death but only at doses that also cause maternal toxicity (ATSDR 2005). Arsenic can cross the placenta and has been found in fetal tissues (ATSDR 2005).

A2.4 Carcinogenicity

The International Agency for Research on Cancer has considered arsenic a carcinogen since 1980 (IARC 1980). The earliest reports linking it to cancer involved elevated lung cancer rates among arsenic-exposed miners and elevated skin cancer associated with ingestion of arsenic-based medicines (Bates *et al.* 1992). Several epidemiological studies of workers in smelters and arsenical pesticide production facilities have demonstrated excess lung cancer mortality associated with exposure to arsenic (Pinto *et al.* 1977; 1978; Axelson *et al.* 1978; Wall 1980; Lee-Feldstein 1983; Higgins *et al.* 1986;Enterline et al. 1987; Jarup *et al.* 1989) After reviewing these studies, CEPA (1993) concluded that the influence of possible confounders, such as concomitant exposure to sulphur dioxide or cigarette smoking, could not explain the excess lung cancer observed in these studies. Exposure was most extensively characterised in three cohorts of workers employed at smelters in Washington, Montana and Sweden and in these studies there was a clear exposure-response relationship between airborne arsenic levels and mortality due to lung cancer (Enterline *et al.* 1987; Higgins *et al.* 1986; Jarup *et al.* 1989; CEPA 1993). Other cancers have not been as well studied, but some studies suggest that cancers of the stomach, colon, liver and urinary system may also be associated with occupational exposure to arsenic (Gibb and Chen 1989). Inhalation exposure to arsenic in occupational settings is significantly higher than exposure to arsenic in the general population, where it is generally considered to be insignificant in non-smokers (CCME 1997)

Several studies have been published to data with respect to carcinogenicity and select studies are briefly summarized below according to the region in which they were conducted.

A2.4.1 Taiwan

In 1968 and 1977, studies were published reporting an increased prevalence of skin cancer in Taiwanese populations exposed to elevated concentrations of arsenic in their drinking water (Tseng *et al.* 1968; Tseng 1977). Subsequently, several epidemiological studies were conducted in Taiwan indicating significant associations between arsenic in drinking water and increased risk of skin cancer as well as cancers of the internal organs, namely the bladder, kidneys and lungs (Chen *et al.* 1985; 1986; 1992; 2004; Wu *et al.* 1989; Morales *et al.* 2000).

Since 1985, research on the association between arsenic in drinking water and cancer has been conducted in other parts of the world, including South America, Europe and the United States (discussed below) but the region of south western (SW) Taiwan known uniquely for Blackfoot Disease (BFD) has been regarded as the single most significant source of data on this relationship (Lamm and Kruse 2005). Reasons cited include the long exposure and follow-up period (since the 1920s), homogeny between lifestyles of the population and a large population size (approximately 40,000 people) (U.S. NRC 2001; Health Canada 2006b). The key dataset consists of population and mortality data from 42 villages in SW Taiwan for the years 1973 to 1986. Arsenic levels were measured in wells from these villages in 1964 to 1966 when researchers investigating BFD found that the artesian wells used for drinking water since the 1920s had very high levels of arsenic. Components of this dataset have been analysed by numerous authors to assess the health effects of arsenic through ingestion of arsenic-contaminated drinking water (Tseng 1977; Chen *et al.* 1985; 1986; 1992; 2004; Wu *et al.* 1989; U.S. NRC 1999; 2001).

Among the 42 Taiwanese villages, there were 21 villages with arsenic levels in water below 300 µg/L, 15 villages with levels from 300 to 590 µg/L and 6 villages with levels greater than or equal to 600 µg/L (Health Canada 2006b). Ecological analyses indicated significant dose-response relationships between the arsenic levels and mortality from cancer of the liver, lung, bladder and kidney in most age groups of both males and females (Wu *et al.* 1989; Chen *et al.* 1992; Health Canada 2006b). Potency indices for excess lifetime risk due to an intake of 10 µg/kg/d of arsenic indicated that the greatest risks were for development of bladder and lung cancer (1.5×10^{-2} for male and females) (Chen *et al.* 1992). At the highest level of exposure, approximately 800 µg/L, relative risk estimates for bladder cancer mortality were estimated to be as high as 28.7 for men and 65.4 for women (Chen *et al.* 1988; Smith *et al.* 1992). Linear extrapolation from high dose risks established in Taiwan as well as in Chile (described below) indicated that at 50 µg/L, 1 in 100 exposed people could die of arsenic related cancer (U.S. NRC 1999).

With respect to lung cancer, a recent cohort study in Taiwan suggested that the threshold below which arsenic does not increase cancer risk could be as high as 640 µg/L. Guo (2004) examined dose response relationships between ingestion of arsenic in drinking water and lung cancer deaths (1971 to 1990) in 10 townships in Taiwan. Data on arsenic levels in drinking water were available for 138 villages from a census survey conducted by the government. After adjusting for age, multivariate regression indicate that arsenic levels above 640 µg/L were associated with a significant increase in the mortality of lung cancer in both genders, but no significant effect was observed at lower levels. Post-hoc analyses were noted to confirm such a dose-response relationship.

Several ecological analyses of the Taiwan data have been published, but the key study that formed the basis of carcinogenic risk assessments by the U.S. EPA (2001; 2005ab), the National Research Council (U.S. NRC 1999; 2001) and Health Canada (2006b) was by Morales *et al*. (2000). Morales *et al*. (2000) published a detailed analysis of data from the 42 villages previously reported in Wu *et al. (*1989) and Chen *et al*. (1992) but rather than using the three or four-level arsenic strata methodology, they used village-specific census and mortality data and village-specific median well water arsenic levels (Lamm *et al.* 2006). They used the median village arsenic level as the sole explanatory variable for estimating risk and assumed a linear no-threshold model for extrapolation to low doses. A quantitative description of these cancer potencies and risks associated with specific doses is found in Section 3.0.

Several criticisms have been put forth regarding the use of the SW Taiwan data for estimating cancer risks at the low doses more typical of those found in the U.S.A or Canada, including:

- Taiwan, being the only BFD-endemic area of the world, is clearly distinguished from other high arsenic regions of the world where BFD has not been found (*e.g.*¸ large parts of China). This suggests there may be an unknown environmental component in this region that might be related to the observed bladder cancer mortality; recent analyses support this theory (Lamm and Kruse 2005; Lamm *et al.* 2006).

- The SW Taiwanese population is not representative of populations in North America due to the compromised nutritional status of the population (Gradient Corporation 2006).

- The results of several recent epidemiological studies that examined arsenic health risks at low or moderate levels of arsenic exposure have not found elevated cancer risks in non-smokers. Results in smokers have been mixed.

The first criticism, that a unique environmental component in Taiwan has likely influenced the arsenic-cancer associations, comes from recent re-analyses of the SW Taiwanese dataset by Lamm and Kruse (2005) and Lamm *et al*. (2006).

Lamm and Kruse (2005) noted that the two common features of the regulatory risk analyses based on this data are: that it is presumed that the variation in cancer rates among the villages can be meaningfully expressed in terms of arsenic exposure alone; and secondly - it is assumed that there is no level below which arsenic exposure does not demonstrate a cancer risk – *i.e.*, that there is no threshold below which it doesn't increase cancer risk. Yet linear regression analysis performed by Lamm *et al*. (2006) showed that arsenic levels in drinking water account for only 21% of the variance in the village Standard Mortality Ratios (SMRs) for bladder and lung cancer. It was also noted that the influence of confounders such as township, BFD prevalence and dependence on artesian wells *versus* shallow-water wells had been reported previously (Chen *et al.* 1985; Lamm and Kruse 2005) but they had never been introduced into a quantitative assessment.

Lamm *et al.* (2006) re-analysed the SW Taiwan data, first by categorizing villages as either "low dose" or "high dose" villages, with the high dose group being indicative of dependency on artesian wells. Chen *et al*. (1985 1986) and Wu *et al*. (1989) reported that the median arsenic concentration of individual artesian wells in the study areas was in the range of 350 to 1,100 µg/L, and that the median concentration in individual shallow wells was between 0 and 300 µg/L. Lamm *et al.* (2006) found significant differences in dose-response between the low dose villages (n=18) and the high dose villages (n=24). In the low dose villages there was a non-significant *negative* response for bladder and lung cancer SMR, while the high dose villages showed a significant positive dose-response relationship with arsenic exposure (Lamm *et al.* 2006). When township was examined as a potential source of cancer risk variability for the low dose villages, it was discovered that this had a significant impact. Only three of the six townships showed a significant positive dose-response with arsenic exposure (2, 4 and 6), while the other three townships demonstrated significant bladder and lung cancer risks that were independent of arsenic exposure. For townships 2, 4 and 6 the data for cancer mortality (lung and bladder) fit an inverse linear regression model ($p<0.001$) with an estimated threshold at 151 µg/L (95% CI: 42 to 229 µg/L). Lamm *et al*. (2006) concluded that if the SW Taiwan data are to be used in formal risk analysis in association with arsenic ingestion, analysis should be restricted to the data from townships 2, 4 and 6. He further noted that the finding of no increased risks at concentrations below 150 µg/L is consistent with other epidemiological data (see further).

The identity of this township factor is not known, but it likely relates to the BFD prevalence township factor previously reported by Chen *et al*. (1985). Previously, it was concluded that higher BFD prevalence rates within townships, were associated with greater SMRs for cancers of bladder, kidney, skin and lung (Chen *et al.* 1985). It is important to note that SW Taiwan is the only area in the world where BFD has been found. This is not likely due to genetics since among nearly 300,000 arsenicosis patients identified in mainland China, not a single case of BFD has been reported (Sun 2004). The rate of bladder cancer mortality for residents of the BFD endemic regions of SW Taiwan is very high relative to the rest of Taiwan (10-fold higher), even in those villages exposed to relatively low arsenic concentrations (0 to 50 µg/L) (Lamm and Kruse 2005). There is only a 3-fold difference between relative risk for the highest arsenic exposure to the lowest arsenic exposures, whereas the relative risk from living in the area in general is 10-fold that of the whole of Taiwan. This suggests another major environmental factor may be driving the bladder cancer risk in this area, and inferences about arsenic risks may not be applicable in other regions (Lamm and Kruse 2005).

The analysis by Lamm and Kruse (2005) was published in the *Journal of Human and Ecological Risk Assessment* (HERA) and selected by HERA's editors as the Human Risk Assessment Paper of the Year in 2005 based on its outstanding contribution to the field of risk assessment.

Another criticism of the use of the SW Taiwan dataset for risk analyses is the suggestion that poor nutrition and limited access to health care may have played a role in the susceptibility of the Taiwanese to arsenic in the endemic area (Beck *et al.* 1995; Schoen *et al.* 2004). Their diet was nutrient poor, consisting largely of rice and yams, with limited intake of fresh vegetables or protein. Some support for this comes from studies in Bangladesh and Taiwan that suggest arsenicosis cases were more likely to have low serum levels of beta carotene (Hsueh *et al.* 1997) or low body mass index (BMI) indicative of malnutrition (Milton *et al.* 2004).

It has also been shown that dietary protein intake and possibly other nutritional deficiencies (*e.g.*, iron, zinc and niacin) can affect arsenic methylation and metabolism in humans (Steinmaus *et al.* 2005a) and in animals (Tice *et al.* 1997; Vaher and Marafante 1987). There is strong evidence suggesting that individual differences in arsenic methylation patterns and the factors (environmental or genetic) that cause those differences play an important role in susceptibility to arsenic-caused disease (Steinmaus *et al.*; 2005a; Chen *et al.* 2003a,b; Hsueh *et al.* 1997; Del Razo *et al.* 1997; Maki-Paakkanen *et al.* 1998). For example, Chen *et al*. (2006) recently demonstrated that subjects in Taiwan with lower arsenic methylation ability have a substantially increased risk of bladder cancer, especially when combined with high cumulative arsenic exposure values. The impact of dietary protein and other nutrients on arsenic methylation does not appear to be large, however, and it is likely that genetic factors play a more important role (Steinmaus *et al.* 2005a; Chung *et al.* 2002). A study that investigated the role of protein and other nutrients on arsenic-induced skin lesions in West Bengal, India found a doubling of risk in subjects with low intakes of calcium, fibre, folate and animal protein, but this could not explain the very large increases in cancer risk observed in SW Taiwan (Mitra *et al.* 2004).

A2.4.2 Chile and Argentina

The U.S. NRC (1999; 2001) and other regulatory agencies evaluating arsenic concluded that there is no evidence of nutritional factors that could account for the high rate of cancer seen in the arsenic-exposed Taiwanese population. They note that increases in cancer risk have been associated with chronic arsenic exposure in other countries, such as Chile and Argentina, where poor nutrition and low protein diets are not issues (U.S. NRC 2001).

An ecological study of a population in Northern Chile exposed to high arsenic levels in drinking water indicated that bladder cancer mortality (1989 to 1993) was significantly elevated in men and women (6- to 8-fold), as was lung cancer mortality (3- to 4-fold) (Smith *et al.* 1998). Significantly elevated skin cancer mortality was observed only in men. Several towns or cities affected by high levels of arsenic in drinking water were studied, including Antofagasta, where concentrations of 870 µg/L were measured from 1955 through 1970. Population weighted average arsenic levels for all the affected towns were approximately 570 µg/L between 1955 and 1969, and decreased to less than 100 µg/L by the end of the1980s. Smoking data and mortality rates from chronic obstructive pulmonary disease indicated that smoking did not contribute to the increased cancer mortality (Smith *et al.* 1998).

A case-control study of newly diagnosed lung cancer (1994 to 1996) in the same region of Chile revealed a clear trend in lung cancer odds ratios with increasing concentration of arsenic in drinking water (Ferreccio *et al.* 2000). The same study also reported considerably higher risks in smokers *versus* non-smokers. As the period of peak exposure was from 1955 to 1975, the increased relative risks of lung cancer were likely related to exposure that predominately occurred 20 to 40 years before cancer diagnosis in association with exposure levels up to 870 µg/L. Significant associations were found with arsenic concentrations in drinking water as low as 30 to 50 µg/L and lung cancer. However, this study has been criticised as a result of the control selection methods used which may have led to under-estimation of risks in the highest exposure group and over-estimation of risks in the lower exposure groups (U.S. NRC 2001). In addition, confidence limits were broad and there was limited low exposure data.

An ecological study in Argentina found increasing trends for bladder, kidney and lung cancer mortality in both men and women during the period 1986 to 1991 (Hopenhayn-Rich *et al.* 1998). High natural arsenic levels in drinking water have been measured in Cordoba province with concentrations often above 100 µg/L and reaching levels over 2,000 µg/L (Nicolli *et al.* 1985). Counties in Cordoba were categorized as low, medium or high arsenic exposure based on available data and SMRs were calculated using the general population of Argentina as the reference population. In the counties with the lowest exposures (<40 µg/L) cancer risks were predominantly either lower than expected (non-significantly) or non-significantly elevated, particularly in the case of bladder and kidney cancer. The incidence of lung cancer in men were found to be significantly elevated for medium and high exposure counties, but not low exposure counties. Levels of arsenic in the medium exposure group were not clearly defined – only that they had at least one measurement over 120 µg/L. A crude average of 178 µg/L was estimated for the high exposure group.

Bates *et al.* (2004) conducted a case-control study of bladder cancer (1996 to 2000) and arsenic exposure in the same region of Argentina, but with the great advantage of individual exposure data. Water samples were collected from each residence and as many of his or her residential sources within the previous 40 years as practicable. During face-to-face interviews, information was collected on residential history, water sources at each residence, consumption of beverages (at the time of the interview and 20 and 40 years earlier) and smoking, occupational and medical histories. Overall and in non-smokers, bladder cancer was not associated with arsenic concentrations in drinking water, even when water consumption rates were accounted for. In smokers, the only significant finding was with the use of well water more than 50 years ago and authors noted that this might be due to chance (Bates *et al.* 2004). The lack of associations observed in this study were unexpected, particularly for the group exposed to > 200 µg/L in their drinking water. There was suggestion of a risk reduction at high arsenic levels, although this was not significant.

Overall, studies in Taiwan, Chile and Argentina have been valuable in establishing the carcinogenicity of arsenic, however, the dose-response relationships that have been observed were at higher exposure levels (> 100 ppb). To date, no epidemiological studies have produced convincing evidence of risks related to drinking water concentrations less than 100 µg/L. Two recent, large cohort studies in Taiwan failed to find statistically elevated risks below 100 µg/L (Chiou *et al.* 2001; Guo 2004). In north eastern Taiwan, Chiou *et al.* (2001) evaluated the association between ingested arsenic from drinking water and the risk of bladder cancer in 8,012 residents from 18 villages with well water samples from each home, as well as information on smoking, water consumption, *etc*. Arsenic levels in well water ranged from < 0.15 to 3,486 µg/L and exposure was categorized into four groups: < 10.0, 10.1 to 50.0, 50.1 to 100.0, and > 100.0 µg/L. Overall, there was a 2-fold increased incidence of urinary cancers (1991 to 1994) relative to the general Taiwan population but when results were broken down by exposure levels there were no significant risks associated with arsenic levels lower than 100 µg/L (Chiou *et al.* 2001). Although a significant trend toward a dose-response relationship was observed, the confidence intervals were very wide, which limits interpretation of this finding. This study had the advantage of individual exposure data and a wide variation in exposure levels but the follow-up period for identifying cancer cases was small (1991-1994).

A2.4.3 United States

In addition to the Taiwanese and South American data, several U.S. and European studies have been conducted that examined health risks associated with arsenic exposure less than 100 µg/L. These studies are examined in the sections below.

A case-control study of bladder cancer in a region of Utah with arsenic exposures in drinking water ranging from 0.5 to 160 µg/L (mean of 5.0 µg/L) indicated no associations in non-smokers (Bates *et al.* 1995). In smokers, positive trends in risk were found for exposures decades prior to diagnosis but the observation was not consistent with respect to latency and statistical significance was established using a 90% confidence interval rather than a 95% confidence interval, raising the possibility that chance could have accounted for the findings (Schoen *et al.* 2004). For cumulative arsenic exposures greater than 53 mg, an odds ratio of 3.31 was identified in smokers (CI: 1.1 to 10.3).

A large cohort mortality study among members of the Mormon Church in Utah (Lewis et al. 1999a) found no relationship between exposure to arsenic in drinking water and bladder or lung cancer. Surprisingly, SMRs for lung cancer were significantly *decreased* in both the high and the low exposure categories. Median drinking water concentrations in the towns in question ranged from 14 to 166 µg/L, and averaged approximately 100 µg/L. A statistically significant increase in prostate cancer mortality was observed (SMR=1.45, CI=1.07 to 1.91) but this occurred only in the middle exposure level and thus, could not be used to confirm a dose-response relationship (Schoen *et al.* 2004). This study has been criticized because the comparison group (the general population of Utah) does not have the same religious prohibitions against drinking and smoking as does the Mormon Church (U.S. NRC 2001). The authors point out, however, that as 70% of Utah is Mormon, these lifestyle factors are not likely to have contributed significantly to their findings (Lewis *et al.* 1999b)

A case-control study of bladder cancer and drinking water in counties of Nevada and California (1994 to 2000) involved collection of individual data on water sources, water consumption patterns, smoking and other factors for 181 cases and 328 controls. The counties were noted to contain the largest populations historically exposed to drinking water arsenic at concentrations near 100 µg/L. No increased risks were identified for arsenic intakes as high as 80 µg/d or above(odds ratio = 0.94; CI = 0.56 to 1.57). When the analysis focused on exposures over 40 years ago, a significant association was identified for smokers exposed to greater than 80 µg/d (median intake of 177 µg/day) (Steinmaus et al. 2003).

A very large ecological analysis (Lamm et al. 2004) of arsenic in drinking water and bladder cancer mortality in the U.S. from 1950 to 1979 found no associations over the exposure range of 3 to 60 µg/L. In this study, county-specific groundwater arsenic concentration data were obtained for 133 U.S. counties (in 26 States) known to be exclusively dependent on groundwater for their public drinking supply. The data was collected in U.S. Geological Surveys between 1973 and 1998, with most of the measurements obtained after 1980. This study has been criticized by the U.S. EPA (2005b), as it was assumed that groundwater arsenic concentrations for the counties were stable over time. However, several other epidemiological studies have made the same assumption, including the Taiwanese studies. In addition, a recent study in western Nevada found a strong correlation between arsenic measurements taken from the same wells over a period of 1 to 20 years, indicating that a single or limited number of arsenic measurements per well can be used to predict arsenic exposures over a period of many years (Steinmaus *et al.* 2005b).

Another major limitation of this study is that 84% of the 133 counties studied had median exposure levels of 3 to 5 µg/L, so there was little variance across the counties. This would have limited its power to detect any true associations. It has also been noted that the analysis of bladder cancer mortality data in North America is limited, as bladder cancer generally does not result in mortality (Health Canada 2006b; Frost 2004).

A similar study to Lamm *et al. (*2004) was published by the U.S. American Water Works Association Research Foundation, but in this study all 33 counties examined had mean drinking water arsenic levels of 10 µg/L or greater, with 12 counties having measured concentrations of 20 µg/L or greater (Frost 2004). They evaluated the relationships between lung and bladder cancer mortality (1950 to 1999) and incidence (1973 to 1999) using multi-level, hierarchical statistical models. Three approaches were employed by the research team: 1) combining all cancer deaths for all ages across the decades for which data was available, 2) conducting a sub analysis limited to the population age 50 years or older, and 3) combining cancer deaths for those decades (1960 to 1999) for which comparable census variables were available.

The results indicated that arsenic in drinking water at levels > 10 µg/L was not associated with greater mortality from bladder or lung cancer, nor was it associated with greater incidence of these cancers. The principal investigator noted that the findings are consistent with other recent studies of the health effects of low dose arsenic exposure and are not consistent with the U.S. EPA predictions of excess cancer risk from low dose exposure (Frost 2004). The analysis did detect other predictors of elevated cancer mortality, demonstrating the power of the modeling technique used to detect geographic relationships between exposures and health outcomes (Frost 2004).

Health Canada (2006b) dismissed the findings of Frost (2004) its recent "*Guidelines for Canadian Drinking Water Quality: Guideline Technical Document*". Health Canada noted that the authors of the report cautioned that it is possible for elevated risks of lung and bladder cancer mortality or incidence to be present but not apparent in the analysis, as the analysis of cancer risks from bladder cancer mortality data is limited, given that people with bladder cancer do not generally die from it. However, this statement is unclear, however, since the study examined both mortality and incidence. The caution could not apply to the lack of association with bladder cancer *incidenc*e. Moreover, the study examined lung cancer as well as bladder cancer and most people with lung cancer do die from it. Health Canada further noted that this was an ecological study that relates exposures and outcomes in groups of individuals that may not be representative of individual responses to arsenic exposure (Health Canada 2006b). While this is true, it is also true of the study in SW Taiwan which was chosen as the basis of their cancer potency calculation.

An unpublished study of arsenic in drinking water and cancer mortality in 226 U.S. counties primarily dependent on groundwater and with populations of 25,0000 or more indicated significant negative correlations between arsenic level and various cancers, particularly for colon and lung cancer (Williams and Longbrake 2005). Among the 226 counties, 145 were classified as "low arsenic" (< 3 ppb), 44 as "medium arsenic" (3 to 10 ppb) and 37 as "high arsenic" (> 10 ppb).

Michaud *et al*. (2004) examined the relation between toenail arsenic levels and bladder cancer risk in a nested case-control study among a cohort of male smokers in the Alpha-Tocopherol, Beta-Carotene Cancer Prevention Study. Two hundred eighty incident bladder cancer cases between baseline (1985 to 88) and April 1999. A control was matched to each case on the basis of age, toenail collection date, intervention group and smoking duration. Trace element measurements from toenail clippings reflect internal exposure 9 to 18 months prior to collection, depending on the length of the toenail and studies have shown that arsenic levels measured in toenails remain relatively constant over spans of up to 6 years (Garland *et al.* 1993; Karagas *et al.* 2001). The primary advantage of this exposure measurement is that it provides an integrated measure of internal inorganic arsenic exposure, reflecting all sources of exposure (*i.e.*, diet, drinking water, occupational).

No associations were observed between inorganic arsenic concentration and bladder cancer risk, even at the 95^{th} percentile of toenail arsenic levels (Michaud *et al.* 2004). Another study validating toenails as biomarkers of arsenic ingestion from water found a correlation of 0.65 when arsenic levels in water were equal or greater than 1 µg/L (Karagas *et al.* 2000). Michaud *et al.* (2004) used the linear regression from this analysis to estimate that the 50^{th}, 75^{th}, 90^{th} and 95^{th} percentiles of toenail arsenic levels in their study correspond roughly to 2, 10, 50 and 100 µg/L, respectively. It was concluded that their results suggest arsenic exposure levels of around 50 µg/L do not increase the risk of bladder cancer. However, given the small sample size in the top percentiles, they could not exclude the possibility that exposure levels of about 100 µg/L may be associated with bladder cancer risk in smokers (Michaud et al. 2004).

To examine the effects of lower levels of arsenic exposure on bladder cancer incidence, Karagas *et al.* (2004) conducted a case–control study in New Hampshire, USA where levels of arsenic above 10 µ/L are commonly found in private wells. They identified 383 cases of transitional cell carcinoma of the bladder (1994 to 1998) and 641 general population controls. Individual exposure to arsenic was determined in toenail clippings using instrumental neutron activation analysis. Among smokers, an elevated odds ratio (OR) for bladder cancer was observed but only for the uppermost category of arsenic and this was not statistically significant (OR: 2.17, 95% CI: 0.92 to 5.11 (greater than 0.330 µg/g compared to less than 0.06 µg/g). Among non smokers, there was no association between arsenic and bladder cancer risk.

A2.4.4 Europe

A case-control study of bladder (n=61) and kidney cancer (n=49) cases diagnosed in Finland (1981 to 1995) suggested an association between arsenic in drinking water and bladder cancer risk at very low exposure levels. Arsenic concentrations in the well samples (1967 to 1980) ranged from 0.05 to 64 µg/L, although the median concentration was only 0.1 µg/L. Information on residential history, drinking water consumption at the selected addresses and potential confounders such as smoking and occupation were collected using a mail questionnaire. Two latency periods were evaluated, short (3 to 9 years prior to cancer diagnosis) and long (> 10 years). For the short latency period only, statistically significant increased risks of bladder cancer (approximately 2-fold) were reported for arsenic concentrations > 0.5 µg/L. Exploration by smoking status revealed no increased risks in non-smokers but a 10-fold elevated risk in smokers exposed to arsenic greater than 0.5 µg/L (Kurttio et al. 1999).

The results of this study are questionable, as the majority of exposure to inorganic arsenic among subjects would likely have been food rather than water, similar to the situation in Canada. The finding of elevated risk for shorter but not longer latency is also inconsistent with the results of other studies on arsenic and cancer latency (Steinmaus *et al.* 2003; Bates *et al.* 2004; Chen *et al.* 1986; Chiou *et al.* 2001). Kurttio *et al.* (1999) noted that the relative risk observed was not expected at such low concentrations, and may be due to either bias or chance.

A2.5 Meta-Analysis of Low Level Studies

One of the criticisms of the "low level" arsenic studies is that they had insufficient power to detect the small increases in risk that would be expected with exposure to low levels of arsenic. Very large studies are required to detect small risks at a statistically significant level.

In an attempt to answer this criticism and increase study power, Exponent (2005) recently conducted a review and meta-analysis of low-level arsenic exposure in drinking water and bladder cancer. Eight case-control or cohort studies were identified as eligible for the meta analysis (Bates *et al.* 1995; Lewis *et al.* 1999a; Chiou *et al.* 2001; Steinmaus *et al.* 2003; Karagas *et al.* 2004; Michaud *et al.* 2004). A mean relative risk (mRR), a 95% confidence interval, and p-value for heterogeneity were generated for each analysis. The meta-analysis indicated that bladder cancer was not significantly associated with low-level exposure to arsenic in drinking water. In non-smokers, the mRRs were consistent, robust and less than 1.0. For smokers, the results were heterogeneous, although there was no consistent evidence of increased risk or effect modification. The main results of the meta-analysis were not consistent with and were below the range of relative risks (1.2 to 2.5) predicted by the U.S. NRC (2001).

A2.6 Mechanistic Data and Mode of Action

An important consideration in predicting cancer risks at low doses and the shape of the dose-response curve is evidence regarding mechanisms and mode of action (*i.e.*, how does arsenic increase cancer risk). Despite a large body of research, however, this is still not clearly understood with respect to arsenic. Much of the difficulty is that the effects of arsenic at a cellular level are strongly dose-, time- and species-dependent (Barchowsky *et al.* 1999; WHO 2001; Andrew *et al.* (2006). It is likely that more than one mechanism is involved (Liu *et al.* 2005; Rossman 2003).

Overall, the evidence suggests that inorganic arsenic and specifically its trivalent metabolites, MMA^{III} and DMA^{III}, are clastogenic; *i.e.*, they cause chromosome breakages (Kligerman *et al.* 2003). This is likely mediated by cytotoxicity resulting from high dose exposures, but it may also be a secondary result of diminished DNA repair capacity and oxidative damage (Rossman 2003). Inorganic arsenic has not been shown to mutate bacterial strains and it does not induce point mutations or directly damage DNA (Kligerman *et al.* 2003; Schoen *et al.* 2004).

Some studies have reported increased chromosome aberrations (*i.e.*, elevated micronuclei) in the lymphocytes of people exposed to high levels of arsenic in drinking water, but it is difficult to distinguish whether this is caused by clastogenicity or aneuploidy (Basu *et al.* 2001; Mahata *et al.* 2003). Aneuplody is an error of cell division and evidence suggests this may be a threshold type of response (Elhajouji *et al.* 1995).

As arsenic appears to be genotoxic by causing chromosomal mutations, it has been suggested that it may act, in a latter stage of carcinogenesis (tumour progression), rather than as a classical initiator or promoter (Moore *et al.* 1995; U.S. EPA IRIS 1998). Arsenic has been shown to enhance the onset and growth of malignant skin tumours induced by ultraviolet radiation in mice (Rossman *et al.* 2001). It has also been shown to potentiate the genotoxicity of other carcinogens, such as polycyclic aromatic hydrocarbons (Rossman 2003). This suggests that arsenic may act as a co-carcinogen and require a partner in causing cancer. This type of interaction may explain the finding in some epidemiological studies that smokers are more susceptible to arsenic caused cancer (Chen *et al.* 2004).

There is considerable evidence that oxidative stress and inhibition of DNA repair play important roles in the carcinogenicity of arsenic (Andrew *et al.* 2006; Mo *et al.* 2006). Cellular proliferation, altered gene expression, altered DNA methylation and modulation of signal transduction pathways have also been associated with arsenic toxicity (Schoen *et al.* 2004). The metabolism of inorganic arsenic results in the generation of free radicals, which cause oxidative stress and studies suggest oxidative DNA damage and DNA-protein cross links may be the major DNA lesions induced by arsenic (Bau *et al.* 2002; Kitchin and Ahmad 2003; Rossman 2003). Antioxidants such as Vitamin E and glutathione have been shown to protect again arsenite genotoxicity, which is suggestive of an oxidative mechanism for arsenic (Huang *et al.* 1993; Rossman 2003).

A recent study of residents chronically exposed to arsenic in drinking water in Inner Mongolia found a strong correlation between water arsenic concentrations and expression of a gene involved in DNA repair (OG1) (Mo *et al.* 2006). Maximum gene expression was observed at an arsenic exposure level of 149 µg/L with expression levelling off or decreasing when arsenic exposure was above this concentration. Increased expression of this gene is considered an adaptive response, and was noted to be in agreement with reports in cultured human cells that low doses (0.1 to 1 µM) arsenic induces significant up-regulation of adaptive responses (*e.g.*, DNA repair, enhanced cell proliferation, telomerase activity), whereas at high concentrations (> 1 µM), these processes are down-regulated. In another recent study of people exposed to arsenic in drinking water, arsenic was associated with decreased expression of a gene associated with DNA repair (ERCC1) (Andrew *et al.* 2006).

In a review of the mechanisms of arsenic carcinogenicity, Rossman (2003) stated that*: "It is becoming increasingly clear that high dose exposure to arsenic compounds differs from low dose exposure with regard to genotoxicity, types of reactive species formed, signal pathways activated and gene expression. Many "stress proteins" seem to be induced only at high dose"*.

Similarly, Snow *et al.* (2005) note that the changes in tissue response seen at low doses are different not only in magnitude, but in character from those seen at higher, more toxic doses and are biphasic in nature, possibly indicative of a hormetic effect. For example, treatment of human keratinocyte and fibroblast cells with 0.1 to 1 µM arsenic produces a low dose protective effect against oxidative stress and DNA damage caused by other oxidative agents (Smith *et al.* 2001; Hirano *et al.* 2003). At higher concentrations (> 10 µM), down regulation of DNA repair, oxidative DNA damage and apoptosis were observed (Snow *et al.* 2003). A low dose adaptive or protective response by a toxic agent is known as hormesis and is characterised by many agents that cause oxidative stress (Calabrese and Baldwin 2003; Parsons 2003; Snow *et al.* 2005). High concentrations of arsenite may result in its sudden accumulation in cells and may have effects that differ from a slower accumulation, which would allow tolerance mechanisms to come into play (Rossman 2003).

As arsenic is not a direct acting carcinogen, the default to a linear low dose extrapolation model when calculating risks from arsenic ingestion is likely not appropriate. However, cellular and biological effects have been demonstrated at very low levels. Although many of them are likely to be adaptive or protective, the significance of them in vivo is not yet fully understood (Kaltreider *et al.* 2001; Andrew *et al.* 2006; Snow *et al.* 2005; U.S. NRC 2001). Some of the responses seen at low doses, such as increased cell proliferation can be both cyto-protective and pro-carcinogenic (Snow *et al.* 2005).

Animal studies are considered to provide limited evidence of the carcinogenicity of inorganic arsenic but sufficient evidence for the carcinogenicity of one of its metabolites, dimethylarsinic acid (DMA^{III}) (IARC 2004). On the whole, laboratory animals appear to be less susceptible to the effects of arsenic; even after doses 100 times greater than experienced by humans, they often show little response (U.S. NRC 1999; Smith and Smith 2004).

A2.7 Essentiality and Therapeutic Effects

Several animal studies suggest that arsenic may be an essential element in rats, mice, goats, chicks and mini pigs (U.S. NRC 1989). Animals fed a diet with unusually low concentrations of arsenic did not gain weight normally, became pregnant less frequently and had offspring that were smaller than normal or died early (reviewed in Uthus 1992; ATSDR 2005). Adding arsenic back to the diet reversed these effects. ATSDR (2005) noted that: "*Despite all of the adverse health effects associated with inorganic arsenic exposure, there is some evidence that the small amounts of arsenic in the normal diet (10-50 ppb) may be beneficial to your health*." No cases of arsenic deficiency have ever been reported in humans, likely because there are small amounts in almost all food, water, air and soil (DTMRP 2006). In the event that health effects associated with arsenic deficiency exist, effects such as those observed in animal studies would be hard to detect and characterize in humans (DTMRP 2006). A study of haemodialysis patients suggested that arsenic homeostasis is disturbed by haemodialysis treatment and found correlations between markedly decreased serum arsenic and various diseases, particularly injuries of the central nervous system (CNS), vascular diseases, and cancer (Mayer *et al.* 1993). The authors concluded that desirable arsenic concentrations in the body seem to be reasonable.

There is no known biological function for arsenic, however, some research indicates that it has a physiological role in methionine metabolism (Uthus 2003). A research chemist with the U.S. Department of Agriculture has shown that animals deprived of arsenic have decreased methionine in their plasma and a decreased liver content of an important metabolite of methionine, S-adenosylmethionine (SAM). Animals with low amounts of methionine in their diet, or with decreased stores of SAM are prone to tumour development. Decreased amounts of SAM are also associated with hypomethylation and recent studies suggest that arsenic status can affect DNA methylation in animal and cell culture models (Davis *et al.* 2000; Uthus 2003; Uthus 2005). Uthus (2005) determined the effects of feeding various concentrations of arsenic (50, 0.5 and < 10 ng/g in the diet) on rats treated with a colon carcinogen, dimethylhydrazine (Uthus 2005). Rats fed a diet high in arsenic had significantly higher numbers of preneoplastic lesions in the colon compared to rats fed a small amount of arsenic. DNA was also hypomethylated (a state normally associated with cancer) in rats that had the increased number of colon lesions. Rats fed a diet devoid in arsenic (*i.e.*, < 10 ng/g in diet) responded similarly as those fed the high-arsenic di*et al*though the differences with the low dose group were not statistically significant (Uthus 2005)

Arsenic trioxide (AS_2O_3) has been used successfully as a chemotherapeutic agent for acute promyelotcytic leukemia (APL) and recent research suggests it may have therapeutic use in the treatment of other types of cancer as well (Douer and Tallman 2005). In randomized clinical trials, predominantly mild side effects were reported with controlled intravenous dosing of 50 to 150 µg/kg bw/d arsenic trioxide for 12 to 39 days (12 patients reported by Soignet *et al.* 1998; 40 patients reported by Soignet *et al.* 2001; 540 patients reported by Dombret *et al.* 2002). Tsuji *et al.* (2004) note that most patients in these trials experienced effects such as fatigue, nausea, elevated blood glucose, headache, edema; only a few had more serious effects such as peripheral neuropathy or cardiac arrhythmia and these appeared to be manageable. In a study of arsenic trioxide treatment in newly diagnosed children with APL, George *et al. (*2004) reported minimal toxicity after eight cycles of As_2O_3 (150 µg/kg/day) administered over a period of 12 months. With a median follow-up of 30 months, they achieved remission rates of more than 90%, which is similar to data from children treated with the more conventional therapy of ATRA and chemotherapy. The authors note, however, that long-term follow-up is required to assess long-term remission and late side effects in children (George *et al.* 2004).

A3.0 ARSENIC EXPOSURE LIMITS

The determination of allowable or “safe” exposures to chemicals typically relies on the principle that the dose of a chemical dictates the nature and magnitude of any health effects that are expressed. For the purpose of risk assessment, however, chemicals are commonly categorized according to the nature of their toxic response. Threshold chemicals comprise the largest category, which are chemicals for which a threshold needs to be exceeded for toxicity to be expressed. The magnitude of the toxic response then increases with increasing dose. The threshold phenomenon applies to most toxic responses and chemicals, with the exception of some carcinogens and some forms of cancer. A no-observed-adverse-effects-level (NOAEL) can be determined for a chemical that exhibits a threshold-type dose-response relationship. A NOAEL represents the dose of the chemical that produces no obvious response in the most sensitive test species and test endpoint. The NOAEL may then be used to derive an exposure limit associated with minimal risk of adverse effects through the application of uncertainty or safety factors that provide an added level of protection (*e.g.*, protection of sensitive individuals). The exposure limit represents the dose of the chemical that is expected to be safe to the most sensitive subjects following exposure for a prescribed time period.

Non-threshold chemicals comprise substances that potentially can produce cancer through a genetically mediated mechanism for which a threshold level cannot be determined. Non-threshold chemicals are assessed using a unit-risk approach, based on the assumption that zero risk can only occur with the absence of exposure. Risk, therefore, increases with each unit of exposure. Exposure limits for carcinogenic chemicals are based on an incremental lifetime cancer risk (ILCR) of one in 100,000 (AENV 2001). This level of risk is considered an essentially negligible.

In the case of arsenic, both threshold-based and non-threshold based exposure limits have been recommended by regulatory agencies. Threshold-based limits are typically referred to either as “Reference Doses” (RfDs), “Reference Concentrations” (RfCs), “Tolerable Daily Intakes” (TDIs), or “Minimum Risk Levels” (MRLs). Non-threshold based exposure limits are usually referred to as “Risk-specific Doses” (RsDs) and are based on a measure of carcinogenic potency known as a slope factor or unit risk. Some agencies have developed both RfDs and RsDs for inorganic arsenic, with an RfD based on non-carcinogenic effects and an RsD based on carcinogenicity (*e.g.*, U.S. EPA IRIS 1998). Other agencies, such as the Health Council of the Netherlands, have recommended only a threshold-based TDI, as they concluded arsenic compounds damage DNA through a non-genotoxic mechanism (HCON 2000). The recommended exposure limits and their basis for each agency are described below for both oral and inhalation exposure.

A3.1 Oral Exposure Limits

Table A-3 below summarizes the oral exposure limits evaluated by CEI. Additional detail and discussion regarding these limits is providing the sections below.

Table A-3. Summary of Oral Exposure Limits for Arsenic

Agency/Organization	Limit	Reference
Health Canada – Food Directorate	TDI: 1 µg/kg bw/day	Personal Communication – Lo 2006
Health Canada – Water Quality and Health Bureau	RsD: 0.3 µg/L	Health Canada 2006b
Health Canada – Contaminated Sites Division	SF: 2.8 $(mg/kg\text{-}d)^{-1}$	Health Canada 2004
World Health Organization	PMTDI: 2 µg/kg bw/day	WHO 1983, JECFA/WHO 1989, WHO 2003
Health Council of the Netherlands	TDI: 1 µg/kg bw/day	HCON 2000
Agency for Toxic Substances and Disease Registry	MRL(acute): 5 µg/kg bw/day MRL (chronic): 0.3 µg/kg bw/day bw/day	ATSDR 2005
United States Environmental Protection Agency	RfD: 0.3 µg/kg bw/day SF: 1.5 $(mg/kg\text{-}d)^{-1}$	U.S. EPA IRIS 1998

TDI: tolerable daily intake

RsD: risk specific dose associated with 10^{-5} ICLR

SF: slope factor

PMTDI: provisional maximum tolerable daily intake

MRL: minimal risk level

RfD: reference dose

A3.1.1 Health Canada

Health risks from arsenic in food are assessed by the Food Directorate using an interim TDI of 1 µg/kg body weight per day (Personal Communication - Lo 2006). This was derived based on a recent review of epidemiological data available in the scientific literature, although the background document for this derivation is not yet available. Until recently, the Food Directorate had used the TDI of 2 µg/kg body weight/day for inorganic arsenic recommended by JECFA/WHO (1989). For example, Indian and Northern Affairs Canada recently assessed arsenic intake in Arctic populations assuming a TDI of 2 µg/kg body weight per day, below which no health effects would be expected (CACAR II 2003).

Recently, Health Canada has decreased its recommended drinking water guideline for inorganic arsenic from 25 µg/L to 10 µg/L (Health Canada 2006b). As part of the assessment, they determined a unit risk associated with ingestion of 1µg/L arsenic in drinking water based on linear extrapolation from the estimated risks of bladder, liver and lung cancer in the SW Taiwan cohort (Morales *et al.* 2000; Wu *et al.* 1989; Chen *et al.* 1992). Based on this unit risk calculation and conservative assumptions regarding differences between the Taiwan and Canadian populations (*e.g.*, arsenic metabolism, drinking rates or body weights), a target concentration of 0.3 µg/L was estimated to be associated with essentially negligible cancer risks (1.4 in 100,000 to 1.9 in a million). The estimated lifetime cancer risk associated with ingestion of drinking water with arsenic at 10 µg/L is greater than the range that is considered “essentially negligible”, but treatment costs and the ability to measure and remove arsenic from drinking water supplies were also considered in the determination of a guideline value. (Health Canada

2006b). It was noted that the use of a linear quantitative risk assessment model may have lead to an overestimate of the risk of internal organ cancers (Health Canada 2006b).

Assuming an adult weighs 70 kg and drinks 1.5 litres of water/day, the Health Canada drinking water RsD of 0.3 µg/L translates to an RsD of 0.006 µg/Kg bw/day.

Health Canada (2006b) chose the study population of SW Taiwan to estimate low dose cancer risks, as it was recommended for quantitative risk assessment by the U.S. EPA (2001) and the U.S. NRC (2001). Reasons cited by the U.S. NRC (2001) for this include the sufficiently long-term exposure to arsenic and follow-up, extensive pathology data, homogeny between lifestyles of the population, and a large population size (approximately 40,000 people). The difficulties associated with the use of this cohort were described earlier (section 2.0). The negative findings of several epidemiological studies of populations with low dose exposure (reviewed earlier), together with the evidence that arsenic acts in a non-linear fashion to increase cancer risk (Rossman 2003; Schoen *et al.* 2004) suggests that the unit cancer risk recommended by Health Canada (2006b) is highly conservative.

In Health Canada's *Guidelines for Federal Contaminated Site Risk Assessment in Canada* (Health Canada 2004), it is recommended that arsenic be assessed as a non-threshold carcinogen, with an oral slope factor of 2.8 $(mg/kg\text{-}d)^{-1}$ corresponding to a 5% increase in the prevalence of skin cancer in the Taiwanese cohort (Tseng *et al.* 1968; Tseng 1977; CEPA 1993). The slope factor translates to an RsD of 0.0036 µg/kg body weight per day. This is however, based on an older assessment of arsenic carcinogenicity (CEPA 1993) that does not incorporate recent literature or data on the association between arsenic exposure and internal cancers, notably lung and bladder cancer.

A3.1.2 World Health Organisation (WHO)

The Joint FAO/WHO Expert Committee on Food Additives (JECFA) proposed a provisional maximum tolerable daily intake (PMTDI) of 2 µg/kg bw/d for inorganic arsenic in 1983 (WHO 1983). Based on a review of the epidemiological evidence at the time, they noted that a dose of 200 µg/L arsenic was associated with a 5% increase in the lifetime risk of skin cancer. It was concluded that arsenicism could be associated with water supplies containing an upper arsenic concentration of 1,000 µg/L or greater and that a concentration of 100 µg/L may give rise to presumptive signs of toxicity (*i.e.*, effects on the skin). The PMTDI was proposed based on the LOAEL of 100 µg/L, a daily intake of drinking water of 1.5 L/d and a body weight of 70 kg. No uncertainty factors were employed so it was noted that marginal effects at the TDI level of 2 µg/kg/d cannot totally be excluded (JECFA/WHO 1989).

In 1989, JECFA reviewed its evaluation and established a provisional tolerable weekly intake (PTWI) for inorganic arsenic of 15 µg/kg bw/week based on the previous PMTDI of 2 µg/kg bw/day. They acknowledged that there was a narrow margin between the PTWI and intakes reported to have toxic effects in epidemiological studies.

WHO has also established a provisional drinking water guideline for arsenic of 10 µg/L, based upon treatment technology and human health risk, which was maintained upon a re-evaluation in 2003 (WHO 2003).

A3.1.3 The Health Council of the Netherlands

In 2000, the Health Council of the Netherlands recommended a TDI of 1 µg/kg bw/d based on a review of the literature. The TDI was derived based on the dermal effects associated with ingestion of 100 µg/L arsenic in drinking water noted by JECFA/WHO (1989), and incorporation of a 2-fold uncertainty factor (HCON 2000). They noted that mild hyper-pigmentation was reported in one study at a dose level of 0.8 µg/kg bw/day, but in other studies NOAELs for dermal effects were determined to be 0.9 to 3 µg/kg/day.

The Health Council concluded that inorganic arsenic does not damage DNA by a genotoxic mechanism but rather it has clastogenic (chromosome damaging) properties. Accordingly, the existence of a toxic threshold was assumed and the TDI was derived from NOAELs/LOAELs rather than a cancer potency calculation (HCON 2000).

A3.1.4 Agency for Toxic Substances and Disease Registry (ATSDR)

For acute (*i.e.*, short-term) oral exposure, ATSDR derived a MRL of 5 µg/kg/d inorganic arsenic based on a LOAEL of 50 µg/kg bw/d associated with gastrointestinal effects and facial edema in Japanese people who ingested arsenic-contaminated soy sauce for 2 to 3 weeks (Mizuta *et al.* 1956; ATSDR 2005). An uncertainty factor of 10 for the use of a LOAEL was applied to this dose.

A chronic duration oral MRL of 0.3 µg/kg bw/d was derived based on a NOAEL of 0.8 µg/kg/d for dermal effects in a Taiwanese farming population exposed to arsenic in well water (Tseng 1977; Tseng *et al.* 1968). An uncertainty factor of 3 (for human variability) was applied.

A3.1.5 United States Environmental Protection Agency (U.S. EPA)

The U.S. EPA IRIS (1998) calculated an oral RfD of 0.3 µg As/kg bw/d based on the absence of effects on the skin at a dose of 0.8 µg/kg bw/d (Tseng *et al.* 1968; Tseng 1977). Hyper-pigmentation, keratosis and possible vascular complications were calculated to be associated with a lowest-observed-adverse-effect-level (LOAEL) of 14 µg As/kg bw/d. The RfD was based on a NOAEL of 0.8 µg As/kg bw/d, with the application of an uncertainty factor of 3 to account for lack of data on reproductive toxicity in humans, and for differences in individual sensitivity. The U.S. EPA IRIS (1998) noted some limitations of the studies, in that the exposure levels were not well characterized and other risk factors were present.

The reference doses (or TDIs/MRLs) derived based on the studies by Tseng *et al. (*1968) and Tseng (1977) are likely to be conservative because they assumed no or minimal exposure to arsenic through food. Recent assessments of human exposure to arsenic in Bangladesh indicate that when rice is a major component of the diet and is cooked in the same water used for drinking, it contributes almost as much arsenic as drinking water itself contributes (45% versus 55%; assuming 2 L of drinking water per day and 150 kg of rice per year ingested per person). If ingestion of rice is similar in Taiwan to Bangladesh, this means that the subjects in Tseng *et al.* (1968) and Tseng (1977) may have received almost double the intake of arsenic per day that was estimated based on drinking water alone. By contrast, the U.S. EPA IRIS assumed that arsenic intake from food was only 2 µg/d or 0.036 µg/kg body weight, translating to less than 5% of total intake.

In the carcinogenic assessment on U.S. EPA IRIS (1998) arsenic exposure *via* the oral route was considered to be carcinogenic to humans, based on the incidence of skin cancers in epidemiological studies examining human exposure through drinking water (Tseng *et al.* 1968; Tseng 1977). Based on the application of a multistage mathematical model to the data from these studies, they calculated an oral q_1^* of 0.0015 (µg As/kg bw/d)$^{-1}$. It was assumed that the Taiwanese individuals had a constant exposure from birth, and that males consumed 3.5 L drinking water per day and females consumed 2.0 L per day. Doses were converted to equivalent doses for U.S. males and females based on differences in body weights and differences in water consumption and it was assumed that skin cancer risk in the U.S. population would be similar to the Taiwanese population. The multistage model was used to predict dose-specific and age-specific skin cancer prevalence rates associated with ingestion of inorganic arsenic; both linear and quadratic model fitting of the data were conducted. The q_1^* of 0.0015 (µg As/kg bw/d)$^{-1}$, corresponds to an RsD of 0.0067 µg As/kg bw/d for an acceptable risk level of 1 in 100,000.

In 2001, the U.S. EPA revised the drinking water guideline for arsenic from 50 µg/L down to 10 µg/L, largely based on new estimates of carcinogenic risk from the ingestion of inorganic arsenic (U.S. EPA 2001). Their estimate of cancer risk was based on a re-analysis of the data on the SW Taiwanese cohort published by Morales *et al.* (2000). Based on the SW Taiwan data set reported by Morales *et al.* 2000 and Wu *et al.* 1989 and a linear no-threshold model, they estimated mean population cancer risks of 0.63 to 2.99 in 10,000 to be associated with exposure to 10 µg/L. Essentially negligible health risks (1 in 100,000 or less) would be associated with a water arsenic level of approximately 1 µg/L or less. This translates to a daily intake of 0.03 µg/kg/day assuming a body weight of 65 kg and a water intake of 2 L per day. Similar analyses of the SW Taiwan data set of Morales *et al.* (2000) by the U.S. NRC (2001) using different assumptions and modeling estimated a 1 in 1000 risk of developing bladder or lung cancer in ones lifetime associated with exposure to 3 µg/L inorganic arsenic in drinking water (U.S. NRC 2001). The models are highly influenced by factors such as drinking water rates, comparison populations employed and levels of arsenic assumed in food.

The U.S. EPA is also currently revising the assessment for inorganic arsenic for IRIS based on the more recent data on internal cancers associated with exposure to arsenic in drinking water in the SW Taiwan population (Morales *et al.* 2000; Wu *et al.* 1989; Chen *et al.* 1992). It has published a draft report that is currently under review by a Science Advisory Board (SAB). In it, a slope factor is recommended that corresponds to an RsD of 0.002 µg/kg/day for a 1 in 100,000 cancer risk. The first draft of the SAB comments is available online and one of the significant comments is that given its limitations, the SW Taiwan datas*et al*one is not sufficient for estimating cancer risk in humans (SAB 2005). They recommended that the U.S. EPA do an integrated risk analysis that takes into account multiple epidemiology studies, including the low dose studies that in and of themselves may have limited statistical power or insufficient data at high doses. In addition, they note that the studies should be judged by the same set of criteria, with the comparative assessment of those criteria across studies clearly laid out in tabular format. There are several precedents for formally integrating health outcome information from a number of epidemiology studies (SAB 2005). Integrative analyses improve statistical power and precision of the estimates. Exponent (2005) note that in the U.S. EPA assessments, there has been a lack of systematic discussion of studies from US and other similar populations with low level exposures to arsenic. Further, exclusion criteria applied to low-level epidemiological studies were not applied to the SW Taiwan data. No formal quantitative evaluation of validity or the impact of bias has been conducted.

The SAB also noted recent data (Chowdury *et al.* 2000; Watanabe *et al.* 2004) that suggests dietary intake from food may have been substantially underestimated for the Taiwanese population relative to intake in the U.S. Adjustment for background arsenic intake from food is extremely important because the total exposure dose is the important value in terms of toxicity and cancer induction (SAB 2005).

A3.2 Inhalation Exposure Limits

Table A-4. Summary of Inhalation Exposure Limits for Arsenic

Agency/Organization	Limit	Reference
Health Canada – Existing Substances Division	URE: 6.4 $(mg/m^3)^{-1}$	CEPA 1993
Ontario Ministry of the Environment	AAQC: 0.3 $\mu g/m^3$	OMOE 2005
United States Environmental Protection Agency	URE: 0.0043 $(\mu g/m^3)^{-1}$	U.S. EPA IRIS 1998

URE: unit risk estimate

AAQC: ambient air quality criterion

A3.2.1 Health Canada

Health Canada has not recommended an inhalation exposure limit based on non-carcinogenic endpoints. As part of its assessment of arsenic for its "Priority Substance List Assessment Report: Inorganic Arsenic" (CEPA 1993), they calculated estimates of cancer potency corresponding to a 5% increase in mortality due to respiratory cancer using three different cohorts of arsenic exposed miners. These included workers at the Tacoma copper smelter in Washington (Enterline *et al.* 1987), the Anaconda smelter in Montana (Higgins *et al.* 1986) and the Ronnskar smelter in Sweden (Jarup *et al.* 1989). These cohorts were chosen to estimate cancer potency because they reported adequate exposure information. Mathematical models were employed to estimate the cancer potency corresponding to 5% increases in lung cancer mortality (*i.e.*, TC_{05}s) from these studies. The calculated TC_{05} cancer potencies for inhaled arsenic ranged from 7.83 to 50.5 $\mu g/m^3$ (CEPA 1993). The lowest potency estimate corresponds to a unit risk for lung cancer of 6.4 $(mg/m^3)^{-1}$. This was recommended as the Inhalation Unit Risk for the assessment of arsenic in Health Canada's recent Guidelines for Federal Contaminated Site Risk Assessment in Canada (Health Canada 2004). The unit risk can be converted to an RsD of 0.0005 (µg/kg BW/day) for an acceptable risk level of 1 in 100,000 assuming a 70 kg adult breathes 23 m^3/day.

The Ontario Ministry of the Environment and Energy (OMOE 2005) recommended a 24-hour Ambient Air Quality Criterion (AAQC) of 0.3 µg As/m^3 (equivalent to 0.1 µg As/kg body weight/day assuming a 70 kg adult breathes 23 m^3/day).

A3.2.2 United States Environmental Protection Agency (U.S. EPA)

The U.S. EPA has not recommended an inhalation exposure limit based on non-carcinogenic endpoints. The U.S. EPA Integrated Risk Information System (IRIS), last updated in 1998, considers arsenic to be a non-threshold carcinogen. Based on this assumption, they calculated an inhalation q_1^* of 0.0043 (µg As/m^3)$^{-1}$, based on studies by Brown and Chu 1983a,b; Lee-Feldstein 1983; Higgins *et al*. 1986; and Enterline and Marsh 1982 which indicated increased mortality due to lung cancer in of exposed occupational populations. A geometric mean was obtained for data sets obtained with distinct exposed populations (Anaconda smelter and ASARCO smelter); the final estimate was the geometric mean of those two values (U.S. EPA IRIS 1998). It was assumed that the increase in age-specific mortality rate of lung cancer was a function only of cumulative exposures. The q_1^* was converted to an inhalation RsD of 0.0008 (µg/kg BW/day) for an acceptable risk level of 1 in 100,000 assuming a 70 kg adult breathes 23 m^3/day.

A3.3 Recommended Exposure Limits for the Risk Assessment of Arsenic

A3.3.1 Oral Exposure

Substantial uncertainties remain in the risk assessment of oral ingestion of arsenic, particularly in relation to its carcinogenic potency at low doses. Several lines of evidence summarised in this review indicate that existing exposure limits based on cancer risks are likely very conservative. Until more research and analysis is available, however, the use of a range of exposure limits is appropriate, with the lower end of the range represented by a cancer-based limit (*i.e.*, RsD) and the upper end, a threshold based TDI or RfD. It is recommended that the interim TDI currently used by the Food Directorate (1 µg/kg/day) be used as the upper end of a health-based exposure limit, with the RsD derived from Health Canada's Guidelines for Canadian Drinking Water (Health Canada 2006b) (0.006 µg/kg/day) as the lower end of the exposure limit range.

Health Canada's assessment of arsenic carcinogenicity in its 2006 drinking water quality guideline document is the most recent official regulatory assessment that examined not only skin cancer but internal cancers associated arsenic ingestion (Health Canada 2006b). Although the U.S. EPA is currently revising its assessment of oral exposure to inorganic arsenic for IRIS, this is currently under review by a Science Advisory Board and has not yet been finalized.

A3.3.2 Inhalation Exposure

Inhalation RsDs based on Health Canada's assessment of inorganic arsenic (CEPA 1993) and the U.S. EPA IRIS (1998) assessment are based on studies reporting excess mortality due to respiratory cancer among cohorts of arsenic exposed miners. The RsDs corresponding to an essentially negligible incremental risk in cancer from these assessments are 0.0005 and 0.0008 µg/kg/day, respectively (Health Canada 2004; U.S. EPA IRIS 1998). To be conservative, it is recommended that the Health Canada RsD be employed in the risk assessment of inhalation arsenic exposure. It should be kept in mind, however, that the occupational cohorts in question were exposed to high levels of arsenic in air and the RsDs were developed through a no-threshold, linear extrapolation model to estimate risks associated with much lower air concentrations. Given the evidence that arsenic causes cancer through a non-linear mechanism, the inhalation RsDs developed for inorganic arsenic are likely to be highly conservative.

As outlined in the Terms of Reference within the main report, inhalation effects were not evaluated as part of the CEI assessment. Thus, this inhalation exposure limit has not been incorporated into the cancer risk assessment in the main document and is provided here for information purposes only.

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APPENDIX B
EXPOSURE MODEL CALCULATIONS

Table B-1. Estimated Exposure and Risk Based on Drinking Water Risk-specific Dose (Health Canada 2006)

Soil Ingestion Pathway

	Inorganic Concentration [mg/kg]		Inorganic		IR	EF	ED	BW	AT	EDI (μg/kg/day)		
Life Stage	**Baseline**	**Incremental**	**Baseline**	**Incremental**	**g/day**	**days/year**	**yrs**	**kg**	**days**	**Baseline**	**Incremental**	**Combined**
Infant	1.30E+00	5.33E-03	1.30E+00	5.33E-03	0.02	182.5	0.5	8.2	182.5	0.002	0.000007	0.002
Toddler	1.30E+00	5.33E-03	1.30E+00	5.33E-03	0.08	182.5	4.5	16.5	1642.5	0.0032	0.000013	0.0032
Child	1.30E+00	5.33E-03	1.30E+00	5.33E-03	0.02	182.5	7	32.9	2555	0.00040	0.0000016	0.00040
Teen	1.30E+00	5.33E-03	1.30E+00	5.33E-03	0.02	182.5	8	59.7	2920	0.00022	0.00000089	0.00022
Adult	1.30E+00	5.33E-03	1.30E+00	5.33E-03	0.02	182.5	61	70.7	22265	0.00018	0.00000075	0.00018
Composite							81			0.00038	0.0000016	0.00038

Water Ingestion Pathway (Athabasca River Water Quality)

	Arsenic Concentration [mg/L]		Inorganic		IR	EF	ED	BW	AT	EDI (μg/kg/day)		
Life Stage	**Baseline**	**Incremental**	**Baseline**	**Incremental**	**L/day**	**days/year**	**yrs**	**kg**	**days**	**Baseline**	**Incremental**	**Combined**
Infant	1.18E-03	1.00E-04	1.18E-03	1.00E-04	0.3	182.5	0.5	8.2	182.5	0.022	0.0018	0.023
Toddler	1.18E-03	1.00E-04	1.18E-03	1.00E-04	0.6	182.5	4.5	16.5	1642.5	0.021	0.0018	0.023
Child	1.18E-03	1.00E-04	1.18E-03	1.00E-04	0.8	182.5	7	32.9	2555	0.014	0.0012	0.016
Teen	1.18E-03	1.00E-04	1.18E-03	1.00E-04	1	182.5	8	59.7	2920	0.0099	0.00084	0.011
Adult	1.18E-03	1.00E-04	1.18E-03	1.00E-04	1.5	182.5	61	70.7	22265	0.013	0.0011	0.014
Composite							81			0.013	0.0011	0.014

Water Ingestion Pathway (Ell's River Water Quality)

	Arsenic Concentration [mg/L]		Inorganic		IR	EF	ED	BW	AT	EDI (μg/kg/day)		
Life Stage	**Baseline**	**Incremental**	**Baseline**	**Incremental**	**L/day**	**days/year**	**yrs**	**kg**	**days**	**Baseline**	**Incremental**	**Combined**
Infant	1.17E-03	1.00E-04	1.17E-03	1.00E-04	0.3	182.5	0.5	8.2	182.5	0.021	0.0018	0.023
Toddler	1.17E-03	1.00E-04	1.17E-03	1.00E-04	0.6	182.5	4.5	16.5	1642.5	0.021	0.0018	0.023
Child	1.17E-03	1.00E-04	1.17E-03	1.00E-04	0.8	182.5	7	32.9	2555	0.014	0.0012	0.015
Teen	1.17E-03	1.00E-04	1.17E-03	1.00E-04	1	182.5	8	59.7	2920	0.010	0.00084	0.011
Adult	1.17E-03	1.00E-04	1.17E-03	1.00E-04	1.5	182.5	61	70.7	22265	0.012	0.0011	0.013
Composite							81			0.013	0.0011	0.014

Berry Ingestion Pathway

	Arsenic Concentration [mg/kg-ww]		Inorganic		IR	EF	ED	BW	AT	EDI (μg/kg/day)		
Life Stage	**Baseline**	**Incremental**	**Baseline**	**Incremental**	**g/day**	**days/year**	**yrs**	**kg**	**days**	**Baseline**	**Incremental**	**Combined**
Infant	2.37E-02	1.07E-03	8.78E-03	3.96E-04	3	365	0.5	8.2	182.5	0.0029	0.00013	0.0030
Toddler	2.37E-02	1.07E-03	8.78E-03	3.96E-04	5	365	4.5	16.5	1642.5	0.0029	0.00013	0.0030
Child	2.37E-02	1.07E-03	8.78E-03	3.96E-04	11	365	7	32.9	2555	0.0029	0.00013	0.0030
Teen	2.37E-02	1.07E-03	8.78E-03	3.96E-04	19	365	8	59.7	2920	0.0029	0.00013	0.0030
Adult	2.37E-02	1.07E-03	8.78E-03	3.96E-04	23	365	61	70.7	22265	0.0029	0.00013	0.0030
Composite							81			0.0029	0.00013	0.0030

37% Assumed inorganic content

Aboveground Traditional Plants Ingestion Pathway

	Arsenic Concentration [mg/kg-ww]		Inorganic		IR	EF	ED	BW	AT	EDI (μg/kg/day)		
Life Stage	**Baseline**	**Incremental**	**Baseline**	**Incremental**	**g/day**	**days/year**	**yrs**	**kg**	**days**	**Baseline**	**Incremental**	**Combined**
Infant	2.37E-02	1.07E-03	8.78E-03	3.96E-04	0.3	365	0.5	8.2	182.5	0.00037	0.000017	0.00039
Toddler	2.37E-02	1.07E-03	8.78E-03	3.96E-04	1	365	4.5	16.5	1642.5	0.00037	0.000017	0.00039
Child	2.37E-02	1.07E-03	8.78E-03	3.96E-04	1	365	7	32.9	2555	0.00037	0.000017	0.00039
Teen	2.37E-02	1.07E-03	8.78E-03	3.96E-04	3	365	8	59.7	2920	0.00037	0.000017	0.00039
Adult	2.37E-02	1.07E-03	8.78E-03	3.96E-04	3	365	61	70.7	22265	0.00037	0.000017	0.00039
Composite							81			0.00037	0.000017	0.00039

37% Assumed inorganic content

Table B-1. Estimated Exposure and Risk Based on Drinking Water Risk-specific Dose (Health Canada 2006)

Root Ingestion Pathway

	Arsenic Concentration [mg/kg-ww]		Inorganic		IR	EF	ED	BW	AT	EDI (µg/kg/day)		
Life Stage	**Baseline***	**Incremental**	**Baseline**	**Incremental**	**g/day**	**days/year**	**yrs**	**kg**	**days**	**Baseline**	**Incremental**	**Combined**
Infant	1.90E+00	2.00E-05	7.03E-01	7.40E-06	0.3	365	0.5	8.2	182.5	0.030	0.00000031	0.030
Toddler	1.90E+00	2.00E-05	7.03E-01	7.40E-06	1	365	4.5	16.5	1642.5	0.030	0.00000031	0.030
Child	1.90E+00	2.00E-05	7.03E-01	7.40E-06	1	365	7	32.9	2555	0.030	0.00000031	0.030
Teen	1.90E+00	2.00E-05	7.03E-01	7.40E-06	3	365	8	59.7	2920	0.030	0.00000031	0.030
Adult	1.90E+00	2.00E-05	7.03E-01	7.40E-06	3	365	61	70.7	22265	0.030	0.00000031	0.030
Composite							81			0.030	0.00000031	0.030
* cattail measured- AHW data			37%	Assumed inorganic content								

Moose Ingestion Pathway

	Arsenic Concentration [mg/kg-ww]		Inorganic		IR	EF	ED	BW	AT	EDI (µg/kg/day)		
Life Stage	**Baseline***	**Incremental**	**Baseline**	**Incremental**	**g/day**	**days/year**	**yrs**	**kg**	**days**	**Baseline**	**Incremental**	**Combined**
Infant	2.05E-02	1.37E-04	7.59E-03	5.06E-05	0	365	0.5	8.2	182.5	0	0	0
Toddler	2.05E-02	1.37E-04	7.59E-03	5.06E-05	25	365	4.5	16.5	1642.5	0.011	0.000076	0.011
Child	2.05E-02	1.37E-04	7.59E-03	5.06E-05	49	365	7	32.9	2555	0.011	0.000076	0.011
Teen	2.05E-02	1.37E-04	7.59E-03	5.06E-05	90	365	8	59.7	2920	0.011	0.000076	0.011
Adult	2.05E-02	1.37E-04	7.59E-03	5.06E-05	106	365	61	70.7	22265	0.011	0.000076	0.011
Composite							81			0.011	0.000075	0.011
*WBR moose muscle - AHW data			37%	Assumed inorganic content								

Fish Ingestion Pathway

	Arsenic Concentration [mg/kg-ww]		Inorganic		IR	EF	ED	BW	AT	EDI (µg/kg/day)		
Life Stage	**Baseline**	**Incremental**	**Baseline**	**Incremental**	**g/day**	**days/year**	**yrs**	**kg**	**days**	**Baseline**	**Incremental**	**Combined**
Infant	1.00E-01	1.00E-02	3.70E-02	3.70E-03	0	365	0.5	8.2	182.5	0	0	0
Toddler	1.00E-01	1.00E-02	3.70E-02	3.70E-03	22	365	4.5	16.5	1642.5	0.049	0.0049	0.054
Child	1.00E-01	1.00E-02	3.70E-02	3.70E-03	40	365	7	32.9	2555	0.045	0.0045	0.049
Teen	1.00E-01	1.00E-02	3.70E-02	3.70E-03	47	365	8	59.7	2920	0.029	0.0029	0.032
Adult	1.00E-01	1.00E-02	3.70E-02	3.70E-03	51	365	61	70.7	22265	0.027	0.0027	0.029
Composite							81			0.030	0.0030	0.033
			37%	Assumed inorganic content								

Summary HQ

	Composite Inorganic EDI (µg/kg/day)			HQ (1 in 100,000 Risk)			Pathway Contribution	
Pathway	**Baseline**	**Incremental**	**Combined**	**Baseline**	**Incremental**	**Combined**	**Baseline**	**Incremental**
Soil	0.00038	0.0000016	0.00038	0.063	0.00026	0.063	0%	0%
Water	0.026	0.0022	0.028	4.3	0.37	4.7	26%	41%
Berry	0.0029	0.00013	0.0030	0.48	0.021	0.50	3%	2%
Plants	0.00037	0.000017	0.00039	0.062	0.0028	0.065	0%	0%
Root	0.030	0.00000031	0.030	5.0	0.000052	5.0	30%	0%
Moose	0.011	0.000075	0.011	1.9	0.013	1.9	11%	1%
Fish	0.030	0.0030	0.033	4.9	0.49	5.4	30%	55%
Total	0.10	0.0054	0.11	16.7	0.90	17.6	100%	100%
RsD (µg/kg/day)	0.006							

Table B-2. Estimated Exposure and Risk Based on Contaminated Site Risk-specific Dose (Health Canada 2004)

Soil Ingestion Pathway

	Inorganic Concentration [mg/kg]		**Inorganic**		**IR**	**EF**	**ED**	**BW**	**AT**	**EDI (µg/kg/day)**		
Life Stage	**Baseline**	**Incremental**	**Baseline**	**Incremental**	**g/day**	**days/year**	**yrs**	**kg**	**days**	**Baseline**	**Incremental**	**Combined**
Infant	1.30E+00	5.33E-03	1.30E+00	5.33E-03	0.02	182.5	0.5	8.2	182.5	0.002	0.000007	0.002
Toddler	1.30E+00	5.33E-03	1.30E+00	5.33E-03	0.08	182.5	4.5	16.5	1642.5	0.0032	0.000013	0.0032
Child	1.30E+00	5.33E-03	1.30E+00	5.33E-03	0.02	182.5	7	32.9	2555	0.00040	0.0000016	0.00040
Teen	1.30E+00	5.33E-03	1.30E+00	5.33E-03	0.02	182.5	8	59.7	2920	0.00022	0.00000089	0.00022
Adult	1.30E+00	5.33E-03	1.30E+00	5.33E-03	0.02	182.5	61	70.7	22265	0.00018	0.00000075	0.00018
Composite							81			0.00038	0.0000016	0.00038

Water Ingestion Pathway (Athabasca River Water Quality)

	Arsenic Concentration [mg/L]		**Inorganic**		**IR**	**EF**	**ED**	**BW**	**AT**	**EDI (µg/kg/day)**		
Life Stage	**Baseline**	**Incremental**	**Baseline**	**Incremental**	**L/day**	**days/year**	**yrs**	**kg**	**days**	**Baseline**	**Incremental**	**Combined**
Infant	1.18E-03	1.00E-04	1.18E-03	1.00E-04	0.3	182.5	0.5	8.2	182.5	0.022	0.0018	0.023
Toddler	1.18E-03	1.00E-04	1.18E-03	1.00E-04	0.6	182.5	4.5	16.5	1642.5	0.021	0.0018	0.023
Child	1.18E-03	1.00E-04	1.18E-03	1.00E-04	0.8	182.5	7	32.9	2555	0.014	0.0012	0.016
Teen	1.18E-03	1.00E-04	1.18E-03	1.00E-04	1	182.5	8	59.7	2920	0.0099	0.00084	0.011
Adult	1.18E-03	1.00E-04	1.18E-03	1.00E-04	1.5	182.5	61	70.7	22265	0.013	0.0011	0.014
Composite							81			0.013	0.0011	0.014

Water Ingestion Pathway (Ell's River Water Quality)

	Arsenic Concentration [mg/L]		**Inorganic**		**IR**	**EF**	**ED**	**BW**	**AT**	**EDI (µg/kg/day)**		
Life Stage	**Baseline**	**Incremental**	**Baseline**	**Incremental**	**L/day**	**days/year**	**yrs**	**kg**	**days**	**Baseline**	**Incremental**	**Combined**
Infant	1.17E-03	1.00E-04	1.17E-03	1.00E-04	0.3	182.5	0.5	8.2	182.5	0.021	0.0018	0.023
Toddler	1.17E-03	1.00E-04	1.17E-03	1.00E-04	0.6	182.5	4.5	16.5	1642.5	0.021	0.0018	0.023
Child	1.17E-03	1.00E-04	1.17E-03	1.00E-04	0.8	182.5	7	32.9	2555	0.014	0.0012	0.015
Teen	1.17E-03	1.00E-04	1.17E-03	1.00E-04	1	182.5	8	59.7	2920	0.010	0.00084	0.011
Adult	1.17E-03	1.00E-04	1.17E-03	1.00E-04	1.5	182.5	61	70.7	22265	0.012	0.0011	0.013
Composite							81			0.013	0.0011	0.014

Berry Ingestion Pathway

	Arsenic Concentration [mg/kg-ww]		**Inorganic**		**IR**	**EF**	**ED**	**BW**	**AT**	**EDI (µg/kg/day)**		
Life Stage	**Baseline**	**Incremental**	**Baseline**	**Incremental**	**g/day**	**days/year**	**yrs**	**kg**	**days**	**Baseline**	**Incremental**	**Combined**
Infant	2.37E-02	1.07E-03	8.78E-03	3.96E-04	3	365	0.5	8.2	182.5	0.0029	0.00013	0.0030
Toddler	2.37E-02	1.07E-03	8.78E-03	3.96E-04	5	365	4.5	16.5	1642.5	0.0029	0.00013	0.0030
Child	2.37E-02	1.07E-03	8.78E-03	3.96E-04	11	365	7	32.9	2555	0.0029	0.00013	0.0030
Teen	2.37E-02	1.07E-03	8.78E-03	3.96E-04	19	365	8	59.7	2920	0.0029	0.00013	0.0030
Adult	2.37E-02	1.07E-03	8.78E-03	3.96E-04	23	365	61	70.7	22265	0.0029	0.00013	0.0030
Composite							81			0.0029	0.00013	0.0030

37% Assumed inorganic content

Aboveground Traditional Plants Ingestion Pathway

	Arsenic Concentration [mg/kg-ww]		**Inorganic**		**IR**	**EF**	**ED**	**BW**	**AT**	**EDI (µg/kg/day)**		
Life Stage	**Baseline**	**Incremental**	**Baseline**	**Incremental**	**g/day**	**days/year**	**yrs**	**kg**	**days**	**Baseline**	**Incremental**	**Combined**
Infant	2.37E-02	1.07E-03	8.78E-03	3.96E-04	0.3	365	0.5	8.2	182.5	0.00037	0.000017	0.00039
Toddler	2.37E-02	1.07E-03	8.78E-03	3.96E-04	1	365	4.5	16.5	1642.5	0.00037	0.000017	0.00039
Child	2.37E-02	1.07E-03	8.78E-03	3.96E-04	1	365	7	32.9	2555	0.00037	0.000017	0.00039
Teen	2.37E-02	1.07E-03	8.78E-03	3.96E-04	3	365	8	59.7	2920	0.00037	0.000017	0.00039
Adult	2.37E-02	1.07E-03	8.78E-03	3.96E-04	3	365	61	70.7	22265	0.00037	0.000017	0.00039
Composite							81			0.00037	0.000017	0.00039

37% Assumed inorganic content

Table B-2. Estimated Exposure and Risk Based on Contaminated Site Risk-specific Dose (Health Canada 2004)

Root Ingestion Pathway

	Arsenic Concentration [mg/kg-ww]		Inorganic		IR	EF	ED	BW	AT	EDI (µg/kg/day)		
Life Stage	**Baseline***	**Incremental**	**Baseline**	**Incremental**	**g/day**	**days/year**	**yrs**	**kg**	**days**	**Baseline**	**Incremental**	**Combined**
Infant	1.90E+00	2.00E-05	7.03E-01	7.40E-06	0.3	365	0.5	8.2	182.5	0.030	0.00000031	0.030
Toddler	1.90E+00	2.00E-05	7.03E-01	7.40E-06	1	365	4.5	16.5	1642.5	0.030	0.00000031	0.030
Child	1.90E+00	2.00E-05	7.03E-01	7.40E-06	1	365	7	32.9	2555	0.030	0.00000031	0.030
Teen	1.90E+00	2.00E-05	7.03E-01	7.40E-06	3	365	8	59.7	2920	0.030	0.00000031	0.030
Adult	1.90E+00	2.00E-05	7.03E-01	7.40E-06	3	365	61	70.7	22265	0.030	0.00000031	0.030
Composite							81			0.030	0.00000031	0.030
* cattail measured- AHW data			37%	Assumed inorganic content								

Moose Ingestion Pathway

	Arsenic Concentration [mg/kg-ww]		Inorganic		IR	EF	ED	BW	AT	EDI (µg/kg/day)		
Life Stage	**Baseline***	**Incremental**	**Baseline**	**Incremental**	**g/day**	**days/year**	**yrs**	**kg**	**days**	**Baseline**	**Incremental**	**Combined**
Infant	2.05E-02	1.37E-04	7.59E-03	5.06E-05	0	365	0.5	8.2	182.5	0	0	0
Toddler	2.05E-02	1.37E-04	7.59E-03	5.06E-05	25	365	4.5	16.5	1642.5	0.011	0.000076	0.011
Child	2.05E-02	1.37E-04	7.59E-03	5.06E-05	49	365	7	32.9	2555	0.011	0.000076	0.011
Teen	2.05E-02	1.37E-04	7.59E-03	5.06E-05	90	365	8	59.7	2920	0.011	0.000076	0.011
Adult	2.05E-02	1.37E-04	7.59E-03	5.06E-05	106	365	61	70.7	22265	0.011	0.000076	0.011
Composite							81			0.011	0.000075	0.011
*WBR moose muscle - AHW data			37%	Assumed inorganic content								

Fish Ingestion Pathway

	Arsenic Concentration [mg/kg-ww]		Inorganic		IR	EF	ED	BW	AT	EDI (µg/kg/day)		
Life Stage	**Baseline**	**Incremental**	**Baseline**	**Incremental**	**g/day**	**days/year**	**yrs**	**kg**	**days**	**Baseline**	**Incremental**	**Combined**
Infant	1.00E-01	1.00E-02	3.70E-02	3.70E-03	0	365	0.5	8.2	182.5	0	0	0
Toddler	1.00E-01	1.00E-02	3.70E-02	3.70E-03	22	365	4.5	16.5	1642.5	0.049	0.0049	0.054
Child	1.00E-01	1.00E-02	3.70E-02	3.70E-03	40	365	7	32.9	2555	0.045	0.0045	0.049
Teen	1.00E-01	1.00E-02	3.70E-02	3.70E-03	47	365	8	59.7	2920	0.029	0.0029	0.032
Adult	1.00E-01	1.00E-02	3.70E-02	3.70E-03	51	365	61	70.7	22265	0.027	0.0027	0.029
Composite							81			0.030	0.0030	0.033
			37%	Assumed inorganic content								

Summary HQ

	Composite Inorganic EDI (µg/kg/day)			HQ (1 in 100,000 Risk)			Pathway Contribution		
Pathway	**Baseline**	**Incremental**	**Combined**	**Baseline**	**Incremental**	**Combined**	**Baseline**	**Incremental**	**Combined**
Soil	0.00038	0.0000016	0.00038	0.13	0.00052	0.127	0.38%	0.03%	0.36%
Water	0.026	0.0022	0.028	8.6	0.73	9.3	26%	41%	27%
Berry	0.0029	0.00013	0.0030	0.95	0.043	0.99	3%	2%	3%
Plants	0.00037	0.000017	0.00039	0.12	0.0056	0.130	0.37%	0.31%	0.37%
Root	0.030	0.00000031	0.030	9.9	0.00010	9.9	30%	0.01%	28%
Moose	0.011	0.000075	0.011	3.8	0.025	3.8	11%	1%	11%
Fish	0.030	0.0030	0.033	9.9	0.99	10.9	30%	55%	31%
Total	0.10	0.0054	0.11	33	1.79	35	100%	100%	100%
RsD (µg/kg/day)	0.003								

APPENDIX C
AHW SAMPLING DATA

Sample ID	Category	Sample Species	Sample Tissue	Description	Lab Results As Mean (ug/kg ww)	Std. Dev.	n	Results (nd = 1/2 DL) As Mean (ug/kg ww)	Std. Dev.	n
SWC 1A	Outside EDM	Cattail	Whole plant with mud	stem: half green, half dry	49	2		49	2	
SWC1B	Outside EDM	Cattail	Whole plant with mud	stem: half green, half dry	939	140		939	140	
SWC1C	Outside EDM	Cattail	Whole plant with mud	stem: half green, half dry	32	1		32	1	
SWC2A	Outside EDM	Cattail	Whole plant with mud	stem: half green, half dry	833	66		833	66	
SWC2B	Outside EDM	Cattail	Whole plant with mud	stem: half green, half dry	1969	180		1969	180	
SWC2C	Outside EDM	Cattail	Whole plant with mud	stem: half green, half dry	466	25		466	25	
SWC 3A	Outside EDM	Cattail	Whole plant with mud	stem: half green, half dry	22	22		22	22	
SWC3B	Outside EDM	Cattail	Whole plant with mud	stem: half green, half dry	22	22		22	22	
SWC3C	Outside EDM	Cattail	Whole plant with mud	stem: half green, half dry	23	23		23	23	
SWC4A	Outside EDM	Cattail	Whole plant with mud	with cattail, stem: half green, half dry	4	3.8		4	4	
SWC4B	Outside EDM	Cattail	Whole plant with mud	stem: half green, half dry	5	5.1		5	5	
SWC4C	Outside EDM	Cattail	Whole plant with mud	stem: half green, half dry	14	14		14	14	
LP1A	Outside EDM	Cattail	Whole plant with mud	stem: total dry	746	116		746	116	
LP1B	Outside EDM	Cattail	Whole plant with mud	stem: total dry	188	4		188	4	
LP1C	Outside EDM	Cattail	Whole plant with mud	stem: total dry	4726	247		4726	247	
LP4A	Outside EDM	Cattail	Whole plant with mud	stem: total dry	280	1		280	1	
LP4B	Outside EDM	Cattail	Whole plant with mud	stem: total dry	523	73		523	73	
LP4C	Outside EDM	Cattail	Whole plant with mud	stem: total dry	1325	176		1325	176	
LP2	Outside EDM	Cattail	Whole plant with mud	with cattail, stem total dry	301	301		301	301	
LP3	Outside EDM	Cattail	Whole plant with mud	with cattail, stem total dry	228	228		228	228	
#1	WBR	Cattail	washed root	frozen						
#2	WBR	Cattail	washed root	frozen						
#3	WBR	Cattail	washed root	frozen						
12U0461938, UTM6331577, #3	WBR	Cattail	stem with firm core	Fresh	<1			0.5		
12U0444775, UTM6323659, #1	WBR	Cattail	whole root	Fresh	596	23		596.0		

					Lab Results			Results (nd = 1/2 DL)		
Sample ID	Category	Sample Species	Sample Tissue	Description	As Mean (ug/kg ww)	Std. Dev.	n	As Mean (ug/kg ww)	Std. Dev.	n
12U0466439, UTM6321709, #4	WBR	Cattail	stem with firm core	Fresh	2.1	0.2		2.1		
12U0468836, UTM6313564, #2	WBR	Cattail	whole root	Fresh	422	43		422.0		
12U0464384, UTM6349673, #2	WBR	Cattail	whole root	Fresh	69	2.7		69.0		
12U0488385, UTM6358757, #3	WBR	Cattail	whole root	Fresh	79	9.2		79.0		
12U0463294, UTM6340453, #1	WBR	Cattail	root	Fresh	ns			ns		
12U0471525, UTM6309725, #1	WBR	Cattail	stem with firm core	Fresh	4.1	0.7		4.1		
12U0467170, UTM6311217, #2	WBR	Cattail	stem with firm core	Fresh	8.2	0.4		8.2		
12U0458945, UTM6348825, #3	WBR	Cattail	root skin	Fresh	277	41		276.7		
12U0458945, UTM6348825, #3	WBR	Cattail	root starch	Fresh	9.6	**		9.6		
12U0452518, UTM6347782, #4	WBR	Cattail	root hair	Fresh	262	12		262.1		
12U0452518, UTM6347782, #4	WBR	Cattail	root skin	Fresh	228	13		228.1		
12U0452518, UTM6347782, #4	WBR	Cattail	root starch	Fresh	25	0.3		25.1		
12U0460019, UTM6344695, #2	WBR	Cattail	root hair	Fresh	1939	293		1938.7		
12U0460019, UTM6344695, #2	WBR	Cattail	root skin	Fresh	947	31		947.5		
12U0460019, UTM6344695, #2	WBR	Cattail	root starch	Fresh	33	0.4		33.2		
12U0461109, UTM6339896, #1	WBR	Cattail	stem with firm core	Fresh	3.6	0.1		3.6		
12U0451460, UTM6349372, #4	WBR	Cattail	stem	Fresh, dry	6.0	0.3		6.0		
12U0454067, UTM6352908, #3	WBR	Cattail	stem with firm core	Fresh, dry	3.5	0.1		3.5		
12U0457134, UTM6352864, #2	WBR	Cattail	stem with firm core	Fresh, dry	10	1.3		10.0		
12U0458878, UTM6349146, #1	WBR	Cattail	stem with firm core	Fresh, dry	8.0	0.5		8.0		
12U0466444, UTM6340475, #2	WBR	Cattail	stem with firm core	Fresh	11	0.0		11.3		
12U0463298, UTM6340452, #4	WBR	Cattail	stem	Fresh, dry	6.2	0.4		6.2		
12U0468685, UTM6340846, #1	WBR	Cattail	stem with firm core	Fresh, dry	12	0.7		11.8		
12U0464256. UTM6339149, #3	WBR	Cattail	stem with firm core	Fresh	2.1	0.3		2.1		
12U0483991, UTM6347016, #3	WBR	Cattail	root hair	Fresh	71	6.1		71.4		
12U0483991, UTM6347016, #3	WBR	Cattail	root skin	Fresh	16	1.3		16.0		
12U0483991, UTM6347016, #3	WBR	Cattail	root starch	Fresh	2.1	0.3		2.1		
12U0484058, UTM6347381, #2	WBR	Cattail	root hair	Fresh, washed	2082	169		2082.3		
12U0484058, UTM6347381, #2	WBR	Cattail	root skin	Fresh, washed	1139	13		1139.2		
12U0484058, UTM6347381, #2	WBR	Cattail	root starch	Fresh, washed	82	11		81.8		
12U0484820, UTM6351639, #1	WBR	Cattail	root hair	Fresh, washed	68	5.6		67.9		
12U0484820, UTM6351639, #1	WBR	Cattail	root skin	Fresh, washed	24	4.3		23.9		
12U0484820, UTM6351639, #1	WBR	Cattail	root starch	Fresh, washed	3.2	0.0		3.2		
12U0460372, UTM6352822, #1	WBR	Cattail	whole root	Fresh, washed	61	**		60.8		
12U0460333, UTM6354160, #2	WBR	Cattail	whole root	Fresh, washed	129	2.0		129.1		
12U0462874, UTM6364016, #3	WBR	Cattail	whole root	Fresh, washed	37	2.8		36.7		

Sample ID	Category	Sample Species	Sample Tissue	Description	Lab Results As Mean (ug/kg ww)	Std. Dev.	n	Results (nd = 1/2 DL) As Mean (ug/kg ww)	Std. Dev.	n
12U0461106, UTM6339947, #1	WBR	Cattail	root hair	Fresh, washed	2618	**		2618.1		
12U0461106, UTM6339947, #1	WBR	Cattail	root skin	Fresh, washed	791	68		790.8		
12U0461106, UTM6339947, #1	WBR	Cattail	root starch	Fresh, washed	9.6	**		9.6		
12U0489241, UTM6255291, #3	WBR	Cattail	root hair	Fresh, washed	803	79		802.7		
12U0489241, UTM6255291, #3	WBR	Cattail	root skin	Fresh, washed	346	17		345.8		
12U0489241, UTM6255291, #3	WBR	Cattail	root stach	Fresh, washed	41	3.6		41.5		
12U0460019, UTM6344699, #2	WBR	Cattail	root skin	Fresh, washed	1409	3.5		1409.0		
12U0460019, UTM6344699, #2	WBR	Cattail	root starch	Fresh, washed	31	**		30.9		
06-10487	PR-GP	Deer	Meat	Road Kill, female, adult, mule deer	<1			0.5		
06-10976	WBR	Deer	Meat	Road Kill, female, adult, white tailed deer	2.3	0.2		2.341970472		
05-27599, 01	WBR	Deer	Meat	Problem Deer, female, adult, white tailed deer	4.0	0.0		4.027826545		
05-27599, 02	WBR	Deer	Meat	Problem Deer, female, adult, white tailed deer	2.8	0.3		2.849340489		
05-27599, 03	WBR	Deer	Meat	Problem Deer, female, adult, white tailed deer	4.1	0.3		4.117378848		
06-11041	WBR	Deer	Meat	Road Kill, female, adult, white tailed deer	2.0	0.1		2.047228538		
06-11046	WBR	Deer	Meat	Road Kill, female, adult, white tailed deer	1.8	0.1		1.826903163		
#2	EDM	White tail Deer	Striploin	White tailed deer, male, frozen	<1			0.5		
#3	EDM	White tail Deer	Liver	White tailed deer, male, frozen	3.6	0.5		3.552360486		
#1	PR-GP	White tail Deer	Leg	White tailed deer, male, 3-4yrs,frozen	<1			0.5		
06-11853, WMU 519, exhibit#8	WBR	White tail Deer	muscle	White tailed deer, male, adult, frozen	14	2.2		14.03600132		
06-11853, WMU 518, exhibit#9	WBR	White tail Deer	muscle,Neck	White tailed deer, female, adult, frozen	4.8	0.2		4.768714851		
06-11853, WMU 519, exhibit#10	WBR	White tail Deer	muscle, hind Leg	Road Kill, White tailed deer, female, yearling, fresh	20	0.8		19.59158421		
06-11853, WMU 519, exhibit#11	WBR	White tail Deer	muscle, Front Leg	White tailed deer, male, adult	11	0.3		10.79217762		
06-11853, WMU 531, exhibit#16	WBR	0	0	0				0		
#1	PR-GP	Moose	Striploin	moose, male, 3-4yrs, frozen				0		
#2	EDM	Moose	Shoulder	White tailed deer, male, 2-3yrs,frozen				0		
25660	YUK	Moose	kidney	male, frozen	<1			0.5		
25660	YUK	Moose	liver	male, frozen	4.2	0.0		4.224609214		
25660	YUK	Moose	muscle	male, frozen	<1			0.5		
25708	YUK	Moose	liver	male, frozen	1.4	0.2		1.412837672		

					Lab Results			Results (nd = 1/2 DL)		
Sample ID	Category	Sample Species	Sample Tissue	Description	As Mean (ug/kg ww)	Std. Dev.	n	As Mean (ug/kg ww)	Std. Dev.	n
25708	YUK	Moose	muscle	male, frozen	1.5	0.0		1.511716969		
26452	YUK	Moose	kidney	male, frozen	4.6	0.5		4.635660737		
26452	YUK	Moose	liver	male, frozen	2.2	0.2		2.19438945		
26452	YUK	Moose	muscle	male, frozen	1.1	0.0		1.133355794		
26515	YUK	Moose	Liver	male, frozen	131	9.6		131.4209009		
26607	YUK	Moose	Liver	male, frozen	2.0	0.1		2.001655804		
26621	YUK	Moose	kidney	male, frozen	2.0	0.1		2.011297583		
26621	YUK	Moose	liver	male, frozen	2.4	0.4		2.367869503		
26622	YUK	Moose	kidney	male, frozen	6.6	0.0		6.579616147		
26622	YUK	Moose	liver	male, frozen	12	0.0		11.64053293		
26622	YUK	Moose	muscle	male, frozen	7.1	0.5		7.088718797		
26625	YUK	Moose	Liver	male, frozen	37	2.5		36.68208982		
26629	YUK	Moose	kidney	male, frozen	20	1.9		20.03125747		
26629	YUK	Moose	liver	male, frozen	44	1.3		43.97182395		
26632	YUK	Moose	liver	male, frozen	40	5.8		39.51195866		
26632	YUK	Moose	muscle	male, frozen	24	0.4		24.14088885		
OHE 984	YUK	Moose	muscle	male, frozen	7.7	0.3		7.735504488		
23486	YUK	Moose	muscle	male, frozen	18	0.6		17.63782859		
24147	YUK	Moose	muscle	male, frozen	43	1.2		43.46156558		
24161	YUK	Moose	muscle	male, frozen	18	0.2		17.58052702		
24162	YUK	Moose	muscle	male, frozen	<1			0.5		
24179	YUK	Moose	muscle	male, frozen	5.2	0.1		5.152309922		
24181	YUK	Moose	muscle	male, frozen	3.7	0.2		3.736447716		
24184	YUK	Moose	muscle	male, frozen	1.3	0.1		1.33579589		
26174	YUK	Moose	muscle	male, frozen	13	0.3		13.02905935		
26306	YUK	Moose	muscle	male, frozen	3.6	0.1		3.608286077		
26535	YUK	Moose	muscle	male, frozen	<1			0.5		
26550	YUK	Moose	muscle	male, frozen	9.3	0.1		9.323929221		
26595	YUK	Moose	muscle	male, frozen	1.2	0.1		1.197877173		
26614	YUK	Moose	muscle	male, frozen	15	1.8		14.68553618		
27234	YUK	Moose	muscle	male, frozen	2.4	0.2		2.38897178		
27239	YUK	Moose	muscle	male, frozen	4.5	0.4		4.501187312		
27240	YUK	Moose	muscle	male, frozen	<1			0.5		
91009885	WBR	Moose	Meat	Male, 4 yrs, frozen	67.81503136	3.05755		67.81503136		
91009885	WBR	Moose	kidney	Male, 4 yrs, frozen	1.463500528	0.14632		1.463500528		
06-11853, 01	WBR	Moose	Meat	Male, 5 yrs, frozen	<1			0.5		
06-11853, 2	WBR	Moose	Meat	Male, 5 yrs, frozen	5.1	0.8		5.057694364		
06-11853, 03	WBR	Moose	Meat	Male, 5 yrs, frozen	11	1.2		10.83925177		
06-11853, WMU 530, exhibit#6	WBR	Moose	Meat	Male, adult	5.9	0.4		5.868643599		
06-11853, WMU 530, exhibit#7	WBR	Moose	Meat	Male, adult	1.1	0.1		1.111913486		
06-11853, WMU 530, exhibit#12	WBR	Moose	Meat, Thigh	Female, adult, frozen	2.8	0.0		2.838073511		
06-11853, WMU 532, exhibit#13	WBR	Moose	Meat	Female, adult (5-6yrs), frozen	5.5	0.7		5.49737923		
06-11853, WMU 519, exhibit#50	WBR	Moose	Hind round Muscle	moose, male, adult	<1			0.5		
06-11853, WMU 519, exhibit#51	WBR	Moose	Hind round Muscle	moose, male, yearling	<1			0.5		

					Lab Results			Results (nd = 1/2 DL)		
Sample ID	Category	Sample Species	Sample Tissue	Description	As Mean (ug/kg ww)	Std. Dev.	n	As Mean (ug/kg ww)	Std. Dev.	n
06-11853, WMU 530, exhibit#18	WBR	Moose	Liver	moose, female, 2-3yrs, thawed	9.7	0.3		9.69866342		
06-11853, WMU 530, exhibit#15	WBR	Moose	Liver	moose, female, 2 yrs, frozen	27	0.9		26.64442877		
06-11853, WMU 530, exhibit#17	WBR	Moose	Thigh	moose, male, 3 yrs, fresh	2.1	0.1		2.074672573		
06-11853, WMU 530, 607-860-129	WBR	Moose	Thigh	moose, male, 3 yrs, frozen	6.5	0.7		6.518612038		
06-11853, WMU 532, exhibit#14	WBR	Moose	Muscle	no information provided	2.5	0.6		2.465548385		
06-11853, WMU 519, exhibit#52	WBR	Moose	Liver	roadkill, moose, female, adult, fresh	<1			0.5		
06-11853, WMU 519, exhibit#53	WBR	Moose	Neck	moose, male, 3yrs, frozen	<1			0.5		
06-11853, WMU 519, exhibit#54	WBR	Moose	Neck	moose, male, 7-8yrs, frozen	9.6	1.1		9.57128829		
06-11853, WMU 532, exhibit#55	WBR	Moose	Shoulder	moose, male, 4 yrs, frozen	3.0	0.2		2.992245934		
06-11853, WMU 532, exhibit#56	WBR	Moose	Shoulder	moose, male, 2yrs, frozen	27	0.9		27.32355912		
06-11853, exhibit#57	WBR	Moose	Back Strps	moose, male, 4yrs, frozen	4.4	0.8		4.382914641		
06-11853, WMU 530, exhibit#58	WBR	Moose	Thigh	moose, male, young, frozen	3.0	0.2		3.014596291		
06-11853, WMU 518, exhibit#59	WBR	Moose	muscle	male, frozen	38	4.0		38.42883274		
06-11853, WMU 530, exhibit#60	WBR	Moose	shoulder	moose, female, calf, fresh	1.1	0.1		1.086637019		
06-11853, WMU 529, exhibit#62	WBR	Moose	muscle	moose, male, 5yrs, frozen	<1			0.5		
06-11853, WMU 529, exhibit#61	WBR	Moose	heart	moose, male, 2-3yrs, frozen	<1			0.5		
06-11037	WBR	Moose (originally identified as deer; check sample results)	Meat	Road Kill, female, adult	1.7	0.1		1.746755738		

APPENDIX D
VALIDATION OF GAME MEAT MODEL

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APPENDIX D - VALIDATION OF GAME MEAT MODEL

D1.0 VALIDATION OF GAME MEAN MODEL

In order to validate the predictive models used to determine future game meat concentrations, the baseline assessment used measured concentrations in soil and surface water, and predicted concentrations in forage to estimate baseline moose meat concentrations for comparison. The values calculated using this model for baseline were **not** employed in the CEI risk assessment. Rather, this exercise is intended to compare the model developed by CEI for the Future Scenario with the measured analytical data to ensure that this model is sufficiently accurate in predicting tissue concentrations in game meat.

Estimates of the inorganic arsenic concentrations in game meats are predicted with a biotransfer model based on algorithms developed by the U.S. EPA (2005). For modelling purposes, moose meat was chosen as a "surrogate" to represent all types of game meat. The choice reflected the fact that: i) large game meat (*i.e.*, moose meat and venison) is typically consumed in greater quantities and on a more regular basis by indigenous people compared to other types of game meat (*i.e.*, small mammals, waterfowl, upland game birds) (Wein 1989); and, ii) large animals, such as moose, live for longer periods and are more likely to accumulate arsenic in the muscle and other edible tissues compared to smaller animals. The model accounted for exposure of the moose to arsenic *via* the following pathways:

- Consumption of above-ground plants
- Incidental ingestion of soil while foraging for plants
- Consumption of surface water

Inorganic arsenic content of above-ground forage resulting from root uptake that is consumed by game animals in the baseline scenario may be predicted using predictive models presented in Equations D-1 and D-2 below.

BCF Model

$$C_p = C_s \times BCF_s \qquad \textbf{\textit{Equation \#D-1}}$$

Where,

C_p = concentration in plant (0.052 mg/kg dry weight, total arsenic)
C_s = concentration in soil (1.3 mg/kg, as inorganic arsenic);
BCF_s = soil-to-plant bioconcentration factor (0.04)

Regression Model

$$\ln(C_p) = m\ln(C_s) + C \qquad \textbf{\textit{Equation \#D-2}}$$

Where,

C_p	=	concentration in plant (0.16 mg/kg dry weight, total arsenic)
m	=	slope of regression model (0.564 unitless);
C_s	=	concentration in soil (1.3 mg/kg);
C	=	Y-Intercept (-1.992 mg/kg)

The Regression model provides the highest prediction of total arsenic in forage plants from root uptake (0.16 mg/kg dry weight).

The total arsenic concentration in the plant from airborne deposition may be predicted using Equation #D-3 below. This calculation was based on a deposition rate of 0.00001 kg As/ha/yr, which was deemed to represent the arsenic loading rate from existing facilities in the region (Suncor 2005).

$$Pd = \frac{CF \times DR \times R_p \times \left[1.0 - \exp\left(-kp \times Tp\right)\right]}{Yp \times kp} \qquad \textbf{\textit{Equation \#D-3}}$$

Where,

Pd	=	plant tissue concentration due to deposition
DR	=	deposition rate (0.000000001 kg/m2/year)
CF	=	conversion factor (1,000,000 mg/kg)
Rp	=	intercept fraction of edible portions of plant (0.5 unitless)
kp	=	plant surface loss coefficient (18 year-1)
Tp	=	length of plant exposure to deposition per harvest of the edible portion of the ith plant group (0.12 unitless)
Yp	=	crop yield or productivity (0.24 kg DW/m2)

The total arsenic concentration in forage from surficial deposition is about 0.0001 mg/kg dry weight. By combing the total arsenic concentration from root uptake (0.16 mg/kg dry weight) with the predicted total arsenic concentration from deposition (0.00001 mg/kg dry weight), the overall concentration of total arsenic in forage is about 0.1601 mg/kg dry weight.

The arsenic concentration in game meat may be predicted using Equation D-4.

$$C_m = BTF \times MF \times \left[\left(C_p \times FIR\right) + \left(C_s \times SIR \times BA_s\right) + \left(Cw \times WIR \times \mathrm{BA}_s\,(\mathrm{w})\right)\right]$$

Equation #D-4

Where,

Cm	=	concentration of total arsenic in moose muscle (calculated to be 0.0034 mg/kg wet-weight or 3.4 g/kg wet weight)
BTF	=	bio-transfer factor (0.002 day/kg wet-weight) (U.S.EPA 2005)
MF	=	metabolism factor (Assumed to be 1.0; unitless)
Cp	=	concentration of total arsenic in plants (0.16 mg/kg dry-weight)
FIR	=	food ingestion rate for moose (9.0 kg dry weight / day; U.S.EPA 1993)
Cs	=	1.3 mg/kg dry-weight (*i.e.*, measured concentration of arsenic in soils).
SIR	=	soil ingestion rate (0.132 kg/day = 2%×FIR; Suter *et al.* 2000)
BAs	=	bioavailability of arsenic in soil (Assumed to be 1.0; unitless)

Cw = 0.0012 mg/L (*i.e.*, estimated arsenic content of surface waters, assumed to be inorganic)
WIR = water ingestion rate (24 L/day)
BAs(w)` = bioavailability of arsenic in drinking water (Assumed to be 1.0; unitless)

The calculation revealed the total arsenic content of the moose meat to be 0.0034 mg/kg wet-weight (equivalent to an inorganic arsenic content of 0.0013 mg/kg wet weight, assuming the meat contains 37% inorganic arsenic).

The method above provides an estimate of the average likeliest game meat concentration for arsenic. However, based on a study with the transfer of arsenic in sheep (Beresford *et al*. 2001), bio-transfer factors were measured to range with a 5^{th} and 95^{th} percentile of 0.0012 and 0.0038 days/kg, respectively. The central estimate was determined to be 0.0025 days/kg, which is similar to the value used in Equation #6. Substituting the 5^{th} and 95^{th} percentile values into Equation #6 provides a lower and an upper estimate in total arsenic concentrations of 0.002 to 0.0064 mg/kg wet weight, respectively. These predicted concentrations are similar in range to the measured concentrations provided in Table 19 in the main report (AHW 2006 data). Figure 1 in the main report provides a graphical comparison of the range in predicted and measured baseline game meat concentrations. The range of predicted concentrations are similar in range, as measured by the central estimate and 5^{th} and 95^{th} percentile, to the measured concentrations validating that the predictive model used in the **future** scenario should be appropriate.

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