SUDBURY AREA RISK ASSESSMENT

VOLUME II – CHAPTER 7: LIMITATIONS AND UNCERTAINTIES IN THE HUMAN HEALTH RISK ASSESSMENT

Table of Contents

Tables

This page left blank intentionally

7.0 LIMITATIONS AND UNCERTAINTIES IN THE HUMAN HEALTH RISK ASSESSMENT

7.1 Introduction

There is no prescribed "off the shelf" model or single approach to conduct a comprehensive human health risk assessment such as the current assessment developed to evaluate health risks in the GSA. As such, many decisions are made along the way that can influence the outcome of the assessment. Seemingly simple, yet critical, decisions such as, which COC to evaluate, which communities of interest, which receptor groups, *etc*., all have considerable influence on how the HHRA will progress. In addition, the quantitative, or numerical, risk assessment requires the input of large amounts of data and numerical variables. Some of these input variables can be obtained from the general published literature, while other information must be Sudbury-specific and were obtained from the various surveys conducted under the auspices of the Sudbury Soils Study. It must be realized that the goal of quantitative exposure assessment is to produce a conservative model to ensure that risks are never underestimated.

Each of the decisions and input variables contain some element of variability and uncertainty and can affect the outcome of the assessment to some degree. This leads to some amount of "uncertainty" with the final results and conclusions. Risk managers need to know the uncertainties surrounding the study conclusions so that they can make recommendations accordingly (*e.g*., ask for more experimentation or monitoring, hedge decisions away from large losses). An uncertainty analysis can pinpoint the priorities for obtaining new information, so that uncertainty can be reduced and the decision-maker can have increased confidence in the decision ultimately taken.

The traditional approach to dealing with uncertainties is to make the risk assessment conservative through the use of extreme assumptions and point estimates, and large uncertainty factors. There are, however, costs to this approach (Moore and Elliott, 1996). In regulatory programs in which worst case assumptions are the norm, expensive risk mitigation measures may be enacted for chemicals that pose little threat to human health or the environment. Conversely, in programs that rely on best-guess values or so-called reasonable conservative values, chemicals having low likelihoods of causing effects may be ignored. This would be a mistake if the effects were potentially catastrophic (*e.g.*, stratospheric ozone depletion). In some cases, this results in the need to consider information beyond those generated by the quantitative risk assessment. This "weight-of-evidence" evaluation has been utilized for arsenic and lead in the current HHRA.

This chapter discusses the topic of uncertainty analysis, and the related issue of sensitivity analysis. Uncertainty and sensitivity analysis both focus on the output of a model and are, therefore, closely related. The purposes of the two types of analyses, however, are different. An uncertainty analysis assesses the uncertainty in model outputs that derives from uncertainty (and variability) in the inputs. A sensitivity analysis assesses the contributions of the inputs to the total uncertainty in the output, and can evaluate the "leverage" a given variable may have on the overall assessment results. The general concept of uncertainty analysis is described first in this chapter, followed by a discussion of specific areas of uncertainty attached to the Sudbury HHRA.

7.2 Uncertainty Analysis

Quantitative HHRA generally involves assigning numerical values to input parameters in an appropriate exposure or risk model to obtain a quantitative estimate of risk. Numerical values are required for parameters describing contaminant concentrations in environmental media, contaminant fate and transport, human exposure and toxic response. These values may be measured, assumed, prescribed or based on published literature. Variability and uncertainty in the input parameters or risk model result in variability and uncertainty in the estimate of risk.

Uncertainty is a widely recognized aspect of HHRA, but it is often ignored in regulatory applications. In decision-making for contaminated sites, there are compelling reasons to characterize uncertainties as part of the risk assessment to avoid the mistaken impression that model results are precise and well understood (Reckhow, 1994; Finkel, 1994). A balanced discussion of conclusions and uncertainties enhance the overall credibility of the assessment.

Chao *et al.* (1994) provide an excellent example of how consideration of uncertainties about the consequences of ground level ozone can lead to a more cost-effective decision-making process. In their example, they considered uncertainties in emissions inventories, ozone formation processes, the transport of ozone and its precursors, and impacts on human health and ecological systems. They then created a decision analytic tool to assess the effects of these uncertainties in the development of an optimal abatement strategy. Their analysis showed that a flexible strategy which involves the use of less capital intensive measures initially, and which takes advantage of new information in the future, reduces the expected total costs for meeting air quality goals when compared to the inflexible strategies initially considered. Thus, uncertainty analysis helps to discriminate among management options, identifies critical information needs, and, as shown in this example, "can spur on the iterative search for new decision options that may outperform any of the initial ones offered" (Finkel, 1994).

7.2.1 Deterministic *versus* Probabilistic Modelling

Traditional deterministic methods of quantitative risk assessment use single, or "point estimate" values for input parameters and produce a single estimate of risk or hazard. While input parameters may be selected with some knowledge of their variability or uncertainty, a deterministic analysis does not normally provide any information on the variability of the resulting risk estimate. For example, although input values are often selected to represent either average or reasonable maximum exposure conditions, the location of the point estimate of risk in the context of its potential range and distribution cannot be determined directly. A discrete, or deterministic, sensitivity analysis may provide some indication of the potential range of estimated risk values, but the variability of, and hence confidence in, the risk estimate remains unknown.

Probabilistic risk assessment uses probability distributions to characterize variability and uncertainty in input parameters and produces a probability distribution of estimated exposure or risk. The exposure distribution can be directly compared to a toxicity benchmark to estimate the probability of exceedance.

In the case of the current assessment, both methodologies were evaluated to derive estimates of risk to GSA residents. However, based upon the recommendations of the International Expert Review Panel, many of the underlying probabilistic distributions used to represent key assumptions within the assessment were replaced with more standardized regulatory-endorsed deterministic (*i.e.*, single point) values. As such, the usefulness of applying a probabilistic approach to the current assessment was greatly diminished (*i.e.*, any variability left within the results of the probabilistic assessment were based upon a small number of limited distributions). As noted previously, due to these changes, the deterministic results for the reasonably maximally exposed (RME) individual were used to conservatively evaluate health risks to residents of the GSA.

7.2.2 Conservative Values and Uncertainty Factors

Uncertainty analysis makes clear what is known and what is not, a huge advantage over the use of conservative assumptions and uncertainty factors. Thus, uncertainty analysis provides an objective and transparent means of comparing assumptions, models, and data put forth by stakeholders in an environmental dispute. After understanding the uncertainties with a risk assessment, it may still be agreed that it would be prudent to be conservative. This is appropriate given that the place for applying issues such as "what is an acceptable risk?" is during the risk management stage (the stage at which societal interests are normally considered). Use of conservative assumptions and uncertainty factors in an analysis has the effect of blurring the distinction between science and decision-making, although a

significant amount of work has been done to incorporate science into the establishment of uncertainty factors. Ultimately, the task of assessors is to come up with estimates of what is likely to happen, what might happen, and what is not likely to happen, to identify possible risk management options, but not to make decisions for society. Extending an analogy by Reckhow (1994), a forecast of "it will very likely rain" when rain is highly unlikely is not helpful; rather, we would like to know the true odds and act according to our attitude toward risk. Thus, rather than bring an umbrella to work everyday, we may choose to bring it only when the probability of rain is greater than 30%. The risk assessment approach does not negate a conservative approach, but rather moves it to the more appropriate risk management stage.

7.3 General Sources of Uncertainty in Human Health Risk Assessment

Generating a list of the various sources of uncertainties that affect a human health risk assessment is the first step *en route* to conducting a successful uncertainty analysis. Such a list will help structure the analysis and ensure that major sources of uncertainty are either quantified or explicitly excluded from the study (Finkel, 1990). Uncertainty can be classified in many ways (for examples, see Finkel, 1990; Hoffman and Hammonds, 1994; Rowe, 1994; Hora, 1996; Cullen and Frey, 1999; Paté-Cornell, 2002). In this section, common sources of uncertainty in human health risk assessments of contaminated sites are described and classified according to type of uncertainty. Section 7.4 provides details on how these relate to the HHRA of the Greater Sudbury Area.

There are many sources or components of uncertainty in a typical risk assessment. In an HHRA of a contaminated site, we may be uncertain about the identity of the sub-population at highest risk of exposure, possible routes of exposure, the appropriate multimedia exposure model, ingestion rates, chemical concentration in different media, sensitivity of different age groups to the chemical of interest, importance of modifying factors (*e.g.*, diet, health), *etc.* Despite the long list of possible sources of uncertainty, they all belong to one or several of four general types of uncertainty: i) variability; ii) incertitude arising from lack of knowledge about parameter values; iii) model structure; and, iv) decision rules. For a more in-depth discussion of these types of uncertainty, see Finkel (1990).

Variability refers to the observed differences in a population or parameter attributable to true heterogeneity (Warren-Hicks and Moore, 1998; U.S. EPA, 1999). It is the result of natural random or stochastic processes and stems from, for example, environmental, lifestyle and genetic differences. Examples include variation between individuals in size (*e.g.*, height, weight), physiology (*e.g.*, metabolic rate, food intake rate), and between environments (*e.g.*, soil type, climate, chemical concentration).

Parameter uncertainty refers to our incertitude about the true values of the parameters or variables in a model (Warren-Hicks and Moore, 1998; U.S. EPA, 1999). Parameters are often estimated from laboratory, field or other studies. This type of uncertainty is introduced because the estimated value typically relies on insufficient, unreliable or partially relevant information for the parameter of interest. Several processes contribute to parameter uncertainty including measurement errors, random errors, and systematic errors (Finkel, 1990). Measurement error often arises from the imprecision of analytical devices used, for example, to quantify chemical levels in different media or measure levels of detoxifying enzymes in humans. Errors in measurement, however, are not necessarily restricted to analytical hardware. Reconstructing past releases at a contaminated site may be subject to measurement error because historical data can be faulty or ambiguous.

Random error or sampling error is a common source of incertitude in HHRA and arises when one tries to draw an inference about a quantity from a limited number of observations. For sample means, one can examine the importance of sampling error by calculating the standard deviation of sample means (Sokal and Rohlf, 1981). Sample means based on 3,000 observations will have a standard deviation only one-tenth that of means based on 30 observations. Systematic error occurs when the errors in the data are not truly random, such as might occur when the sample population is not representative of the entire population (*e.g.*, when sampling is biased towards more contaminated areas).

Systematic error, unlike random error, does not decrease with more observations and is not accounted for when calculating sample statistics (*e.g.*, mean, standard deviation). When systematic error is pervasive, sample statistics such as 95% confidence intervals can be quite misleading. For example, nearly half of the 27 measures of the speed of light measured between the years 1875 and 1958 had 95 or 99% confidence intervals that did not bracket the most accurate value available today (c = 299,792.458 km/sec) (Henrion and Fischhoff, 1986).

In risk assessment, mathematical models are used to determine which variables to measure, specify how they relate, and to estimate the values of variables which cannot be measured directly. Model uncertainty is a serious challenge in risk assessment (Finkel, 1990; Reckhow, 1994). For example, different dose-response models commonly lead to two-fold or more differences in estimated low toxic effects doses (*e.g.*, ED5 or LD10), even when the list of models is restricted to those that fit the data equally well and are theoretically plausible (Moore and Caux, 1997). In cancer risk assessment, model uncertainty is further exacerbated by the need to extrapolate to very low levels of effect. Cothern *et al*. (1986) observed that a concentration of 50 µg/L trichloroethylene in drinking water provides a risk estimate of 1×10^{-2}

with a Weibull dose-response model, and 1 x 10^{-10} with a probit model. These estimates provide a range of uncertainty "equivalent to not knowing whether one has enough money to buy a cup of coffee or pay off the national debt" (Cothern *et al.,* 1986). This example illustrates the difficulty of choosing an appropriate model equation even with a simple system, one medium, one species, and constant laboratory conditions.

The problem of model uncertainty is likely to be much more serious with complex models such as regional-scale fate and transport models. Most applications of uncertainty analysis in human health risk assessment do not propagate uncertainties associated with model structure; rather the model structure is assumed reasonable and only parameter uncertainties are propagated. Beck (1987), Reckhow (1994), Oreskes *et al*. (1994) and others discuss the issue of model uncertainty and describe the process for selecting, evaluating, calibrating and validating models that, if followed, can substantially reduce this source of uncertainty in a risk assessment.

Decision rule uncertainty comes into play during risk management (*i.e*., after a risk estimate has been generated). This type of uncertainty arises when social objectives, economic costs, value judgements, *etc*. are part of the decision-making process for deciding on what actions to take to remediate a problem. Individual decision makers are likely to be uncertain about how to best represent the complex preferences of their constituents. Such uncertainty can be quantified by collection of empirical data (*e.g*., opinion polls) and formally treated *via* decision analysis, but rarely is. Even with the availability of formal analytical tools, controversial judgments remain about how to value life, distribute costs, benefits and risks among individuals and groups, and deciding whether to reduce risks now or some time in the future (Finkel, 1990).

7.4 Uncertainties in the Sudbury HHRA

When assumptions are made during the risk assessment process, either because of data gaps or knowledge gaps, each assumption results in some degree of uncertainty in the overall conclusions of the assessment. To understand the uncertainties within the HHRA and to ensure that the impact of these uncertainties is understood, it is important to document and characterize each of these. To ensure that the risk assessment does not underestimate the potential for the occurrence of adverse effects, it is necessary to make assumptions which are conservative. In other words, assumptions should be made that tend to overestimate exposure, toxicity and risk, rather than underestimate these parameters.

The following sections describe areas of uncertainty within the current risk assessment, and discuss the potential impacts of these uncertainties on the conclusions drawn from the assessment. Given the tendency for the assumptions used in this HHRA to overestimate both exposure and toxicity, it is considered extremely unlikely that the overall risk characterization resulted in underestimated potential health risks.

The following discussion identifies uncertainties in the exposure assessment (section 7.4.1) followed by uncertainties in the toxicological assessment and endpoints (section 7.4.2). Specific areas of uncertainty are further discussed in Sections 7.4.3 and 7.4.4.

7.4.1 Uncertainties in the Exposure Assessment

The following section outlines a number of the key uncertainties related to the exposure assessment portion of the HHRA.

Area-Wide Risk Assessment approach versus Site-Specific Approach

It was discussed earlier in this report that no specific regulatory guidance exists in Canada for undertaking an area-wide risk assessment of this scope. However, the process followed for the Sudbury HHRA embraces the basic principles used in site-specific risk assessments (SSRAs) and area-wide risk assessments (AWRAs) conducted in Ontario and elsewhere in Canada. In addition, the Sudbury Technical Committee is comprised of stakeholders knowledgeable in local Sudbury issues, health issues and risk assessment. This provides considerable confidence that the process followed by this area-wide risk assessment and the issues addressed were very appropriate.

Foundation of the HHRA is data generated from the 2001 Study. This study was conducted prior to the involvement of the SARA Group in the HHRA.

The study design was rigorous and encompassed over 8,000 soil samples, and involved a significant degree of QA/QC by numerous stakeholders in the study.

The parameters measured were chosen by scientists from the MOE, Laurentian University and other study stakeholders.

Sampling was conducted throughout the GSA. It was assumed that the data supplied for the risk assessment were representative of concentrations in Sudbury, although it is possible that the methodologies employed might over- or underestimate the site conditions.

It is important to note that only approximately 10% of the residential properties were sampled. As such, it is possible that local areas of higher soil COC concentrations were not captured in the 2001 survey.

Projected chemical concentrations in media used in the exposure modeling were assumed to remain unchanged over time.

Due to the continuing rate of decrease in smelter emissions arising from ongoing efforts by the companies and improving technologies, media concentrations of the chemicals of concern (COC) within the GSA should continue to decrease over time. Therefore, it is expected that the use of existing conditions would be conservative and may actually overestimate future exposure levels. No attempt was made to predict future levels based on current emission rates and characteristics. Metal refining has been on on-going in the Sudbury Basin for more than 100 years. That, coupled with the knowledge that emission have decreased dramatically in recent years and increased stack heights have resulted in wider regional dispersion patterns, has lead to the assumption that GSA soil levels have reached steady-state and will not increase in the future.

Chemical concentrations reported at "below detection level"

There is some uncertainty regarding the "actual" concentration of a chemical for which laboratory analysis indicates a concentration below detection. Theoretically, the value of that concentration could be any value between zero and the detection limit. While the most conservative value to use in the assessment would be the detection limit itself, it was considered to be realistically conservative to employ a value of one-half the detection limit when the exposure concentration was listed as below the level of detection. Depending on the "true" value, this may actually over- or under-estimate the evaluated chemical concentration.

Use of outdoor air concentrations to represent indoor levels

The ambient air monitoring program provides a robust data set, with data collected over an extended time period (one year); the assumption that indoor concentrations are equivalent to outdoor PM_{10} concentrations is considered very conservative based other studies which typically indicate lower indoor air levels as compared to outdoor levels (refer to the discussion in Section 2.1.5). Therefore, this approach would tend to over-predict exposure and potential risk.

Selection of appropriate soil ingestion rate (SIR)

There is some uncertainty and minor controversy regarding the selection of an appropriate soil ingestion rate (SIR) for children in the current assessment. The SARA Group selected a value of 80 mg/day for children recommended in the Federal guidance on human health preliminary quantitative risk assessment (PQRA) recently published by Health Canada (2004). However, the Ontario MOE (and the U.S. EPA) recommends the use of an SIR of 100 mg/day for children, and have incorporated this value into their guidance and regulations for the province of Ontario. Both values are rooted in a similar dataset of soil tracer studies in children, and largely differ due to differing statistical analyses and methodologies used in the development of the SIR. Neither value is incorrect, and both involve appropriate interpretations of the underlying scientific data. The SARA Group ultimately selected the Health Canada regulatory recommended value as it was based upon a more recent evaluation of the scientific literature. However, it is important to note that the conclusions and recommendations of the current assessment would not have changed had the slightly more conservative SIR of 100 mg/day been used in the Sudbury HHRA.

Treatment of replicate samples

Where duplicate soil, or other media, samples were analyzed, the geometric mean of the two samples was used, as opposed to the arithmetic mean or the highest value. This subject is discussed in detail below in Section 7.4.5.

Potential impacts of lead leaching from water distribution pipes

Use of provincial drinking water data based upon distribution system samples could potentially underestimate the concentrations of lead in community drinking water (*i.e.*, the potential for additional lead to leach out of older distribution pipes while *en route* to Sudbury residences, as well as fixtures and fittings within the home itself). This could be addressed through the use of a tap water survey (similar to that conducted for the well water and lake survey). However, results of the exposure assessment modelling for lead indicate that drinking water is not a significant pathway for exposure, compared to other dominant pathways such as market basket exposures.

Indoor dust concentrations (soil to dust ratio)

The concentrations of COC in indoor dust were measured in approximately 90 homes in the study area. Since the levels of COC in outdoor soil were significantly correlated to indoor dust concentrations, the relationship between the two was used to predict indoor dust levels for the wide range of soil concentrations observed in the Sudbury area (see also Appendix M for a full discussion of this issue).

Use of a non-linear model to calculate to calculate soil-to-dust regression relationships

Some concern has been raised with respect to the use of a non-linear model, rather than alternate linear models, to calculate the soil-to-dust regression relationships, as part of the indoor dust survey. The justification for using the natural-log (*ln*) transformed data was provided in Appendix D of the Indoor Dust Survey Report. Visual examination of residuals indicated that linear regressions using the raw data resulted in a violation of at least one of the classical assumptions. A transformation of the raw data is common place and can, in some circumstances, help correct non-normality, nonlinearity and/or lack of homeostatic variances. It is noted that slight deviations from normality are not considered critical; however, the residuals appeared to significantly deviate from normality and for some COC appeared to violate the homogeneity of variance assumption. It was therefore decided to conduct the linear regression analysis on the natural-log transformed data. While this approach does introduce some uncertainty, it was considered consistent with previous work conducted by the various regional U.S. EPA districts, and provides a more robust approach (as illustrated in the dust report) over the use of a concentration ratio (CR) approach.

Sudbury-specific soil-to-dust regression relationship used to calculated TOR indoor dust levels

To be consistent on comparison points within the assessment, the soil-to-dust regression relationship developed as part of the Sudbury Indoor Dust Survey was also used to calculate indoor residential dust levels for the Typical Ontario Resident. While there is a considerable degree of uncertainty in using Sudbury-specific values to represent a generic Ontario soil-to-dust concentration relationship, the survey conducted in Sudbury evaluated residences in obviously industry-impacted areas (*i.e.*, closer to an existing or historic smelter site, and higher soil concentrations), as well as in non-impacted areas (*i.e.*, Hanmer and other outlying areas with very low soil concentrations). The full range of the calculated regression equation accounts for this full range of exposure condition possibilities (*e.g.*, low soil concentrations and higher indoor dust concentrations typically observed in urban non-industrial areas *versus* high soil and lower dust concentrations observed in industrial-impacted areas), and should roughly provide a reasonable microcosm representing the range of potential outdoor soil *versus* indoor dust residential conditions occurring throughout the Province.

Food consumption patterns of Sudbury residents

It was assumed, for the most part, that the typical Sudbury resident has similar eating habitats to other Canadians. To test, and support, this assumption, a food consumption survey (see Appendix K) of Sudbury was undertaken. In addition, separate exposure model runs were conducted for certain known eating preferences for some GSA residents (*i.e.*, blueberry consumption, consumption of wild game and fish by anglers, hunters and members of the First Nation communities).

The levels of COC in Mother's milk

No published methodology for consideration of mother's milk exposures to inorganic compounds is available. However, maternal transfer exposures are considered by the IEUBK model for lead. As the difference between formula exposures and those from mother's milk would be very small, the potential contribution of COC in mother's milk was not considered. A comparison of these two potential sources for infants is presented in Table 2.11 in Chapter 2.

The levels of COC in home grown produce

The concentrations of COC were measured in produce obtained from approximately 70 residential gardens, and 10 local commercial operations (see Appendix E). A variety of produce types were analyzed from gardens with a wide range of soil conditions and COC concentrations. As a result, there is a great deal of confidence that the data are representative of Sudbury produce and helps to reduce the uncertainty with the exposure assessment.

It should be noted that during the Vegetable Garden Survey, the Vale Inco Copper Cliff facility was shut down because of a two-month strike between May 23rd and September 4th, 2003. During this time, due to the facility shutdown, no additional atmospheric inputs from the Vale Inco stack were released. While this certainly would have an impact on COC deposition during this time period, the purpose of the Vegetable Garden Survey was to determine uptake from the soil and not to measure new atmospheric inputs. This shutdown would not have altered the levels of metals analyzed during the Vegetable Garden Survey because below ground vegetables (*i.e.,* potatoes) would not be affected by direct deposition, and all above ground produce was washed or peeled, depending upon variety and convention. With this in mind it could be construed that the shut down actually decreased the uncertainty related to the results of the survey. The metal levels analyzed in the produce collected during this survey were reflecting direct uptake from the soil and not any additional atmospheric inputs.

Levels of COC in local fish tissue

Due to the prevalence of local lakes and popularity of sport fishing, the consumption of local fish was considered a potentially important pathway to be evaluated for the HHRA. Therefore, COC levels were measured in common sport fish species from eight local lakes (see Appendix G). These data were used in the exposure assessment to provide Sudbury-specific input variables. A variety of lakes were sampled, with robust sample sizes of each species for analysis. Some of the lakes sampled were very close to the active smelters and sources of emissions. Therefore, the study team is confident that the data reflects some of the "worst case" exposure conditions, resulting in conservative estimates of risk.

Level of COC in local wildlife (game)

COC concentrations within local wildlife were modelled as part of the ecological risk assessment.

Concentrations were predicted for moose meat only, as moose were found to have higher predicted body burdens than other types of wildlife (primarily due to the moose's higher consumption rate for forage and aquatic plants).

Concentrations were modelled for ERA Zone 2, which encompasses much of the urban region within the GSA. This is considered conservative give that most hunting occurs in more remote locations, which are typically further removed from the emission sources.

Level of COC in Consumer Products

Background concentrations of the COC in consumer products were not evaluated in the current assessment. A detailed literature review was conducted to determine whether this potential route of exposure would be significant. However, this review failed to provided a quantitative value of the contribution of consumer products to total daily exposure for the study COC. While some of the COC are found in several consumer products (*e.g.*, lead in some hair dye and cosmetics; cobalt and nickel in cleaning products and cosmetics, such as eye shadow), the relative concentrations are minor compared to exposure contributions arising from other pathways, such as oral ingestion of food and water.

Level of COC in Cigarettes

Similar to mammals, plants require essential minerals to survive. Through evolution, they have adapted the ability to acquire these nutrients directly from soil. *Nicotiana tabacum* (tobacco) is known to be used effectively in biotechnology for the removal of metals from contaminated soils (Bernhard *et al*., 2005).

Subsequently, when tobacco is dried and processed for cigarettes, it may potentially become a source of daily metal intakes for regular smokers. It has been reported that arsenic, copper, nickel, lead, and selenium can be found in either tobacco, cigarette paper, filters and/or cigarette smoke (Bernhard *et al.*, 2005; Arista, 2003a). Arista Laboratories conducted a study to determine metals' yields in cigarette smoke from 25 brands of cigarettes (Arista, 2003a). All data obtained from their study has been validated and compared to historical values (Arista, 2003b). The findings from this study relating to the COC are summarized in Table 7.1.

Table 7.1 Metal Content in Cigarettes

Chemicals from Cigarette smoke [a]	Metal Content (ng/cigarette)[b]	Standard Deviation (ng/cigarette)	Detection limit (ng/cigarette)	Limit of Quantitation (ng/cigarette)
Selenium	1.2	0.2	0.9	2.3
Arsenic	3.6	0.4	1.0	2.7
Nickel	<detection limit	Na	1.8	4.7
Lead	13.6	1.4	0.7	2.0
Cobalt	Not reported	Not reported	Not reported	Not reported

[a] Adapted from Arista, 2003b.
[b] values attained from automated machinery

Bernhard *et al*. (2004) conducted a critical review on metals in cigarette smoke in 2005. Their results pertaining to the COC are summarized in Table 7.2

Table 7.2 Metal Content in Tobacco

Chemicals from Tobacco [a]	Metal Content (µg/g tobacco)	Serum Concentration [b] of Smokers > 10 cigarettes/day	Serum Concentration of Non Smoker
Copper	156	1.31mg/L	Not reported
Nickel	0.64-1.15 0.078-5 µg/cigarette	Not a significant source	Not reported
Cobalt	Not reported	Not reported	Not reported
Selenium	Not reported	Not reported	Not reported
Lead	1.2 µg/cigarette	143µg/l	101µg/l

[a] Adapted from Bernhard *et al*., 2005
[b] Serum metal concentrations including background serum metal concentrations

From the studies above, it was shown that cigarettes do contribute some arsenic, lead, selenium, and copper to the diet of regular smokers. The daily contribution of metals from cigarettes was further evaluated in an Austrian study conducted by Wolfsperger *et al*. (1994). This study showed that higher levels of cobalt, lead, and nickel were found in the hair of cigarette smokers when compared to their non smoking counterparts. The findings of their study relating to COC are summarized in Table 7.3.

Table 7.3 Metal Content in hair of Cigarette Smokers vs. Non Smokers

Chemicals from Cigarette Smoke [a]	Metal Content in Hair (µg/g hair) (P < 0.05)	
	Cigarette Smokers	Non Smokers
Copper	Not Reported	Not Reported
Nickel	0.64	0.32
Cobalt	0.025	0.010
Selenium	Not Reported	Not Reported
Lead	3.42	1.47

[a] Adapted from Wolfsperger *et al*., 1994

Health Canada recently issued a statement that smoking cigarette may contribute to an additional 0.01 to 0.04 µg/kg bw/day of Arsenic exposure (Health Canada, 2006). Similarly, the Nickel Institute (1997) also concluded that tobacco smoking may be a source of nickel exposure. They cite a study that suggested smoking a pack of 20 cigarettes per day can contribute up to 0.004 mg Ni/day (Grandjean, 1984). Furthermore, the Nickel Institute also issued a statement indicating that smoking cigarettes with nickel contaminated hands may significantly increase the potential for oral nickel exposures. Other researchers have shown that 0.04 to 0.58 µg of nickel is released with the mainstream smoke of one cigarette (WHO, 1991). Smoking 40 cigarettes per day may thus lead to inhalation of 2–23 µg of nickel (WHO, 2000). As for selenium, Olson and Frost (1970) found an average of 0.08 mg selenium/kg (range 0.03 - 0.13 mg/kg) in a variety of cigarette tobaccos. If it is assumed that a cigarette contains 1 g tobacco and that all the selenium in tobacco is volatilized and inhaled during smoking, it can be calculated that a person smoking one pack of 20 cigarettes per day would inhale an average of 1.6 µg from this source (WHO, 1987).

While the data indicates that smoking cigarettes appears to be a potential additional source of some of the COC, the degree of contribution would be highly dependent on the number of cigarettes smoked per day, and the conditions under which they are consumed. As such, the potential contribution from cigarette smoke to COC body burden could not be accurately quantified in the current HHRA, but does add an additional degree of uncertainty for those individuals who are smokers (or are routinely exposed to second-hand smoke).

The resuspended dust pathway was not considered

U.S. EPA recommends that inhalation of resuspended dust be evaluated only if site-specific exposure setting characteristics indicate that this is potentially a significant pathway. This could potentially be the case in areas of tailing or slag piles. However, the air monitoring program was designed to capture dust-

borne contaminants originating from the tailing and or slag piles, and this data was used for the evaluation of indoor air exposures. This assumption is further substantiated by the data provided in Section 2.1.5.

A site-specific bioaccessibility study was conducted as part of the HHRA

Though less uncertainty than assuming 100% bioaccessibility or using non-site-specific literature-based values, the use of bioaccessibility studies within HHRA is an emerging area that introduces several elements of uncertainty into the assessment.

There is no accepted method for conducting a study of this nature and as such professional judgment was used in the development of the methods and the interpretation of results; methodological changes are emerging in the literature on an ongoing basis; and, the methods have not been validated for all COC. The methods and results of this study are further discussed elsewhere.

The purpose of the bioaccessibility study must be kept in context. The purpose of the study was to estimate the relative difference in bioaccessibility between metals in soil and dust from the GSA, and those used in the toxicological studies used to derive the TRVs utilized in the HHRA. The study was NOT intended to measure the absolute bioavailability of metals in soil and dust from the GSA.

Nickel species-specific fingerprints in ambient air

A variety of metal speciation techniques were used to assist in the development of species-specific "fingerprints" to represent typical ambient exposures to the various forms of nickel in air, as well as for instances where air quality has been impacted by fugitive dusts arising from the Vale Inco Copper Cliff facility. A weight-of-evidence approach was used to develop a fingerprint incorporating relative percentages of the various nickel species. While there is potential variability in particulate sources in ambient air which may have implications on the corresponding nickel species observed, an effort was made to err on the side of conservatism in selecting an upper-end estimate percentage value for the more toxicologically relevant nickel species observed (*i.e.*, nickel subsulphide and nickel oxide).

Use of weight-of-evidence approach to reduce overall speciation uncertainties

Each of the speciation techniques used to develop an overall species fingerprint for the COC have their individual distinct advantages and disadvantages (as outlined in Appendix I). However, when one uses a weight-of-evidence approach, incorporating a variety of different speciation analytical techniques, to develop an overall picture of the COC species present within the GSA, it is hoped that this will provide greater confidence in the study conclusions. Nonetheless, many of the speciation techniques are still in

their infancy with respect to their application for environmental forensics, and as such there is inherent uncertainty in their use, and risk managers should be aware of the limitations each of these techniques may have.

Geographical extent of fugitive dust impacts from the Vale Inco Copper Cliff Facility

Fugitive dusts from the Vale Inco Copper Cliff facility were detected at the Sudbury Centre West monitoring station. This station is located very close to the Vale Inco Copper Cliff facility, and does not provide insight to the geographical extent to which fugitive dusts may be blown from the facility property into the Sudbury Centre COI. It is likely, given the nature of the particulate, that this is a localized impact. However, there is no quantitative data available at this time to confirm this assumption. While Vale Inco has indicated that they are aggressively pursuing avenues to reduce fugitive dusts from their property, it may be useful to conduct additional air monitoring in Sudbury Centre at locations further east of the Sudbury Centre West station to obtain a better delineation of the extent of aerial particulate impacts.

Use of biomonitoring as part of the HHRA

The current HHRA should not be considered a health study in which biomonitoring is used to predict potential health risks within the GSA population. Biomonitoring is often viewed as the gold standard for exposure assessment, and ultimately risk management. However, there are many pros and cons to the use of biomonitoring in such health studies. One of the largest challenges is how one interprets the results of biomonitoring work. One needs an accurate benchmark by which to compare measured biomonitoring data. Some biomonitoring data is easier to collect than others (*e.g.*, breast milk, urine, and hair *versus* blood or adipose tissue). Due to ethical reasons, one cannot typically conduct such biomonitoring activity without a clear demonstration of potential risk within the community under study. However, should the HHRA demonstrate potential risk to a particular receptor group due to exposures to one of the COC, one of the recommendations for follow up work could be the gathering of biomonitoring data to test the assessment results.

For the current HHRA, the Sudbury Soils Study benefited from having urinary arsenic data collected as part of a separate study in the Town of Falconbridge (see Appendix N), which could be used in the weight-of-evidence evaluation of arsenic risks throughout the GSA. Potential risks related to exposures to lead were also evaluated with the U.S. EPA IEUBK model, which uses physiological-based pharmacokinetic (PBPK) modelling techniques to predict blood lead concentrations in children exposed

to lead from a variety of environmental media. This model has been validated using biomonitoring data, and provides an additional line of evidence for the evaluation of lead risks to GSA residents for the current study (see Chapter 5 for a discussion of the results of the IEUBK modelling conducted for the current study).

Use of Ontario Typical Range (OTR98) soil concentrations to estimate risks to Typical Ontario Resident

As recommended by U.S. EPA (1999) guidance, risks for all potential scenarios (both CTE and RME) were calculated based upon 95UCLM soil concentrations. In the case of the Typical Ontario Resident (TOR), the raw data are not available on which to calculate the 95UCLM. As such, to evaluate risks to the TOR receptor, soil concentrations representing the MOE's OTR98 were used in the current assessment for both CTE and RME scenarios. The OTR98 statistic represents the 98th percentile of soil concentrations in Ontario Typical Range (*i.e.*, from soil surveys taken from across the province), and is used as the basis of the MOE Table 1 full depth background site condition standards (Province of Ontario, 2004). The MOE considers the OTR98 as a level, which if exceeded, prompts further investigation on a case-by-case basis to determine the significance, if any, of the above normal concentration. The OTR_{98} value is shown with other statistics for each COC in Table 7.4 for comparison.

Table 7.4 Ontario Typical Range Statistics for TOR Receptor (MOE, 2007)

Statistic	Ontario Typical Range Data (µg/g)					
	Arsenic	**Cobalt**	**Copper**	**Lead**	**Nickel**	**Selenium**
n	60	60	59	60	60	60
50^{th} percentile	3.5	6.5	16.2	28.9	36	0.39
Average	5.7	7.6	25.9	51.5	45	0.45
OTR_{98} (98^{th} percentile)	17	17	65	98	32	1.3

Use of the OTR_{98} soil concentration statistic in the Typical Ontario Resident scenarios likely results in a conservative estimation of risks to the TOR receptors, when compared to the GSA-based scenarios using 95UCLM soil concentrations for the various COI. However, given this statistic is used by the MOE as a province specific background standard, it would seem to be a reasonable upper-end value to use for the Typical Ontario Resident exposure in this assessment.

Other key assumptions and related uncertainties include:

Receptors and their characteristics were selected in an attempt to purposely overestimate potential exposures (*e.g.*, it was assumed that the residential receptor, such as the female preschool child, would spend 100% of her time in the GSA while consuming significant amounts of food from the local area).

The residential receptor was assumed to be present in the GSA for 24 hours/day, 7 days/week, 52 weeks/year for their entire lifetime.

Uncertainty in the estimation of exposure in risk assessment is generally related to a lack of specific knowledge about the site itself, the receptors of concern, or the scenarios in which those receptors may be exposed. In order to address these data gaps, data from the literature was employed as a basis for scientific judgment of values which would represent the realistic exposures. This approach was used in cases where data were lacking.

Transplacental transfer of COC was not considered in the assessment. While it is likely that some *in utero* exposure does occur, no method of assessment for this exposure was identified in the literature. As such, this pathway was not considered.

The individual variability in physiological and behavioural parameters may be a source of uncertainty in risk assessment. Where site-specific data were lacking, receptors and their characteristics were selected in an attempt to purposely overestimate potential exposures. An example of this might be soil ingestion by children; while there were no site-specific data describing soil ingestion, or activities leading to soil ingestion, data from various literature sources such as the U.S. EPA were employed. These data were considered comprehensive and conservative; as they were based on fecal soil content, soil and dust ingestion from all sources was included, and it is unlikely that this value would underestimate typical soil ingestion.

Pica children were not singled out as unique receptor groups, nor were these unique behaviours specifically assessed in the HHRA. If a child is known to exhibit pica behaviour, then special attention is generally paid to the child's activities. As a result, it is expected that pica related exposures, while likely in some instances, will only occur on short-term, intermittent occasions. Further discussion on children exhibiting Pica behaviours is provided in Chapter 6.

7.4.2 Uncertainties in the Hazard Assessment

The following assumptions were used in development of toxicological criteria for the COC, all of which contribute to the uncertainties inherent in the HHRA.

Animal models are used as surrogates for humans in the development of TRVs, thereby introducing uncertainties into the risk factors due to the interspecies variability in sensitivity.

For genotoxic carcinogens, it was assumed that no repair of genetic lesions occurs, and therefore, no threshold can exist for chemicals that produce self-replicating lesions. However, the existence of enzymes that routinely repair damage to DNA is well documented in the scientific literature, and the potential adverse effects arising from damage to DNA is only observed if the ability of these repair enzymes to "fix" the damage is exceeded.

In the derivation of limits by regulatory agencies, large uncertainty factors (*i.e.*, 100-fold or greater) were used in the estimation of the reference dose (RfD) for threshold-type chemicals. These uncertainty factors were applied to exposure levels from studies where no adverse effects are observed (*i.e.*, to the NOAEL). Thus, exceeding the toxicological criterion does not mean that adverse effects would occur. Exposures greater than the calculated toxicological criterion may also be without risk (*i.e.*, below the threshold for adverse effects in humans), but this could not be, or was not, determined by the agency which derived the toxicological criterion. Humans were assumed to be the most sensitive species with respect to toxic effects of chemical. However, for obvious reasons, toxicity assays are not generally conducted on humans, so toxicological data from the most sensitive laboratory species were used in the estimation of toxicological criteria for humans.

Different age and gender categories were used as part of the exposure and hazard assessment components of the risk assessment to permit the evaluation of potential risks to sensitive subcategories (such as the female preschool child). As specific toxicity data is typically not available for specific life-stages or genders, this adds an additional layer of uncertainty to the results. In fact, the results of the assessment may distinguish a difference between genders or life-stages which can not be validated based upon existing toxicity data for most chemicals. However, it is considered a conservative approach to use chronic lifetime risk reference values with less-than-lifetime exposures.

The most sensitive toxicological endpoint (for example decreased growth, body weight loss/gain, reproductive effects) was selected for each chemical from the available scientific literature to represent the exposure limit.

TRVs, because of their inherent conservatism, are widely considered protective of sensitive subgroups and lifestages. However, risk assessment, and TRV's and environmental quality guidelines for that matter, can only protect most of the people, most of the time. There can always be those individuals that are hypersensitive, and those situations require special consideration. But, risk assessments do not investigate these situations unless there is clear evidence that such a situation exists in the study area. There is no such evidence of this in the Greater Sudbury Area.

Chemical specific uncertainties are discussed in the individual chemical toxicological profiles (Appendix A).

In the case of arsenic, there is agreement in the published literature that the methods used to estimate the oral toxic potency of arsenic based on exposures of Taiwanese populations to arsenic in drinking water would significantly overestimate cancer risks at lower levels of exposures, such as that experienced by the general North American population. The use of such data would thus result in an overestimation of cancer risk for the populations of Sudbury and Ontario.

In addition, the basis for the inhalation cancer potency factor for arsenic was an air concentration derived from occupational epidemiological studies. It has been suggested that because exposures to airborne arsenic would be mediated by inhalation of particulate matter, and since a higher proportion of particulate matter would be respirable in occupational settings as compared to environmental exposures, the inhalation potency of arsenic is likely overestimated for exposures associated with environmental contamination.

Only Seilkop (2004) provides species specific cancer IURs for airborne nickel; as such, these IURs have been utilized in this assessment as part of the weight-of-evidence approach to evaluating nickel inhalation risks. The following uncertainties regarding these IURs have been identified:

- These values have not been derived or endorsed by regulatory agencies; although MOE has acknowledged that these IUR have been under consideration by MOE and have been the topic of discussion on several instances. These include meetings and presentations by Jacques Whitford Environmental Ltd. (JWEL) and the SARA Group on the Port Colborne CBRA HHRA and the Sudbury HHRA, respectively. Presentations on this topic occurred June 23, 2003, February 10, 2004 (Seilkop's presentation attended by both JWEL and SARA consultants), June 16, 2004 and a further technical discussion with MOE at JWEL's office (August 10, 2005). This approach was also discussed at the SARA technical workshop (December 7, 2005). JWEL cites their version of the

Seilkop and Oller approach in the 2005 public review draft of the Port Colborne CBRA HHRA (and an earlier draft released and then withdrawn in 2003). As the MOE does not have an official mechanism to provide comments on this document prior to its final draft, comments on the current draft CBRA HHRA are still pending. SARA has presented the Seilkop 2004 approach in the October, 2005 draft of the SARA HHRA, which MOE commented on in January, 2006.

- While the Seilkop and Oller approach to quantifying inhalation risks related to exposures to nickel were included in the weight-of-evidence approach, the TRVs generated from this work have not been formally accepted by any recognized regulatory agency to date. While the Seilkop and Oller approach does allow for the generation of TRVs specifically for nickel oxide and nickel subsulphide, like the other TRVs used within the weight-of-evidence approach, they are not without their flaws and criticisms. For example, some reviewers have raised concerns with respect to the use of a 10-4 effect level as a point of departure (POD) in the derivation of the IUR for nickel oxide using the U.S. EPA Benchmark Dose Software (BMDS), as well as related methodological issues. As such, there are some uncertainties related to the use of these TRVs in the current assessment

The other IURs for airborne nickel used in the current assessment have been derived by regulatory agencies such as U.S. EPA, Health Canada and WHO. It is important to note that these IURs are based on occupational cohorts that were exposed to refinery dust and as such the applicability of these IURs to the ambient environment in the GSA is uncertain.

The current assessment of health risks related to oral exposures to nickel is based upon the U.S. EPA RfD of 20 µg/kg bodyweight/day (see Chapter 5 and Appendix A5). It should be noted that, unlike other regulatory values outlined in Appendix A5, this reference value does not address allergic contact dermatitis (ACD) arising from an oral exposure as a specific endpoint of concern. It may be assumed that the selected oral reference value used for the current assessment may not be protective of hypersensitive individuals, which are typically a very small subset of the overall population. Refer to Chapter 6.6 for further discussion.

The toxicological profiles provided with the HHRA (Appendix A) were intended as overviews of the available toxicological information and opinions available at the time of their completion. As such, they relied on secondary reviews by major reputable agencies, which is standard practice in preparing toxicological profiles. It is almost always impractical and unnecessary to review all key primary papers when a number of reputable agencies have already done so. There is essentially no added value for the

considerable costs and time that would be necessary to obtain and review the primary papers. In any event, the primary literature was reviewed up to what was most current at the time the profiles were prepared (which was largely up to the summer of 2005), with some minor revisions to certain COC based upon recent review comments. The purpose of these reviews is also clearly stated at the front of every profile. For example, the lead profile states: "This profile is not intended to provide a comprehensive review of the available toxicological and epidemiological literature on lead compounds. Rather, the purpose of the lead toxicological profile is to: i) summarize the most relevant toxicological and epidemiological information on this substance; ii) outline any recent information that may challenge previous findings; and iii) provide supporting rationale for the lead exposure limits selected for use in the human health risk assessment of the Sudbury area. The following toxicological review of lead is based primarily on secondary sources, such as ATSDR toxicological profiles and other detailed regulatory agency reviews, and is supplemented with recent scientific literature." Furthermore, the toxicological profiles were not used as a means of selecting TRVs; rather, the profiles simply provide supplementary supporting documentation for those readers who may be interested in an overview of the toxicology for each COC, but do not wish to conduct this level of research on their own.

Thus, in our opinion, the level of effort and detail that went into preparation of the profiles is appropriate and adequate for the purpose of the risk assessment. TRVs were selected based on detailed review of several of the most well-known and well-regarded regulatory agencies in the world. A number of considerations went into selecting the TRVs, including the scientific basis, the underlying science policies, the date of last major revision and others. This approach is consistent with the Ministry's October, 2005 Procedures document that indicates a strong preference for TRVs produced by credible regulatory agencies (MOE, 2005).

Nickel dermatitis has not been quantitatively evaluated (Section 6.7). MOE has indicated that the Neilsen *et al*. (1999) study which looked at the flare up of dermatitis in dermally sensitised women given oral doses of soluble nickel was used as the basis for the TRV used by WHO (2005) to derive their drinking water guideline. The TDI used by WHO was 12 :g/kg bw/day based on a LOAEL established after oral provocation of fasted patients with an empty stomach. The drinking water guideline developed from this LOAEL was 70 :g/L (12 :g/kg/day * 70 kg / 2 L/day * 20% source apportionment). This value was not used in the HHRA as absorption from drinking-water on an empty stomach is much greater than that absorbed from food or other media. WHO (2005) notes that it can range between 10- to 40- fold higher than absorption from food. It is believed by the SARA Group that this is not an appropriate TRV to use in an HHRA that investigates nickel exposure from multiple media and sources. Furthermore, no

Canadian regulatory agency has adopted a TRV or guideline value based on the TDI used by WHO (2005).

Several issues have not been included in the quantitative evaluation; rather, these have been discussed in Chapter 6. These include:

- Co-exposure to SO_2 and inhalation of metal fine particulates in air (PM_{10});
- Exposure to mixtures and risk health effects of common non critical endpoints (*e.g.,* cardiovascular system effects, respiratory effects, reproductive and neurological effects); and
- Pica children and other sensitive sub-populations (seniors, pregnant women, people with compromised health and/or low socio-economic status).

7.4.3 Uncertainty Surrounding the Arsenic Analysis in Vegetables

One consideration in the HHRA exposure assessment was the COC levels in locally or home-grown produce. At the request of the Technical Committee, conservative toxicological screening values were developed by the SARA Group to help interpret the significance of the survey data since there are virtually no regulatory guidance levels on metal levels in food items.

A problem arose in that the conservative screening criterion for arsenic was at the routine analytical detection limit (DL). Therefore, any detectable arsenic was cause for concern. To verify the reported arsenic concentrations, replicate vegetable samples were re-submitted to the laboratory for analysis. This QA/QC measure produced variable results which caused concern with regard to the reliability of the arsenic data generated by the laboratory. A series of steps were taken to address this matter.

As part of the QA/QC approach, the SARA Group purchased standard reference material (SRM) to help determine the variability related to the performance of determination of metals, particularly arsenic in the produce samples. The SRM was purchased from the National Institute of Standards and Technology and consisted of spinach leaves which contained certified values for all COC. The spinach sample was sent in triplicate to SGS Lakefield Research (SGS LR) for the analysis of total metals.

The SRM identified that there was inconsistency and false values reported for As in vegetable tissue. The spinach SRM was certified to contain As at levels which were below the detection limit that SGS was able to achieve (0.2 µg/g). The laboratory reported levels which varied widely (below detection, 1.6 µg/g

and 2.5 µg/g). This showed that although the SRM contained arsenic which was below the laboratory detection limit the levels reported were well above and might have exceeded the SARA screening criteria for these vegetables.

The laboratory was alerted to the issue and reran the samples (now aware that the As was certified below DL) and all results for the triplicate CRM returned with the As below DL.

The false positives in the CRM triplicate cast uncertainly over the reported As levels in the vegetable samples. To address this uncertainty eight vegetable tissue samples from the 2003 vegetable garden study were split, relabelled, so that the laboratory was not aware which samples were submitted, and re-sent for total metal analysis. These samples were split so some were duplicates and others were triplicates.

The As levels reported following this analysis were erratic and lower than the previously reported values. As levels which exceeded 3 µg/g remained at this level but other results which were previously between 1 and 2 µg/g were now reported as below detection (0.2 µg/g).

The SARA Group has requested that SGS send them the method validation reports to confirm that the MDLs that are being reported are correct. It is clear from this exercise that there is uncertainty surrounding the analysis of As at levels below 3 µg/g in the vegetable tissue. Some of the samples which were reported to contain As may in fact not contain As at levels which are as high as those reported.

In conclusion, although there is some uncertainty in the vegetable arsenic data, the observed variability tends to increase As levels in the reported data, therefore, resulting in higher exposure concentrations that would over-estimate risk.

7.4.4 Application of the Geometric Mean of Replicate Samples *versus* the Arithmetic Mean for Calculation of Media Concentrations

The Study database of soil samples consists of more than 8,000 samples collected from across the GSA during three separate surveys (MOE, 2004; CEM, 2004; Golder Associates, 2001). As part of the Study soil sampling protocol, original and duplicate soil samples were collected from almost 90% of sample locations. Original soil samples were collected using hand-held corers and consisted of between 15 and 30 soil cores collected in a "W" or "X" pattern at each location. Each soil core was divided into three depth intervals (*i.e.*, 0 to 5 cm, 5 to 10 cm, and 10 to 20 cm). The individual depth intervals from each core were mixed together (*i.e.*, all 0 to 5 cm depths) to form a composite sample to represent the depth

interval at the sample location. Duplicate soil samples were collected by following the soil sampling procedure a second time at the same sample location, shifted slightly to obtain new soil cores.

Several statistics can be used to describe central tendency or "average" concentrations of a data set including the mode, median, arithmetic and geometric means. Typically, concentration data from environmental samples tends to be positively skewed with a log-normal distribution. In these cases, the median and geometric mean better represent the central tendency or "average" value of the data set because they are relatively unaffected by extreme values.

In most soil sampling programs, duplicate samples are collected at one in every 10 sample locations and are split from a thoroughly mixed composite sample made up of two individual samples collected from the site. In those cases, using the arithmetic mean to estimate central tendency is appropriate, as one would be evaluating the systematic or methodological errors (*e.g.*, improper mixing, laboratory errors, *etc*.), which should be normally distributed around the mean. However, the Study soil sample database is made up of almost an equal number of original and duplicate soil samples from locations across the GSA. In this case, each duplicate sample was in fact an additional sample collected from the same location in the same manner as the first. Duplicate samples were not homogenized with the original sample to form a composite; rather they represent a second sample collected at the sample site. Therefore, given any variance would likely be largely due to differences in environmental concentrations at this location. For the purposes of the HHRA, the geometric mean of the original and duplicate soil samples was used to calculate COC concentrations in soil. As not every site had a duplicate sample, it was felt that it would unfairly bias the statistics if each of these original and duplicate samples were considered individual samples, and not combined to represent that particular sampling location.

Summary statistics, presented in the Table 7.5 for comparison, were calculated for all of the soil samples (*i.e.*, each sample is considered as an individual data point) extracted from the Study, for the complete data set. This included the resulting statistics if one treated each of the original and duplicate samples as discrete samples (*i.e.*, the complete data set), as well as those related to the arithmetic mean of the original and duplicate samples, and the geometric mean of the original and duplicate samples. The relative difference between the arithmetic mean and the geometric mean concentration for each COC is also provided, followed by the overall relative difference for all of the COC in the HHRA.

Based upon the results of this analyses, the use of a geometric mean over that of an arithmetic mean, results in an average difference of 0.55% on the mean and 0.50% on the 95% UCLM.

Table 7.5 Summary of Surface Soil Concentrations Extracted from Database

COC	No. of Samples (n)	Mean	Standard Deviation	95% UCLM
ARSENIC				
Complete Data Set	2137	17.3	33.1	21.8
Arithmetic Mean	1124	17.0	33.2	23.2
Geometric Mean	1124	16.7	32.9	22.9
Relative Difference between Means	-	0.64%	-	0.60%
COBALT				
Complete Data Set	2137	19.8	21.6	21.8
Arithmetic Mean	1124	19.3	21.1	22.0
Geometric Mean	1124	19.2	21.0	21.9
Relative Difference between Means	-	0.27%	-	0.27%
COPPER				
Complete Data Set	2137	427.3	669.8	517.8
Arithmetic Mean	1124	411.0	654.4	532.9
Geometric Mean	1124	407.4	651.6	528.7
Relative Difference between Means	-	0.44%	-	0.39%
LEAD				
Complete Data Set	2136	45.8	56.9	53.5
Arithmetic Mean	1124	44.4	55.5	54.7
Geometric Mean	1124	43.8	55.0	54.1
Relative Difference between Means	-	0.60%	-	0.57%
NICKEL				
Complete Data Set	2137	398.1	543.2	471.5
Arithmetic Mean	1124	383.7	531.2	482.6
Geometric Mean	1124	380.3	528.2	478.7
Relative Difference between Means	-	0.43%	-	0.40%
SELENIUM				
Complete Data Set	2137	2.25	3.58	2.74
Arithmetic Mean	1124	2.18	3.46	2.82
Geometric Mean	1124	2.14	3.43	2.78
Relative Difference between Means	-	0.89%	-	0.78%
OVERALL RELATIVE DIFFERENCE	**-**	**0.55%**	**-**	**0.50%**

7.5 Sensitivity Analysis

The purpose of a sensitivity analysis is to identify how variation in the output of a model (*e.g.*, total daily intake of a chemical) is influenced by uncertainty in the input variables. If the output variance precludes effective decision making, sensitivity analysis may be used to identify the input variables that contribute the most to the observed output variance. Subsequently, research efforts may be initiated to reduce uncertainty in those input variables in which it can be. Sensitivity analysis can also be used to simplify model structure by identifying those input variables that contribute little to the output (*e.g.*, a minor route of exposure) and thus can be removed from the analysis.

Sensitivity analysis methods may be classified into three groups: i) screening methods; ii) methods for local sensitivity analysis; and, iii) methods for global sensitivity analysis. Screening methods are generally used to separate influential input variables from non-influential ones, rather than quantify the impact that an input variable has on the output of the model. Screening methods are useful for models with large numbers of input variables. They are able to identify important input variables with little computational effort, but at a cost of losing quantitative information on the importance of the input variables. In contrast, local and global sensitivity measures provide quantitative estimates of the importance of each input variable. The difference between them is that the former focuses on estimating the impact of small changes in input variable values on model output, while the latter addresses the contribution to model output variance over the entire range of each input variable distribution.

In the case of a probabilistic risk assessment, in order to identify those assumptions (*e.g.,* body weight, breathing rate, food intake rate, *etc*.) that have the most influence on a particular forecast of interest (*e.g.,* HQ, EDI, *etc.*), a series of sensitivity charts can be produced by Crystal Ball®. Crystal Ball® calculates sensitivity by determining rank correlation coefficients between input variables and the forecast of interest. Crystal Ball® determines the contribution to variance by squaring all of the rank correlation coefficients and normalizing them to 100%. The "Contribution to Variance" provide by Crystal Ball® is an approximation and does not necessarily represent the true variance apportionment (Crystal Ball, 2004), particularly when there are correlations between input variables.

However, as noted previously, recommendations from the IERP resulted in the elimination of a number of the key probabilistic distributions previously evaluated probabilistically as part of the draft risk assessment. As a result, probabilistic analyses would not produce useful output given the small number of probabilistic distributions on which it would be based (*i.e.*, largely driven by receptor body weight, and other related properties).

To investigate the relative sensitivity of risk predictions as part of the deterministic risk assessment, the impact of key input variables on the calculated health risk related to exposures of the female preschool child living in Copper Cliff to lead and nickel were tabled. The key variables evaluated included the following:

- The selected oral TRV for lead (i.e., the MOE recommended value of 1.85 versus the U.S. EPA recommended value of 3.7 µg/kg bw/day) and nickel (i.e., the U.S. EPA recommended value of 20 versus the OEHHA recommended value of 11 µg/kg bw/day);

- The soil/dust consumption rate (i.e., the MOE recommended value of 100 mg/day versus the Health Canada recommended value of 80 mg/day);

- The selected food consumption database (i.e., the older Health Canada/Nutrition Canada database versus the more recent USDA data from the Northeastern U.S.);

- In the case of nickel, impact of evaluating risk to the female preschool child versus a hypothetical individual exposed for an entire lifetime;

- In the case of lead, the relationship selected to estimate indoor dust COC concentrations from paired outdoor residential soil COC concentrations (i.e., the soil-to-dust regression equation calculated from the indoor dust survey versus IEUBK default soil-to-dust concentration ratio of 0.7); and

- The bioaccessibility of lead and nickel in soil and dust media (i.e., one-phase results from bioaccessibility testing versus two-phase results from bioaccessibility test versus assuming 100% bioaccessibility, but using the IEUBK bioavailability default value of 50%, for lead).

As would be expected, the output in Tables 7.6 and 7.7, demonstrate that as expected, the variable with the largest leverage impact on the estimated risk is the TRV value. While the other variables have lesser impacts, they are still play a significant role in the calculation of overall risk. In the case of lead (see Table 7.6), the reason these variables have a large influence is that the vast majority of the acceptable daily intake as dictated by the TRV is used up by background sources unrelated to Sudbury (*i.e.*, the market basket). As such, any increase in the acceptable daily intake (*e.g.*, through selection of the U.S. EPA TRV instead of the MOE TRV) or modification of key receptor or chemical parameters (*e.g.*, bioaccessibility, food consumption rates, *etc.*) will result in a significant impact on the amount of lead can be present within the soil and dust before exceeding the selected TRV.

Ultimately the choice of assumptions used in the assessed scenario is based upon an evaluation of the best available science and considers the implications of regulatory policy as it pertains to these assumptions. In the case of the current HHRA, these parameters were selected based upon the expert judgment of the SARA Group in consultation with the Technical Committee and the IERP. As can be observed in the following tables, even minor changes to certain key assumptions can have a significant impact in the calculated risk estimate, and have large implications on the requirement for follow up risk management activities.

Table 7.6 Relative Impact to Predicted Risk from Changing Key HHRA Assumptions for Lead in Copper Cliff

Scenario	Assessed Scenario	MOE Soil Consumption Rate	USDA Food Consumption + MOE soil consumption rate	IEUBK Bioavailability Adjustment	IEUBK Soil-to-Dust Concentration Ratio	USDA Food Consumption	Two-Phase Bioaccessibility	Using Health Canada TRV	USDA Food Consumption + Two-Phase Bioaccessibility + Health Canada TRV
% Change	-	6% increase	3% decrease	9% decrease	10% decrease	10% decrease	11% decrease	49% decrease	60% decrease
Oral TRV (µg/kg bw/day)	1.85 (MOE)	1.85 (MOE)	1.85 (MOE)	1.85 (MOE)	1.85 (MOE)	1.85 (MOE)	1.85 (MOE)	3.7 (HC)	3.7 (HC)
Soil/Dust Consumption Rate (mg/day)	80	100	100	80	80	80	80	80	80
Food Consumption Database	HC	HC	USDA	HC	HC	USDA	HC	HC	USDA
Soil-to-Dust Relationship	Regression Equation	Regression Equation	Regression Equation	Regression Equation	IEUBK Soil-to-Dust Concentration Ratio (0.7)	Regression Equation	Regression Equation	Regression Equation	Regression Equation
Bioaccessibility	Soil = 66% Dust = 83% (one-phase)	Soil = 66% Dust = 83% (one-phase)	Soil = 66% Dust = 83% (one-phase)	Soil = 100% Dust = 100%	Soil = 66% Dust = 83% (one-phase)	Soil = 66% Dust = 83% (one-phase)	Soil = 38% Dust = 43% (two-phase)	Soil = 66% Dust = 83% (one-phase)	Soil = 38% Dust = 43% (two-phase)
Bioavailability	100%	100%	100%	50% (IEUBK)	100%	100%	100%	100%	100%
Receptor group	Female Preschool Child								

Table 7.7 Relative Impact to Predicted Risk from Changing Key HHRA Assumptions for Nickel in Copper Cliff

Scenario	Assessed Scenario	MOE Soil Consumption Rate	Two-Phase Bioaccessibility	USDA Food Consumption + MOE Soil Consumption Rate	USDA Food Consumption	OEHHA Oral TRV	OEHHA Oral TRV + Lifetime	MOE soil consumption rate + Lifetime	Lifetime Exposure
% Change	-	1% increase	1% increase	6% decrease	7% decrease	83% increase	30% decrease	62% decrease	62% decrease
Soil/Dust Consumption Rate (mg/day)	80	100	80	100	80	80	80	100	80
Food Consumption Database	HC	HC	HC	USDA	USDA	HC	HC	HC	HC
Oral TRV (µg/kg bw/day)	20	20	20	20	20	11	11	20	20
Bioaccessibility	Soil = 42% Dust = 30% (one-phase)	Soil = 42% Dust = 30% (one-phase)	Soil = 38% Dust = 43% (two-phase)	Soil = 42% Dust = 30% (one-phase)	Soil = 42% Dust = 30% (one-phase)	Soil = 44% Dust = 31% (one-phase)	Soil = 42% Dust = 30% (one-phase)	Soil = 42% Dust = 30% (one-phase)	Soil = 42% Dust = 30% (one-phase)
Receptor group	Female Preschool Child	Female Preschool Child	Female Preschool Child	Female Preschool Child	Female Preschool Child	Female Preschool Child	Lifetime Receptor	Lifetime Receptor	Lifetime Receptor

7.6 References

Arista Laboratories Europe (Arista). 2003a. U.K. Smoke Constituents Study. Part 11: Determination of Metal Yields in Cigarette Smoke by ICP-MS & CVAAS. Report. Available at: http://www.the-tma.org.uk/benchmark/benchmarkresources/part11.pdf [Oct 30, 2006]

Arista Laboratories Europe (Arista). 2003b. U.K. Smoke Constituents Study. Part 11: Determination of Metal Yields in Cigarette Smoke. Annex B-Validation Data. Available at: http://www.the-tma.org.uk/benchmark/benchmarkresources/part11v.pdf [Oct 30, 2006]

Beck, M.B. 1987. Water quality modeling: A review of the analysis of uncertainty. Water Resources Research 23: 1393-1442.

Bernhard, D., Rossmaan, A. and Wick, G. 2005. Critical Review: Metals in Cigarette Smoke. IUBMB Life, 57(12): 805-809.

CEM. 2004. Metal Levels in the Soils of the Sudbury Smelter Footprint. A Report to: Inco Ltd. and Falconbridge Ltd. Centre for Environmental Monitoring, Laurentian University. July 12, 2004.

CEPN. 1997. Risk Assessment for Nickel and Nickel Compounds in the Ambient Air from Exposure by Inhalation. Lepicard, S., Schneider T., Fritsch P., Maximilien R., and Deloraine A. Centre d'etude sur l'Evaluation de la Protection dans le domaine Nucléaire. Cited In: MOE comment.

Chao, H.P., Peck, S.C., and Wan, Y.S. 1994. Managing uncertainty: The tropospheric ozone challenge. Risk Analysis 14: 465-475.

Cothern, C.R., Coniglio, W.A., and Marcus,W.L. 1986. Estimating risk to human health. Environmental Science and Technology 20: 111-116.

Cullen, A.C., and Frey, H.C. 1999. Probabilistic Techniques in Exposure Assessment. Plenum Press, New York.

Finkel, A.M. 1990. Confronting Uncertainty in Risk Management: A Guide for Decision-makers. Center for Risk Management, Resources for the Future, Washington, D.C.

Finkel, A.M. 1994. Stepping out of your own shadow: A didactic example of how facing uncertainty can improve decision-making. Risk Analysis 14: 751-761.

Grandjean, P. 1984. Human exposure to nickel. In: Sunderman, F. W., Jr., et al., eds. Nickel in the Human Environment: Proceedings of a Joint Symposium, March 1983, Lyon, France. Lyon, France: International Agency for Research on Cancer. (IARC Scientific Publication No. 53). pp. 469-480. Cited In: Nickel Institute, 1997.

Golder Associates. 2001. Town of Falconbridge Soil Sampling Program - Comprehensive Falconbridge Survey. A Report to: Falconbridge Ltd. Golder Associates, Ltd. September 11, 2001.

Health Canada. 2006. Arsenic and its Compounds - PSL1. Available at: http://www.hc-sc.gc.ca/ewh-semt/pubs/contaminants/psl1-lsp1/arsenic_comp/arsenic_comp_3_e.html [Oct 31, 2006]

Henrion, M., and Fischoff, B. 1986. "Assessing Uncertainty in Physical Constants." American Journal of Physics 54: 791-798.

Hoffman, F.O., and Hammonds, J.S. 1994. Propagation of uncertainty in risk assessments: The need to distinguish between uncertainty die to lack of knowledge and uncertainty due to variability. Risk Analysis 14: 707-712.

Hora, S.C. 1996. Aleatory and epistemic uncertainty in probability elicitation with an example from hazardous waste management. Reliability Engineering and System Safety 54: 217–223.

Hsieh T.H., Yu C.P., and Oberdörster G. 1999. Modeling of deposition and clearance of inhaled Ni compounds in the human lung. Regul. Toxicol. Pharmacol. 30:18-28.

MOE. 2007. Memo from Minnie de Jong of Standards Development Branch to Brian Cameron of MOE Sudbury District Office providing information requested by SARA Group related to Average OTR values. January 24, 2007.

MOE. 2004. City of Greater Sudbury 2001 Urban Soil Survey. Environmental Sciences and Standards Division, Ontario Ministry of the Environment. Report No. SDB-008-3511-2003. July 2004.

Moore, D.R.J., and Caux, P.Y. 1997. Estimating low toxic effects. Environmental Toxicology and Chemistry 16: 794-801.

Moore, D.R.J., and Elliott, B.J. 1996. Should uncertainty be quantified in human and ecological risk assessments used for decision-making? Human and Ecological Risk Assessment 2: 11-24.

Nickel Institute. 1997. Safe use of nickel in the workplace. Available at: http://www.nickelinstitute.org/index.cfm/ci_id/13028/la_id/4.htm [Oct 31, 2006]

Olson, O.E. and FROST, D.V. 1970. Selenium in papers and tobaccos. Environ. Sci. Technol., 4: 686-687. Cited IN: WHO, 1987.

Oreskes, N., K. Shrader-Frechette., and Belitz, K. 1994. Verification, validation, and confirmation of numerical models in the earth sciences. Science 263: 641-646.

Paté-Cornell, E. 2002. Risk and uncertainty analysis in government safety decisions. Risk Analysis 22: 633-646.

Province of Ontario. 2004. Soil, Ground Water and Sediment Standards for Use Under Part XV.1 of the *Environmental Protection Act*. March 9, 2004.

Reckhow K.H. 1994. Water quality simulation modeling and uncertainty analysis for risk assessment and decision making. Ecological Modeling 72: 1-20.

Richardson, G.M. 1997. Compendium of Canadian Human Exposure Factors for Risk Assessment. O'Connor Associates 1155-2720 Queensview Dr., Ottawa, Ontario.

Rowe, W.D. 1994. Understanding uncertainty. Risk Analysis 14: 743-750.

Seilkop, S.K. 2004. Estimation of Respiratory Cancer Risks Associated with Exposure to Small Airborne Concentrations of Nickel-Containing Substances. Presention to the Ontario Ministry of the Environment and Inco. February 10th, 2004.

Seilkop, S.K. and Oller, A.R. 2003. Respiratory cancer risks associated with low-level nickel exposure: an integrated assessment based on animal, epidemiological and mechanistic data. Reg Toxicol and Pharma 37:173-190.

Sokal, R.R., and Rohlf, F.J. 1981. Biometry. W.H. Freeman and Company, New York.

U.S. EPA. 1999. Risk Assessment Guidance for Superfund: Volume 3 - (Part A, Process for Conducting Probabilistic Risk Assessment). United States Environmental Protection Agency. Office of Solid Waste and Emergency Response, Washington, D.C.

Warren-Hicks, W., and Moore, D.R.J. (eds.). 1998. Uncertainty Analysis in Ecological Risk Assessment. SETAC Press, Pensacola, Florida.

WHO. 2005. Nickel in Drinking-water. Background document for development of WHO Guidelines for Drinking-water Quality. World Health Organization (WHO). WHO/SDE/WSH/05.08/55.

WHO. 2000. Nickel. Air Quality Guidelines. 2nd eds. Available at: http://www.euro.who.int/document/aiq/6_10nickel.pdf#search='nickel%20intake%20from%20cigarettes' [Oct31, 2006]

WHO. 1991. Nickel. Environmental Health Criteria, No.108. Cited In: WHO, 2000.

WHO. 1987. International Programme on Chemical Safety, Environmetnal Health Criteria 58: Selenium. Available at: http://www.inchem.org/documents/ehc/ehc /ehc58.htm [Oct 31, 2006]

Wolfsperger, M., Hauser, G., Gossler, W. and Schlagenhaufen, C. 1994. Heavy metals in human hair samples from Austria and Italy: influence of sex and smoking habits. Sci Total Envrion, 156(3): 235-242.