Soil Investigation and Human Health Risk Assessment for the Rodney Street Community, Port Colborne

March 2002

⊗ **Ontario**

Ministry of the Environment

Soil Investigation and Human Health Risk Assessment for the Rodney Street Community, Port Colborne

March 2002

Printed on recycled paper

Executive Summary

Conclusions

The Ministry of the Environment's soil investigation and human health risk assessment report for the Rodney Street community has determined that elevated nickel and lead soil contamination on some properties warrants action. This report is the ministry's final report and supercedes the two earlier versions (March 2001, October 2001). The report provides a comprehensive soil investigation and human health risk assessment that examined almost 2,000 soil samples from about 200 properties to determine the level of human health risk posed by metal and arsenic levels in surface soil in the Rodney Street community. The health risk assessment reviewed concentrations of seven metals and arsenic and makes the following final recommendations:

- an intervention level of 8,000 parts per million[1] be set for soil nickel. The intervention level for nickel requires action through remediation of soil.

- an intervention level of 1,000 ppm for lead for play areas on residential properties or in public areas *covered by sod or grass* to which children have access. *The bare soil* intervention level for lead is 400 ppm for these areas. The intervention levels for lead require action through follow-up by individual residents to reduce personal exposure to lead.

The assessment also concludes that no action for the remaining five metals (antimony, beryllium, cadmium, copper, cobalt) and arsenic in soil is required.

In carrying out the investigation and assessment, the Ministry of the Environment identified soil nickel levels in excess of 8,000 ppm, in at least one composite soil sample, in the first 30 cm of soil at 25 properties. The soil nickel results from historical emissions from Inco Limited (Inco). The ministry also found soil lead levels in excess of 1,000 ppm at 11 properties, including two of the 25 properties with elevated soil nickel levels. These soil-lead levels are typical of older urban residential neighborhoods and result from the historical use of lead-based paints, leaded gasoline and discarded lead-acid batteries. The elevated lead levels found on some properties are not caused by either Inco or Algoma emissions.

The following summarizes the report's key findings for the Rodney Street community:

1. A soil nickel intervention level of 8,000 ppm has been established to protect toddler-aged children (7 months to less than 5 years);

2. Soil nickel levels in the community should not pose any immediate or long-term risks to other age groups;

---

[1]Also referred to as ppm, μg/g, micrograms per gram, or, millionths of a gram.

3.   At least one composite soil sample from twenty-five properties had more than 8,000 ppm nickel;

4.   Intervention levels for lead have been established to protect children;

5.   Eleven properties have soil lead levels over 1,000 ppm; and

6.   The soil levels of antimony, beryllium, cadmium, copper, cobalt and arsenic were below the level of concern and therefore no action is needed.

Human Health Risk Assessment

The Ministry of the Environment conducted a human health risk assessment for seven metals (antimony, beryllium, cadmium, cobalt, copper, lead, nickel) and arsenic because these elements were found in the surface soils of the Rodney Street community at concentrations that warranted further investigation. The health risk assessment was peer reviewed by an international panel of experts so the ministry could be assured that the best and most recent science was used in its Rodney Street study. The panel included recognized North American and European experts from the fields of: human health, nickel toxicology, risk assessment and metal bioaccessibility. The peer review experts were:

*   Dr. Ambika Bathija (Washington, D.C.), affiliated with the United States Environmental Protection Agency

*   Dr. Lynne Haber (Cincinnati, Ohio), affiliated with the Toxicology Excellence for Risk Assessment

*   Dr. Robert Jin (Toronto, Ontario), affiliated with the Ontario Ministry of Health and Long-Term Care

*   Dr. Tor Norseth (Oslo, Norway), affiliated with the Norwegian National Institute of Occupational Health

*   Dr. Rosalind Schoof (Seattle, Washington), affiliated with Gradient Corporation

*   Dr. John Wheeler (Atlanta, Georgia), affiliated with the Agency for Toxic Substances and Disease Registry

A human health risk assessment can be triggered when contaminants are found in a community at levels above the ministry's *Guideline for Use at Contaminated Sites in Ontario* (MOE, 1997). The ministry's health risk assessment examined total exposure to contaminants through a number of possible pathways, such as the air we breathe, the soil we may incidentally ingest, the water

we drink and the food we eat.

This human health risk assessment has assessed exposure to soil contaminants in the Rodney Street community. The ministry's report established soil concentration levels that maintain human exposure below health protection criteria. The assessment has also established levels to remediate soil and to reduce exposure to contaminated soil.

Nickel

The report reviewed the toxicological basis for health protection criteria that have been developed by key agencies worldwide responsible for health and environmental protection. These health-based criteria are developed to provide protection against the most sensitive effects found in human or animal studies. For nickel, the most sensitive effects are reproductive effects and reduced organ weight in animals. The health risk reassessment establishes a soil nickel intervention level of 8,000 ppm, which is intended to protect toddler-aged children. The report finds this soil intervention level should not pose immediate or long-term risks to other age groups. However, it is noted in the report that there are insufficient scientific data to determine if the Rodney Street community nickel soil intervention level of 8,000 ppm will prevent nickel dermatitis in sensitized individuals, or prevent people from becoming sensitized to nickel. The Ventana Community Health Assessment Project being undertaken through the Community Based Risk Assessment will further investigate nickel dermatitis in Port Colborne.

In this human health risk assessment, the ministry considered contaminant exposures from a variety of sources including: indoor and outdoor air; soil and dust; food from the supermarket and home gardens; and the municipal drinking water supply. To protect residents, and especially toddlers, the soil nickel intervention level was developed to ensure that all of these nickel exposures did not exceed a value that is well below any potential health risk.

The ministry's *Guideline for Use at Contaminated Sites in Ontario* (MOE, 1997) references a human health value for nickel in soil of 310 ppm. However, the 310 ppm value is only used for a generic clean-up of soil or as a trigger value above which soil nickel levels require a detailed, scientific, human health risk assessment. Soil levels above 310 ppm do not necessarily constitute a health risk, rather they trigger the need for further study. The 310 ppm value should not be used for site specific health risk assessments, such as the study done by the ministry for the Rodney Street community, or the Community Based Risk Assessment that is being done for the broader community.

The form of nickel in the soil can have an important impact on its availability and its toxicity for both the natural ecosystem and human health.

The predominant form of nickel in the soil in the Rodney Street community is nickel oxide (80% of the total nickel, on average). This is consistent with known emissions from the metallurgical process employed by Inco, especially in its historic operations. This is also acknowledged by

Inco.

After the shutdown of the refinery in 1984, nickel injury on sensitive species of vegetation has rarely been observed in Port Colborne. This corroborates the low soil nickel plant bioavailability results (the ability of plants to absorb nickel from the soil) and suggests that most of the nickel injury observed on vegetation up to 1984 was likely from deposition and absorption of nickel from the ambient air and not from uptake of soluble nickel from nickel-contaminated soil by plants. There were no symptoms of nickel related injury to plants observed in the Rodney Street community during the most recent investigations.

Nickel levels in air fell continuously from 1992 to 1995, the most recent time period for which the ministry has complete air data. Limited ambient air data for the Rodney Street community obtained by the MOE in 2001 shows that the current nickel levels in air are about 1/3 of what they were from 1992 to 1995 and are in the concentration range found in other Ontario cities.

Using the 2001 air nickel data, and taking a cautious approach that assumes all of the nickel is in the form of nickel oxide (the predominant form of nickel found in the soil), the life-time cancer risk is estimated to be in the range of 3.1×10^{-6} to 1.6×10^{-5}, a range which is considered to be very low. This is an interim estimate since risks for carcinogens are usually based on an average annual air concentration. Data for the summer and fall months are available for the Rodney Street community in 2001. The ministry will continue to monitor ambient air in 2002 to confirm the declining trends in air nickel levels. In addition, the ministry will determine the form of nickel present in the air to better estimate the risk. Inco, at the ministry's request, is also assessing air emissions from their Port Colborne refinery.

Lead

Lead in soil has long been recognized as posing potential risks, particularly to children up to five years of age, who were considered the most sensitive to exposures for direct soil/dust ingestion.

The average soil lead level in surface soil in the Rodney Street community is about 200 ppm, which is similar to other older urban residential communities in Ontario. As a result, estimated exposures (and hence blood lead levels) are predicted to be similar to those for other urban Ontario populations. The Regional Niagara Public Health Department undertook blood lead screening clinics in Port Colborne between April and June 2001. A key conclusion from the clinics was that children under seven years and pregnant women in the Eastside community are not at increased risk of lead exposure compared to other communities in Ontario, even considering the localized elevated soil lead levels on some properties.

It is prudent, however, to conclude that the 11 properties with reported soil lead levels higher than 1,000 ppm may have some possibility of elevating blood lead levels in children who routinely play in these areas.

As a result, the report proposes an intervention level of 1,000 ppm for lead for play areas on residential properties or in public areas *covered by sod and grass* to which children have access. *The bare soil* intervention level for lead is 400 ppm for these areas. Residents at properties exceeding 1,000 ppm lead in soil were advised in March and again in October 2001 of their soil lead levels and provided information on reducing environmental lead exposure. Additional ways to reduce exposure to the lead in soil are presented in the ministry's fact sheet, *"Frequently Asked Questions About Lead Contamination"*.

Arsenic

People everywhere in North America are exposed to low levels of arsenic in the environment. Exposures can occur by a number of different pathways including normal diet and drinking water. The measured soil-arsenic levels in the Rodney Street community were compared to the levels found in other Ontario communities with elevated levels of soil arsenic. In the case of these communities, no adverse health effects were predicted to be associated with the arsenic in the soil.

This report concludes that the measured levels of arsenic in Rodney Street community soil are unlikely to pose an undue health risk to residents of this community, based on consideration of: the measured availability of the arsenic in these soils; comparison to typical levels elsewhere; and knowledge of health study outcomes involving arsenic soil exposure in other Ontario communities.

Antimony, Beryllium, Cadmium, Cobalt and Copper

Taking the same approach as used for nickel, estimates were modeled using the maximum reported levels of each metal in the Rodney Street community surface soil and backyard produce, Port Colborne municipal drinking water, ambient air and supermarket food.

For the metals antimony, beryllium, cadmium, cobalt and copper, estimated total daily intakes for all age groups were well below stringent oral or breathing exposure limits from major recognized jurisdictions, such as the US Environmental Protection Agency, World Health Organization and Health Canada. No adverse health effects are anticipated to result from exposure to antimony, beryllium, cadmium, copper or cobalt, in soils in the Rodney Street community.

Therefore, soil intervention levels were not developed and no action is required for these metals for the Rodney Street community.

Background

From 1918 to 1984 Inco operated a nickel refinery in the city of Port Colborne. Between the years 1972 and 1991, the Ontario Ministry of the Environment conducted numerous investigations to document the impact of Inco's emissions on soil and vegetation in and around

Port Colborne. These investigations concluded that emissions from more than a half a century of of nickel refining had resulted in metal contamination of soil in various locations throughout the Port Colborne area. Nickel, copper and cobalt concentrations in surface soil (0 to 5 cm depth) were elevated in residential communities adjacent to Inco and for a considerable distance downwind (east-northeasterly) of the refinery to levels which could or did cause injury to vegetation (phytotoxicity). Generally, the vegetation impacts were on farm crops and to silver maple trees.

The ministry's effects-based guideline for nickel, copper, cobalt, arsenic and zinc are all based on phytotoxicity (injury to vegetation). The Guideline's criterion for selenium is based on the protection of grazing animals. Numerous ministry studies conducted on Port Colborne farms in the 1970s and 1980s documented toxicity to agricultural crops as a result of ambient air SO_2 fumigations and metal soil contamination. Up to 1991 the highest soil nickel concentration that could be proven to exist through repeat sampling in the Port Colborne area was 9,750 ppm. A human health risk assessment using this maximum soil-nickel level was published by the ministry and Regional Niagara Public Health Department in 1997. It was concluded at the time that based on a multi-media assessment of potential risks, no adverse health effects are anticipated to result from exposure to nickel, copper, or cobalt, in soils in the Port Colborne community.

Additional extensive soil sampling was conducted by the ministry in Port Colborne and the surrounding area in 1998 and 1999 and demonstrated that soil nickel concentrations exceeded background levels up to 28 km downwind of the refinery, covering a 345 km^2 area of the Niagara peninsula. Furthermore, soil nickel levels exceeded the ministry's effects-based guideline (the level of nickel in soil that *may* cause injury to plants) for a distance of up to 3 km downwind of Inco over an area of almost 29 km^2. In addition, copper and cobalt also exceed their corresponding effects-based soil guidelines in smaller areas of the community, mainly immediately east, north, and northeast of the refinery.

In September 2000, soil nickel levels from a single property on Rodney Street were found to exceed the soil nickel level used in the 1997 health risk assessment (9,750 ppm). This result caused the ministry to sample and analyze soil from all properties on Rodney Street. Preliminary results of the additional sampling indicated that surface soil nickel levels ranged up to 17,000 ppm, and that the soil metal levels were extremely variable between properties. As a result, in November 2000, the ministry sampled soil from residential properties south of Louis Street to Rodney Street and east of the Welland Canal to Davis Street, to determine the extent of this contamination. Between April 25 and May 5, 2001 the ministry sampled surface soil from additional residential properties as requested by the residents. In all, almost 2,000 soil samples were collected from about 200 properties.

The findings that some properties had high metal levels in soil triggered the ministry to undertake a human health risk assessment of metals and arsenic found at elevated concentrations in the soil.

Soil Investigation Results - Extent and Severity

Ministry investigations have shown elevated soil metals levels around Port Colborne and in residential communities adjacent to the Inco site. Because the community is in close proximity to the refinery, soil would have received stack emissions as well as extensive fugitive emissions, both of which would have been particularly significant early in Inco's operating history.

The ministry sampled the front, back or side yard on each property, depending on the size and nature of each yard. The strategy also included intensive quality control with the taking of duplicate and triplicate soil core samples from selected properties. The strategy was designed to allow the ministry to describe the specific soil levels for each property and to also define with precision, the soil-metal level variability that is present in the community. This information was used in combination with the human health risk assessment to establish a soil intervention level for nickel.

The soil nickel concentration in the Rodney Street community averaged about 2,500 ppm, while the single highest concentration was 17,000 ppm. Ninety per cent of the soil nickel concentrations for all samples collected in the Rodney Street community are below 5,600 ppm. Twenty-five of the properties sampled had at least one soil result that exceeded the soil nickel intervention level of 8,000 ppm developed to be protective of toddler-aged children.

Soil-metal concentrations on average increased slightly with depth to a maximum of between 10 cm to 20 cm, the limit of the majority of residential sampling performed. Based on trench digging, metal concentrations above the proposed soil nickel intervention level are not likely to be found much deeper than 30 cm on most residential properties within the community.

Sources and Mechanisms

Based on the extensive surface soil sampling investigation and laboratory testing, elevated soil levels for nickel, copper, and cobalt in the Rodney Street community are considered directly related to Inco emissions. The company acknowledged this in an open letter to the public in December 2000.

Both Inco and Algoma emitted arsenic, however it is not possible to confidently apportion which company contributed how much arsenic and where it was deposited in the Rodney Street community.

Elevated soil lead levels were found on some properties randomly scattered in the Rodney Street community. This is considered typical of domestic residential lead sources in older urban communities and is not attributed to either Inco or Algoma emissions. The erosion and flaking of old lead-based paint from exterior structures such as house and shed walls, porches, fences, poles and playground equipment is a common source of soil lead contamination in older urban communities. The soil lead levels found in the Rodney Street community are not unusual, either

in extent or concentration, relative to other similar urban communities in Ontario.

Levels of cadmium, chromium, copper, barium, and zinc in soil were often associated with higher lead levels. Along with lead, these elements were common pigment, anti-mildew, or anti-fungal additives in old exterior paint and are frequent co-contaminants in residential soil. Lead and antimony levels observed in the soil are an indication that batteries may have been stored or disposed of on the property, whereas lead, barium and zinc soil contamination is a signature of lead-based paint.

With the exception of one property where elevated beryllium levels were concurrent with high lead and other metals, the marginally elevated soil beryllium concentrations across the Rodney Street community are likely related to the presence of slag and local shale deposits. Emissions from Algoma may have contributed to the marginally elevated soil beryllium levels in the Rodney Street community.

Considerable variability in soil contaminant levels was evident between adjacent properties. This "patchwork" pattern of high-and-low soil contamination on neighbouring lots is likely related to property maintenance and landscaping. Adding topsoil or mulch, re-sodding, building, and cultivating gardens are landscaping practices that, over time, tend to cover or dilute contaminants that are predominantly present in the surface soil. It also indicates that the source of the soil contamination is likely atmospheric and that with recent deposition substantially decreased, newly landscaped properties have not become re-contaminated to the levels of undisturbed properties.

Information from six trenches in the community indicated that the higher levels of metals in soil were to be found in the upper layers, supporting the likelihood that metals in soil in the wider community resulted from atmospheric deposition. However, visual and test findings from three other trench samples indicated that fill materials may have contributed to higher metal levels in soils on some properties, particularly on the south of Rodney Street.

Study Participants

The Rodney Street community study involved many scientists and technicians from the Ministry of the Environment. The following staff of the Standards Development Branch, Ecological Standards and Toxicology Section, participated in sample collection and sample processing: Marius Marsh, Murray Dixon, Bill Gizyn, Bob Emerson, Ron Hall, Danuta Roszak, Deborah Terry, Melanie Appleton, Richard Chong-Kit, Mike Mueller, and Al Kuja. Randall Jones co-ordinated the geo-referencing of the soil samples, managed the soil data base, and prepared the contaminant contour maps. Dave McLaughlin was the principal investigator and author of Part A of the report, with considerable contribution by Murray Dixon.

The Human Health Risk Assessment (Part B) was conducted by toxicologists of the Standards Development Branch, Human Toxicology and Air Standards Section. Brendan Birmingham co-ordinated this effort and is the principal author of Part B of the report. Contributing ministry toxicologists included Scott Fleming, Satish Deshpande, Marco Pagliarulo, Mark Chappel, and Audrey Wagenaar. Elliot Sigal and Glenn Ferguson of Cantox Environmental Inc., and Bryan Leece of Dillon Consulting Ltd. provided technical assistance.

Laboratory Services Branch personnel provided critical laboratory support. Liz Pastorek administered the contract for the private laboratory that conducted the soil analysis. Tender evaluation was conducted by Peter Drouin. The initial quality control check was handled by Sathi Seliah. Rusty Moody and Jim Howden provided data monitoring and data management duties. The in-house laboratory analysis required to ensure data quality was conducted by Lian Liu and Julie Uzonyi (ICO - metals) and Hung Sing Chiu and Regina Pearce (hydrides).

Jacques Whitford Environmental Limited provided 2000 and 2001 residential vegetable garden produce and garden soil data for both the Rodney Street community and Port Colborne in general, and 2001 air monitoring data.

Paul Nieweglowski and Bob Slattery of the Niagara District Office provided liaison between the ministry's Operations Division and the Environmental Science and Standards Division, and community and industry stakeholders. Rick Day was the communications officer.

Neil Buonocore and Frank Dobroff of the Ministry's West Central Region Technical Support installed and operated the air quality monitor in the Rodney Street ball park.

Barry Zajdlik of Zajdlik & Associates provided statistical analysis of the soil data base and the soil sampling procedure.

Ron Pearson of Barenco Inc. and Tom Hutchinson of Trent University provided a critical review of the *Part A Soil Investigation* component of the report.

Expert panel peer reviewers of the *Part B Human Health Risk Assessment* component of the report for nickel were:

- Dr. Ambika Bathija, Washington, DC
- Dr. Lynne Haber, Cincinnati, Ohio
- Dr. Robert Jin, Toronto, Ontario
- Dr. Tor Norseth, Oslo, Norway
- Dr. Rosalind Schoof, Mercer Island, Washington
- Dr. John Wheeler, Atlanta, Georgia

Dr. Robert Willes of Cantox Environmental Inc. was the facilitator of the expert panel consensus meeting, and prepared the expert Panel consensus report.

Samir Konar provided an external editorial review. Mel Plewes provided internal document review.

Project administration was managed by Marny Paget with assistance from Murray Dixon and Robyn Dale.

Dave McLaughlin was the Project Co-ordinator, Jim Smith was the managing Director, and project management support was provided by George Crawford, Dale Henry, John Mayes, and Paul Nieweglowski.

Part A

Soil Investigation: Main Document

Table of Contents
Part A - Soil Investigation: Main Document

1.0 Introduction

The ministry's report entitled *Soil Investigation and Human Health Risk Assessment for the Rodney Street Community, Port Colborne: March 2002*, has two parts. *Part A* describes the results of an extensive soil sampling program in the Rodney Street community of Port Colborne. *Part B* is a human health risk assessment that characterizes the potential risk associated with the elevated soil metal levels, and where warranted, establishes a soil intervention level intended to mitigate the potential health risks. For the purpose of Part A and Part B of this report, the Rodney Street community in Port Colborne is defined as the residential area bounded by the Welland Canal to the west, the International Nickel Company Limited (Inco) refinery to the east, the south side of Louis Street to the north and both sides of Rodney Street to the south. This area is also sometimes referred to as the Eastside community.

1.1 Objectives of Part A

1)   to determine the extent and severity of soil metal and arsenic contamination in the Rodney Street community of Port Colborne;

2)   to characterize soil metal and arsenic contamination in the Rodney Street community;

3)   to determine the source(s) of the soil metal and arsenic contamination in the Rodney Street community; and

4)   to determine the mechanism(s) of soil metal and arsenic contamination in the Rodney Street community.

2.0 Scope of the Study

The objective of the (Part A) sampling program was to obtain soil chemical data that is representative of the over-all metal levels of the sampled residential yards in the Rodney Street community and would therefore be indicative of the environmental exposure that a community resident would receive from long term use of the property. This representative soil data was required for the human health risk assessment (Part B of this report). The risk assessment process integrates the soil chemical data with other relevant data to determine what the over-all exposure would be for people using the property for a lifetime. The scope of the sampling program was not to exhaustively characterize the soil metal status of all possible sample sites on every yard of every residential property, rather it was to collect soil data that was representative of each sampled property. The sampling program is described in more detail in Section 5.0 of Part A.

The soil sampling conducted by the ministry in the Rodney Street community is not necessarily the only sampling that would be conducted to characterize the contaminant status of a particular property. If the soil metal levels on a property exceed the risk-based soil intervention level

determined by the ministry's health risk assessment in Part B of this report, the ministry may order the source of the contamination to conduct additional or more detailed sampling as part of a remediation strategy for the property. This sampling would be conducted according to the ministry's *Guideline for Use at Contaminated Sites in Ontario* (MOE 1997a).

3.0 Background

The ministry has been conducting environmental investigations in the Port Colborne area since the early 1970s. What led the ministry to conduct the intensive soil investigation in the Rodney Street community is explained in Section 3.3.

The Rodney Street community is one of the oldest residential areas in Port Colborne. Archival photographs show that the current road system in the Rodney Street community was in place in 1917. The Port Colborne municipal tax records obtained from the City of Port Colborne indicate that the date of home construction in this community ranges for the late 1800s to the 1970s, with about 40% of the homes built before 1920, about 54% of the homes built between 1920 and 1950, and roughly 6% of the homes built after 1950.

Historically, the Rodney Street community was situated between two large industries; Inco's nickel refinery situated to the east of Davis Street, and the Algoma Steel/Canada Furnace Company (Algoma) iron ore blast furnace situated west of the south end of Welland Street. Although metal deposition patterns in the Port Colborne area are strongly influenced by the predominant west-southwesterly winds, in the Rodney Street community the industries were so close that emissions from both facilities impacted the community. The histories of these two sources and a summary of their processes and possible emissions are briefly summarized in the following two Sections. The source of the information in Sections 3.1 and 3.2 regarding the operational histories of these two industries is largely derived from JWEL (2001a,b,c).

3.1 International Nickel Company Limited

From 1918 to 1984, the International Nickel Company Limited (Inco) operated a nickel refinery in the city of Port Colborne. Over this period of time, five operating periods are distinguishable, based on a report prepared for Inco (JWEL 2001c).

The first operating period from 1918 to 1930 was based on the Orford Process and electro-refining. The Orford Process separated the copper and nickel components of copper-nickel matte (impure copper-nickel sulphide) that was shipped to Port Colborne from Inco's facility in Copper Cliff. Once separated, the copper sulphide was oxidized to create SO_2 and blister copper and the nickel sulphide was roasted and reduced to create nickel oxide and fine refined nickel.

In the second operating period from 1931 to 1938, the Orford Process was discontinued and transferred to the Inco facility in Copper Cliff and nickel sulphide was shipped to the Port

Colborne facility for refining to produce nickel oxide. The crude oxide was further processed through a series of reverberatory-type anode furnaces that reduced the nickel oxide at high temperature to impure nickel metal. The impure nickel then went through electro-refining to produce nickel. The residue from the electro-refining was further processed to extract platinum, palladium, rhodium, ruthenium and iridium.

In the third operating period from 1939 to 1959, nickel sulphide concentrate was brought directly to the refinery where it initially went through a sintering process to aggregate fine particles and drive off the sulphur. Some of the sinter was sold and the rest was refined on site through the reverberatory-type anode furnaces. It was then further refined through a sulfate-chloride electrolyte process to produce nickel, cobalt oxide and residue. As in the second operating period, platinum, palladium, rhodium, ruthenium and iridium were extracted from the residue.

In the fourth operating period from 1960 to 1979, both nickel sulfide concentrate and nickel oxide concentrate were processed at the refinery. The nickel sulfide concentrate went directly to the anode casting, which produced a matte that was further processed by electro-refining. Along with nickel and cobalt oxide, sulphur was produced by the electro-refining process. The precious metals continued to be extracted from the electro-refining residue. In the 1960s, a Cortrell electrostatic precipitator was installed.

In the fifth operating period from 1980 to 1990, electro cobalt refining started in 1983 and the nickel refinery was shut down in 1984.

Emissions were not monitored over the operational life of the Inco refinery and so actual emissions are unknown. However, it has been estimated that approximately 18,000 tonnes of nickel was emitted by Inco over the operational life of the refinery (JWEL 2001c). Over 58% of the nickel released from the refinery was released prior to 1938 and over 97% of the total nickel emissions occurred before 1960. The highest rate of estimated annual nickel emissions, and likely the period during which the greatest amount of nickel deposition occurred, was 1939 to 1959 during which an estimated 11,466 tonnes of nickel was released to the surrounding environment (JWEL 2001c). Figure 1 illustrates the estimated nickel emissions from Inco during the six operating process time periods.

3.2 Algoma / Canada Furnace Company

The only other large industry known to historically operate in the vicinity of the Rodney Street community that had the potential to impact soil quality across the community was the Algoma iron smelter. This smelter operated from 1913 to 1977 and was originally owned by the Canadian Furnace Company Limited. In 1950, the site was purchased by Algoma Steel Corporation Limited and operated as the Canada Furnace Division of Algoma until the facility was closed in 1977. The smelter was subsequently demolished. In this report this iron industry is referred to as Algoma.

Algoma, which was located on the east side of the Welland canal bordering on the southwest corner of the Rodney Street community, had two blast furnaces at the Port Colborne facility. The iron ore or sinter, limestone and coke for the blast furnaces were shipped to Port Colborne and stored in open stockpiles next to the facility (JWEL 2001c). Initially, the ore (if not already sintered) was sintered in a single Greenawalt sintering plant. The sinter was loaded into the top of the blast furnaces along with coke (the reducing and thermal agent) and limestone (for fluxing of the gangue (rock/other minerals in the ore) material). A blast of hot air was introduced at the bottom of the furnace to burn the coke and thus heat, reduce, and melt the sinter and limestone as it descends to the bottom of the furnace. As the coke burns it releases carbon dioxide. Some of the carbon dioxide reacts with coke particles to create carbon monoxide (an endothermic reaction). The carbon monoxide reduces the iron oxides to iron. The molten iron and slag was collected at the bottom of the furnace and periodically poured (tapped) into iron ingots referred to as "pigs".

The waste gas from a blast furnace contains carbon monoxide plus particulate. The particulate was removed and the waste gas was used as fuel for heating the five stoves that Algoma used to heat the air for the two blast furnaces. Emissions from Algoma may have included arsenic, iron, magnesium, possibly beryllium, boron, manganese, calcium, phosphorous, sulphur, silicon, plus substantial particulates (dust).

3.3 Ontario Ministry of the Environment Investigations

Between the years 1972 and 1999, the Ontario Ministry of the Environment (MOE, or the ministry) conducted numerous investigations to document the impact of Inco's emissions on soil and vegetation in and around Port Colborne. These investigations concluded that emissions from over half a century of nickel refining had resulted in elevated levels of heavy metals in soil in various locations throughout the Port Colborne area. Nickel, copper and cobalt concentrations in surface soil (0 - 5 cm depth) were elevated in residential communities adjacent to Inco and for a considerable distance downwind (east-northeasterly) of the refinery to levels which could or did cause injury to vegetation (phytotoxicity). Generally, the observed vegetation impacts were to farm crops east of Inco and to silver maple trees east of the canal and south of Highway 3. Silver maple is a species of tree that is particularly sensitive to many pollutants, including nickel.

With the benefit of hindsight (MOE soil quality guidelines were not available prior to 1996), it is apparent that the levels of nickel, copper and cobalt identified in soil over a large area of Port Colborne as a result of these early MOE investigations are consistently and substantially elevated above MOE effects-based soil guidelines for phytotoxicity published in Table A of the MOE's Guideline (MOE 1997a). By contrast, the soil arsenic, zinc and selenium levels found in those investigations exceed normal Ontario Table F (MOE 1997A) background ranges in a few areas, and even less frequently exceed Table A effects-based guidelines for phytotoxicity (zinc) and effects on grazing animals (selenium). The soil arsenic levels in these early investigations very rarely exceeded the Table F background guidelines. Very occasional exceedences of the Table A

guideline for arsenic occurred in agricultural soil on farms immediately east of Inco. A more detailed description of the MOE soil guidelines, how they are intended to be used in the remediation of contaminated sites, and how the ministry uses them in environmental investigations, is provided in Section 4.0.

The MOE Table A effects-based guidelines for nickel, copper, cobalt, arsenic and zinc are all based on phytotoxicity (injury to vegetation). The Table A criterion for selenium is based on the protection of grazing animals. Numerous MOE studies conducted on Port Colborne farms in the 1970s and 1980s documented toxicity to agricultural crops as a result of ambient air SO_2 fumigations and heavy metal soil contamination (MOE 1977, MOE 1978b, Ontario Ministry of the Environment and Agriculture Canada 1980, MOE 1981; Ontario Ministry of the Environment and Ontario Ministry of Agriculture and Food 1983). Up to 1991, the highest soil nickel concentration that could be proven to exist through repeat sampling in the Port Colborne area was 9,750 µg/g (MOE 1994b). Higher soil nickel levels were very occasionally encountered at a few sample sites close to Inco, however, when the sites were re-sampled these high levels could not be validated. A human health risk assessment using this maximum soil nickel level (9,750 µg/g) was jointly published by the MOE and Regional Niagara Public Health Department in 1997. It was concluded that based on a multi-media assessment of potential risks, *no adverse health effects are anticipated to result from exposure to nickel, copper or cobalt in soils in the Port Colborne area* (MOE 1997d).

Additional extensive soil sampling was conducted by the MOE in the City of Port Colborne and the surrounding area in 1998 and 1999 and demonstrated that soil nickel concentrations exceed the MOE Table F soil background-based guideline of 43 µg/g up to 28 km downwind of the refinery, covering a 345 km² area of the Niagara peninsula (MOE 2000a, MOE 2000b). Furthermore, soil nickel levels exceeded the MOE Table A effects-based guideline of 200 µg/g for phytotoxicity for a distance of up to 3 km downwind of Inco over an area of almost 29 km². In addition, copper and cobalt also exceed their corresponding effects-based Table A soil guidelines in smaller areas of the community, mainly immediately east, north, and northeast of the refinery. The derivation and significance of MOE guidelines are described in Section 4.0.

MOE surface soil sampling conducted in 1991 in the vicinity of the Rodney Street community immediately west and northwest of Inco found that soil nickel concentrations in the general area ranged from about 2,000 µg/g to about 4,000 µg/g (MOE 1994a). However, this soil sampling was incidental to a study of general phytotoxicity impacts to agricultural areas of Port Colborne focussing on areas where earlier work had indicated both high soil concentrations and vegetation impacts. No properties on Rodney Street itself were sampled in these early ministry investigations. As a result, very few surface soil samples were collected and little depth sampling (deeper than 5 cm) was done in this part of Port Colborne (the highest soil nickel concentration at 5 - 10 cm was 2,750 µg/g). Concentrations in this range were confirmed by repeated sampling over several years, and so were believed by the ministry to represent soil metal levels in urban areas of Port Colborne.

During a public information forum held in January 2000 at the Port Colborne city hall, a resident of Rodney Street requested that the ministry sample soil on his property. MOE Phytotoxicology scientists sampled the front and back yards of the property in June 2000. Analysis of the soil samples revealed that soil nickel concentrations at depth (10 - 15 cm) were much higher (~14,000 µg/g) than previously believed to be present in this part of Port Colborne. In addition, soil copper, cobalt, arsenic, lead and zinc concentrations at depth also exceeded their respective MOE Table A effects-based guidelines. MOE human health toxicologists conducted a screening level risk assessment on the new soil data and determined that the health-based nickel reference dose was exceeded for the maximum nickel concentration found in the front yard of this Rodney Street property. The human health reference dose calculations incorporate considerable safety factors, and although an exceedence of the nickel reference dose does not mean that an adverse health effect will occur, it does trigger the need for further investigation.

As a result of the findings for the single Rodney Street property, the Medical Officer of Health requested additional sampling of residential properties on Rodney Street. This additional sampling of front and back yards was completed October 3 and 4, 2000. The results showed a wide variance in soil nickel concentrations from one property to the next, and in some cases between yards on the same property. The highest soil nickel concentration was 17,000 µg/g, but several properties were substantially above the 2,000 µg/g to 4,000 µg/g nickel range that was anticipated for this area of Port Colborne, based on the ministry's 1998 and 1999 studies (MOE 2000a, MOE 2000b). On some properties the nickel concentrations were highest in the surface soil and lower at depth, while on other properties the reverse was observed. Properties with higher soil metal levels were sometimes adjacent to properties with much lower metal concentrations. Soil nickel concentrations tended to be higher in the front yards of Rodney Street properties than the back yards. This may reflect re-entrainment of contaminated dust along the roads or snow plowing and piling of dust-contaminated snow. In addition to unexpectedly high nickel, copper, and cobalt levels, the soil zinc, arsenic and in some cases lead concentrations, were also elevated on some properties and were inconsistent with levels previously observed elsewhere in the Port Colborne area. While collecting soil from the Rodney Street properties it was observed that some areas of some yards had considerable non-soil material, such as concrete, brick, and wood rubble, metal pieces, and what appeared to be cinders, slag and possibly ash. This suggested that some areas of what is now Rodney Street may have historically received fill, possibly residential refuse, or industrial process waste.

In an open letter to the residents of Port Colborne (December, 2000) concerning nickel, copper and cobalt in surface soil in and around Port Colborne, Inco stated that, "*Inco no longer refines nickel in Port Colborne, but we do acknowledge these metal levels are the result of Inco's historic operations in Port Colborne*" (Inco 2000).

If the source of the unexpectedly high soil metal levels encountered on Rodney Street in October 2000 was contaminated fill then the extent of the soil contamination was anticipated to be quite limited (eg., isolated pockets, or isolated properties). If the source of the contamination was

fugitive or stack emissions from local industries then the soil concentrations would be expected to be more widespread and quite high over the general area and decrease with increasing distance from the source(s) (eg., properties beyond Rodney Street). The October 2000 Rodney Street data introduced new environmental information, in that the nickel levels were higher than anticipated and lead, arsenic, zinc, selenium and antimony were encountered above generic guidelines. Although Inco is the known source of nickel in the broader Port Colborne area, Inco may not be the source of these other contaminants.

The variability of the soil metal levels between properties made it difficult to judge the extent of the contamination. Therefore, in order to determine the extent and severity of surface soil metal contamination, all residential properties in the Rodney Street community were sampled by the ministry from November 8 to 17, 2000. In addition to the residential property sampling, soil trenches were dug at several locations in the vicinity of Rodney Street to determine if soil was contaminated at depth. Also, the City of Port Colborne requested that the MOE sample the playground located on the east side of Welland Street north of Nickel Street, as fill had been used in the construction of the berms in this park. The ministry also sampled several areas in the baseball park south of Rodney Street and west of Davis Street.

After the April, 2001 open house meeting hosted by the ministry in Port Colborne, several property owners notified the ministry that their properties, or areas on their properties, had not been sampled in the Rodney Street community-wide sampling program of November 2000. Between April and July 2001, the ministry sampled surface soil from additional residential properties. These additional properties were not sampled the previous November because access was unobtainable after repeated attempts, or they were vacant lots with obvious signs of very recent disturbance, or there was confusion about property boundaries. In at least one case, the property had been sampled in November 2000 but the owner asked that a specific section of the yard be sampled in 2001 that had not been previously sampled.

4.0 MOE Soil Guidelines

Interpretation of soil metal levels in this report is based on comparisons to the MOE *Guidelines for Use at Contaminated Sites in Ontario* (MOE 1997a). In 1996 the ministry published the soil guidelines (revised in 1997) to help owners of contaminated industrial property clean up the soil so that the property could be redeveloped for alternative uses. Although the soil guidelines were developed specifically for remediating single contaminated industrial properties, they have been used by both the ministry and the environmental consulting community to evaluate soil quality on a broader scale.

The use of the ministry's soil clean-up guidelines are voluntary, although the ministry can order property owners to follow them in order to remediate contaminated soil if the contamination has the potential to cause an off-property adverse effect, or the ministry can order the polluter to remediate if the contamination itself constitutes an off-property adverse effect.

The ministry's soil clean-up guidelines, including the SSRA approach, have been used successfully on hundreds of properties across Ontario to restore the economic and social value of previously contaminated lands.

The Table F guidelines represent background soil concentrations obtained from a MOE province-wide parkland sampling program. Soil concentrations that exceed Table F are usually indicative of a pollution source. The Table A soil guidelines are effects-based and were derived to protect both human health and the natural environment, whichever is potentially affected at the lowest concentration. Table A guidelines are not available for all chemical parameters, since for some elements there is insufficient scientific information available to establish effects-based values (i.e., strontium), or the element is considered non-toxic even at high concentrations (i.e., iron), or the element is a plant nutrient (i.e., magnesium).

An exceedence of a Table A guideline does not mean that an adverse effect will occur, it means that an effect may occur if the specific sensitive environmental receptor the guideline is intended to protect is present at the site and the soil conditions are such that the contaminant is readily bio-available (see Part A Section 5.3.3 for an explanation of plant bio-availability as it relates to ecological toxicity; Part B Appendix 5 explains bio-availability as it relates to human toxicology). For example, the nickel Table A guideline of 200 µg/g is intended to protect sensitive agricultural crops, specifically cereal grains such as oats, because these plants are injured at soil nickel levels far below most other plant species. In addition, even sensitive cereal crops will not be injured at 200 µg/g nickel unless the nickel in the soil is readily bio-available. Usually bio-availability of metals increases as: metal concentration increases; the concentration of soluble forms of the metal increase; soil pH decreases; soil cation exchange capacity decreases; soil nutrient levels decrease; soil organic matter decreases; and, soil clay content decreases/soil sand content increases.

The ministry's generic soil nickel guideline of 200 µg/g is based on eco-toxicity (injury to plants), because plants are the most sensitive environmental receptor. The ministry does not have a generic (Table A) soil nickel guideline based on human health. However, when a proponent uses the guideline to evaluate a property, the ministry has a value of 310 µg/g nickel in soil that triggers the need to conduct a human health risk assessment (MOE 1996). For example, if the soil nickel level is higher than 200 µg/g (the generic Table A guideline) but does not exceed 310 µg/g, the SSRA need only include an ecological risk assessment. If the soil nickel level is greater than 310 µg/g, the SSRA must include both an ecological risk assessment and a human health risk assessment. The 310 µg/g human health component value is neither a standard nor a guideline, it is only used as a screening level that triggers the requirement to conduct a human health risk assessment.

Regardless of the sensitivity of the soil and the presence of sensitive environmental parameters, the MOE generic soil guidelines are not used at all if a site specific risk assessment is used to evaluate the potential impact of the soil contamination and derive remedial options. The human

health risk assessment described in Part B of this report is part of a site specific risk assessment, and therefore the MOE generic soil guidelines do not apply to the Rodney Street community. However, the soil guidelines are referenced to allow comparison to background levels and evaluate the potential for adverse ecological and human health effects. For detailed information on the development and use of the ministry's soil quality guidelines, including all of the technical and rationale guideline documents, visit the ministry's soil guideline web page at http://www.ene.gov.on.ca/envision/decomm/index.htm.

5.0 Methods
5.1 Soil Sampling

In October 2000, sampling on 17 properties on Rodney Street was conducted to a depth of 15 cm. Since the results of this sampling showed metal levels were elevated to at least a depth of 15 cm, the sampling depth was increased to 20 cm for the soil sampling in the Rodney Street community in November 2000. Efforts were made to sample all yards on all properties in the Rodney Street community, but in some cases conditions made it impossible to collect an appropriate soil sample. For example, sampling to the 20 cm depth was not possible on every property, as occasionally stony fill was encountered. Also, some yards were covered with gravel, asphalt, or debris (e.g., vehicles, boats, car parts, construction material), which physically prevented the investigators from sampling soil. Generally, soil samples were not collected within one metre of driveways, walkways, building structures, fences and debris to reduce the likelihood of encountering local residential sources of contamination (e.g., driveway spills, eroded paint from painted surfaces). On some very small areas, this one metre buffer disqualified the area resulting in some properties not being sampled during the November 2000 investigation. At the property owner's/occupant's specific request, some of these small areas not sampled in November 2000 were sampled in April and May 2001.

Each property was assessed for sampling. A front and a back yard was usually identified, and on some properties a side yard was also included. Yards were discrete areas usually separated by physical structures such as driveways, fences, and buildings. A hand-held soil corer was utilized to collect a minimum of nine soil cores, while walking a grid, "W", or "X" pattern across the designated sampling area. Soil cores were usually collected from at least nine discrete areas of each yard sampled. Each soil core was divided into three depth intervals (0 - 5 cm, 5 - 10 cm and 10 - 20 cm) and the nine core sections for each of the three sample depths (e.g., 0 - 5 cm) were placed in one labelled polyethylene bag. The nine core sections per bag per yard are referred to as a composite soil sample.

Where only one yard was sampled using the composite single sample procedure, and soil could be obtained from all three depths, then soil would actually have been collected from a total of 27 discrete soil core sections per yard (1 yard X 9 cores X 3 depths = 27 discrete soil core sections). Duplicate sampling means the soil sampling procedure was performed twice over the same yard. A yard sampled in duplicate for which soil could be obtained from all three depths would

actually have had soil collected from a total of 54 discrete soil core sections per yard (1 yard X 9 cores X 2 replicates X 3 depths = 54 discrete soil core sections). Triplicate sampling means the soil sampling procedure was performed three times over the same yard. A yard sampled in triplicate for which soil could be obtained from all three depths would actually have had soil collected from a total of 81 discrete soil core sections per yard (1 yard X 9 cores X 3 replicates X 3 depths = 81 discrete soil core sections). Table 1 indicates how many discrete soil core sections were obtained on a property if soil was collected from all three depths and multiple yards on a property were sampled using single composite, duplicate, or triplicate sampling procedures. Assuming soil could be obtained from all three depths, the minimum number of discrete soil core sections sampled per property ranged from 27, representing 3 composite samples if only one yard was sampled, to as many as 243, representing 27 composite samples, if three yards were sampled in triplicate at all three depths. Figure 2 illustrates how a property was sampled.

The yards of all of the properties on Rodney Street sampled in October 2000 were sampled in duplicate. Of the properties in the Rodney Street community sampled in November 2000, most were sampled using single composites, while 10 percent of the properties were sampled using triplicate composites (typically two properties in each block). Properties in the Rodney Street community sampled in April and July, 2001 were composite sampled in duplicate. Duplicate and triplicate sampling was done to provide a measure of the sampling variability. Sample variability is described in Section 7.1.

5.2 Trench Sampling

Seven trenches, 2 metres long by 0.5 metre wide, were dug using a backhoe supplied by the City of Port Colborne. The trenching was done in October 2000 while the Rodney Street properties were being sampled. The purpose of the trenches was to obtain soil samples at depth to estimate how deep the contamination extended, and to observe the soil profile for signs of fill, refuse, or industrial process waste. Trenches were excavated to a depth at which contact was made with natural clay, which was about one metre in most trenches. Duplicate soil samples were removed from the sides of each trench using a trowel at three depths: 30 - 35 cm, 60 - 65 cm (which were within what appeared to be layers of coarser or discoloured soil and possibly some fill material), and 100 - 105 cm. Soil samples were placed in labelled, polyethylene bags.

The seven trenches were excavated from four areas:

1) Two trenches were excavated in the baseball diamond at the southwest corner of Davis Street and Rodney Street. The first trench was located on the outfield side of second base and the second trench was dug in the middle of the outfield.

2) Two trenches were excavated in the vacant lot situated on the south side of Rodney Street between Welland and Fares Streets. This lot was owned by Algoma from 1920 to 1992 and is currently owned by Inco. The first trench was about ten metres in from the southeast corner of

Welland and Rodney Streets, and the second trench was about ten metres in from the southwest corner of Fares and Rodney Streets.

3) One trench was excavated directly in front of the residence at 124 Rodney Street on the north shoulder of the road.

4) Two trenches were excavated on the west side of the playground located between Welland Street and Fares Street, north of Nickel Street. In addition, 0 - 5 cm, 5 - 10 cm and 10 - 15 cm duplicate soil samples were collected from eight sites along a sod-covered berm running around the north and west perimeter of the basketball court located in the same playground.

5.3 Soil Sample Preparation and Analyses
5.3.1 Metals and Hydrides

All soil samples were stored in locked vehicles while not in view of the investigators until they were delivered to the ministry's Ecological Standards and Toxicology Section for processing using standard MOE chain of custody and laboratory procedures (MOE 1993a). The samples were air dried and passed through a 2 mm sieve where vegetation and stony debris were removed, and then ground to pass through a 355 micron sieve. The fine soil fraction was transferred to the MOE Laboratory Services Branch (LSB). Because of the desire to have the analyses conducted as quickly as possible, LSB arranged to have the Rodney Street community soil samples analysed by Agat Laboratories, an accredited private environmental laboratory. LSB imposed a strict quality management regime on the private lab to ensure data integrity (refer to Section 5.4). The soil samples were analysed for the following metals: aluminum, barium, beryllium, cadmium, calcium, chromium, cobalt, copper, iron, lead, magnesium, manganese, molybdenum, nickel, strontium, vanadium and zinc. For these metals, samples at both the MOE LSB and Agat were analysed using an accredited analytical method comparable to MOE protocol E3073L1. In addition, arsenic, antimony and selenium (hydrides) were also included in the soil analysis. For the arsenic, antimony and selenium, samples at both MOE LSB and Agat were analysed using an accredited analytical method comparable to the MOE protocol E3245L1. Unless otherwise specifically identified, all the soil data in this report are reported as dry weight totals.

5.3.2 Determination of Soil pH

To assist in the interpretation of the plant bioavailability of the soil contaminant concentrations, soil pH was determined for a subset of 36 soil samples collected from across the ten blocks of the Rodney Street community. Soil pH was determined in distilled water using MOE standard protocols (MOE 1985).

5.3.3 Plant-bioavailable Soil Nickel

Between 1969 and 1979, the ministry conducted several investigations and prepared many reports on soil and vegetation chemistry in the Port Colborne area. During this time, soil was collected from urban and rural properties (typically mineral soil) and selected farm fields (both mineral soil and organic "muck" soil) to determine plant-bioavailable soil nickel concentrations using a weak ammonium acetate extraction. Ammonium acetate extraction was not done on samples collected in 2000 or 2001.

In order for the nickel in the soil to potentially cause plant injury the nickel must be dissolved in soil water so it can be taken up by plant roots. The amount of nickel dissolved in soil water depends on the chemical form of the nickel and the chemical and physical properties of the soil and is not always directly related to the amount of nickel in the soil. Therefore, higher soil nickel levels do not necessarily mean a greater risk of plant injury.

The plant-bioavailable nickel data in Part A of this report relates to soil conditions that affect the natural ecosystem, specifically vegetation. The ammonium acetate extraction used on soil is intended to simulate the leaching of nickel from the soil by natural rainfall, soil water movement, and normal soil microbial activities. The extraction process used to estimate the leaching of soil metals into the human gastrointestinal tract to evaluate the potential impact on human health from the ingestion of contaminated soil is an entirely different test and is covered in Appendix 5 of Part B of this report.

5.3.4 Nickel Speciation

Nickel can occur in various forms in the natural environment and industrial processes can produce several types of nickel. Examples of forms of nickel are nickel oxide, nickel sulphide, nickel subsulphide, nickel chloride, and metallic nickel. These various forms are often referred to as nickel species. The species of nickel in the soil can have an important impact on how available the nickel is and therefore its potential toxicity to both the natural ecosystem and human health.

Metal speciation in soil samples is non-routine, and few labs can provide quantitative results. Nickel speciation was conducted on selected soil samples from the Rodney Street community. The ministry shared processed soil composite samples from properties on Rodney Street with Inco and both organizations had qualitative tests conducted at different laboratories. Inco used their own research facility and the MOE used the Ministry of Northern Development and Mines Geoscience Laboratory in Sudbury. In addition, the ministry submitted selected soil samples for quantitative analysis to Lakefield Research in Ontario and to the Stanford Synchrotron Radiation Laboratory in California.

5.4 Data Management and Laboratory Quality Control

In order to expedite the analysis of the more than 1,500 soil samples collected from the Rodney Street community in November 2000, the MOE retained the services of Agat Laboratories, an accredited private laboratory. Agat analysed the samples collected from the Rodney Street community in November 2000. The MOE Laboratory Services Branch analysed the samples collected from the Rodney Street properties in October 2000, and the additional properties in the Rodney Street community collected from April to July, 2001. The management of the contract lab was carried out by senior scientists and managers of the MOE Laboratory Services Branch (LSB) Quality Management Unit. The contract with Agat Laboratories was established only after a thorough review of their proposal and laboratory procedures and a successful analysis of preselected test samples. The MOE analysed the first 100 soil samples from the November 2000 sampling of the Rodney Street community. These same 100 samples were then analysed by Agat Laboratories and the results compared with those obtained by the MOE LSB. This initial comparison was done by staff of the MOE LSB Quality Management Unit. The Agat Laboratory individual element results had to be within 20% of the corresponding MOE results for the same sample. Only after this first quality assurance target was met successfully were the remaining Rodney Street community samples sent to Agat Laboratories. This potential difference ($\pm 20\%$) is the analytical component of the combined sampling and analytical "error" described in Section 7.1.

All sample submissions sent to Agat Laboratories contained at least four "check" samples which had been previously analysed as part of the original 100 samples. Each submission also contained field replicate samples, which could be used to measure repeatability of the sampling and analytical processes. The preliminary acceptance criteria were 20% for the check samples and 50% for the field replicates. The field replicates had a higher acceptance bracket because it was known from previous work in Port Colborne that between-replicate variability increased as the soil contaminant concentration increased. This is a common occurrence for non-homogeneous samples.

Data was quality assured in several ways. Data checking was performed by the manager and a senior scientist of the MOE LSB Spectroscopy Section, as well as Phytotoxicology scientists. If the results for the "check" samples and the replicate data were acceptable, then the rest of the data were checked for outliers. Outliers were determined by reviewing the ratios of elements between samples in each laboratory submission. Several sample submissions were repeated because the ratios of certain elements did not match the observed general trend. In almost all cases, repeat analysis by Agat Laboratories, and in some cases by MOE, confirmed the original result. Repeat analysis was continued until the data either matched the original "check" samples or were confirmed by MOE analysis. Generally, outliers were found to be due to the improper use of dilution factors. Outlier sample results were either re-calculated or Agat Laboratories was required to repeat the analyses. The variability in field replicates that was finally reported was much less than the 50% used as an acceptance criteria. Once all these criteria were met, the data

were released to the principal authors for use in the preparation of this report.

Agat experienced some difficulty consistently obtaining the agreed to detection limits of 0.1 µg/g for selenium and 0.4 µg/g for antimony. However, since identification of samples exceeding the ministry's effects-based guidelines of 10 µg/g for selenium and 13 µg/g for antimony was the critical factor, occasional problems obtaining the agreed upon detection limits did not jeopardize data quality.

6.0 Results
6.1 Soil Results
6.1.1 Data Tables

Differences between summary statistics given in this report and the October 2001 draft report are because the summary statistics in the current report include both the 2000 and 2001 sample data for the Rodney Street community. The analytical results for all soil samples taken from residential properties in the Rodney Street community in 2000 and 2001 are listed in Table 2 (2000) and Table 3 (2001). Table 4 lists the soil results of the trench samples collected in 2000. Tables 2 and 3 are generically coded so that individual property addresses cannot be determined. All the owners/occupants of residential properties sampled in the Rodney Street community in 2000 and 2001 have been contacted and provided with a copy of these tables and the corresponding codes to allow them to identify the data pertaining to their properties.

In addition, the 2000 and 2001 residential soil data are summarized in four tables that list the minimum, maximum, mean and median datum plus the 10th to the 90th percentiles. Table 5 summarizes the 0 to 5 cm sampling depth from all residential properties, Table 6 summarizes the 5 to 10 cm depth data, Table 7 summarizes the 10 to 20 cm depth data and Table 8 summarizes the soil data by all depths combined.

The distribution of nickel in soil in the Rodney Street community is very skewed. The maximum soil nickel level is 17,000 µg/g, which was detected in a soil sample collected in the original Rodney Street soil sampling in October 2000, confirming that these very high soil nickel levels are not wide-spread throughout the community. Only 10% of the samples had soil nickel concentrations greater than 5,592µg/g and only 20% of the samples had soil nickel concentrations greater than 3,784 µg/g (see Table 8). The average soil nickel concentration on residential properties in the Rodney Street community is 2,508 µg/g, which is consistent with the data in the 1998 and 1999 Port Colborne MOE soil investigations that predicted the soil nickel levels in this community are in the range of 2,000 µg/g to 4,000 µg/g. The residential soil data are further discussed in Section 7.

6.1.2 Soil pH

Soil results for the 36 soil samples submitted for pH determination are listed in Table 9. The pH of soil in the Rodney Street community is very consistent, ranging from 6.85 to 7.76 and averaging 7.23. This neutral pH is characteristic of fine-textured mineral soil and common in surface soil in southern Ontario. The soil pH was determined on soil samples collected (mostly from) from the 0 - 5 cm sample depth.

6.1.3 Soil Nickel Plant-Bioavailability

The plant-bioavailability of nickel in Port Colborne pH-neutral mineral soil averaged 0.22%, whereas the plant-bioavailability of nickel in acidic organic (muck) soil averaged 8.49% (MOE 1975, MOE 1977, MOE 1978b). None of the samples analysed for plant-bioavailable nickel were collected from sites where slag or other process waste was observed, and because of the distance from Inco all sites were believed to have been contaminated through atmospheric deposition. The plant-bioavailable nickel levels were much higher in the muck soil. These soils are naturally more acidic, often with a pH less than 6.0, which would place much more nickel in soil water solution and therefore increase its availability to plants. With a neutral to slightly alkali pH (the average soil pH in the Rodney Street community is 7.23) and with most of the nickel being in the form of insoluble nickel oxide, very little of the nickel in the soil in the Rodney Street community is likely to be available for uptake by plants.

These results indicate that the potential for nickel to go into solution in the soil and be available for uptake by vegetation (plant-bioavailability) is very small for the type of mineral soil that predominates in the Port Colborne area. A very low ammonium acetate soil leach is consistent with nickel being in the form of the very insoluble nickel oxide or simply elemental nickel metal. In order for nickel in the soil to injure vegetation it must be dissolved in soil water, be taken up through the roots and translocated throughout the plant. With such low plant-availability there would be very little dissolved nickel in soil water resulting in a small potential for vegetation uptake and injury. This would explain the paucity of characteristic nickel injury symptoms on vegetation throughout Port Colborne, particularly in the Rodney Street community, even though soil nickel levels far exceed the ministry's effects-based Table A nickel soil guideline. After the shutdown of the refinery in 1984, nickel injury on sensitive species of vegetation has rarely been observed in Port Colborne. This corroborates the low soil nickel plant-bioavailability results and suggests that most of the nickel injury observed on vegetation up to 1984 was likely from absorption of nickel deposited on the foliage from ambient air, and not from translocation of soluble nickel from nickel-contaminated soil. No plant injury believed to be caused by nickel toxicity was observed in the Rodney Street community during the course of sampling in 2000 and 2001.

The low soil plant-bioavailability would also explain the generally poor relationship between soil nickel levels and nickel levels in residential garden produce observed in Port Colborne. The

nickel levels in garden produce were not consistently higher from properties that had high soil nickel concentrations. In fact, a linear relationship between plant nickel levels and soil nickel levels would not be expected. Plants growing in soil with high soil nickel levels may actually have lower tissue nickel levels than plants growing in soil with moderately high nickel levels, because nickel is toxic to plants and injured plants are less physiologically active and so less able to take up compounds dissolved in soil water. The relationship between nickel levels in soil and nickel levels in garden vegetables is given in greater detail in Part B, Appendix 4 of this report.

6.2 Nickel Speciation
6.2.1 Qualitative Nickel Speciation Results and Mineralogy

Nickel concentrations reported in Tables 2 to 4 are all total concentrations, with no distinction made regarding the form (species) of the nickel. Nickel is present in the soil in various forms (e.g., nickel oxide, nickel chloride, nickel sulphide, nickel subsulphide). In this section *Qualitative* nickel speciation results are presented. *Qualitative* means that the analytical techniques used can only determine whether a particular nickel species is present in the soil and not the concentration of the nickel species. *Quantification* of nickel species in soil is given in the next section entitled *Quantitative Nickel Speciation*. *Quantitative* means both the presence and the concentration of the various nickel species in soil can be determined.

Speciation of nickel in soil is non-routine, requiring specialized laboratory equipment and specially trained technicians/scientists. This type of analysis is not routinely conducted in environmental investigations, and was not previously done on Port Colborne soil samples. In previous MOE reports pertaining to soil contamination in Port Colborne, the metal concentrations have been reported as the "total" (refer to Section 5.3.1) amount of nickel in the soil. Prior to 2000, the MOE soil studies in Port Colborne did not speciate the various metal compounds in local soil. The main issue arising from the health related concerns expressed by the community was the species of nickel present in Rodney Street community soils. Of particular concern were the relative amounts of nickel oxide in the soil, as nickel oxide is a component of nickel refinery dust. Nickel refinery dust is a mixture of nickel compounds that is a potential lung carcinogen if inhaled.

Nickel speciation was conducted on selected soil samples collected from properties on Rodney Street (not the Rodney Street community, but just properties on Rodney Street) independently by both the Ontario Ministry of Northern Development and Mines (MNDM) Geoscience Laboratory in Sudbury (MNDM 2001) and by Inco (Inco 2001a). Jacques Whitford Environmental Ltd. (JWEL) also submitted selected soil samples from across Port Colborne for metal speciation as part of their sampling program to determine the contaminants of concern for the Community Based Risk Assessment currently underway in Port Colborne (Enpar 2001).

The MNDM report concluded that nickel oxide was detected in the magnetic fraction of each sample, and that no other nickel form or species was detected in either the magnetic or non-

magnetic fractions of any of the samples. The Inco report had similar conclusions: the only forms of nickel identified in soil samples collected from Rodney Street were elemental nickel, nickel alloys (i.e., nickel-copper alloy), and nickel oxide, but specifically neither sulphidic or halide forms of nickel were detected. The results from the more recent work conducted by JWEL concurred with both the MNDM and Inco reports, in that nickel oxide was the only nickel compound detected, with no evidence of either sulphate or sulphide nickel forms.

Some of the Rodney Street property soil samples examined for nickel speciation by MNDM and Inco were very heterogeneous, containing up to 40% non-soil material, such as wood, construction rubble, metal, and slag. The most abundant non-soil phase was iron oxide, but included metallic iron, carbonaceous particles that could be coke or coal dust (probably coke because of the presence of gas-bubbles), and the occasional arsenic grain hosted in iron oxide and associated with copper, nickel, antimony and molybdenum. The iron oxide was often in the form of dendritic iron oxide crystals, suggesting an artificial origin. Precious metal inclusions with platinum and palladium were identified in several particles in one sample, which were alloyed with copper and hosted in nickel oxide (Inco 2001a).

The origin of the non-soil material in these samples may be inferred from its chemistry. The presence of nickel, copper, cobalt and precious metals suggests an Inco origin, because Inco's emissions are known to have included nickel, copper and cobalt, and Inco currently re-processes it's waste to recover precious metals. However, the presence of quantities of manganese slag with high silicate and iron concentrations in some samples is more typical of blast furnace slag, and suggests some of the iron-based material found in soil from Rodney Street properties may have originated from Algoma.

Some areas of some of the properties in the Rodney Street Community may have received industrial fill from either Inco or Algoma, and that fill may have contributed to the elevated soil metal and arsenic levels on those properties. The current location and history of slag and related process waste on the former Algoma site is not currently known, however Inco has information of this nature for slag waste on its property. Orford slag is a waste from the Orford process used by Inco from 1918 to about 1938 to separate copper from nickel. A single Orford slag sample was examined by Inco from their on-site Orford slag pile and the results made available to the ministry (Inco 2001b). This Orford slag sample contained soda and sulphur, and had cobalt and copper levels that were about ten times higher than the average of the Rodney Street soil samples examined by Inco and the ministry. The Rodney Street soil iron levels are comparable to the slag sample and the soil nickel levels are about twice as high as in the slag sample. In addition, the nickel and copper-bearing phases present in the Orford slag are mostly sulfides, which is in contrast to the Rodney Street soil samples in which nickel oxide is the most common nickel form. These data suggest that the non-soil material collected from a few properties on Rodney Street is not primarily Orford slag from Inco. Because the non-soil or fill material does not resemble Orford slag does not mean it did not come from Inco, since it is not known when the fill was deposited in this area and Inco only used the Orford process for a portion of the refinery's

operating period. Over the operating life of the Inco refinery nickel oxide has been both a feedstock and product and so its association with wastes from Inco is not unexpected.

On Rodney Street and in the Rodney Street community, many factors have affected the soil composition and chemistry, including the possible dumping of waste materials from both Inco and Algoma, deposition of atmospheric emissions from both Algoma and Inco, domestic sources of contaminants, landscaping practices, and natural soil processes associated with soil microbial activity. Therefore, the soil chemistry of some properties in the Rodney Street community that appear to have received fill would not be expected to exactly match the chemical composition of a particular waste from any particular industrial source. Specifically, the nickel, nickel-copper alloys and nickel oxide, would be expected to have originated from Inco processes, and the manganese slag with high iron concentrations and coke-like material are more likely to have originated from Algoma processes. The qualitative speciation results and mineralogy data suggests that some soil on the from Rodney Street properties contain a mixture of waste materials from both Inco and Algoma.

6.2.2 Quantitative Nickel Speciation Results

The preceding section on qualitative nickel speciation reported that Rodney Street soil samples examined for nickel speciation for the ministry by MNDM and by Inco both confirmed the presence of nickel oxide but no other nickel species. These analyses could not quantify the concentration of nickel oxide. Therefore, the ministry contracted two additional laboratories to conduct further nickel speciation testing in an attempt to quantify the various nickel forms in the soil.

Quantification of metal speciation in soil samples is non-routine, leading edge science. The ministry sent 20 soil samples from the broader Rodney Street community to Lakefield Research in Ontario and six samples to the Stanford Synchrotron Radiation Laboratory in California. The results of these analyses are summarized in Table 10. The ministry's Laboratory Services Branch, in addition to analysing these same 20 samples for total nickel, also provided technical and scientific guidance on the speciation methods used by Lakefield and they sought independent input from the Federal government regarding Lakefield protocols.

Composite soil samples were collected from ten sites in the Rodney Street community. Each sample was split in two with half the sample processed according to standard MOE soil preparation protocols (drying, grinding and sieving) and the other half submitted as a dried unprocessed bulk sample. Sub-samples of all of these 20 samples were sent to Lakefield Laboratories for determination of total nickel and nickel species by a sequential leaching process. Sub-samples of the ten processed samples were sent to the MOE laboratory for total metal analysis. Two sub-samples of the processed samples were sent to Saint Mary's University for total metal analysis and to the Stanford laboratory for speciation analysis. Lakefield used a sequential leaching process to isolate and then quantify the various metal species groups. The

Stanford lab used SEM-XAFS (scanning electron microscope - X-ray absorption fine structure).

The Lakefield and Stanford laboratories were both able to provide quantitative estimates of speciated nickel. Lakefield identified four fractions of nickel in the processed and unprocessed samples; soluble nickel, nickel sulfide, nickel metal and nickel oxide. The processing status made no appreciable difference in the results. On average, nickel oxide comprised just over 80% (range from 54.6% to 87.7%) of the total nickel in the Rodney Street soil samples analysed by Lakefield. Lakefield found nickel metal present at 11.30%, nickel sulphide at 7.66%, and soluble nickel at 0.37% of the total nickel detected in the processed soil. By comparison, the six samples analysed by the Stanford lab averaged 89.5% nickel oxide, but this lab did not observe any other nickel species. Inco's ongoing analytical work in Port Colborne indicates that the majority of the nickel present in soil in the Rodney Street community is in the form of nickel oxide with traces of nickel metal and nickel:copper and nickel:cobalt alloys. However, Inco has not identified nickel sulphide or other nickel species in any of the Rodney Street soil samples it has examined.

The analytical procedures employed by the Lakefield and Stanford labs are very different, and at this time there is not a weight of evidence to suggest that one process is more accurate or more reliable than the other. In this case there are three labs (Inco, Enpar and Stanford), all using Scanning Electron Microscopy technology, that detected only nickel oxide, and one lab (Lakefield) that detected traces of other nickel species in addition to nickel oxide. All labs concur that nickel oxide is the most abundant nickel species present in the soil. Therefore, the conclusions to be drawn from the new nickel speciation tests reaffirm the position originally stated by the ministry that the majority of the nickel in the soil in the Rodney Street community is in the form of nickel oxide. Considering the uncertainty associated, at this time, with this leading edge analytical technology, the presence of nickel species other than nickel oxide in trace concentrations in soil in Port Colborne is speculative until it can be confirmed with repeat analysis of samples from multiple locations using different laboratories.

Table 11 summarizes data from a 1978 report of analyses of Port Colborne refinery dusts (MOE 1978a). This report provides elemental analysis of dust samples collected from the Cottrell Precipitator, the Cobalt Multiclone Stack, the Tumblast Stack, and the Submerged Combustion Evaporator Stack. It must be noted that these data reflect results from one sample from each of the precipitator or stacks collected on a single day and therefore may not be representative of emissions from Inco over its operating life. As mentioned in this 1978 report, the data from an onsite Cottrell Precipitator captured dust from the Anode Reverb Furnaces during charging, smelting, and on-stream periods of operation, and so should reflect the particulates present in stack and fugitive emissions at the time of sampling in 1978. The precipitator dust was 38.7% nickel, 10.5% lead, 7.6% copper, 7.1% sulphur, 0.66% cobalt, 0.61% iron, 0.38% arsenic and 0.14% zinc. The main nickel component was reported as nickel oxide and the main lead component was reported as lead sulphate. In addition, "minor phases" (not quantified) of hydrated nickel and copper sulphate were identified. Therefore, most of the nickel in precipitator dust, and so likely in the stack and fugitive emissions at the time of sampling in 1978, was nickel

oxide.

It is important to note that the MOE Table A effects-based guideline for nickel was developed for the total nickel concentration in the soil and is not based on nickel oxide; i.e., it is a nickel guideline, not a nickel oxide guideline. The ministry does not have a nickel oxide guideline for soil, nor is the ministry aware of a nickel oxide soil guideline for any other environmental agency.

7.0 Discussion
7.1 Sample Variability and Data Confidence

Section 5.1 discussed the strategy used to sample the properties in the Rodney Street community. The sample strategy used in the Rodney Street community was appropriate for its intended purpose, which was to collect information on soil contaminant levels that reflect representative environmental exposures to people using the property. All sampling strategies, no matter how thorough, have inherent variability because they can only estimate, not measure, the soil chemical concentrations. The term used to identify this inherent variability is "sampling error". It is a statistical term used to identify uncertainty, it does not imply the data are wrong.

Statistical analysis was carried out on the replicated samples collected from the two properties on each block that were sampled in triplicate (single samples were collected from all other properties in the Rodney Street community and the properties on Rodney Street were sampled in duplicate). Within-site sampling and analytical variability was acceptable for all elements excluding antimony and selenium, in that the standard deviation of the replicates was less than 20% of the mean value for the property. The standard deviations of the replicate samples for antimony and selenium, expressed as percentages of the mean concentration, were 24.2% and 24.6%, respectively. The concentrations of both antimony and selenium are naturally very low in soil, usually less than 0.2 $\mu g/g$. The high variability between sample replicates for these two elements was related to the difficulty that the contract laboratory had in consistently obtaining detection limits that were in the 0.2 $\mu g/g$ range. A low detection limit for these elements was not a selection criteria for the contract lab, and since the concern was not to miss high soil levels, the problem of detection limit consistency did not jeopardize data quality.

7.2 Soil Results for Residential Properties

Summary statistics presented in this section for soil results from residential properties are based on samples collected in both 2000 and 2001. Summary statistics given in the October 2001 draft document are slightly different because they were based on only the 2000 sampling results.

MOE Table A effects-based generic criteria (residential/parkland land uses - medium/fine textured soils) were exceeded in soil on one or more of the residential properties in the Rodney Street community for the following ten elements: antimony, arsenic, beryllium, cadmium, cobalt,

copper, lead, nickel, selenium and zinc. The Table A guideline (refer to Part A Section 4.0) for lead, antimony and beryllium are based on human health. These are intended for generic clean-up or as a trigger for a detailed human health risk assessment. The criterion for selenium is based on the protection of grazing animals. The criteria for arsenic, cadmium, cobalt, copper, nickel and zinc are based on ecological protection, specifically plant growth. Soil nickel concentrations exceed the Table A effects-based generic criterion of 200 μg/g on almost all of the properties in the Rodney Street community. It was evident from the condition of the few properties that did not exceed the nickel guideline that they had undergone extensive landscaping, so the contaminated soil had either been buried below the 20 cm sampling depth, or been removed and replaced with clean soil.

Elevated soil nickel levels were expected in the Rodney Street community, since the contaminant contour maps prepared for the 1998 and 1999 MOE Port Colborne soil investigations indicated that soil nickel concentrations in the area could range up to 5,000 μg/g. Similarly, soil cobalt and copper concentrations were expected to be high, and about 70% of the properties in the Rodney Street community exceeded the Table A effects-based criteria for these two elements (Table 12). A high percentage (approximately 70%) of the properties sampled in this investigation also had soil lead levels above the Table A effects-based criterion. Soil beryllium levels on almost 50% of the properties exceeded the Table A effects-based criterion. The effects-based criteria for arsenic and zinc were exceeded on about 50% and 40% of the properties, respectively. Table A guideline exceedences were rare for antimony, cadmium, and selenium, occurring on only three properties (about 2 %) for antimony, and one property each (less than 1%) for cadmium and selenium.

7.3 Contaminant Contour Maps
7.3.1 Map Preparations and Precautions

To illustrate the spatial distribution of soil contamination in the Rodney Street community, contaminant contour maps were created for selected elements using Surfer™ and ArcView™ computer mapping programmes. Because of the technical complexities associated with creating contour maps from a very large data base, and because no spatial pattern was evident for some elements, maps were created only for the elements of specific interest and those that exceeded the MOE Table A effects-based guidelines for which the guideline rationale was health-based. Zinc was excluded, because with few exceptions, the exceedences of the Table A effects-based criteria were marginal, and the rationale for the guideline is not health-based. Iron was included because it is a potential emission from both Inco and Algoma, even though environmentally it is essentially a benign contaminant, a spatial pattern may assist in contaminant source allocation. Therefore, maps were prepared for the following ten elements: antimony, arsenic, beryllium, cadmium, cobalt, copper, lead, nickel, selenium and iron (Maps 1 - 30). A separate contaminant contour map was produced for each of the three sampling depths (0 - 5 cm, 5 - 10 cm and 10 - 20 cm) for each element. All the soil data collected in both 2000 and 2001 from the Rodney Street community, excluding the trench samples, was included in the contaminant contour maps. Therefore, the contour maps in this report may appear slightly different than the contour maps in

the October 2001 draft report, which were created using only the 2000 soil data.

Two software packages were used to generate the maps. The data analysis and creation of the concentration contours were produced using Surfer™ (Version 7.0 for Windows 95/NT, by Golden Software Inc.). The output from Surfer™ was then imported into ArcView™ GIS (Version 3.2, by Environmental Systems Research Institute Inc.) and combined with base maps to produce the final maps. Details concerning the process used to generate the maps are provided in Part A Soil Contour Maps and Figures.

These maps are statistical approximations of the spatial distribution of the different contaminants. Soil concentrations are only known with certainty at those sites for which soil was actually sampled and chemically analysed. The contours produced by the program are significantly affected by the spatial distribution of the sampling sites, the accuracy of the position information of the sampling sites, and the program options used to generate the contours. The accuracy of the contours diminishes at the edges of the map and in large areas where there are no or very few sample sites. In this case, the sampling density was almost uniform across the community inside Welland, Rodney, Davis and Louis Streets, therefore the contours should accurately reflect the residential soil conditions for the purposes used in the report. The contours may be less accurate in the area south of Rodney Street in the vicinity of the vacant lot to the southwest and the baseball park to the southeast because the density of the sampling was much lower in these two areas then elsewhere in the community. In addition, contours indicating high metal concentrations at the edge of the map (e.g., Map 24) give the impression that the area of high contamination extends beyond the edges of the map, which may not be the case because there were no sample sites on the other side of the streets at the east, north, and west map edges or beyond the vacant lot and ball park at the south end of the maps.

These maps are very helpful tools for identifying spatial trends, particularly for very large data sets. Although useful and reasonably accurate, particularly with a high sampling density as used in this study, the contour maps are still only estimates of soil concentrations based on a statistical model. The actual soil concentration is known with certainty only at the sites where the samples were collected. Therefore, the maps by themselves should not be used to "prove" a contaminant source, but should be considered relative to all available data to assist with determination of source allocation.

7.3.2 Soil Contaminant Patterns

It is evident from the contaminant contour maps, and the data in Tables 2 to 4, that soil contamination in the Rodney Street community, although extensive for some elements, tends to be patchy. Properties with much lower soil contaminant levels were often encountered between properties with much higher concentrations. Conversely, occasionally single properties with significantly elevated concentrations of some elements were surrounded by properties with much lower contaminant levels. The mechanism for the observed patchwork pattern of contamination

is related to the interaction between atmospheric deposition, placement of contaminated fill and property specific landscaping and construction activities.

Atmospheric deposition, either from fugitive or stack emissions, would result in a decreasing soil contamination gradient relative to increasing distance from the source. Fugitive emissions can be considered a series of small point sources from vents, windows, doors or other openings in the refinery or other buildings. Fugitive emissions can be a significant component of the total emissions, but they are difficult to quantify. The particulates from fugitive sources would be carried by wind currents a short distance into the surrounding community and would not necessarily be deposited evenly due to wind currents eddying around structures like trees and buildings. Particulates emitted from the stack drop out of the plume as their weight overcomes the buoying effect of the hot rising gases. Depending on the wind speed, particle size, and stack gas temperature and velocity, the particles in stack emissions can travel considerable distances before they reach the ground. For both fugitive and stack emissions, the larger denser particles will tend to be deposited closest to the source and the finer particles will tend to be carried the greatest distance. Because it is so close to both Inco and Algoma, the Rodney Street community would tend to get more deposition of larger, denser particles and be affected more by fugitive emissions than more distant areas, which would be impacted relatively more by stack emissions.

Atmospheric deposition by both stack and fugitive emissions was not constant over the operating life of Inco and Algoma. Releases to the local environment, particularly through fugitive emissions, were much greater in the early years of operation at both Inco and Algoma. An estimated 97% of the total nickel emitted from Inco was released before 1959 (Figure 1) and about 75% of the total amount of iron released from Algoma was emitted before 1959 (JWEL 2001c). Subsequent to the cessation of emissions (Inco ceased operations in 1984 and Algoma in 1977) and over time through property landscaping or redevelopment, on some properties the contaminated soil would be either diluted by coverage with clean soil or removed and replaced by clean soil. Landscaping need not be elaborate to substantially alter the surface soil (top few cm) contaminant levels. Simply filling low spots in a lawn with topsoil or re-sodding can add enough clean soil to dilute the residual surficial contamination. The contamination status of undisturbed/unlandscaped properties would remain relatively unchanged in the years since the sources closed, to create the soil contamination patchwork pattern that was observed across the Rodney Street community.

With time, even on undisturbed properties, soil contamination deposited on the surface can work its way downwards into the lower soil horizons where it is subject to further mixing and redistribution both up and down by normal soil processes (ants, earthworms, water movement, etc.). Also, industrial process waste could have been used to fill low areas, and since the waste is usually aggregate in nature, some residents may have deliberately brought it onto their property to improve drainage around homes or structures, or as construction material. Atmospheric deposition over the decades would add to the general contaminant levels on properties that did not receive fill material.

Regardless of the patchiness, some overall contaminant contour gradients and patterns are evident. The most consistent are nickel, copper, cobalt, arsenic and selenium. The concentrations of these five elements tended to be highest in the easterly and southeasterly areas of the Rodney Street community, adjacent to the Inco refinery. The patterns of nickel (Maps 22 - 24), copper (Maps 16 - 18) and cobalt (Maps 13 - 15) soil contamination were particularly similar, with the higher concentrations restricted to properties along Rodney, Davis and Mitchell Streets. The maximum soil nickel level was 17,000 µg/g detected in the 5 - 10 cm depth of a property on Rodney Street. The maximum soil copper concentration was 2,720 µg/g in a sample from the 10 - 20 cm depth of a Mitchell Street property. The highest soil cobalt concentration, also from a Mitchell Street property, was 262 µg/g in the 5 - 10 cm soil profile. Although properties with high nickel levels also had elevated copper and cobalt concentrations, the maxima for these elements did not occur on the same property.

The patterns of soil arsenic (Maps 4 - 6) and soil selenium (Maps 25 - 27) contamination were similar. The contours are driven by the elevated arsenic and selenium levels in soil on properties along Rodney Street, with the remaining contaminated properties tending to be scattered in the eastern part of the community on properties along Mitchell Street, and a few on Davis Street. Unlike nickel, copper, and cobalt, which exceed their respective Table A effects-based guidelines on the majority of properties in the Rodney Street community, the extent of arsenic and selenium contamination was much more restricted, with concentrations that were proportionately much lower. The maximum soil arsenic concentration was 350 µg/g in the 0 to 5 cm depth from a property on Rodney Street. However, most soil arsenic levels were much less, with 70% of the soil samples collected in the Rodney Street community being below the Table A effects-based guideline of 20 µg/g and about 60% of the soil samples collected being within normal arsenic background concentrations (Table 8). The property with the highest soil arsenic level was not the property with the highest soil nickel level.

Although a soil selenium gradient toward Inco was evident, only one property had selenium levels that exceeded the Table A effects-based guideline, with a maximum concentration of 19.4 µg/g in soil collected from the 5 - 10 cm depth of a property on Mitchell Street. Soil selenium levels are naturally low and therefore any elevation above background is noticeable, a fact that allowed for a contaminant gradient to be evident.

Even though soil beryllium levels exceeded the Table A effects-based guideline of 1.2 µg/g on about 47% of the properties in the Rodney Street community, unlike the other nine elements for which maps were constructed, the beryllium contaminant contour maps (Maps 7 - 9) did not indicate a consistent spatial pattern. There was a weak trend of generally slightly higher levels toward the west side of the community, but the highest individual levels occurred toward the east and northeast. The highest soil beryllium level was 4.6 µg/g, which was detected in the 10 - 20 cm depth at a property on Mitchell Street. Like the other metals, soil beryllium levels tended to be slightly higher at depth.

The contaminant contour maps for lead (Maps 19 - 21), and to a lesser degree for cadmium (Maps 10 - 12) and antimony (Maps 1 - 3), did not illustrate a consistent spatial pattern relative to either Inco or Algoma or orient to specific streets, but rather identified numerous apparently random "hot spots". Soil lead levels exceeded the MOE Table A effects-based guideline of 200 μg/g on about 73% of the properties, whereas cadmium and antimony exceeded the MOE guidelines only on about 1% of the properties (Table 12). Even though the three elements appeared to be spatially related to each other (same general patterns on the contour maps) the soil lead concentrations were far higher than either the cadmium or antimony levels and the maximum concentrations did not occur on the same properties. For example, the maximum soil lead level was 1,800 μg/g, which occurred on a property on Mitchell Street. The maximum soil antimony level was 91 μg/g, encountered on a Louis Street property. The maximum soil cadmium concentration was 35 μg/g, which occurred on a Davis Street property. Like the other metals, these elements tended to be slightly higher at depth.

The soil iron concentrations in the Rodney Street community are quite variable and in some areas are substantially elevated. The mean iron concentration was 29,706 μg/g, which is within a normal range for Ontario, but the maximum iron level was 140,000 μg/g. The pattern of iron distribution in soil (Maps 28 - 30) most closely resembles the nickel pattern, with the highest concentrations occurring toward the south and east, obviously centred on the homes on Rodney Street east of Fares Street. Although both Algoma and Inco were potential iron sources, Algoma was a pig iron blast furnace, which are characteristically "dusty" operations. The Inco ore mined in Sudbury was iron-based (Pyrrhotite, Pentlandite, Chalcopyrite, Pyrite, Magnetite, Bornite) and some of the iron would have remained in the matte refined at Port Colborne. JWEL (2001c) estimated the total iron emissions from Algoma at just over 8,900 tonnes, compared to an estimated 111 tonnes of iron from Inco (Table 13).

7.4 Statistical Analysis of Chemical Relationships

The concentrations of some of the chemicals in soil tended to be slightly higher in the lower sample depths. Table 14 summarizes the Rodney Street community 2000 and 2001 soil concentrations by sample depth and lists the upper and lower 95% confidence limits around the mean values. These data clearly illustrate that the difference between depths is statistically significant for many elements.

Results of Pearson Product Correlation tests on the soil data for each depth and all depths combined for all chemicals are summarized in Tables 15, 15.1, 15.2 and 15.3. Due to the very large number of degrees of freedom (1,500 plus) all *r* values greater than 0.088 are significantly correlated at the 95% level, and *r* values greater than 0.115 are significant at 99%. Statistical tests are one tool that can be used to evaluate these data. The *r* values provide a measure of the strength of relationship between the elements, with the higher the *r* value the stronger the correlation. Negative *r* values indicate an inverse relationship (i.e., one soil concentration increases as the other decreases). Similar Pearson Product correlation tables were prepared for

each of the three sample depths: Table 15.1 (0 - 5 cm), Table 15.2 (5 - 10 cm) and Table 15.3 (10 - 20 cm). Since the Rodney Street community is very close to two historic industrial sources, and both sources are towards the south end of the community, atmospheric deposition from both sources could contaminate the community and the contaminants may appear through statistical testing to be spatially correlated even though the contaminants originated from different sources. Relational statistics are a useful tool for evaluating source allocation, but like the contaminant contour maps they should not be used in isolation.

Nickel, copper and cobalt contamination in surface soil in the broader Port Colborne community is associated with Inco emissions. Of these three elements, nickel can be considered a "signature" contaminant for Inco, since the Port Colborne facility was a nickel refinery and elevated nickel concentrations have been found over an extensive area around the refinery and there is a consistent soil nickel concentration gradient relative to distance and direction from Inco. Elements in soil that are highly statistically correlated with nickel are likely to be related to Inco emissions. Similarly, iron can be considered a signature contaminant for Algoma, because the Port Colborne operation was a pig iron blast furnace and emitted large quantities of iron. Elements in soil that are highly statistically correlated with iron are likely to be related to Algoma emissions. Some elements may have been emitted from both Inco and Algoma. Using the common sense approach that nickel is from Inco and iron is from Algoma, if the element has a higher correlation coefficient with nickel then Inco is probably the principal source, if it has a higher r value with iron then Algoma is probably the principal source.

Soil nickel concentrations in the Rodney Street community are very highly correlated with soil cobalt (r=0.929), copper (r=0.878), iron (r=0.798), selenium (r=0.764), zinc (r=0.733) levels, and highly correlated with soil arsenic (r=0.629) levels, suggesting that these elements are associated with Inco emissions. The high statistical correlation is corroborated by the contaminant contour maps which strongly imply a spatial relationship relative to Inco and between nickel, copper, cobalt, and to a lesser extent arsenic, selenium and iron (zinc was not mapped).

Previous MOE soil sampling in the Port Colborne area identified elevated soil nickel, copper and cobalt levels as having originated from Inco (MOE 2001a, MOE 2001b). However zinc, arsenic, selenium and iron levels in soil in areas other than the Rodney Street community have not been shown to be consistently elevated above MOE guidelines, suggesting additional or other sources than Inco may have contributed to these levels in the Rodney Street community. Arsenic levels in soil in the Inco regional plume zone are spatially and statistically related to nickel levels in soil, even though the soil arsenic levels are generally not elevated above either the MOE generic Table A or Table F guidelines. In the MOE report on re-sampling of soil at Humberstone School, the relationship between soil arsenic and soil nickel levels was so consistent that soil arsenic levels could be predicted by soil nickel levels to within a few parts per million (MOE 2001a). The relationship between soil arsenic and soil nickel levels in the Rodney Street community, although statistically highly correlated, is not quite as predictive (less consistent) as in the wider Port Colborne area downwind of the Inco stack. This suggests there may be a secondary arsenic

source in the Rodney Street community.

Figure 3 illustrates arsenic concentrations in surface soil across the regional Port Colborne area. This map was not prepared for the 1998 MOE reports because the soil samples were not analysed for arsenic, as the arsenic concentrations in soil collected during the MOE studies through the 1970s and 1980s indicated that arsenic levels rarely exceeded background. Archived 1998 and 1999 MOE samples were analysed by JWEL as part of the CBRA program and Figure 3 was created using the same Surfer and ArcView settings as the 1998/1999 MOE regional soil contour maps. MOE 2000 and 2001 Rodney Street community soil arsenic data were not used to create Figure 3. Figure 3 clearly illustrates that, even though the concentrations are below the MOE generic soil guideline of 20 µg/g, there is an arsenic plume downwind of Inco that is remarkably similar to the contours of nickel in surface soil. Figure 3 also illustrates that in the Rodney Street community there appears to be a secondary arsenic plume originating in the vicinity of Algoma and extending well into the Rodney Street community.

Soil iron levels are highly correlated (® greater than 0.6) with cobalt, nickel, arsenic, copper, selenium and zinc. With the exception of arsenic, the coefficients of correlation are slightly lower between iron and these metals than they are between these metals and nickel, suggesting a somewhat stronger/more consistent relationship with Inco that Algoma. In the Rodney Street community, the correlation between iron and arsenic (r=0.636) is marginally greater than between nickel and arsenic (r=0.629), reaffirming the apparent spatial relationship in Figure 3 that suggests Algoma was an arsenic source in the Rodney Street community.

Soil lead levels are highly correlated with zinc (r=0.753) and barium (r=0.724). These three elements are common components of lead-based paint. Also, the historic use of leaded gasoline has substantially added to the soil lead levels in all urban areas. Even though lead was likely emitted from Inco (lead made up about 10% of a single precipitator dust sample collected in 1978 (MOE 1978a)) the complete lack of a soil lead spatial pattern relative to either Inco or Algoma, suggests that the lead in the soil in the Rodney Street community is associated with general urban and domestic residential sources (lead based exterior paint, disposal of battery and automotive parts, pesticide use, and leaded gasoline). Any lead deposited in the Rodney Street community by Inco or Algoma cannot be distinguished from general urban and residential sources.

7.5 Results of Trench Samples

The results of chemical analysis of soil samples removed from the walls of the various trenches are summarized in Table 4. All seven of the trenches contained some non-soil or fill-like material, which included construction debris, brick pieces, metal and in some cases possibly coal, coal ash, cinders and slag. Some trenches had much more of this material than others. Natural undisturbed clay was encountered at about one metre in all trenches. The main contaminants in the trench soil are nickel, copper, cobalt, zinc, iron and to a lesser degree lead, arsenic and

beryllium. The iron concentrations are quite elevated in some trench samples, ranging almost to 17% (168,000 µg/g at 60 cm depth from the trench on the shoulder of Rodney Street). In addition to iron enriched fill, these high iron levels in soil samples from some of the trenches may also be from oxidized metal debris observed in some soil layers.

The two trenches from the baseball park at the south end of Rodney Street were contaminated with nickel to the bottom of the trench, a depth of about one metre, with concentrations ranging from 304 µg/g to 6,680 µg/g. The maximum arsenic level was 33.1 µg/g, the maximum copper level was 524 µg/g, and the maximum cobalt concentration was 88.8 µg/g. The nickel, copper, cobalt and arsenic levels all tended to be higher at depth. Most other elements, notably lead, were quite low, at least relative to elsewhere in the Rodney Street community.

The trench excavated on the shoulder of Rodney Street, and the two trenches excavated in the vacant lot south of Rodney Street between Fares and Welland Streets, were similar to each other and different from the baseball park trenches in that the maximum contaminant levels tended to be closer to the surface. For example, in the trench at the shoulder of the road near 124 Rodney Street, the soil nickel levels ranged from 8,900 µg/g to 9,730 µg/g to a depth of approximately 35 cm and then decreased to 204 µg/g at a depth of approximately 60 cm. Similarly, the arsenic concentrations ranged from 30.7 µg/g to 43.1 µg/g in the top 65 cm, then fell to background below this depth. The contaminant levels in the trenches from the vacant field tended to be lower than in the Rodney Street and baseball park trenches. Unlike the baseball park trenches, which had high nickel levels at all depths, the trench on the shoulder of Rodney Street and both trenches in the vacant field had the highest metal levels near the surface, with the layer of nickel contamination abruptly ending between 30 and 60 cms.

The two trenches excavated in the parkette on the east side of Welland Street tended to have lower soil contaminant levels than the other trenches. Although nickel levels were elevated to the bottom in the west trench, all other contaminants were confined to the top 65 cm. Similarly, in the east trench all the contamination was confined to the top 65 cm, falling to virtually background levels below this depth. By comparison, the soil from the two sodded berms located on the perimeter of the parkette's basketball court was much cleaner than the trenches. Only a few samples exceeded the MOE Table F background-based guidelines, and only a single sample exceeded the Table A effects-based guideline for beryllium.

Soil contamination was deepest in the baseball park. Based on the depth of metal contamination and the physical appearance of the trench faces, it is clear that the ball park had received at least one metre of metal-contaminated fill. Anecdotal accounts obtained from community residents during the MOE's April open house suggested that most of the area of what is now the baseball park had been filled. Judging by the presence of debris in the other trenches, it was evident these areas had also received some fill material, although metal contamination was mostly confined to the upper 30 - 60 cm. This suggests that outside of the baseball park, metal-contaminated material may have been used more as top dressing rather than as fill, perhaps to level the ground

in preparation for or subsequent to building. It is also possible that, outside of the obvious deep fill in the baseball park, the bulk of the soil metal contamination in the area of the trenches in the vacant field south of Rodney Street is from atmospheric deposition because the contamination is largely confined to the near surface layer and this area is very close to and directly between both the Inco refinery and the Algoma foundry. If this is the case, and most of the soil metal levels in the broader Rodney Street community are also related to atmospheric deposition, then the soil contamination in the Rodney Street community can be expected to extend to at least 30 cm in depth. However, the only way to confidently characterize the extent of soil contamination at depth is to conduct bore hole or trench sampling at multiple locations throughout the community.

As part of the ongoing environmental sampling for the CBRA, JWEL conducted some test pit sampling across the community in 2001. Two of these test pits (front yard 106 Rodney Street and front yard 91 Rodney Street) were clearly on residential properties. Evidence of slag was detected in the top 40 cm of lawn soil at these two Rodney Street residences, indicating at least a portion of these properties may have been top-dressed or levelled with fill (JWEL 2001b). In both of these test pits the soil nickel levels were highest at about 10 cm and the greatest concentration gradient was between 15 and 30 cm, where the nickel levels fell rapidly from many thousands of ppm to hundreds of ppm or lower. This is consistent with the limited trench sampling conducted by the ministry, corroborating the conclusion that the elevated soil metal and arsenic levels in the Rodney Street community are not likely to be deeper than about 30 cm, unless specific properties have received abundant fill material.

7.6 Contaminant Movement in Soil

If the amount of atmospheric metal deposition were constant and ongoing, the upper most soil layer would have the highest metal concentration because the rate of accumulation at the surface exceeds the rate of downward movement. Fugitive and stack emissions from Inco in the early years of operation, particularly before the 152 m stack was constructed in 1935 (there were two 91 m stacks in place from 1918), would have caused higher levels of atmospheric metal loading and subsequent deposition closer to Inco, particularly in the Rodney Street community, and resulted in the rapid accumulation of metals in surface soil. The pattern of slightly higher soil metal levels on front yards may reflect the influence of re-entrainment by vehicles, surface runoff and/or contaminated snow from the roads plowed onto the shoulders or narrow front yards. With no change in production levels at the refinery the impact of stack emissions and fugitive emissions on the Rodney Street community would have been reduced after the tall stack was constructed. However, Figure 1 indicates that in the period 1939-1959, the amount of nickel emitted increased over four fold, and therefore the rate of deposition in the Rodney Street community may not have decreased at all. With a reduction in production and further reductions in emissions, eventually the rate of accumulation in the surface soil fell below the rate of downward movement resulting in a slow but consistent downward migration of the metal contamination out of the top five cm of the surface soil and into the near-surface and sub-surface soil layers between 10 and 30 cm in depth.

A downward movement of metals from the surface to subsurface soil layers after the cessation of nickel deposition in Port Colborne is consistent with the pattern observed in soil lead levels in Toronto. In the 1970s lead from leaded gasoline combustion was ubiquitous in the Toronto airshed, resulting in high ambient air lead levels and subsequent deposition and accumulation of lead in surface soil. In 1971 the MOE established a baseline soil sampling network throughout Toronto and determined that the soil lead levels were highest at the surface and decreased quickly with depth. The ratio of lead in the 0 - 5 cm surface soil compared to the 10 - 15 cm soil depth was 1.6:1. Leaded gasoline was phased out in the early 1980s resulting in substantial reductions in ambient air lead levels and a virtual cessation of lead deposition to soil. A repeat sampling of the same sites in 1991 showed that with the elimination of lead deposition from the air, the lead had moved down into the soil so that it was consistently higher at depth than near the surface. The ratio of lead in 0 - 5 cm soil compared to 10 - 15 cm soil changed from 1.6:1 in 1971 to 0.6:1 in 1991.

With the cessation of atmospheric deposition, contaminants should no longer accumulate at the soil surface and the fact that soil contaminant levels in the Rodney Street community tend to be higher in subsurface soil layers is a further indication that the main deposition ceased many years ago and the sources of contamination are historic. However, this does not imply that over time the contaminants will continue to move downwards in the soil profile and eventually be deep enough so that they no longer pose a potential ecological or human health concern. Soil is a dynamic chemical, mechanical, and biological system, and at the microcosm scale, soil is constantly in flux. Limited MOE studies in other communities where soil has been contaminated by historic industrial air emissions have indicated that soil contaminants can move downwards through the soil by gravity and soil water percolating through soil pores, root and insect channels. In addition, soil contaminants can be brought back to the surface from a moderate depth as a result of plant uptake and tunnelling by ants, earthworms, and other soil macro and microorganisms. Earthworms alone can completely turnover the top 10 cm of soil in 100 years. The result is that over time, likely many decades, soil contaminants that originated on the surface tend to first move deeper and eventually get mixed into the top 30 or so centimetres of soil.

8.0 Soil Contamination: Source Allocation
8.1 Sources of Contamination

The elevated metals in soil in the Rodney Street community occurred by some combination of stack emissions, fugitive emissions, and aggregate process waste landfilling. Both Inco and Algoma had substantial stack emissions. Both Algoma and Inco had fugitive emissions that escaped from windows, doors, roof vents and from on-site contamination that was re-entrained and dispersed by traffic. Fugitive emissions were uncontrolled, can be as great or greater in magnitude then stack emissions, and tend to have the most significant impact on the local environment adjacent to the industry. In addition, it was not uncommon in the past for process waste to get placed locally to fill low areas or level land for subsequent building, or for employees to take aggregate-like waste for home construction projects. These three emission

pathways are not discrete and elevated soil metal levels in the Rodney Street community probably resulted from a combination of these three mechanisms.

8.1.1 Contaminant Groups

As described in Section 7.4, relational statistics are a useful tool for evaluating source allocation, but like the contaminant contour maps they should not be used in isolation. Since the Rodney Street community is literally sandwiched between two historic industrial sources, emissions from both industries impacted the community and the soil metal levels are spatially correlated even though the contaminants originated from different sources.

It is accepted that nickel is a signature contaminant for Inco. Similarly, iron can be considered a signature contaminant for Algoma because Algoma was a pig iron blast furnace and therefore certainly emitted iron. In the absence of compelling evidence that neither Inco nor Algoma emitted substantial amounts of lead, and in light of the known relationship between urban domestic residential soil lead and the historic use of leaded paint, lead could be considered a signature contaminant for urban domestic residential sources. Based on this common sense approach, three contaminant groups can logically be developed; 1) the Inco nickel group, 2) the Algoma iron group, and 3) the urban domestic residential lead group. Elements in soil that are highly statistically correlated with nickel are likely to be related to Inco emissions. Elements in soil that are highly statistically correlated with iron are likely to be related to Algoma emissions. Elements in soil that are highly correlated with lead are likely to be related to domestic, property specific, domestic residential sources.

Using this common sense grouping in combination with the correlative statistics in Table 15 the various elements can be ranked such that some confidence can be expressed as to their likely origin. It should be emphasized that for some elements it is not possible to definitively allocate them to a specific source, rather the ranking expresses the probability that it is more likely one source is the principal source and another is a secondary or contributing source. This is a grouping based on degrees of confidence rather than certainty. For example, if an element has a higher correlation coefficient with nickel then Inco is probably the principal source, if it has a higher r value with iron then Algoma is probably the principal source. Elements that are highly correlated to both nickel and iron and have very similar r values cannot be confidently allocated to either Inco or Algoma and may have been emitted by both industries.

Table 16 ranks the elements, using only those with positive r values in Table 15, relative to the three signature elements nickel, iron, and lead. Recognizing the limitations of correlative statistics where three separate sources are active in the same geographic area and some of the elements may be emitted from more than one source, all that can confidently be concluded about the source of any specific element is that the further to the right side of Table 16 it occurs the greater the likelihood that Inco, Algoma, or urban domestic residential properties are the principal sources. For example, cobalt has an r value of 0.929 for nickel, and so it is placed on

the far right side of the Inco row in Table 16. The r value between cobalt and iron is 0.760, so it is positioned to the right of centre in the Algoma row. By comparison, the r value between cobalt and lead is 0.380, so it is placed to the left side of the urban domestic residential lead row. The conclusions to be drawn are that cobalt is most likely to have been emitted by Inco, less likely to have been emitted by Algoma, and least likely to be associated with urban domestic residential sources.

Based on the relationships summarized in Table 16, the elements most likely associated with Inco, in addition to nickel, are cobalt, copper, selenium, zinc, and arsenic. The elements most likely associated with Algoma, in addition to iron, are arsenic and manganese. The relationship between nickel and arsenic (r=0.629) and between iron and arsenic (r=636) is too close to confidently allocate either Inco or Algoma as the principal source of elevated soil arsenic levels on some Rodney Street community properties. The Port Colborne soil arsenic map (Figure 3) and the Rodney Street community soil arsenic maps (Maps 4 to 6) clearly illustrate that both Inco and Algoma emitted arsenic. The elements most likely associated with urban domestic residential sources, in addition to lead, are barium, cadmium, antimony, chromium, and strontium.

8.2 Nickel, Copper, Cobalt, Selenium and Zinc

The soil nickel, copper and cobalt contamination documented in Port Colborne and the surrounding area in the 1998 and 1999 MOE investigations (MOE 2000a, MOE 2000b) is related to long term atmospheric deposition of Inco's stack emissions. Stack dynamics and prevailing wind patterns make the area to the northeast of Inco the zone of maximum deposition from stack emissions. The nickel:copper and nickel:cobalt soil ratios from the area to the northeast of Inco in the maximum downwind deposition area are 9.9:1 and 56:1 respectively, and are remarkably consistent to soil ratios from the average of all samples collected in the Rodney Street community in 2000 and 2001; which are 10.1:1 (nickel:copper) and 51:1 (nickel:cobalt), and the Rodney Street community trench samples, 9.5:1 (nickel:copper) and 44:1 (nickel:cobalt) (see Table 17). By comparison, the ratio of these elements in un-contaminated soil is very different. For the natural background levels in Ontario soil (Table F in the MOE *Guideline for Use at Contaminated Sites in Ontario* (MOE, 1997) and the 98th percentile and actual mean background soil values, MOE 1993) the ratios for these three elements range from 0.5:1 to 0.9:1 (nickel:copper) and from 1.8:1 to 2.4:1(nickel:cobalt). This clearly illustrates the soil contaminant signature of Inco's Port Colborne refinery. The nickel, copper and cobalt contamination detected in the Rodney Street community is completely consistent with the soil contamination in the broader Port Colborne area downwind (to the northeast) of Inco, which is commonly accepted, and admitted by Inco, to be from atmospheric stack emissions from the nickel refinery.

Table 18 is a summary of selected soil metal levels observed by the ministry around specific types of heavy industry in Ontario. It clearly illustrates that in Ontario the only industries associated with substantially elevated soil nickel levels even remotely similar to levels found in

Port Colborne are nickel refineries and smelters, and the only other nickel operations in the province are Inco and Falconbridge in Sudbury. Specifically, elevated soil nickel levels have not been detected around foundries similar to the Port Colborne Algoma blast furnace. The only other industry where measurably elevated soil nickel levels have been detected is stainless steel manufacturing, as nickel is used in the production of stainless steel. In addition, Table 18 illustrates that soil iron levels are elevated not only around iron foundries but also nickel refining and smelting centres.

Because of the spatial distribution of selenium in soil in the Rodney Street community and the very high correlation coefficient between nickel and selenium, and because the correlation coefficient is higher between nickel and selenium than between iron and selenium, Inco is likely a more significant source of selenium than Algoma. Figure 4 is a regional-scale contour map of selenium in surface soil in the greater Port Colborne area produced in the same manner as the arsenic regional map (Figure 3). Like arsenic, the soil selenium concentrations in the greater Port Colborne area are mostly below the Table A effects-based guideline, and so selenium was not mapped in the 1998/1999 MOE Port Colborne reports. At the regional scale, the selenium pattern in soil is very similar to the "classic Inco nickel pattern", which strongly suggests Inco is the source. Small areas of marginally higher soil selenium levels are apparent immediately adjacent to the northwest of Inco and downwind to the east of the refinery, but a small plume off of Algoma, similar to the arsenic plume in Figure 3, is not evident for selenium, indicating that Algoma is not principal source of elevated soil selenium levels on some properties in the Rodney Street community.

The elevated concentrations of zinc in soil on some properties in the Rodney Street community are believed to have originated principally from Inco. Zinc was detected in Inco refinery dust (Table 11) and so it was likely emitted by the refinery. However, like arsenic, the statistical correlation between nickel and zinc in soil ($r= 0.733$) is very similar to the relationship between iron and zinc ($r= 0.677$), making it difficult to confidently allocate a primary source. Based on the statistics and the spacial distribution of soil zinc levels on some properties, Algoma may be a secondary, contributing source of zinc. In addition, there are many potential domestic sources of zinc, such as paint, galvanized fences, roofs and eaves troughs.

The spatial distribution of the nickel, copper, cobalt, selenium and zinc soil contamination is consistent with the principal source to the south and east of the Rodney Street community, as the soil concentrations are higher on Rodney Street, Davis Street and Mitchell Street.

8.3 Arsenic

Of the various soil contaminants, arsenic is one of the most difficult to confidently attribute to a specific source in the Rodney Street community. Arsenic is strongly correlated to both nickel and iron, with the correlation coefficient being slightly higher with nickel. However, the property with the highest soil arsenic level is not the property with the maximum nickel concentration, and

there are several properties with elevated soil arsenic and disproportionately elevated soil nickel levels (i.e., five properties with soil arsenic concentrations between 75 and 150 µg/g and a soil nickel level less than 2,000 µg/g).

Figure 3 suggests there is an arsenic plume associated with Algoma in the vicinity of the Rodney Street community, but the same figure clearly indicates Inco is an arsenic source affecting the greater Port Colborne area. This is corroborated by the 1978 data that identified arsenic in Inco refinery dust at a concentration of 0.38% (MOE 1978a).

Arsenic is frequently associated with iron ore bodies and iron mineralization. Substantially elevated soil arsenic levels are associated with Algoma iron ore sintering operations in Wawa (see Table 18). The Port Colborne Algoma blast furnace used Wawa ore for part of its operating history. Therefore, both Algoma and Inco were sources of arsenic to the Rodney Street community and the data available to date is insufficient to confidently apportion their relative contributions.

8.4 Iron

The emissions of iron from Algoma over its operating life are estimated to have exceeded 8,900 tonnes compared to an estimated 111 tonnes of iron released from Inco (JWEL 2001c, see Table 13). Therefore, there is little doubt that Algoma is the principal source of the very high soil iron levels encountered on some properties in the Rodney Street community. Unlike nickel, the background concentration of iron in soil is quite high (OTR$_{98}$ of 33,000 µg/g for old urban parkland, MOE 1993c). Although pre-Algoma background iron concentrations in the Rodney Street community are unknown, the average soil iron concentration at the Port Colborne schools was determined to be 17,443 µg/g (median of 17,500 µg/g, MOE 2000c). This is about 9,000 µg/g lower than the average soil iron concentration in surface soils (0 to 5 cm) in the Rodney Street community of 26,252 µg/g (median of 24,800 µg/g). Since the soil type is similar, the "normal" soil iron level in Rodney Street community should be similar to the schools. Therefore, it is not unreasonable to assume that the difference in average soil iron levels between the schools and the Rodney Street community, about 9,000 µg/g, is at least in part the result of deposition of Algoma iron particulate emissions. Nevertheless, iron levels across a large part of the Rodney Street community are still within the normal range of background concentrations for Ontario soils. For example, the soil iron 80th percentile for the Rodney Street community is 35,500 µg/g compared to the 98th percentile of the provincial background of 33,000 µg/g (OTR$_{98}$, MOE 1993c).

The soil iron contour maps (Maps 28 - 30) show varying iron concentrations in the Rodney Street community but there is no a obvious gradient relative to either Algoma or Inco. The very high iron concentrations on the south side of Rodney Street are considerably above the general loading in the Rodney Street community. This area is closest to and directly downwind of Algoma so atmospheric deposition would have been greatest in this vicinity. However, because the iron

levels are so high (up to 140,000 µg/g, or 14%) it is likely that some of the iron is from Algoma fill.

Even though the soil iron levels are very high on some properties they are not expected to have any adverse ecological or health effects, because iron is a plant macro-nutrient (required in large concentrations) and is an essential requirement for human health.

8.5 Antimony

Antimony in soil in the Rodney Street community was not spatially or consistently statistically associated with nickel or iron in soil. Neither Inco nor Algoma are believed to have had significant antimony emissions. Only three properties in the Rodney Street community had soil antimony levels above the MOE Table A effects-based guideline. On these few properties the elevated soil antimony levels are spatially correlated with high soil lead concentrations. Antimony is commonly alloyed with lead as a hardening agent, and was used extensively in battery manufacture, particularly automotive lead acid batteries. Phytotoxicology investigations around secondary lead smelters that used lead acid batteries in their feed stock and around battery manufacturers, routinely identified soil lead and antimony contamination. The soil lead and soil antimony Rodney Street community contaminant contour maps illustrate a very consistent spatial relationship between these two elements (compare antimony Map 3 with lead Map 21). The antimony contamination on these three properties is likely associated with lead batteries.

8.6 Beryllium

Although the average soil beryllium concentration in the Rodney Street community was 1.0 µg/g, which is consistent with typical Ontario background levels, a significant number of properties (47%) had soil beryllium concentrations that exceeded the Table A effects-based guideline. The source of the beryllium contamination is not known with certainty, but it could be related to slag deposited or used in the Rodney Street community, natural sources, and on one property from leaded paint.

The ministry has limited information, based on the manufacture of rockwool insulation spun from slag, that beryllium concentrations in slag can range up to 10 µg/g. Beryllium is also associated with coal ash. Anecdotal information suggests that slag was a common material for roadbed construction in the Rodney Street community. A historic photograph of the Rodney Street community shows most of the roads in place by 1917, after the start up of Algoma but before Inco began operations. Therefore, if slag was used in local road construction, Algoma slag would have been an attractive construction material. The 1917 photograph also illustrates that at that time the area that is now the Rodney Street community appeared quite flat, and so wide-scale filling is unlikely to have been necessary, although "top-dressing" or grade-levelling as previously discussed remains a possibility on some properties. In the 2000 MOE investigations, slag was observed on road shoulders, in some of the trench samples, and was occasionally

encountered while sampling the residential properties. Slag-like particles were identified in the scanning electron microscope photographs of soil samples collected from several Rodney Street properties. It is evident that slag is present in soil at the surface in some areas of the Rodney Street community, and its presence may account for the generally higher than expected soil beryllium levels.

The MOE recently became aware of circumstances where elevated concentrations of naturally occurring beryllium were found to be associated with shale deposits. In view of the suspected toxicity of the metal, the presence of numerous deposits of shale in Ontario, and a practice of using shale as fill material, in 1997 MOE Phytotoxicology scientists undertook a province wide sampling program of representative shale deposits in Ontario. Seven of the twelve shale formations sampled, or 58%, had beryllium concentrations in the shale rock and the adjacent soil overburden that exceeded the MOE Table A effects-based guideline of 1.2 µg/g (MOE 1997c).

The highest beryllium concentration found in the province wide shale study was 3.4 µg/g, detected in samples collected from the Animikie-Gunflint shale formation in the Thunder Bay area. The Queenston and Rockcliffe shale formations, closer to Port Colborne, had beryllium concentrations ranging up to 2.3 µg/g. Only two soil samples of the almost 2,000 samples collected from the Rodney Street community had beryllium levels greater than 2.3 µg/g. The marginally elevated soil beryllium levels in this community are consistent with naturally occurring beryllium in soil derived from shale, although the number of properties with beryllium concentrations higher than the provincial background was unexpected. In addition, the soil beryllium concentrations in the Rodney Street community are very highly correlated with soil aluminum levels ($r=0.804$), which implies the beryllium may be natural in origin.

The highest soil beryllium concentration detected in the Rodney Street community was 4.6 µg/g, which occurred on a property that had significantly elevated soil lead levels (877 µg/g). This property also had high arsenic, barium, nickel, cobalt, copper and zinc concentrations. Although soil lead levels and soil beryllium levels across the Rodney Street community are not highly correlated ($r=0.289$), the spatial relationship between beryllium and lead at this single property is not likely coincidental (compare beryllium Maps 7, 8 and 9 with lead Maps 19, 20 and 21). It is certain that the beryllium levels on this property are not related to Inco emissions because the statistical relationships between soil beryllium and soil nickel ($r=0.102$), soil beryllium and copper ($r=0.183$), as well as soil beryllium and cobalt ($r=0.124$) are less significant than the soil beryllium and lead relationship and there is no consistent soil beryllium contour gradient relative to Inco. In addition, beryllium and arsenic soil levels are actually inversely related (negative correlation coefficient, $r=-0.030$, i.e., as arsenic levels increase beryllium levels decrease, and vice versa). Soil beryllium levels are more highly correlated with barium ($r=0.615$) than with antimony ($r=0.097$), which suggests that the elevated lead and beryllium levels on this property are related to paint rather than batteries. Even though the high beryllium levels in soil on this property appear to be related to leaded paint, this is not the case elsewhere in the Rodney Street community. Other than this single property, there is no consistent spatial relationship between

soil beryllium and soil lead concentrations. With the exception of this one property, the marginally elevated beryllium levels in soil in the Rodney Street community are believed to be a combination of natural levels of some local shale inclusions or soil morphologically associated with local shale, surficially scattered slag, and possibly coal ash.

8.7 Lead

The MOE lead contour maps in this report (Maps 19 - 21) display no consistent spacial gradient to either Algoma or Inco. High lead levels are randomly scattered throughout the community with little or no consistent spacial similarity to the contour maps for nickel, copper, cobalt, arsenic or iron. Instead, lead levels in soil are highly correlated with barium and zinc (r>0.700), both elements associated with paint. The maps do suggest that on a few properties both copper and lead levels are elevated. Copper is an antifungal agent and was used in some paint and pesticides, which may explain the lead and copper relationship on some properties.

MOE Phytotoxicology investigations in other communities have clearly linked residential soil contaminated with lead to the erosion, weathering, and/or removal of exterior leaded paint. Paint chips from flaking paint are often visible on the soil. Analysis of these chips collected from residential yards of older urban homes in Toronto showed that the paint contained up to 31%, or 310,000 µg/g, lead and 12.4%, or 124,000 µg/g, zinc (MOE 1995). The soil lead and zinc concentrations of these Toronto yards ranged up to 890 µg/g and 445 µg/g, respectively. MOE Phytotoxicology scientists have assisted MOE District Environmental Officers and local health unit inspectors in the investigation of blood lead poisoning of very young children. In most cases the lead source is found to be either ingestion of soil contaminated with lead from flaking or eroded exterior lead-based paint, or the direct ingestion of paint chips. Lead paint chips have a particular attraction to young children because the chips can be brightly coloured so they are often clearly visible in the soil, and lead has a distinctly sweet taste.

In a recent MOE Phytotoxicology soil-blood lead investigation on a Sarnia property very similar in age and construction to many homes in the Rodney Street community, the soil lead concentration averaged 629 µg/g across the property, 1,150 µg/g within one metre of painted exterior house walls, and ranged from 170 µg/g in the corner of the property away from any structures to 3,400 µg/g adjacent to the front porch (MOE 2000d). In similar investigations of several properties in Welland the soil lead levels averaged between 237 µg/g and 329 µg/g generally across the properties and ranged up to 1,796 µg/g in close proximity to painted exterior walls (MOE 2000e, MOE 2000f, MOE 2000g). The Welland homes also were very similar in age and construction to many homes in the Rodney Street community. Although not community wide studies, these recent MOE investigations in Sarnia and Welland indicate that soil lead levels like those detected in the Rodney Street community are not anomalous for older urban residential areas and can occur in the absence of a local lead source.

Lead is a ubiquitous soil contaminant in old urban environments. Historically, the use of lead additives in gasoline resulted in the release of lead in automobile exhaust and the contamination of soil near the roadways. Soil lead contamination from leaded fuel was particularly acute in urban communities because they have more roads and more cars. Lead was also a component of paint for houses, automobiles, road markings, fire hydrants, bridges and other structures. Lead leached from the paint by rain and removed from structures by peeling, flaking, scrapping, sand blasting and grinding results in the surrounding soil becoming lead contaminated. Lead-based pesticides, particularly lead-arsenate, were commonly used on fruit trees before the 1970s, which could contaminate the soil beneath the trees with both lead and arsenic. Also, very localized areas were contaminated by lead containing debris such as automobile batteries, glassware, ceramics, TV tubes, solder, fishing sinkers and lead shot. Due to the recognition of the detrimental effects of lead on human health and the natural environment the use of lead has been dramatically reduced in recent years. Lead is no longer added to gasoline or most house paint. Because older urban communities have been exposed to more domestic lead sources for a longer period of time, they tend to have substantially higher soil lead levels than younger urban, suburban, or rural communities.

Current levels of lead in soil in Port Colborne in general, and the Rodney Street community specifically, have no consistent spatial relationship relative to Inco or Algoma. Although Inco and Algoma emissions may have contributed to the overall soil lead burden in the Rodney Street community, historic vehicle emissions from the combustion of leaded gasoline and residential sources, such as weathered exterior lead-based paint, are both far more significant and known lead sources that could account entirely for the soil lead levels encountered in this study, and so any Inco or Algoma lead contributions cannot be measured above the normal urban domestic residential lead loading.

Generally, the older and more urban the community, the greater the likelihood that some properties will have elevated lead levels. The Rodney Street community is among the oldest in Port Colborne (average house age is about 78 years, based on Port Colborne municipal tax records) and therefore elevated soil lead levels on some properties expected. Soil lead levels from newer urban/suburban communities and from outlying rural residential areas in Port Colborne would not necessarily have similar soil lead levels as the Rodney Street community. Therefore, soil lead data collected from boulevards and large suburban or rural lawns around Port Colborne in earlier MOE soil investigations cannot be used to gauge normal residential soil lead burdens for the Rodney Street community.

8.7.1 Comparison of Lead Levels in the Rodney Street Community with Other Urban Communities

Generally, the older and more urban the community the greater the likelihood that some properties will have elevated soil lead levels. In the Rodney Street community of Port Colborne about 70% of the properties exceed the MOE Table A generic effects-based soil lead guideline of

200 µg/g. Also, the MOE has adapted from the US EPA lead intervention levels of 400 µg/g for children's play areas with bare soil and 1000 µg/g for all other areas to which children have regular access and the soil is sodded or covered. The average lead level of the combined 2000 and 2001 Rodney Street community residential soil is 217 µg/g and the 90th percentile is 406 µg/g.

In Toronto, the MOE has been monitoring environmental lead levels for 25 years in a community that has no known industrial source of lead pollution. In this community, which is similar to the Rodney Street community in age and style of home construction, 78% of the residential properties exceed the MOE Table A effects-based criterion and the average soil lead level is 486 µg/g. Spuriously elevated soil lead levels are an artifact of older "residential urban-core" communities. Soil lead concentrations in the 1,000 µg/g range, such as detected at a few scattered properties in the Rodney Street community, are entirely consistent with urban domestic residential lead sources and do occur in older urban communities in Ontario.

Table 19 summarizes the Rodney Street soil lead data and compares it to other communities in Ontario and American urban lead studies where there are no known industrial lead sources. The soil lead levels from these other communities are similar to, and in some cases, substantially higher than in Port Colborne. It is also apparent that soil lead levels tend to be much higher directly adjacent to exterior painted walls. In the Toronto communities the soil lead levels from older residential homes routinely averaged from 202 µg/g to 509 µg/g, the 90th percentiles were up to 930 µg/g, and soil lead levels ranged up to 3,400 µg/g (MOE 1974, MOE 1971-1991). In a survey of 1,984 urban residential properties in the United States the soil lead level adjacent to the house wall averaged three times higher than the midpoint of the adjacent yard (324 µg/g vs 105 µg/g) and the 90th percentile ranged from 243 µg/g to 633 µg/g for samples collected from the midyard compared to samples collected nearer walls. The higher lead level near the wall was concluded to be associated with weathering of exterior leaded paint (U.S. H.U.D. 2001). In a study of almost 1,000 "high risk" homes in three communities in California (risk defined by wood construction, homes greater than 50 years, weathered paint evident, and young children present, but no industrial lead sources) the average soil lead levels were 188 µg/g in Los Angles, 234 µg/g in Sacramento, and 897 µg/g in Oakland (Sutton 1995). In a detailed study of lead contamination on a residential property caused by the removal of leaded paint by sandblasting, the soil lead levels were found to range from 360 µg/g at distances of more than about one metre from the walls being treated to 3,900 µg/g within one metre of the walls (Mielke 2001).

There is a consistent relationship between the age of the home and the likelihood of elevated soil lead levels. This has been observed in numerous MOE investigations and is supported by the published literature. In a California study approximately 85% of the residential properties built before 1920 had maximum soil lead levels greater than 500 µg/g, compared to about 11% of the homes built after 1970 (Sutton et al., 1995). Similarly, a study of urban communities in the United States found that 67% of the homes built before 1940 had maximum soil lead levels above 400 µg/g compared to only 9% built after 1960 (U.S. H.U.D. 2001). This study also found

that 26% of the properties with houses built before 1940 had maximum soil lead levels greater than 1,600 $\mu g/g$, which is in the range of the maximum soil lead level (1,800 $\mu g/g$) found in the Rodney Street community.

Soil lead levels in a California study of 933 residential properties in three communities found that the age of the housing was the best predictor of lead in soil; homes built before 1920 were 10 times more likely to have soil lead levels greater than 500 $\mu g/g$ compared to post 1950 homes (Sutton et al., 1995, and see Figure 5).

Table 20 summarizes the soil lead levels of Rodney Street properties broken down by the year of house construction. In the Rodney Street community, about 40% of the houses were built before 1920, about 54% were constructed between 1920 and 1949, and only about 6% of the houses were built after 1950. The mean soil lead level was highest (260 $\mu g/g$) from the oldest group of homes and lowest (125 $\mu g/g$) from the youngest group of homes.

Lead was likely emitted from Inco since lead comprised 10.5% of a single Cottrell Precipitator dust sample collected at the Inco refinery in 1978 (MOE 1978a). However, based on data from soil samples collected in the Port Colborne area downwind to the east-northeast of Inco in the area of greatest atmospheric deposition, of those soil samples with soil nickel levels greater than 1,000 $\mu g/g$, the average soil nickel level was 2,120 $\mu g/g$ and the average soil lead concentration was 98 $\mu g/g$ (MOE 2000a, MOE 2000b), which gives a nickel:lead soil ratio of 21.6:1. Similarly, using the soil data from the Rodney Street community that was collected in 2000 and 2001 (excluding the trench data) the average soil nickel level was 2,508 $\mu g/g$ and the average soil lead level was 217 $\mu g/g$, which gives a nickel:lead ratio of 11.6:1. Since the nickel:lead ratio of 21.6:1 for the Port Colborne area downwind of Inco is not similar at all to the nickel:lead ratio of 11.6:1 for the Rodney Street community, the lead is unlikely from the same source as the nickel contamination. Also, for the Rodney Street community soil data, since the statistical correlation between iron and lead (r=0.377) is even less than between nickel and lead (r=0.426) the lead is unlikely from the same source as the iron contamination. Furthermore, based on almost 2,000 soil samples from almost every yard of almost every property in the Rodney Street community, there is no consistent soil lead concentration gradient relative to either Inco or Algmoa.

The preceding data indicate that the lead levels in soil in the Rodney Street community can be accounted for by general urban domestic residential lead sources. Although Inco and Algoma likely emitted some lead and deposition from these two industries impacted the Rodney Street community, the lead contribution from these sources is not measurable above the normal urban domestic residential lead loading.

9.0 Conclusions
9.1 General Conclusions

The average soil nickel concentration in the Rodney Street community is 2,508 µg/g and the predominant form of nickel in the soil in the Rodney Street community is nickel oxide (80% of the total nickel, on average).

Property by property sampling revealed substantial variation in both the numbers of contaminants and the soil contaminant concentrations. Of the approximately 2,000 samples collected from about 200 properties, 99% of the properties had soil nickel levels that exceeded the MOE generic Table A effects-based criterion of 200 µg/g. The maximum soil nickel level was 17,000 µg/g. In addition to nickel, the MOE Table A effects-based guidelines were exceeded for lead on approximately 73% of the properties, cobalt on 72% of the properties, copper on 66% of the properties, beryllium on 47% of the properties, arsenic on 46% of the properties, zinc on 37% of the properties, antimony on 1.5% of the properties, and selenium and cadmium on less than 1% of the properties. For many elements on most properties, the soil contaminant concentrations tended to increase with depth. If the trenches excavated in the vacant lot south of Rodney Street and the park east of Welland Street, and the test pits dug by JWEL on two Rodney Street properties are representative of the soil profiles across the community, then soil contamination on most residential properties in the Rodney Street community may extend to 30 cm, but should decrease very rapidly below that depth.

Inco is the source of soil nickel, copper, and cobalt contamination in the Rodney Street community. The elevated soil arsenic levels are a result of emissions from both Inco and Algoma. Because of the degree of spatial and statistical relationship of selenium and zinc with nickel, copper and cobalt, Inco is likely the principal source of the elevated soil zinc and selenium levels. However, there is almost as strong a relationship between iron, zinc and selenium as there is between nickel, zinc and selenium, and therefore Algoma may be a secondary or contributing source of soil selenium and zinc concentrations in the Rodney Street community.

The baseball park at the southwest corner of Rodney and Davis Streets was, at least partially, created from metal contaminated fill. Similarly, because of the long history of industry ownership of the land behind the residential properties on the south side of Rodney Street, some of these residential properties may also have been affected by fill, particularly in the back yards. Not withstanding the possibility that isolated properties may have imported aggregate process waste for home construction projects or grading and subsequently added to the soil contaminant burden on these properties, the primary mechanism of the soil nickel, copper, cobalt, arsenic, selenium and zinc soil contamination across the broader Rodney Street community is believed to atmospheric deposition of stack and fugitive emissions and not fill. Regardless of the mechanism of contamination (fill or atmospheric deposition) the nickel, copper and cobalt originated from Inco.

The height of the tall stack, constructed in 1935, in conjunction with the strong southwesterly prevailing winds in the non-snow season, dispersed most of the stack emissions to the northeast of the Inco refinery after the stack was built, resulting in the soil contaminant pattern across the regional Port Colborne area documented in the 1998 and 1999 MOE soil investigations. However, fugitive emissions from the refinery, particularly in the early years of the refinery's operation, would have substantially impacted the Rodney Street community. At the MOE open house in April 2001, many anecdotal observations were offered by long time Rodney Street community residents and refinery employees about the chronically dusty conditions in the Rodney Street community and in and around the Inco refinery during the earlier years of operation. Since the community was literally sandwiched between two large industries (Inco and Algoma) that were dusty by nature, particularly in the past before the technology was available and implemented to control fugitive emissions, there is no reason to doubt the anecdotal comments about the Rodney Street community being a dusty neighbourhood.

The highest soil nickel, copper, cobalt, arsenic, selenium and zinc soil concentrations occurred on properties in the south and eastern areas of the Rodney Street community along Rodney, Mitchell and Davis Streets. Based on the contaminant contour maps it is likely that elevated soil metal levels may extend slightly further along Davis Street north of Louis Street. Further soil sampling is also warranted in the residential communities immediately adjacent to the north-northwest, north, and north-northeast of Inco.

In Ontario, the only industries associated with substantially elevated soil nickel levels even remotely similar to levels found in Port Colborne are nickel refineries and smelters, and the only other nickel refineries and smelters in the province are the Inco and Falconbridge operations in Sudbury. Elevated soil nickel levels have not been detected around foundries similar to the Algoma foundry that operated in Port Colborne to the west of the Rodney Street community.

The randomly scattered soil lead contamination observed in the Rodney Street community is primarily related to general urban domestic residential lead sources and not to Inco or Algoma emissions. The erosion and flaking of old lead-based paint from exterior structures such as house and shed walls, porches, fences, poles and playground equipment is a common source of soil lead contamination in older urban communities. The soil lead levels found in the Rodney Street community are not unusual, either in extent or concentration, relative to other similarly aged older urban communities in Ontario. On properties where the soil lead levels were elevated, the concentrations of cadmium, chromium, copper, barium and zinc sometimes were proportionately elevated. Along with lead, these elements were common pigment, anti-mildew or anti-fungal additives in old exterior paint and are frequent co-contaminants with lead in residential soil.

Antimony was another element that was highly correlated with lead on some properties, although it exceeded MOE guidelines on only three properties. Antimony is commonly alloyed with lead, particularly in lead acid batteries. Lead and antimony soil contamination is an indication that batteries may have been stored or disposed of on the property, whereas lead and barium, lead and

zinc, or lead and copper soil contamination is a signature of lead-based paint.

Although the average soil beryllium level in the Rodney Street community was comparable to the provincial soil background concentration, almost one half of the properties exceeded the MOE Table A effects-based guideline. Soil beryllium levels marginally above the guideline are not unusual, because the guideline and the upper end of the background range are the same (1.2 µg/g). Also, MOE investigations have documented that shale, and soil derived from shale, regularly exceed the guideline. In addition, slag and coal ash can have a beryllium concentration that is above the guideline, and slag is present on some properties in the Rodney Street community. Slag was believed to have been used as roadbed material. With the exception of one property where elevated beryllium levels were concurrent with high lead and other heavy metals, the marginally elevated soil beryllium concentrations across the Rodney Street community are likely related to the presence of slag and local shale deposits. Emissions from Algoma may have contributed to the generally marginally elevated soil beryllium levels in the Rodney Street community.

Recent or ongoing atmospheric deposition results in soil contaminants accumulating in the upper most soil layers and decreasing quite abruptly with depth. Where atmospheric deposition is the only source of contamination, measurably elevated soil contaminant levels are usually confined to the top 20 - 30 cm of soil. For most of the Rodney Street community, and across Port Colborne, the highest soil contaminant levels tended to occur not right at the surface but between 10 and 20 cm, and then fell rapidly to near background concentrations below about 25 - 30 cm. This pattern, which has been observed by the ministry in other communities that have been impacted by historic industrial emissions, is consistent with deposition that was much higher in the past and was much lower or abated entirely in more recent years. Test pits dug by JWEL on Inco property at undisturbed sites confirm the pattern of high levels at the surface, highest levels between 10 and 20 cm, lower levels below 20 cm, and very low (almost background) soil contaminant concentrations below about 30 cm (JWEL 2001b). Figure 1 confirms that Inco nickel emissions were substantially higher in the past (97% of the total nickel emitted was released before 1960). Algoma ceased operations in 1977 but 75% of the total amount of iron emitted by Algoma was released before 1959. Based on these data it is evident that most of the heavy metals and arsenic that are currently present in soil in the Rodney Street community, and elsewhere in the Port Colborne area, are a result of historic industrial emissions that occurred up to or before 1960. Some properties in the Rodney Street community, particularly those on the south side of Rodney Street, in addition to being impacted by atmospheric deposition also likely have received industrial process waste or fill from either or both Inco and Algoma.

Other than the properties on the south side of Rodney Street that may have received process waste or fill from either or both Inco and Algoma, the "patchwork" pattern of high and low soil contamination on neighbouring lots is likely related to fugitive emissions and property maintenance and landscaping. Adding topsoil or mulch, re-sodding, building, and cultivating gardens are landscaping practices that, over time, tend to cover or dilute contaminants that are

predominantly present in the surface soil. It also indicates that the source of the soil contamination is likely atmospheric and that with deposition abated, newly landscaped properties have not become re-contaminated to the levels of undisturbed properties.

The plant-bioavailability of nickel in soil in Port Colborne is very low, in the range of less than 1% for mineral soil (this refers to the plant bio-availability in Part A Section 5.3.3, not the human stomach leach bio-availability discussed in Part B Appendix 5). This means the nickel has a low mobility in the soil, which means it would not be readily taken up by plants. The low plant-bioavailable nickel levels account for the remarkably minor amount of nickel injury observed on species of vegetation known to be sensitive to nickel in areas of Port Colborne where the soil nickel levels are substantially above the MOE Table A ecotox-based guidelines.

9.2 Conclusions Related to the Stated Objectives

There were four specific objectives of Part A of the report stated in Section 1.0.

1) *To determine the extent and severity of soil metal and arsenic contamination in the Rodney Street community of Port Colborne.*
>Although an overall spatial pattern was evident for some contaminants, particularly nickel, copper, cobalt and arsenic (see the soil contaminant contour maps) there was considerable property by property variability. Soil contamination above the 8,000 µg/g nickel intervention level on some properties in the Rodney Street community is at least 20 cm deep but is not likely to extend much deeper than 30 cm. Additional sampling is warranted north of Louis Street to find any additional properties with soil nickel levels greater than 8,000 µg/g.

2) *To characterize metal soil contamination in the Rodney Street community.*
>The metal contaminants can be divided into the following three logical groups (see Table 21):

>>1) the Inco nickel group that includes nickel, cobalt, copper, selenium, zinc and arsenic;

>>2) the Algoma iron group that includes iron, arsenic, manganese, and possibly selenium and zinc; and

>>3) the urban domestic residential lead group that includes lead, zinc, barium, cadmium, antimony, chromium and strontium.

3) *To determine to the extent possible the source(s) of the soil contamination in the Rodney Street community.*
>Inco is the source of the soil nickel, copper and cobalt contamination. Inco is likely the

principal source of the elevated soil zinc and selenium levels. Algoma is the principal iron and manganese source and is likely a contributor to the soil zinc and selenium levels. Both Inco and Algoma emitted arsenic and it is not possibly to confidently apportion the soil arsenic levels in the Rodney Street community between these two sources. Although both Inco and Algoma likely emitted lead, their contribution cannot be measured above the general urban domestic residential lead sources. Soil lead levels in the Rodney Street community are consistent with soil lead levels in older urban communities in Ontario and the United States. The sources of the lead are numerous and property specific (i.e., lead-based paint, pesticide use, storage, maintenance, and disposal of vehicles and vehicle parts, particularly batteries, historic use of leaded gasoline). Slightly elevated soil beryllium levels are likely related to natural shale deposits with a contribution from Algoma slag and particulate emissions.

4) *To determine to the extent possible, the mechanisms(s) of soil contamination in the Rodney Street community.*

Not withstanding the possibility that isolated properties may have imported aggregate process waste for home construction projects or grading and subsequently added to the soil contaminant burden on these properties, the predominant mechanism of soil nickel, cobalt, copper and to a lesser extent iron, selenium, arsenic and zinc contamination in the Rodney Street community is believed to be atmospheric deposition of Inco and Algoma emissions from both fugitive and stack sources. The contamination in the Rodney Street baseball park is predominantly from Inco fill with contributions from atmospheric deposition. Some of the properties on Rodney Street, particularly the back yards of the properties on the south side, likely have been influenced by shallow filling or spreading of Inco or Algoma aggregate process waste. Regardless of the mechanism(s) of contamination, Inco is the only source of the substantially elevated soil nickel concentrations in the Rodney Street community.

9.3 Calculated Soil Nickel Levels

Nickel deposition over Inco's operating life-time was estimated by JWEL (2001c) to range from approximately 5,000 g/m^2 in the vicinity of the Welland Canal to about 20,000 g/m^2 adjacent to the refinery. The Rodney Street community falls within this estimated deposition range. Table 21 calculates the resultant soil nickel concentration at various depths across the 5,000 g/m^2 to 20,000 g/m^2 range of estimated nickel deposition. If all the nickel stayed where it fell on the soil (no wash-off or leaching), at a deposition rate of 20,000 g/m^2 the resultant soil nickel concentration would be 266,667 µg/g if the nickel all stayed in the top 5 cm of soil. At a deposition rate of 5,000 g/m^2 the resultant soil nickel concentration could be 11,111 µg/g if the nickel were evenly mixed in the top 30 cm of soil. Based on JWEL (2001c), the highest soil nickel level found in the Rodney Street community of 17,000 µg/g through to a depth of 20 cm could be accounted for by a deposition rate as low as 5,000 g/m^2 (see Table 21). Therefore,

atmospheric deposition alone could account for all the nickel currently present in soil in the Rodney Street community.

9.4 Application of the Soil Nickel Intervention Level

In consideration of the variability inherent in sampling soil and laboratory testing, the ministry used the maximum nickel concentration found in any soil sample at any depth in the front, back or side yard, to determine if a property exceeded the soil intervention level derived from the human health risk assessment. If the maximum soil nickel concentration on the property exceeded the intervention level, the property becomes a candidate for soil remediation. Even though the maximum concentration is a worst case estimate of the overall property concentration, because of sampling and analytical variability a property owner/occupant could still have concerns that the maximum nickel concentration found on the property is an underestimate of the true (but unknown) nickel concentration. By determining the variability around the maximum measured concentration a confidence interval for any specific concentration can be calculated.

The soil data from all of the sampled properties in the Rodney Street community were statistically evaluated to determine the confidence interval around the maximum value of all the samples collected from any single property. Given the large data base and the log-normal distribution of the soil nickel levels across the Rodney Street community, the asymptotic distribution (also known as the Gumbel distribution - see Johnson and Kotz, 1995) of the maximum likelihood estimates (normal) was used to construct confidence intervals around the maximum concentration for any property. From the Human Health Risk Assessment in Part B of this report it was determined that at a soil nickel concentration of 9,061 µg/g (refer to Part B, Section 7.1) the nickel exposures from all sources, including soil in the Rodney Street community, is less than the nickel reference dose (RfD) for all age groups including the toddler. Using the confidence intervals from the Gumbel distribution of the combined 2000 and 2001 Rodney Street residential soil data, it can be shown that, if the maximum soil nickel level of any single sample at any depth from a property is 8,139 µg/g or less, it is 99% certain that no sample on the property would exceed 9,061 µg/g. Based on these calculations the soil intervention level is set at 8,000 µg/g nickel.

9.5 The Effect of Remediation of Properties Above the 8,000 µg/g Intervention Level

Properties with soil nickel concentrations at and above 8,000 µg/g are to be remediated. Some of these properties may also have elevated levels of other metals or arsenic, which will be removed with the nickel contaminated soil during remediation. After the remediation there will be fewer properties in the Rodney Street community that exceed the MOE guidelines (Table 12). Specifically many of the properties with elevated arsenic levels in soil will be remediated as a result of the nickel clean-up. Table 22 illustrates that after remediating the nickel contaminated properties the maximum soil arsenic level in the Rodney Street community drops from 350 µg/g to 62 µg/g and the 90th percentile drops to 22 µg/g, which is below the MOE Table A guideline

of 25 µg/g for medium/fine textured soil. After remediation, the maximum soil arsenic level in the Rodney Street community is about 1/4 the maximum level in Port Hope, about 1/10th the maximum level in Deloro, and about 1/17th the maximum concentration of arsenic in soil in Wawa. Wawa and Deloro have undertaken health studies and a health risk assessment was conducted in Port Hope. These studies concluded:

Wawa

> *This study did not produce evidence that Wawa residents are at an increased risk of cancer due to exposure to arsenic* (Goss Gilroy 2000).

Deloro

> *Estimated arsenic exposures are not measurably higher than those of typical Ontario residents* (Cantox 1999).

Port Hope

> *Estimated intakes from contact with these soils yield risk estimates in the range generally considered negligible and below the WHO permissible intake* (MOE 1991).

Conclusions Summary

!   Inco is the source of nickel, copper, and cobalt soil contamination in the Rodney Street community.

!   Inco is the principal source of selenium and zinc soil contamination in the Rodney Street community.

!   Both Inco and Algoma emitted arsenic; it is not possible to confidently apportion which source contributed how much arsenic or where it was deposited in soil in the Rodney Street community.

!   Algoma is the principal source of iron.

!   The predominant mechanism of the soil nickel, copper, cobalt, arsenic, iron, selenium and zinc contamination in the Rodney Street community is atmospheric deposition from both stack and fugitive sources.

!   The soil lead contamination in the Rodney Street community is typical of older urban residential communities in Ontario; the sources are domestic, numerous and property specific (e.g., paint, pesticide use, the storage, maintenance, and disposal of vehicles and vehicle parts (particularly batteries); historic use of leaded gasoline).

!   The surficial soil contamination in the Rodney Street community is between 20 and 30 cm deep (may be deeper on the south side of Rodney Street).

!   The mechanism of the soil contamination at depth in the baseball park at the southwest corner of Rodney and Davis Streets is industrial process waste/fill.

!   Industrial process waste/fill has contributed to the soil contamination on some properties (particularly the back yards) on the south side of Rodney Street.

!   Process waste/fill that is high in iron and low in nickel is from Algoma, if it is high in nickel and relatively low in iron it is from Inco, and if it has high concentrations of both iron and nickel it is likely a mix of both Algoma and Inco waste/fill.

!   Both Inco and Algoma likely emitted lead, and some lead in soil in the Rodney Street community is probably from industrial emissions, but the contribution can not be measured above the general urban domestic residential soil lead burden.

!   The generally elevated soil beryllium concentrations are associated with natural shale deposits and/or dust/slag probably from Algoma.

!   Most of the surficial soil contamination in the Rodney Street community occurred earlier in Inco's and Algoma's operating history, likely before 1960.

10.0 References

Cantox Environmental Inc. 1999. *Deloro Village Exposure Assessment and Health Risk Characterization for Arsenic and Other Metals.* Final Report Prepared for the Ontario Ministry of the Environment, and CH2H Gore & Storie Ltd., December 1999.

Enpar Technologies Inc. 2001. *Scanning Electron Microscopy and Energy Dispersive X-Ray Analyses of Four Soil Samples from the Port Colborne Area.* Project No. 30029, prepared for Jacques Whitford Environmental Ltd. January 24, 2001.

Goss Gilroy Inc. 2001. *Survey of Arsenic Exposure for Residents of Wawa.* Submitted to the Wawa Environmental Steering Committee, January 2001.

Inco. 2000. *An Open Letter to the Residents of Port Colborne.* Del Fraiport, Manager, Inco Port Colborne Refinery.

Inco Technical Services Limited Research. 2001a. *Mineralogy Report Project #55-813*, January 15, 2001.

Inco Analytical Services. 2001b. *Mineralogy Report, Project Number 55-813*, February 19, 2001.

Jacques Whitford Environment Ltd. 2001a. *Draft Report on Potential CoC Identification Using Statistical Analysis.* JWEL Project No. Ont34647. November 16, 2001.

Jacques Whitford Environment Ltd. 2001b. *Draft Report on Potential CoC Identification Using Soil Chemical Concentration Data in Exceedence of MOE Generic Guidelines.* JWEL Project No. Ont34645. November 23, 2001.

Jacques Whitford Environment Ltd. 2001c. *Draft Report on Potential CoC Identification Using Emission Inventories and Dispersion Modelling of Inco and Algoma Operations.* JWEL Project No. Ont34648. November 23, 2001.

Johnson, N. and S. Kotz. 1995. *Extreme Value Distributions In: Continuous Univariate Distributions.* John Wiley & Sons, Toronto.

Massachusetts Department of Environmental Protection. 1994. *Background Documentation for the Development of the MCP Numerical Standards.* April 1994, Bureau of Waste Site Cleanup and Office of Research and Standards.

MOE. 1971 to 1991. *Ongoing Study of Heavy Metals in Soil and Vegetation in Toronto,* unpublished, Ontario Ministry of the Environment.

MOE. 1974. Memo to C.J. MacFarlane from S.N. Linzon, Subject: *Soil Lead Survey for the Toronto Board of Health Control Area*, January 15, 1974.

MOE. 1975. *Phytotoxicology Surveys Conducted in the Vicinity of International Nickel Company, Port Colborne, Ontario, 1969-1974*. Phytotoxicology Section, Air Resources Branch, Ontario Ministry of the Environment.

MOE. 1977. *Effect of Heavy Metals on the Growth of Lettuce, Celery, and Onion, Groetelaars Farm, Port Colborne*. Phytotoxicology Section, Air Resources Branch, Ontario Ministry of the Environment.

MOE. 1978a. Letter dated June 1, 1978, citing Inco J. Roy Gordon Research Laboratory results *Analysis of Dusts from the Port Colborne Nickel Refinery*. February 10, 1978, Ontario Ministry of the Environment.

MOE. 1978b. *Investigations of the Effects of Heavy Metals on Muck Farms East of International Nickel Company, Port Colborne, Ontario. 1976-1977*. Phytotoxicology Section, Air Resources Branch, Ontario Ministry of the Environment.

MOE. 1980. *Lead and Other Metals in Vegetation and Soil in the Vicinity of Tonolli Company of Canada Ltd. and Exide Canada Inc., Dixie Road, Mississauga - 1980*. Phytotoxicology Section, Air Resources Branch, Ontario Ministry of the Environment.

MOE. 1981. *Phytotoxicology Surveys in the Vicinity of the Inco Nickel Refinery, Port Colborne - 1979, 1980*. Phytotoxicology Section, Air Resources Branch, Ontario Ministry of the Environment.

MOE. 1985. *Procedures Manual for Vegetation and Soils Processing Laboratory*. Air Resources Branch, Phytotoxicology Section, Ontario Ministry of the Environment.

MOE. 1986. *Results of Phytotoxicology Sampling Programs Conducted in the Vicinity of Ivaco L'Orignal during 1984 through April 1985*. Author: R. Emerson. Phytotoxicology Section, Air Resources Branch, Report No. ARB-82-86-Phyto, Ontario Ministry of the Environment.

MOE. 1990. *Phytotoxicology Assessment Surveys Conducted in the Vicinity of the Ivaco Metal Recycling Plant, Longueuil Township May, 1986 Through January, 1988*. Author: R.N. Emerson. Phytotoxicology Section, Air Resources Branch, Report No. ARB-114-88-Phyto, Ontario Ministry of the Environment.

MOE 1991. *Assessment of Human Health Risk of Reported Soil Levels of Metals and Radionuclides in Port Hope.* Hazardous Contaminants Branch, November 1991, ISBN 0-7729-9065-4.

MOE. 1993a. *Field Investigation Manual Part 1: General Methodology.* Authors: W.D. McIlveen and D.L. McLaughlin. Phytotoxicology Section, Hazardous Contaminants Branch, Report Number HCB-014-3511-93, Ontario Ministry of the Environment.

MOE. 1993b. *Crowe Foundry - Cambridge (1992).* Phytotoxicology Section, Hazardous Contaminants Branch, Report No. HCB-049-3512-1993TM, Ontario Ministry of the Environment.

MOE. 1993c. *Ontario Typical Range of Chemical Parameters in Soil, Vegetation, Moss Bags, and Snow.* Hazardous Contaminants Branch, Report No. HCB-3512-93, PIBS No. 2792, ISBN 0-778-1979-1, Ontario Ministry of the Environment.

MOE. 1994. *Phytotoxicology Survey Report: International Nickel Company Limited Port Colborne - 1991.* Authors: D. McLaughlin and S. Bisessar. Phytotoxicology Section, Standards Development Branch, ISBN 0-7778-2721-1, Report No. SDB-003-3512-94, Ontario Ministry of the Environment.

MOEE. 1994. *Atlas Specialty Steels - Welland (1993),* Phytotoxicology Section, Standards Development Branch, Report No. SDB-042-3512-94TM, Ontario Ministry of the Environment and Energy.

MOE. 1995. *Soil Contamination of Residential Properties in Metropolitan Toronto by Lead and Other Metals from Paint.* Authors: S. Bisessar and D. McLaughlin. Standards Development Branch, Phytotoxicology Section, unpublished, Ontario Ministry of the Environment.

MOE. 1996. *Rationale for the Development and Application of Generic Soil, Groundwater and Sediment Criteria for Use at Contaminated Sites in Ontario.* Standards Development Branch. PIBS 3250E01, ISBN 0-7778-5906-8, Ontario Ministry of the Environment.

MOE. 1997a. *Guideline for Use at Contaminated Sites in Ontario.* Revised February 1997, PIBS 3161E01, ISBN 0-7778-6114-3, Ontario Ministry of the Environment.

MOE. 1997b. *Phytotoxicology 1995 Investigation: Atlas Specialty Steels Welland,* Standards Development Branch, Phytotoxicology Section, Report No. SDB-014-3511-1997, Ontario Ministry of the Environment.

MOE 1997c. *Investigation into the Chemical Composition of Shales in Ontario.* Author: W.D. McIlveen. Standards Development Branch, Ecological Standards and Toxicity Section, Report No. SDB-023-3511-1998, Ontario Ministry of the Environment.

MOE 1997d. *Technical Report: Assessment of Potential Health Risks of Reported Soil Levels of Nickel, Copper and Cobalt in Port Colborne and Vicinity. May 1997.* Authors: B. Leece and S. Rifat. Standards Development Branch and the Regional Niagara Public Health Department, Report No. SDB-EA054.94-3540-1997, Ontario Ministry of the Environment.

MOE. 1999a. *Studies of the Terrestrial Environment in the Wawa Area, 1998-1999.* Phytotoxicology and Soil Standards Section, Standards Development Branch. Report No. SDB-047-3511-1999, Ontario Ministry of the Environment.

MOE. 1999b. *Phytotoxicology 1998 Investigation: The Village of Deloro, Ontario.* Standards Development Branch, Phytotoxicology and Soil Standards Section, Report No. SDB-056-3511-1999, Ontario Ministry of the Environment.

MOE. 2000a. *Phytotoxicology Soil Investigation: Inco - Port Colborne (1998).* Authors: Kuja, A., D. McLaughlin, R. Jones and W. McIlveen. Ecological Standards and Toxicity Section, Standards Development Branch, Report No. SDB-031-3511-1999, Ontario Ministry of the Environment.

MOE. 2000b. *Phytotoxicology Soil Investigation: Inco-Port Colborne (1999).* Authors: Kuja, A., R. Jones, and W. McIlveen. Ecological Standards and Toxicity Section, Standards Development Branch, Report No. SDB-031-3511-2000, Ontario Ministry of the Environment.

MOE. 2000c. *Results of Soil Sampling in School Yards and Beaches in the Port Colborne Area, April 2000.* Authors: W. McIlveen and D. McLaughlin. Ecological Standards and Toxicology Section, Standards Development Branch, Report No. SDB-031-3511-2000, Ontario Ministry of the Environment.

MOE. 2000d. *Complaint Investigation Report to Property Owners/Occupiers.* Privileged and confidential. Standards Development Branch, Report No. SDB-035-3511-2000, Ontario Ministry of the Environment.

MOE. 2000e. *Complaint Investigation Report to Property Owners/Occupiers.* Privileged and confidential. Standards Development Branch, Report No. SDB-055-3511-2000, Ontario Ministry of the Environment.

MOE. 2000f. *Complaint Investigation Report to Property Owners/Occupiers*. Privileged and confidential. Standards Development Branch, Report No. SDB-078-3511-2000, Ontario Ministry of the Environment.

MOE. 2000g. *Complaint Investigation Report to Property Owners/Occupiers*. Privileged and confidential. Standards Development Branch, Report No. SDB-079-3511-2000, Ontario Ministry of the Environment.

MOE. 2001a. *Phytotoxicology 2001 Investigation: Re-sampling of Soil at Humberstone School, and Arsenic in Soil at all Schools - Port Colborne*. Ecological Standards and Toxicology Section, Standards Development Branch, Report No. SDB-043-3511-2001, April 2001, Ontario Ministry of the Environment.

MOE. 2001b. *Metals in Soil and Vegetation in the Sudbury Area (1970-2000)*. Ecological Standards and Toxicology Section, Standards Development Branch, Report No. SDB-045-3511-2001, Ontario Ministry of the Environment.

Mielke, Howard W., Eric T. Powell, Aila Shas, Christopher R. Gonzales, and Paul W. Mielke. 2001. *Multiple Metal Contamination from House Paints: Consequences of Power Sanding and Paint Scraping in New Orleans*. Environmental Health Perspectives, Vol 109 No. 9, September 2001.

Ontario Ministry of the Environment, and Agriculture Canada. 1980. *Effects of Heavy Metals and Root Nematode on Celery Grown on Organic Soil in the Vicinity of International Nickel Company, Port Colborne, Ontario, 1980*. Air Resource Branch, Phytotoxicology Section, and Agriculture Canada Vineland Research Station, Ontario Ministry of the Environment.

Ontario Ministry of the Environment and Ontario Ministry of Agriculture and Food. 1983. *Joint Report by Ontario Ministry of the Environment (MOE) and Ontario Ministry of Agriculture and Food (OMAF) Regarding Nickel Contamination of Soil on Muck Farms East of Inco Metals Company, Port Colborne,* Ontario Ministry of the Environment.

Ontario Ministry of Northern Development and Mines. 2001. *Mineralogy Report Geoscience Laboratory, Job #00-0590,* Ontario Ministry of the Environment.

Sutton, Patrice M., Marcos Athanasoulis, Peter Flessel, Guirguis Guirguis, Mary Haan, Robert Schlag, and Lynn R. Goldman. 1995. *Lead Levels in the Household Environment of Children in Three High-Risk Communities in California*. Environmental Research 68, 45-57.

U.S. Department of Housing and Urban Development. 2001. *National Survey of Lead and Allergens in Housing Final Report, Volume I: Analysis of Lead Hazards.* HUD Contract Number C-OPC-21356 U.S. Department of Housing and Urban Development.

Part A

Soil Investigation: Tables

Table of Contents
Part A - Soil Investigation: Tables

Table 1: Minimum Number of Soil Cores Collected for Every Yard Sampled

Number of Yards per Property	Single Composite Sample	Duplicate Sample	Triplicate Sample
1 Yard	27 (1 yard X 9 cores X 3 depths)	54 (1 yard X 9 cores X 2 replicates X 3 depths)	81 (1 yard X 9 cores X 3 replicates X 3 depths)
2 Yards	54 (2 yards X 9 cores X 3 depths)	108 (2 yards X 9 cores X 2 replicates X 3 depths)	162 (2 yards X 9 cores X 3 replicates X 3 depths)
3 Yards	81 (3 yards X 9 cores X 3 depths)	162 (3 yards X 9 cores X 2 replicates X 3 depths)	243 (3 yards X 9 cores X 3 replicates X 3 depths)

Assumes 9 cores per yard (this was minimum number), and all three depths could be sampled.

Table 2: Results of Chemical Analysis (µg/g) of 2000 Residential Soil Samples, Port Colborne

Al = aluminum, Sb = antimony, As = arsenic, Ba = barium, Be = beryllium, Cd = cadmium, Ca = calcium, Cr = chromium, Co = cobalt, Cu = copper, Fe = iron, Pb = lead, Mg = magnesium, Mn = manganese, Mo = molybdenum, Ni = nickel, Se = selenium, Sr = strontium, V = vanadium, Zn = zinc. Data in bold and underlined exceed MOE Table A effects-based guideline. na - data not available

Site / Location	Soil Depth	Al	Sb	As	Ba	Be	Cd	Ca	Cr	Co	Cu	Fe	Pb	Mg	Mn	Mo	Ni	Se	Sr	V	Zn
2022013 (Front yard)	0-5 cm	5200	na	35	100	<0.25	0.50	11000	35	120	510	58000	290	3200	800	2.1	6400	5.70	41	24	660
	0-5 cm	5200	na	37.0	110	<0.25	0.70	12000	37	110	470	53000	300	3400	800	2.2	5500	5.70	38	24	620
	5-10 cm	5500	na	65.0	130	0.6	<0.10	9900	49	220	990	130000	400	3200	1200	3.7	16000	6.90	37	34	1100
	5-10 cm	5800	na	110.0	140	0.6	0.40	11000	44	150	770	90000	480	3300	1100	3.0	9200	8.70	41	29	990
	10-15 cm	7300	na	30.0	99	<0.25	<0.10	11000	19	57	430	38000	190	4000	560	<0.25	4900	3.90	36	24	420
	10-15 cm	7500	na	42.0	140	0.6	<0.10	12000	36	84	660	51000	240	3900	740	1.4	7100	5.10	42	25	620
	15-20 cm	7100	na	18.0	90	<0.25	0.50	17000	20	30	200	26000	100	4400	400	1.3	2100	2.50	45	21	260
	15-20 cm	6800	na	13.0	78	<0.25	0.30	16000	15	18	130	17000	140	4000	300	<0.25	1200	2.10	42	19	170
2022014 (Back yard)	0-5 cm	7800	na	15.0	67	<0.25	0.50	14000	22	48	210	30000	93	4800	470	0.8	2400	2.80	49	24	340
	0-5 cm	6700	na	20.0	68	<0.25	0.60	14000	24	49	240	30000	99	4700	500	1.2	2600	3.80	49	21	330
	5-10 cm	5900	na	20.0	74	<0.25	0.40	15000	23	58	260	31000	110	4500	490	1.1	2800	3.90	59	21	390
	5-10 cm	5500	na	32.0	87	<0.25	<0.10	16000	28	96	420	53000	170	4400	640	1.5	5100	8.70	64	24	520
	10-15 cm	6800	na	28.0	100	0.6	0.50	20000	29	110	390	45000	180	5400	650	1.9	4300	5.20	86	28	550
	10-15 cm	5600	na	29.0	99	<0.25	0.40	16000	27	100	410	47000	220	3700	650	1.5	4400	5.50	82	26	550
	15-20 cm	7900	na	39.0	150	0.7	0.50	25000	35	150	580	56000	260	5700	930	2.3	5600	5.80	140	32	720
	15-20 cm	5500	na	38.0	100	<0.25	<0.10	18000	24	110	410	50000	240	3700	610	1.0	5500	5.50	87	27	600
2022501 (Front yard)	0-5 cm	7100	na	14.0	90	<0.25	0.90	16000	19	36	160	26000	98	6800	530	0.8	1700	2.70	42	23	320
	0-5 cm	8100	na	13.0	80	0.6	1.00	17000	20	39	170	27000	110	7200	550	0.6	1700	2.80	45	26	330
	5-10 cm	7100	na	16.0	72	<0.25	1.10	17000	23	41	180	30000	95	7000	550	0.9	1800	2.80	41	25	340
	5-10 cm	8400	na	15.0	82	0.6	1.10	18000	21	44	190	29000	110	7400	580	0.8	1800	2.60	47	27	360
	10-15 cm	8700	na	18.0	87	0.6	0.90	20000	22	56	250	35000	120	7700	670	<0.25	2800	2.80	49	29	430
	10-15 cm	8800	na	18.0	93	0.7	1.10	20000	26	50	230	32000	120	7600	650	1.2	2300	3.30	51	28	410
2022502 (Back yard)	0-5 cm	10000	na	23.0	100	0.6	1.10	8400	30	58	260	40000	130	3900	700	1.4	3100	4.60	26	32	410
	0-5 cm	9500	na	19.0	84	0.6	0.90	8800	29	51	240	36000	120	3800	620	<0.25	2700	4.20	27	29	360
	5-10 cm	10000	na	26.0	89	0.6	0.80	7500	30	57	290	42000	120	3900	690	1.1	3300	5.10	23	31	400
	5-10 cm	10000	na	23.0	86	0.6	0.50	8700	32	57	290	44000	130	3900	720	1.4	3300	5.00	26	31	430
	10-15 cm	16000	na	16.0	110	0.8	0.80	6900	30	42	180	35000	84	4800	700	0.8	2200	3.40	23	39	270
	10-15 cm	13000	na	16.0	93	0.7	0.70	9700	31	49	220	38000	110	4800	660	0.9	2800	2.80	27	34	330

Table 2: Results of Chemical Analysis (µg/g) of 2000 Residential Soil Samples, Port Colborne

Al = aluminum, Sb = antimony, As = arsenic, Ba = barium, Be = beryllium, Cd = cadmium, Ca = calcium, Cr = chromium, Co = cobalt, Cu = copper, Fe = iron, Pb = lead, Mg = magnesium, Mn = manganese, Mo = molybdenum, Ni = nickel, Se = selenium, Sr = strontium, V = vanadium, Zn = zinc. Data in bold and underlined exceed MOE Table A effects-based guideline. na - data not available

Site / Location	Soil Depth	Al	Sb	As	Ba	Be	Cd	Ca	Cr	Co	Cu	Fe	Pb	Mg	Mn	Mo	Ni	Se	Sr	V	Zn
2022601 (Front yard)	0-5 cm	8700	na	11.0	83	<0.25	0.40	15000	17	38	170	24000	100	5700	400	0.8	1900	2.30	71	25	250
	0-5 cm	8500	na	10.0	77	<0.25	0.40	14000	17	34	150	22000	94	5400	370	<0.25	1600	2.40	58	23	230
	5-10 cm	6600	na	22.0	87	<0.25	0.60	18000	25	63	280	36000	120	6700	540	1.3	3100	3.70	88	27	350
	5-10 cm	8600	na	20.0	100	0.6	0.60	18000	23	70	300	34000	130	6800	590	1.1	3200	3.90	84	29	340
	10-15 cm	6100	na	43.0	88	<0.25	0.30	16000	34	110	500	55000	160	5700	750	1.9	5700	4.70	78	28	590
	10-15 cm	7300	na	45.0	120	0.6	0.40	18000	36	150	640	72000	180	6200	870	2.6	8400	5.40	80	32	610
2022602 (Back yard)	0-5 cm	10000	na	5.6	78	<0.25	0.60	35000	16	20	61	17000	61	11000	430	<0.25	330	1.00	120	26	110
	0-5 cm	10000	na	4.2	73	<0.25	0.50	32000	16	18	50	16000	53	10000	390	<0.25	300	1.00	120	26	98
	5-10 cm	8700	na	5.9	66	<0.25	0.60	30000	15	29	82	15000	77	11000	380	<0.25	580	1.60	97	26	110
	5-10 cm	8500	na	5.4	62	<0.25	0.50	29000	15	17	55	15000	57	10000	320	<0.25	380	1.20	92	26	91
	10-15 cm	9700	na	6.1	83	0.6	0.40	35000	17	25	85	16000	79	12000	350	<0.25	570	1.30	130	28	110
	10-15 cm	14000	na	10.0	120	0.9	1.50	37000	25	51	170	25000	170	13000	490	1.1	1200	2.70	250	40	260
2022701 (Front yard)	0-5 cm	8800	na	15.0	88	<0.25	0.80	11000	20	41	190	28000	150	3900	430	<0.25	2200	3.40	46	23	350
	0-5 cm	8400	na	15.0	86	<0.25	0.70	10000	23	41	180	28000	140	3900	410	1.3	2200	3.50	45	23	330
	5-10 cm	9100	na	18.0	88	<0.25	0.60	9400	27	46	220	32000	150	3700	470	0.9	2700	3.80	41	24	360
	5-10 cm	9500	na	24.0	120	0.6	0.80	11000	32	68	320	44000	200	4100	550	1.6	4300	5.20	47	27	500
	10-15 cm	6700	na	27.0	110	<0.25	0.70	13000	21	47	280	32000	210	4300	470	1.5	3200	5.20	48	20	440
	10-15 cm	7000	na	39.0	120	<0.25	1.00	13000	27	60	340	42000	250	3800	600	0.9	4100	5.90	53	21	590
2022702 (Back yard)	0-5 cm	9100	na	14.0	110	<0.25	0.90	6800	21	47	210	23000	170	2600	350	0.8	2400	3.70	39	24	370
	0-5 cm	8300	na	13.0	100	<0.25	0.60	6000	18	36	160	19000	130	2300	280	0.8	1700	3.60	36	20	330
	5-10 cm	12000	na	21.0	130	0.6	1.10	7100	29	61	270	32000	180	3000	450	1.1	3000	4.60	43	30	460
	5-10 cm	12000	na	15.0	130	0.6	0.90	6900	25	49	220	27000	150	2900	390	1.1	2500	3.70	42	28	390
	10-15 cm	12000	na	20.0	130	0.6	1.20	7500	27	58	280	33000	180	3100	440	<0.25	3000	4.40	47	28	440
	10-15 cm	12000	na	20.0	150	0.7	1.20	7700	29	60	280	34000	190	3300	470	0.8	3200	4.30	49	30	450

Table 2: Results of Chemical Analysis (µg/g) of 2000 Residential Soil Samples, Port Colborne

Al = aluminum, Sb = antimony, As = arsenic, Ba = barium, Be = beryllium, Cd = cadmium, Ca = calcium, Cr = chromium, Co = cobalt, Cu = copper, Fe = iron, Pb = lead, Mg = magnesium, Mn = manganese, Mo = molybdenum, Ni = nickel, Se = selenium, Sr = strontium, V = vanadium, Zn = zinc. Data in bold and underlined exceed MOE Table A effects-based guideline. na - data not available

Site / Location	Soil Depth	Al	Sb	As	Ba	Be	Cd	Ca	Cr	Co	Cu	Fe	Pb	Mg	Mn	Mo	Ni	Se	Sr	V	Zn
2022801 (Front yard)	0-5 cm	8400	na	11.0	75	<0.25	0.40	13000	18	33	140	24000	90	5400	460	0.8	1700	2.00	40	25	200
	0-5 cm	8000	na	9.5	76	<0.25	0.50	14000	16	33	140	23000	86	5500	420	1.1	1600	2.10	41	24	190
	5-10 cm	7600	na	26.0	92	0.6	0.60	16000	26	66	300	45000	170	6000	650	1.6	3800	4.10	61	26	460
	5-10 cm	7800	na	23.0	95	0.6	0.30	17000	25	71	300	45000	170	6200	610	1.4	4100	4.00	52	27	400
	10-15 cm	7700	na	34.0	97	0.6	<0.10	17000	31	91	410	61000	180	5700	710	2.3	6200	4.80	59	28	540
	10-15 cm	8900	na	24.0	110	0.7	0.30	19000	20	59	300	36000	150	6400	550	0.7	4000	3.90	64	28	340
2022802 (Back yard)	0-5 cm	15000	na	13.0	120	1	0.70	35000	27	56	190	27000	130	14000	490	1.0	1700	2.80	170	39	210
	0-5 cm	14000	na	15.0	120	1	0.60	38000	25	59	210	29000	140	14000	530	<0.25	1900	3.10	190	37	240
	5-10 cm	18000	na	13.0	140	1.1	0.50	37000	29	44	160	28000	120	15000	460	0.6	1400	2.70	190	41	190
	5-10 cm	18000	na	12.0	140	1.1	0.60	46000	30	41	170	29000	120	17000	500	1.0	1300	2.40	240	40	180
	10-15 cm	19000	na	10.0	140	1.1	0.50	43000	69	29	120	27000	89	15000	420	9.8	940	2.10	260	43	140
	10-15 cm	20000	na	11.0	150	1.2	0.70	49000	28	31	140	27000	99	18000	470	0.9	1100	2.00	210	42	170
2022901 (Front yard)	0-5 cm	8200	na	14.0	100	<0.25	0.50	7200	22	36	140	20000	96	2900	290	<0.25	1700	1.70	27	22	240
	0-5 cm	7900	na	13.0	81	<0.25	0.50	7300	16	34	130	20000	87	3000	280	0.6	1600	1.90	27	22	230
	5-10 cm	7000	na	18.0	85	<0.25	0.30	8400	19	52	200	29000	120	3300	390	1.0	2600	2.80	27	23	320
	5-10 cm	8800	na	14.0	99	<0.25	0.50	8200	19	48	180	24000	120	3300	380	1.0	2300	1.80	29	24	300
	10-15 cm	7500	na	62.0	130	0.6	0.40	14000	32	97	470	55000	230	4700	710	1.8	5800	5.60	46	28	690
	10-15 cm	7300	na	60.0	130	0.6	0.40	13000	33	100	480	59000	230	4400	720	2.2	6000	6.30	42	27	710
2023001 (Front yard)	0-5 cm	6700	na	16.0	140	<0.25	0.90	79000	45	47	220	29000	240	5900	430	1.3	2400	2.90	580	22	400
	0-5 cm	7500	na	16.0	140	0.6	1.10	91000	21	45	210	29000	200	5800	450	0.8	2200	3.20	690	24	380
	5-10 cm	6200	na	24.0	150	0.6	0.30	77000	26	72	340	48000	220	5600	580	1.6	4700	4.30	590	26	480
	5-10 cm	6800	na	33.0	160	0.6	0.60	74000	25	73	400	48000	250	6200	670	1.4	4500	4.90	560	26	550
	10-15 cm	5700	na	39.0	180	0.6	0.50	39000	28	79	480	50000	320	5400	690	1.6	5500	5.10	270	25	650
	10-15 cm	5800	na	40.0	190	0.6	<0.10	34000	32	110	550	68000	330	6700	830	2.0	8000	4.10	220	28	730

Table 2: Results of Chemical Analysis (µg/g) of 2000 Residential Soil Samples, Port Colborne

Al = aluminum, Sb = antimony, As = arsenic, Ba = barium, Be = beryllium, Cd = cadmium, Ca = calcium, Cr = chromium, Co = cobalt, Cu = copper, Fe = iron, Pb = lead, Mg = magnesium, Mn = manganese, Mo = molybdenum, Ni = nickel, Se = selenium, Sr = strontium, V = vanadium, Zn = zinc. Data in bold and underlined exceed MOE Table A effects-based guideline. na - data not available

Site / Location	Soil Depth	Al	Sb	As	Ba	Be	Cd	Ca	Cr	Co	Cu	Fe	Pb	Mg	Mn	Mo	Ni	Se	Sr	V	Zn
2023101 (Front yard)	0-5 cm	4800	na	14.0	77	<0.25	0.40	12000	17	33	180	18000	130	4100	310	1.2	**1700**	2.60	31	17	280
	0-5 cm	4300	na	14.0	68	<0.25	1.00	10000	17	29	150	18000	110	3500	300	0.8	**1500**	2.40	27	16	250
	5-10 cm	4400	na	19.0	92	<0.25	<0.10	14000	19	**52**	260	28000	170	4400	420	0.8	**2800**	3.00	34	19	380
	5-10 cm	4400	na	15.0	94	<0.25	0.40	14000	19	**55**	280	32000	180	4400	440	1.1	**3200**	2.90	36	22	380
	10-15 cm	5000	na	18.0	120	<0.25	<0.10	16000	23	**72**	**360**	48000	200	4800	570	1.5	**5100**	3.40	43	33	470
	10-15 cm	4300	na	17.0	120	<0.25	<0.10	15000	20	**58**	**330**	34000	180	4500	460	1.0	**4000**	3.00	38	23	400
2023102 (Back yard)	0-5 cm	4100	na	22.0	100	<0.25	0.30	9500	21	**64**	**330**	38000	**250**	2600	490	1.4	**4000**	4.40	39	20	500
	0-5 cm	3700	na	22.0	100	<0.25	<0.10	8300	18	**63**	**320**	33000	**270**	2100	470	1.3	**3900**	5.80	36	17	480
	5-10 cm	4200	na	**36.0**	120	<0.25	<0.10	10000	26	**91**	**460**	52000	**330**	2700	610	1.6	**6100**	5.50	43	24	610
	5-10 cm	4400	na	**37.0**	120	<0.25	0.30	9700	28	**95**	**480**	56000	**310**	2400	640	2.4	**6100**	5.60	42	25	630
	10-15 cm	4200	na	**39.0**	130	<0.25	<0.10	11000	31	**88**	**460**	48000	**360**	2500	630	2.3	**6100**	5.80	51	22	600
	10-15 cm	3900	na	**44.0**	110	<0.25	<0.10	9900	21	**91**	**420**	45000	**310**	2400	540	1.6	**6400**	5.40	41	20	540
2023201 (Front yard)	0-5 cm	10000	na	**30.0**	120	0.7	0.30	19000	27	**79**	**350**	41000	160	6200	640	1.6	**4200**	5.80	66	31	420
	0-5 cm	9600	na	**26.0**	120	0.7	0.40	19000	28	**84**	**360**	46000	170	6400	680	3.2	**4400**	5.50	57	31	450
	5-10 cm	10000	na	**37.0**	120	0.7	<0.10	18000	29	**86**	**410**	48000	160	5500	690	1.5	**5800**	6.50	64	29	460
	5-10 cm	9700	na	**49.0**	120	0.7	<0.10	17000	31	**110**	**480**	61000	200	5400	750	1.7	**7700**	5.80	56	31	520
	10-15 cm	11000	na	20.0	180	1.1	<0.10	32000	17	50	300	36000	130	6100	680	<0.25	**4300**	3.90	97	28	320
	10-15 cm	13000	na	28.0	240	**1.5**	<0.10	40000	18	**63**	**430**	39000	180	6500	850	0.6	**5400**	4.80	120	28	420
2023202 (Back yard)	0-5 cm	8900	na	11.0	96	<0.25	0.50	13000	17	30	140	21000	100	3800	440	0.9	**1500**	2.60	37	27	240
	0-5 cm	8700	na	9.9	110	<0.25	0.60	14000	17	26	120	19000	110	3800	440	0.8	**1300**	2.90	38	25	220
	5-10 cm	7300	na	12.0	92	<0.25	0.30	17000	18	36	170	24000	140	4000	460	<0.25	**2000**	2.50	44	28	260
	5-10 cm	7300	na	10.0	80	<0.25	0.40	18000	16	29	120	22000	110	3600	440	<0.25	**1500**	2.10	42	26	190
	10-15 cm	5800	na	**30.0**	100	<0.25	0.40	12000	21	**51**	240	33000	160	3900	410	0.9	**3000**	5.00	37	29	370
	10-15 cm	5700	na	18.0	93	<0.25	0.60	14000	22	40	190	24000	**210**	3600	370	0.8	**2200**	4.00	39	26	280

Table 2: Results of Chemical Analysis (µg/g) of 2000 Residential Soil Samples, Port Colborne

Al = aluminum, Sb = antimony, As = arsenic, Ba = barium, Be = beryllium, Cd = cadmium, Ca = calcium, Cr = chromium, Co = cobalt, Cu = copper, Fe = iron, Pb = lead, Mg = magnesium, Mn = manganese, Mo = molybdenum, Ni = nickel, Se = selenium, Sr = strontium, V = vanadium, Zn = zinc. Data in bold and underlined exceed MOE Table A effects-based guideline. na - data not available

Site / Location	Soil Depth	Al	Sb	As	Ba	Be	Cd	Ca	Cr	Co	Cu	Fe	Pb	Mg	Mn	Mo	Ni	Se	Sr	V	Zn
2023301 (Front yard)	0-5 cm	5900	na	**37.0**	99	0.6	<0.10	16000	31	**140**	**540**	67000	**220**	5800	830	2.4	**7000**	5.90	40	30	590
	0-5 cm	6200	na	**61.0**	110	0.6	0.40	16000	42	**170**	**670**	78000	**240**	5600	1000	3.2	**8000**	8.10	43	34	700
	5-10 cm	6100	na	**85.0**	120	0.6	<0.10	14000	46	**230**	**970**	130000	**360**	5500	1100	3.9	**17000**	7.90	37	30	**1000**
	5-10 cm	5900	na	**78.0**	94	<0.25	0.30	16000	32	**120**	**640**	66000	**290**	5900	800	2.9	**8800**	7.40	37	24	700
	10-15 cm	5900	na	25.0	70	<0.25	<0.10	21000	19	**72**	**350**	45000	130	6700	500	1.6	**5100**	3.70	44	22	360
	10-15 cm	5700	na	25.0	67	<0.25	<0.10	19000	15	51	280	32000	95	6700	420	0.8	**3500**	4.40	37	19	290
2023302 (Back yard)	0-5 cm	4700	na	19.0	68	<0.25	<0.10	7300	22	**84**	290	39000	140	2000	490	1.2	**3500**	4.40	25	26	360
	0-5 cm	3900	na	**29.0**	65	<0.25	<0.10	6200	22	**84**	280	30000	120	1400	450	1.7	**3100**	4.80	24	17	310
	5-10 cm	4700	na	**55.0**	100	<0.25	0.30	9400	34	**110**	**500**	66000	**330**	2100	660	2.6	**6800**	6.20	31	23	630
	5-10 cm	3900	na	**30.0**	69	<0.25	<0.10	5500	25	**66**	**330**	41000	140	1400	470	2.2	**4300**	4.10	22	16	400
	10-15 cm	4200	na	20.0	46	<0.25	<0.10	5300	17	44	200	27000	120	1300	300	1.0	**2800**	3.20	21	19	260
	10-15 cm	3200	na	9.5	30	<0.25	<0.10	3900	9	19	97	12000	59	990	180	<0.25	**1100**	1.70	15	12	130
2023401 (Back yard)	0-5 cm	9100	na	**350.0**	180	0.7	<0.10	18000	150	**160**	**860**	57000	**280**	3800	860	2.0	**6600**	3.40	66	33	710
	0-5 cm	7300	na	**77.0**	130	0.7	<0.10	11000	54	**160**	**580**	68000	160	2400	860	2.2	**7000**	6.30	44	31	590
	5-10 cm	12000	na	**69**	310	1	0.50	15000	63	**220**	**970**	99000	**390**	4100	1300	3.7	**11000**	7.8	67	44	**1200**
	5-10 cm	10000	na	**52**	150	0.8	<0.10	9500	49	**150**	**680**	98000	190	3400	1000	3.7	**8700**	7.5	41	37	780
	10-15 cm	13000	na	**69**	160	0.9	<0.10	13000	47	**120**	**630**	72000	**230**	5100	900	2.4	**7600**	6.6	53	40	650
	10-15 cm	14000	na	32	160	0.8	<0.10	13000	31	**91**	470	55000	160	4800	640	0.9	**5700**	6	50	37	470
2023501 (Front yard)	0-5 cm	9000	na	**35.0**	180	0.7	0.30	29000	33	**120**	**530**	49000	**310**	11000	850	2.1	**6200**	7.30	100	32	670
	0-5 cm	8500	na	**37.0**	180	0.8	<0.10	28000	40	**150**	**660**	69000	**370**	11000	1000	2.6	**8500**	7.50	85	35	**810**
	5-10 cm	11000	na	**67.0**	210	1	<0.10	30000	57	**200**	**1000**	90000	**400**	10000	1200	3.4	**14000**	8.60	95	39	**1100**
	5-10 cm	12000	na	**53.0**	200	1	<0.10	29000	45	**180**	**840**	93000	**370**	10000	1000	3.7	**13000**	8.40	100	41	**1000**
	10-15 cm	12000	na	**48.0**	200	0.9	<0.10	33000	36	**130**	**1000**	60000	**300**	10000	960	1.7	**12000**	7.10	100	36	**830**
	10-15 cm	9500	na	**48.0**	190	0.8	<0.10	29000	27	**140**	**980**	62000	**350**	8400	1100	2.3	**11000**	6.00	110	32	**840**
2023502 (Back yard)	0-5 cm	9800	na	22.0	240	0.7	0.70	17000	28	**70**	450	38000	**460**	3900	510	1.7	**5000**	5.20	90	29	720
	0-5 cm	9700	na	21.0	220	0.6	0.60	15000	26	**65**	400	37000	**400**	3700	470	1.9	**4400**	4.90	81	28	680
	5-10 cm	11000	na	**19.0**	240	0.7	0.30	17000	27	**68**	430	37000	**410**	3900	490	1.8	**4700**	5.20	90	30	660
	5-10 cm	9900	na	20.0	200	0.6	0.40	16000	25	**64**	**390**	34000	**380**	3600	450	1.0	**4300**	4.60	82	28	600
	10-15 cm	10000	na	20.0	230	0.7	0.60	25000	28	**69**	**390**	40000	**380**	4800	510	1.5	**4400**	6.80	94	30	620
	10-15 cm	12000	na	17.0	210	0.7	<0.10	39000	26	**54**	300	35000	**310**	6700	540	1.0	**3400**	3.50	110	31	480

Table 2: Results of Chemical Analysis (µg/g) of 2000 Residential Soil Samples, Port Colborne

Al = aluminum, Sb = antimony, As = arsenic, Ba = barium, Be = beryllium, Cd = cadmium, Ca = calcium, Cr = chromium, Co = cobalt, Cu = copper, Fe = iron, Pb = lead, Mg = magnesium, Mn = manganese, Mo = molybdenum, Ni = nickel, Se = selenium, Sr = strontium, V = vanadium, Zn = zinc. Data in bold and underlined exceed MOE Table A effects-based guideline. na - data not available

Site / Location	Soil Depth	Al	Sb	As	Ba	Be	Cd	Ca	Cr	Co	Cu	Fe	Pb	Mg	Mn	Mo	Ni	Se	Sr	V	Zn
2023601 (Rodney St ball diamond)	0-5 cm	5100	na	29.0	72	<0.25	<0.10	10000	20	140	560	43000	110	2700	440	1.4	7700	6.20	42	25	400
	0-5 cm	5300	na	29.0	71	<0.25	<0.10	10000	20	130	540	39000	110	2700	450	1.1	6400	5.40	46	25	390
	5-10 cm	5000	na	33.0	72	<0.25	<0.10	13000	21	150	630	49000	110	3600	460	1.3	8600	6.20	52	27	420
	5-10 cm	5500	na	32.0	74	<0.25	<0.10	14000	21	140	640	42000	110	3800	460	1.1	7500	6.50	62	25	430
	10-15 cm	5500	na	33.0	75	<0.25	<0.10	16000	22	130	610	42000	100	4500	410	1.7	7400	6.70	74	26	400
	10-15 cm	6100	na	35.0	74	<0.25	<0.10	20000	21	130	600	41000	99	5600	430	1.3	7300	7.00	97	27	370
2023701 (Front yard)	0-5 cm	10000	na	16.0	110	0.6	<0.10	20000	23	71	290	29000	160	7200	480	<0.25	3900	3.90	54	30	370
	0-5 cm	10000	na	16.0	120	0.7	<0.10	23000	23	85	340	31000	170	8200	530	1.0	4200	4.60	61	30	420
	5-10 cm	12000	na	22.0	130	0.7	<0.10	23000	25	85	340	32000	200	7500	510	1.2	4500	4.30	64	32	430
	5-10 cm	9700	na	23.0	120	0.6	<0.10	25000	23	96	400	34000	200	7500	530	1.6	5600	4.70	68	30	440
	10-15 cm	10000	na	73.0	160	0.8	<0.10	22000	42	210	1000	77000	350	6600	980	3.0	14000	8.30	68	34	930
	10-15 cm	10000	na	56.0	150	0.7	<0.10	23000	29	160	780	48000	310	6500	720	2.5	11000	8.80	81	31	690
2023702 (Back yard)	0-5 cm	9000	na	13.0	110	0.7	<0.10	23000	18	53	230	23000	180	7400	400	0.9	2600	5.20	120	29	320
	0-5 cm	7300	na	11.0	90	<0.25	<0.10	18000	14	48	190	25000	130	6200	370	0.8	2700	2.90	73	26	270
	5-10 cm	7900	na	12.0	100	0.6	<0.10	24000	17	45	190	24000	200	7800	400	0.8	2600	2.20	120	27	280
	5-10 cm	9000	na	15.0	110	0.7	<0.10	27000	16	45	210	25000	130	8000	440	1.1	2500	2.70	100	29	310
	10-15 cm	8100	na	12.0	120	0.6	0.30	27000	15	39	180	23000	280	8000	380	<0.25	2300	2.30	140	27	280
	10-15 cm	9200	na	14.0	120	0.7	0.30	26000	16	53	230	25000	200	7200	430	1.0	2700	2.70	98	30	340
2023801 (Front yard)	0-5 cm	18000	na	12.0	120	0.9	0.70	9900	27	47	150	25000	91	6000	390	1.0	1800	2.30	37	40	190
	0-5 cm	20000	na	130.0	120	0.9	0.60	7900	27	43	130	24000	83	5000	360	0.8	1600	2.10	36	41	180
	5-10 cm	21000	na	100.0	120	0.9	0.40	7100	26	29	89	25000	50	5900	410	<0.25	1000	1.50	29	43	140
	5-10 cm	22000	na	82.0	130	0.9	0.70	5500	28	19	61	26000	39	5600	400	<0.25	600	1.10	27	45	110
	10-15 cm	22000	na	110.0	130	0.9	0.50	6100	27	25	90	27000	46	5900	400	<0.25	1000	1.30	29	45	130
	10-15 cm	21000	na	130.0	130	0.9	<0.10	6200	27	24	93	27000	43	6000	400	0.8	1100	1.20	28	45	130
2023802 (Back yard)	0-5 cm	16000	na	16.0	190	0.9	0.90	14000	26	79	320	30000	320	5800	480	1.2	4300	3.80	51	36	520
	0-5 cm	17000	na	18.0	200	0.9	0.70	13000	30	83	340	32000	300	5700	480	1.3	4600	5.30	55	39	530
	5-10 cm	19000	na	15.0	200	1	0.40	12000	30	75	310	33000	280	6300	520	1.0	4200	3.20	44	43	470
	5-10 cm	18000	na	20.0	210	0.9	0.50	13000	28	84	460	34000	300	6300	550	0.6	4700	0.30	48	41	540
	10-15 cm	15000	na	32.0	230	1	0.30	18000	27	110	530	41000	410	6200	580	1.7	7500	0.30	66	37	690
	10-15 cm	13000	na	42.0	270	1.1	0.50	23000	37	140	740	49000	550	5700	640	2.1	11000	0.40	110	35	940
2023901 (Back yard)	0-5 cm	12000	na	6.5	80	0.6	<0.10	24000	18	46	120	18000	36	9200	460	<0.25	1100	<0.1	42	30	110

Table 2: Results of Chemical Analysis (µg/g) of 2000 Residential Soil Samples, Port Colborne

Al = aluminum, Sb = antimony, As = arsenic, Ba = barium, Be = beryllium, Cd = cadmium, Ca = calcium, Cr = chromium, Co = cobalt, Cu = copper, Fe = iron, Pb = lead, Mg = magnesium, Mn = manganese, Mo = molybdenum, Ni = nickel, Se = selenium, Sr = strontium, V = vanadium, Zn = zinc. Data in bold and underlined exceed MOE Table A effects-based guideline. na - data not available

Site / Location	Soil Depth	Al	Sb	As	Ba	Be	Cd	Ca	Cr	Co	Cu	Fe	Pb	Mg	Mn	Mo	Ni	Se	Sr	V	Zn
	0-5 cm	12000	na	10.0	84	0.6	<0.10	21000	17	47	170	20000	38	7700	510	<0.25	1400	<0.1	39	30	130
	5-10 cm	19000	na	7.7	110	0.9	0.70	24000	24	29	88	23000	27	12000	460	<0.25	780	<0.1	56	40	97
	5-10 cm	20000	na	11.0	120	0.9	0.50	22000	26	40	130	25000	35	12000	540	<0.25	1200	<0.1	49	42	110
	10-15 cm	18000	na	11.0	110	0.9	0.40	30000	42	39	150	24000	39	13000	480	0.8	1500	<0.1	60	39	110
	10-15 cm	20000	na	11.0	120	1	0.30	17000	29	64	240	29000	48	9800	540	<0.25	2300	<0.1	43	44	140
2024101 (Back yard)	0-5 cm	11000	na	13	120	0.6	<0.10	14000	22	70	250	21000	140	5500	360	0.9	3000	4.2	43	26	310
	5-10 cm	13000	na	11	130	0.7	1.00	15000	25	70	260	23000	150	6200	410	<0.25	3100	3.1	43	30	300
	5-10 cm	14000	na	14	140	0.8	0.40	20000	26	64	270	27000	160	9200	420	0.9	3200	3.7	47	34	350
	5-10 cm	12000	na	14	130	0.7	<0.10	21000	23	61	260	24000	130	8200	390	<0.25	3500	3.2	45	30	260
	10-15 cm	12000	na	34	180	0.8	0.30	19000	28	87	450	43000	280	6900	640	1.3	5900	3.9	57	33	520
	10-15 cm	14000	na	19	160	0.8	0.30	28000	25	60	330	31000	140	11000	480	<0.25	3600	3	60	36	370
	15-20 cm	8900	na	27	120	0.6	<0.10	17000	24	80	490	37000	160	5600	550	0.9	6100	5.2	62	33	380
	15-20 cm	9200	na	38	260	0.6	<0.10	16000	26	97	670	38000	210	5500	520	1.6	7800	5.2	58	33	450
2024201 (Side yard)	0-5 cm	13000	na	29.0	190	1	0.50	30000	38	84	400	34000	420	9200	620	1.4	5000	4.40	89	33	540
	5-10 cm	13000	na	28.0	180	1	0.50	32000	36	90	410	35000	330	9800	620	1.3	5300	4.20	85	33	540
	5-10 cm	10000	na	27.0	200	0.8	<0.10	28000	31	110	530	38000	440	7800	590	1.3	8200	5.30	74	27	610
	5-10 cm	12000	na	29.0	200	0.9	<0.10	30000	34	96	450	38000	440	8700	620	1.1	7600	5.80	81	30	540
	10-15 cm	12000	na	29.0	190	0.9	<0.10	32000	26	96	450	39000	330	8100	630	1.2	8600	5.00	77	28	610
	10-15 cm	18000	na	20.0	200	1.2	<0.10	33000	29	55	250	31000	230	11000	600	<0.25	3800	3.50	84	37	380
	15-20 cm	19000	na	18.0	200	1.4	0.40	31000	26	45	220	31000	340	8300	550	<0.25	3500	3.40	100	35	370
	15-20 cm	18000	na	18.0	190	1.2	<0.10	37000	35	54	250	33000	300	11000	620	<0.25	3200	2.60	90	35	360
2024601 (Back yard)	0-5 cm	12000	na	17.0	390	0.8	2.20	24000	36	38	370	31000	650	6500	450	1.3	2300	3.80	130	33	1200
	5-10 cm	12000	na	21.0	420	0.8	2.40	25000	41	41	380	31000	740	6900	480	0.9	2200	4.50	140	34	1300
	5-10 cm	11000	na	17.0	400	0.8	2.30	26000	36	36	360	30000	600	6900	410	1.3	1900	3.60	140	32	1100
	5-10 cm	11000	na	19.0	360	0.9	2.20	22000	34	42	430	38000	650	6400	430	1.6	3000	4.80	130	33	1200
	10-15 cm	12000	na	19.0	580	0.8	2.60	29000	44	35	430	40000	890	8700	500	1.8	2000	4.40	170	36	1300
	10-15 cm	10000	na	20.0	340	0.9	2.20	22000	34	38	410	34000	670	6500	400	1.5	2400	4.50	150	33	1000
	15-20 cm	14000	na	19.0	400	1.2	2.60	22000	38	35	420	38000	700	6400	470	1.1	2200	4.20	180	42	960
	15-20 cm	10000	na	23.0	380	0.9	2.60	21000	34	48	410	46000	870	5700	460	1.9	3200	5.10	160	33	1100
2292316 (Front	0-5 cm	15000	<0.4	9.0	154	1.1	0.75	27600	23	22	104	22500	213	13000	461	4.5	401	<0.3	113	33	183
2292317 (Front yard)	0-5 cm	17000	<0.4	10.5	150	1	1.41	17700	32	48	235	22500	236	7740	459	3.7	1680	<0.3	63	34	394

Part A - Soil Investigation: Tables

Table 2: Results of Chemical Analysis (µg/g) of 2000 Residential Soil Samples, Port Colborne

Al = aluminum, Sb = antimony, As = arsenic, Ba = barium, Be = beryllium, Cd = cadmium, Ca = calcium, Cr = chromium, Co = cobalt, Cu = copper, Fe = iron, Pb = lead, Mg = magnesium, Mn = manganese, Mo = molybdenum, Ni = nickel, Se = selenium, Sr = strontium, V = vanadium, Zn = zinc. Data in bold and underlined exceed MOE Table A effects-based guideline. na - data not available

Site / Location	Soil Depth	Al	Sb	As	Ba	Be	Cd	Ca	Cr	Co	Cu	Fe	Pb	Mg	Mn	Mo	Ni	Se	Sr	V	Zn
	5-10 cm	19100	<0.4	9.7	159	1.2	1.37	17100	32	48	246	24700	**206**	7850	463	3.7	**1670**	<0.3	64	39	368
	10-20 cm	25100	<0.4	10.5	201	1.5	1.29	17500	37	43	**337**	30100	188	9150	420	3.7	**1570**	<0.3	75	50	379
2292318 (Back yard)	0-5 cm	19300	<0.4	10.0	184	1.2	1.36	18100	32	35	164	26800	192	6820	446	3.9	**1320**	<0.3	71	38	314
	5-10 cm	21100	<0.4	9.4	179	1.2	1.17	22900	32	31	139	27300	154	7290	437	3.7	**1080**	<0.3	70	41	263
	10-20 cm	20200	<0.4	12.2	196	1.2	1.51	16600	33	36	174	28900	184	6940	412	3.7	**1670**	<0.3	69	40	318
2292319 (Front yard)	0-5 cm	17500	<0.4	10.9	145	1.1	1.34	19400	28	49	231	24700	**201**	8220	520	3.7	**1850**	<0.3	58	35	291
	5-10 cm	19800	<0.4	11.4	153	1.1	1.37	23100	29	50	236	26800	**203**	9020	555	3.8	**1920**	<0.3	65	39	287
	10-20 cm	18700	<0.4	15.1	174	1.2	1.56	22500	28	**57**	**301**	28300	**249**	8880	538	4.0	**2700**	0.50	75	37	327
2292320 (Back yard)	0-5 cm	20800	<0.4	17.3	288	**1.6**	2.17	19400	35	**51**	**333**	29400	**377**	7320	466	3.9	**2300**	<0.3	114	43	593
	5-10 cm	19900	<0.4	17.8	293	**1.5**	2.13	20300	34	**52**	**391**	30700	**369**	7220	497	4.2	**2670**	0.30	116	40	610
	10-20 cm	19600	<0.4	21.2	267	**1.5**	2.10	24500	31	46	**390**	30700	**336**	7400	486	3.9	**2790**	0.60	125	39	498
2292321 (Front yard)	0-5 cm	16000	<0.4	8.8	118	0.9	1.39	14000	26	**61**	242	20300	159	6300	397	3.5	**1580**	<0.3	41	35	248
	5-10 cm	16700	<0.4	6.7	106	0.9	0.95	11200	22	40	156	20000	124	5570	351	3.2	**1200**	<0.3	35	34	198
	10-20 cm	18000	<0.4	6.7	119	1.1	0.99	18800	23	32	124	20400	170	6710	361	3.6	976	<0.3	50	36	224
2292322 (Back yard)	0-5 cm	17700	<0.4	8.6	165	1.1	1.32	20600	25	33	152	22700	**231**	9120	382	3.8	**1210**	<0.3	407	35	339
	5-10 cm	19500	<0.4	7.8	172	**1.7**	1.23	21200	27	33	154	26400	**314**	9310	438	3.8	**1130**	<0.3	87	37	404
	10-20 cm	19200	<0.4	8.4	231	1.2	0.97	34700	27	26	119	24600	**589**	13000	484	4.3	974	<0.3	97	36	588
2292323 (Front yard)	0-5 cm	19900	<0.4	9.7	169	1.2	1.35	15300	30	38	211	26000	165	7020	578	3.6	**1380**	<0.3	59	40	298
	0-5 cm	19100	<0.4	9.5	158	1.2	1.09	13100	28	37	196	24100	143	6560	484	3.8	**1280**	<0.3	50	38	268
	0-5 cm	21000	<0.4	8.0	155	1.2	1.05	13500	30	35	179	26700	155	6700	588	3.9	**1310**	<0.3	53	42	299
	5-10 cm	19300	<0.4	9.9	158	1.1	1.19	12900	27	39	217	25500	156	6360	494	3.6	**1520**	<0.3	51	37	272
	5-10 cm	19800	<0.4	10.8	162	1.2	1.13	12300	30	39	219	27100	161	6410	517	3.7	**1550**	<0.3	48	38	265
	5-10 cm	21900	<0.4	8.6	168	1.2	1.09	12500	30	39	218	27300	167	6410	517	3.6	**1480**	<0.3	52	38	263
	10-20 cm	17800	<0.4	14.1	153	1.2	1.50	15900	26	38	245	29200	144	6420	633	3.5	**1850**	<0.3	56	34	396
	10-20 cm	20600	<0.4	11.9	150	1.2	1.13	14100	29	40	236	27000	146	6600	559	3.4	**1790**	<0.3	49	40	261
	10-20 cm	19800	<0.4	11.8	152	1.2	1.20	14000	27	39	223	29800	137	6340	697	3.6	**1840**	<0.3	51	38	314

Table 2: Results of Chemical Analysis (μg/g) of 2000 Residential Soil Samples, Port Colborne

Al = aluminum, Sb = antimony, As = arsenic, Ba = barium, Be = beryllium, Cd = cadmium, Co = cobalt, Cu = copper, Fe = iron, Pb = lead, Mg = magnesium, Mn = manganese, Mo = molybdenum, Ni = nickel, Se = selenium, Sr = strontium, V = vanadium, Zn = zinc. Data in bold and underlined exceed MOE Table A effects-based guideline. na - data not available

Site / Location	Soil Depth	Al	Sb	As	Ba	Be	Cd	Ca	Cr	Co	Cu	Fe	Pb	Mg	Mn	Mo	Ni	Se	Sr	V	Zn
2292324 (Back yard)	0-5 cm	13600	<0.4	13.2	185	1.1	2.07	14500	23	42	210	22900	256	5770	541	3.7	1820	0.30	78	31	394
	0-5 cm	15100	<0.4	1.3	188	1	1.45	13600	25	39	203	23600	224	5230	464	3.7	1920	<0.3	69	33	364
	0-5 cm	15700	<0.4	14.4	194	1.2	1.84	15000	27	44	227	25500	263	5610	438	3.8	2060	0.50	76	35	384
	5-10 cm	15700	<0.4	15.5	216	1.2	2.12	17000	27	48	246	27800	276	6630	611	4.0	2340	0.60	89	33	446
	5-10 cm	15900	0.4	13.8	211	1.1	1.57	15700	27	43	223	25900	266	6080	503	3.9	2230	0.50	77	35	418
	5-10 cm	15900	<0.4	18.4	219	1.3	2.37	17000	28	50	266	26800	344	6000	492	3.9	2650	0.60	87	35	424
	10-20 cm	15600	1.0	20.0	266	1.8	1.78	21900	26	42	241	24400	263	6220	504	4.1	2320	0.30	170	34	433
	10-20 cm	16700	<0.4	17.8	207	1.2	1.58	19500	28	41	230	26400	260	6700	494	3.8	2360	0.70	92	35	412
	10-20 cm	15500	<0.4	18.0	224	1.2	1.72	22000	28	43	245	28000	237	6570	431	4.2	2400	0.80	109	35	391
2292325 (Front yard)	0-5 cm	13500	<0.4	7.2	99	0.7	0.81	18900	20	25	120	16600	95	7760	321	4.0	894	<0.3	59	30	158
	5-10 cm	13500	<0.4	7.9	94	0.7	0.83	13500	19	32	143	15800	104	5380	278	3.6	1070	<0.3	46	29	163
	10-20 cm	13100	<0.4	9.8	92	0.7	0.87	12000	20	38	184	17800	88	4780	293	3.7	1700	<0.3	40	27	183
2292326 (Back yard)	0-5 cm	11300	<0.4	10.4	111	0.6	1.18	16000	23	28	160	16000	148	6380	269	3.8	939	<0.3	43	24	294
	5-10 cm	10400	<0.4	10.0	109	0.6	1.11	13500	19	28	158	16100	143	5740	246	3.6	1130	<0.3	43	23	315
	10-20 cm	11900	<0.4	11.2	141	0.7	0.94	15000	20	31	242	16700	173	5890	244	3.6	1170	<0.3	49	24	329
2292327 (Front yard)	0-5 cm	28200	<0.4	12.1	218	1.3	1.63	20700	37	72	303	28900	210	10500	453	4.8	3340	<0.3	64	52	351
	5-10 cm	29500	<0.4	15.0	234	1.3	2.03	27100	42	96	438	32100	216	12200	497	4.7	4710	0.45	69	54	432
	10-20 cm*	20200	0.6	21.2	183	1	0.90	47625	31	76	416	33100	175	18675	590	3.2	5785	2.58	83	39	331
2292328 (Back yard)	0-5 cm	26900	<0.4	12.2	192	1.3	1.76	30200	33	61	266	32700	212	11700	724	4.8	2820	<0.3	73	48	331
	5-10 cm	26100	<0.4	9.4	161	1.2	1.28	35500	31	48	207	29700	133	15600	604	5.0	2280	<0.3	70	46	255
	10-20 cm	21500	<0.4	9.5	141	1	0.77	51700	27	32	155	26100	94	21200	550	5.0	1820	<0.3	81	39	175
2292329 (Front yard)	0-5 cm	24600	<0.4	13.7	203	1.3	1.85	21200	36	71	292	28800	261	9280	596	4.8	3310	<0.3	73	52	430
	5-10 cm	25200	<0.4	14	205	1	1.90	21200	36	82	344	30500	228	9270	596	5	4120	1	75	53	406
	10-20 cm*	25025	0.5	17.5	208	1.2	1.21	17725	35	71	328	30450	199	8538	709	2.7	3593	1.41	65	51	344
2292330 (Back yard)	0-5 cm	25900	<0.4	8.6	162	1.3	0.90	17900	32	35	136	27800	109	8120	459	4.4	1440	<0.3	69	48	213
	5-10 cm	31100	<0.4	8.2	182	1.5	0.87	32700	36	35	128	32700	93	10000	538	4.6	1310	<0.3	81	55	192
	10-20 cm*	20400	0.5	13.8	143	1.1	0.79	15425	28	42	196	25700	99	5823	444	2.6	2088	1.18	71	43	218

Table 2: Results of Chemical Analysis (µg/g) of 2000 Residential Soil Samples, Port Colborne

Al = aluminum, Sb = antimony, As = arsenic, Ba = barium, Be = beryllium, Cd = cadmium, Ca = calcium, Cr = chromium, Co = cobalt, Cu = copper, Fe = iron, Pb = lead, Mg = magnesium, Mn = manganese, Mo = molybdenum, Ni = nickel, Se = selenium, Sr = strontium, V = vanadium, Zn = zinc. Data in bold and underlined exceed MOE Table A effects-based guideline. na - data not available

Site / Location	Soil Depth	Al	Sb	As	Ba	Be	Cd	Ca	Cr	Co	Cu	Fe	Pb	Mg	Mn	Mo	Ni	Se	Sr	V	Zn
2292331 (Side yard)	0-5 cm	23800	<0.4	13.0	119	1.1	1.04	24400	34	**67**	243	30000	169	9020	774	4.7	**2880**	<0.3	36	49	270
	5-10 cm	26600	<0.4	12	138	1	0.90	49500	34	49	188	29900	**337**	9310	744	5	**2100**	<0.3	43	51	245
	10-20 cm	25700	<0.4	9.0	109	1.1	0.58	80100	28	23	63	25200	73	8080	682	5.3	**524**	0.34	49	46	111
2292332 (Back yard)	0-5 cm	19400	<0.4	8.7	122	0.8	0.83	14600	28	27	107	19300	91	7490	386	4.0	**1130**	<0.3	49	35	194
	5-10 cm*	19400	0.5	10.9	124	0.8	0.74	15325	28	29	127	18375	94	7445	355	2.4	**1248**	0.60	53	36	212
	10-20 cm*	16050	0.5	13.8	121	0.8	0.77	33750	24	32	156	17275	123	17425	323	3.0	**1615**	0.70	83	31	243
2292333 (Front yard)	0-5 cm	18700	<0.4	13.1	141	0.9	1.44	12600	32	**70**	**305**	26500	**261**	6760	619	4.1	**3320**	1.00	42	40	412
	5-10 cm*	16700	0.6	16.4	131	0.8	0.99	12200	29	**65**	**318**	25800	**246**	6555	605	2.5	**3650**	1.48	37	38	380
	10-20 cm*	16275	0.5	18.2	135	0.8	0.91	13075	27	**58**	**315**	26175	**234**	6495	612	2.3	**3693**	2.35	38	36	341
2292334 (Back yard)	0-5 cm*	16475	0.4	11.0	104	0.7	0.69	9180	24	35	149	19875	90	4635	315	2.2	**1620**	0.65	36	34	215
	5-10 cm	18500	<0.4	9	101	1	0.78	9600	23	32	132	21700	78	4950	340	4	**1530**	<0.3	36	37	183
	10-20 cm*	17125	0	14	112	1	0.66	13125	24	32	173	20775	89	5543	326	2	**1703**	1	46	35	214
2292335 (Front yard)	0-5 cm	21300	<0.4	8.6	125	0.9	0.99	12100	32	**64**	208	22700	179	6160	427	4.1	**2140**	<0.3	44	42	267
	5-10 cm	21100	<0.4	8.4	123	0.9	0.98	13300	30	**76**	237	23600	172	6640	439	4.1	**2600**	<0.3	43	42	273
	10-20 cm*	20550	0.5	22.3	121	1	0.85	10363	32	**100**	441	29425	164	5645	500	2.5	**5913**	3.15	42	41	307
2292336 (Back yard)	0-5 cm*	13100	0.4	11.3	102	0.6	1.27	13400	43	**65**	239	21775	170	5835	416	2.6	**2598**	2.23	45	34	349
	5-10 cm	15800	<0.4	13	104	1	1.55	12000	31	**89**	**376**	36400	171	5660	482	4	**4670**	3	43	36	430
	10-20 cm*	13600	0.3	6.2	75	0.5	0.50	30010	19	21	90	21475	55	4580	325	1.9	**1165**	0.56	30	36	142
2292337 (Front yard)	0-5 cm	21200	<0.4	18.6	157	1.1	1.60	11900	34	**112**	**384**	28800	**208**	6850	615	4.0	**4680**	1.56	39	48	401
	5-10 cm	18000	<0.4	14	116	1	1.10	9130	27	**57**	242	26900	101	6260	636	4	**3090**	0	28	42	227
	10-20 cm*	17650	0.4	14.4	104	0.8	0.58	9745	27	48	231	25125	86	5995	569	2.2	**3133**	1.58	28	39	204
2292338 (Back yard)	0-5 cm	22700	<0.4	12.5	186	1.1	1.26	11100	33	**55**	249	28200	153	5770	555	4.1	**2820**	0.30	53	47	328
	5-10 cm	18500	<0.4	12	138	1	0.92	9200	26	40	178	21700	95	4780	485	4	**2000**	<0.3	43	37	230
	10-20 cm*	18800	0.4	14.9	168	1	0.67	11058	28	45	226	23625	112	5068	434	2.3	**2698**	0.92	49	40	250

Table 2: Results of Chemical Analysis (µg/g) of 2000 Residential Soil Samples, Port Colborne

Al = aluminum, Sb = antimony, As = arsenic, Ba = barium, Be = beryllium, Cd = cadmium, Ca = calcium, Cr = chromium, Co = cobalt, Cu = copper, Fe = iron, Pb = lead, Mg = magnesium, Mn = manganese, Mo = molybdenum, Ni = nickel, Se = selenium, Sr = strontium, V = vanadium, Zn = zinc.   Data in bold and underlined exceed MOE Table A effects-based guideline.   na - data not available

Site / Location	Soil Depth	Al	Sb	As	Ba	Be	Cd	Ca	Cr	Co	Cu	Fe	Pb	Mg	Mn	Mo	Ni	Se	Sr	V	Zn
2292339 (Front yard)	0-5 cm	24700	<0.4	16.5	339	1.2	1.46	14000	36	**99**	**345**	26700	**425**	7590	474	0.8	**3390**	0.40	56	52	694
	0-5 cm	22800	0.5	21.4	348	1.2	1.68	14400	36	**152**	**497**	25700	**431**	7610	464	0.9	**4730**	1.51	58	50	717
	0-5 cm	23900	<0.4	17	356	1	1.38	12600	34	**114**	**380**	26000	**465**	7240	490	1	**3650**	1	53	50	661
	5-10 cm	29900	1	31	326	1	2.07	16000	43	**222**	**694**	32800	352	8870	494	1	**7000**	2	66	59	697
	5-10 cm	28000	1	27	250	1	1.55	15900	38	**188**	**616**	30300	**251**	8740	493	1	**6330**	1	62	56	523
	5-10 cm	28600	<0.4	24.4	305	**1.4**	1.63	14500	42	**179**	**561**	29500	**312**	8070	491	0.8	**5610**	0.88	63	55	553
	10-20 cm*	28675	1.1	**30.2**	243	**1.4**	1.28	17650	42	**162**	**654**	32275	**224**	8638	464	2.0	**7540**	3.58	68	54	600
	10-20 cm*	25800	1.2	**36.9**	269	**1.3**	1.19	17225	45	**214**	**871**	35200	**282**	8295	500	2.1	**9025**	5.83	69	52	**914**
	10-20 cm	28075	1.1	**31.2**	243	**1.4**	1.24	19500	45	**174**	**718**	33825	**249**	8930	486	2.1	**8363**	3.65	69	53	605
2292340 (Back yard)	0-5 cm	15700	<0.4	**26.5**	143	0.9	1.12	15000	25	31	165	20700	184	5840	466	0.4	**1230**	<0.3	62	37	273
	0-5 cm*	13475	0.3	12.4	97	0.7	0.58	10048	21	24	116	19175	76	4945	516	1.2	935	0.65	41	32	211
	0-5 cm*	11700	0.4	11.6	101	0.7	0.60	12250	19	26	148	18425	113	4860	441	1.3	**857**	0.69	46	30	231
	5-10 cm*	15075	0.5	**32.5**	120	0.8	0.78	15525	24	32	175	21150	184	5828	473	1.3	**1480**	0.58	52	36	227
	5-10 cm*	11375	<0.47	7.8	62	0.5	0.34	8195	15	16	63	17700	39	4250	568	1.0	486	0.45	28	27	104
	5-10 cm*	11750	0	11	79	1	0.54	12500	17	23	110	18325	78	4883	519	1	865	1	39	29	169
	10-20 cm*	13825	0.4	**27.5**	128	0.9	0.69	14325	23	32	180	19725	156	5323	426	1.3	**1665**	0.50	61	34	235
	10-20 cm*	12950	0.4	17.5	115	0.8	0.69	12925	20	31	167	19525	96	4938	476	1.2	**1425**	0.60	56	32	220
	10-20 cm*	11375	0.4	15.8	117	0.7	0.70	14000	19	35	221	19050	152	4745	428	1.0	**1680**	0.63	53	30	261
2292341 (Front yard)	0-5 cm	25600	<0.4	23.2	235	**1.7**	2.45	19400	38	**66**	320	26900	**244**	8540	489	5.4	**2970**	6.30	77	53	422
	5-10 cm	28700	<0.4	16	205	**2**	1.52	29600	37	49	208	31500	153	12800	613	6	**2060**	0	77	56	294
	10-20 cm*	24500	0.6	**44.3**	227	**1.4**	2.42	24575	38	**100**	**566**	36400	**321**	9285	777	2.9	**6733**	2.65	77	48	546
2292342 (Back yard)	0-5 cm	26100	<0.4	**31.8**	261	**1.6**	2.37	17700	40	**64**	**372**	25700	**283**	7300	401	5.1	**3110**	1.20	90	54	434
	5-10 cm*	23500	<0.4	**44**	253	**2**	2.83	18100	39	**69**	**392**	25200	**315**	7290	390	5	**3710**	1	92	51	479
	10-20 cm*	22850	0.8	**47.6**	287	**1.4**	1.97	19275	38	**67**	**433**	27400	**367**	6773	281	2.6	**4570**	1.68	102	46	525
2292343 (Front yard)	0-5 cm	17900	<0.4	18.8	282	**1.4**	2.16	27100	37	**107**	**469**	29900	**622**	10300	589	6.3	**5230**	2.40	92	44	618
	5-10 cm	16700	<0.4	**32.1**	288	**1.5**	2.44	32000	35	**116**	**621**	34600	**596**	10400	704	6.5	**7740**	<0.3	109	36	638
	10-20 cm*	18700	0.8	**25.3**	277	**1.4**	1.30	32400	33	**60**	423	30600	**511**	7990	635	2.7	**5670**	1.84	123	37	472

Part A - Soil Investigation: Tables

Table 2: Results of Chemical Analysis (µg/g) of 2000 Residential Soil Samples, Port Colborne

Al = aluminum, Sb = antimony, As = arsenic, Ba = barium, Be = beryllium, Cd = cadmium, Ca = calcium, Cr = chromium, Co = cobalt, Cu = copper, Fe = iron, Pb = lead, Mg = magnesium, Mn = manganese, Mo = molybdenum, Ni = nickel, Se = selenium, Sr = strontium, V = vanadium, Zn = zinc. Data in bold and underlined exceed MOE Table A effects-based guideline. na - data not available

Site / Location	Soil Depth	Al	Sb	As	Ba	Be	Cd	Ca	Cr	Co	Cu	Fe	Pb	Mg	Mn	Mo	Ni	Se	Sr	V	Zn
2292344 (Back yard)	0-5 cm	23300	<0.4	13.5	188	1.4	1.44	16700	33	43	220	24900	159	6880	432	5.5	2080	0.50	80	48	325
	5-10 cm	23400	<0.4	14.1	188	1.4	1.47	16300	33	47	236	25900	176	6900	431	5.3	2330	<0.3	80	48	331
	10-20 cm	25100	<0.4	17.0	200	1.5	1.58	18000	36	50	265	26900	177	7410	434	5.3	2580	<0.3	87	51	334
2292345 (Front yard)	0-5 cm	20200	<0.4	14.5	179	1.4	1.59	30800	32	58	288	27900	179	12000	612	5.7	2770	0.40	82	43	387
	5-10 cm	24200	<0.4	21	209	2	1.86	30600	36	74	406	36400	185	12500	985	6	4380	1	79	47	438
	10-20 cm*	24325	1	22	270	2	2.04	38000	35	52	311	48400	211	11425	1648	3	3620	2	112	44	699
2292346 (Front yard)	0-5 cm	17000	9.2	6.3	160	1.1	1.83	34400	32	40	142	21600	169	15300	819	6.2	999	<0.3	117	42	402
	5-10 cm	18900	9.3	8.0	177	1.3	2.45	36200	32	47	166	26100	217	13700	1540	6.0	1240	<0.3	139	44	610
2292347 (Front yard)	0-5 cm	17700	6.9	10.8	152	1	1.55	24600	33	55	272	25900	196	9980	489	5.7	2650	<0.3	76	41	374
	5-10 cm	19500	8	14	170	1	1.74	23300	33	64	314	28000	260	9790	479	6	3070	<0.3	78	43	395
	10-20 cm*	18550	3	16	163	1	1.55	24075	31	53	274	26550	184	9155	475	3	2655	1	73	39	333
2292348 (Back yard)	0-5 cm	23100	7.2	11.0	194	1.2	2.90	24500	48	66	318	27200	241	12600	457	5.8	2620	<0.3	69	49	536
	5-10 cm	26700	7.7	14.9	210	1.4	3.31	22000	48	84	389	30900	267	11000	480	5.7	3680	<0.3	65	53	631
	10-20 cm*	24700	2.9	16.8	189	1.3	2.14	26650	40	55	300	30375	164	11575	445	3.3	2963	1.35	75	48	405
2292349 (Back yard)	0-5 cm*	16200	1.9	7.1	156	0.8	1.22	22050	25	24	134	20925	134	7238	409	3.0	859	0.65	106	3227	
	5-10 cm	21900	8	8	201	1	1.40	23300	33	33	178	25700	189	7800	456	5	1110	<0.3	136	40	351
	10-20 cm*	21200	3	12	312	1	1.82	30500	34	44	345	28450	270	8108	437	3	1960	1	179	39	509
2292350 (Front yard)	0-5 cm*	17375	1.3	6.2	96	0.7	0.77	7375	25	25	89	19750	87	4893	430	2.5	832	0.63	28	36	154
	5-10 cm*	16750	1.3	5.6	89	0.7	0.71	6853	23	25	89	19450	77	4525	428	2.4	811	0.58	25	35	133
	10-20 cm*	17325	1.0	8.6	98	0.7	0.84	7020	24	36	148	22075	92	4890	474	2.3	1465	0.80	25	36	162
2292351 (Back yard)	0-5 cm*	18250	1.9	9.6	157	1	0.97	14525	31	31	124	23000	167	8130	446	2.9	1285	0.68	57	40	232
	5-10 cm*	21250	2	10	162	1	0.98	14800	32	33	134	25775	198	8368	478	3	1328	1	63	42	234
	10-20 cm*	21450	2.2	10.0	162	1	0.85	16375	30	32	126	26150	196	8383	476	3.0	1275	0.68	65	42	215
2292352 (Back yard)	0-5 cm	19300	0.8	12.7	128	0.8	1.25	14000	38	64	455	25100	164	6360	591	1.1	2300	<0.3	45	44	343
	5-10 cm	19500	0.8	14.4	123	0.9	1.13	13200	29	69	569	25800	122	6310	624	0.8	2570	0.90	44	44	316
	10-20 cm	21600	<0.4	13.2	120	0.9	0.98	11900	33	54	516	26700	100	6360	612	0.8	2040	<0.3	45	47	284

Table 2: Results of Chemical Analysis (µg/g) of 2000 Residential Soil Samples, Port Colborne

Al = aluminum, Sb = antimony, As = arsenic, Ba = barium, Be = beryllium, Cd = cadmium, Ca = calcium, Cr = chromium, Co = cobalt, Cu = copper, Fe = iron, Pb = lead, Mg = magnesium, Mn = manganese, Mo = molybdenum, Ni = nickel, Se = selenium, Sr = strontium, V = vanadium, Zn = zinc. Data in bold and underlined exceed MOE Table A effects-based guideline. na - data not available

Site / Location	Soil Depth	Al	Sb	As	Ba	Be	Cd	Ca	Cr	Co	Cu	Fe	Pb	Mg	Mn	Mo	Ni	Se	Sr	V	Zn
2292353 (Front yard)	0-5 cm	10400	3.2	13.5	166	0.5	2.33	66300	30	77	299	29400	562	31900	478	2.3	2590	0.70	104	27	721
2292354 (Front yard)	0-5 cm	18900	0.7	12.1	137	0.8	1.01	10800	31	118	342	26000	123	6180	553	0.9	3900	1.50	54	46	290
	5-10 cm	22600	<0.4	13.0	131	1	0.93	8720	29	92	303	27300	86	6200	537	0.7	3190	<0.3	56	50	225
	10-20 cm	14300	<0.4	7.2	64	0.5	0.40	3550	21	32	120	21000	30	3470	564	0.2	1420	<0.3	24	36	131
2292355 (Front yard)	0-5 cm	13500	<0.4	8.3	61	0.4	0.52	6690	17	51	148	21900	63	3300	328	0.5	2150	<0.3	22	33	164
	5-10 cm	15400	<0.4	5.4	48	0.4	0.36	4910	17	30	85	17100	34	2980	269	0.5	974	<0.3	16	34	92
	10-20 cm	15200	1.7	43.0	162	1	2.04	15800	32	167	792	59700	333	6200	627	2.0	12000	6.10	56	43	743
2292356 (Back yard)	0-5 cm	16100	0.4	12.7	176	0.8	1.15	10200	29	46	193	22600	166	3750	319	0.9	2070	0.40	54	38	350
	5-10 cm	19700	1.0	18.8	290	1.4	1.87	9100	34	60	284	28800	282	3730	343	1.7	3100	0.40	101	44	542
	10-20 cm	23300	5.3	38.6	569	2.4	2.30	15100	53	87	619	42600	540	4440	477	2.8	6300	2.20	219	55	923
2292357 (Front yard)	0-5 cm	14100	1.1	25.5	212	0.7	1.77	26400	47	143	581	34800	327	10800	577	1.5	6770	4.30	59	39	467
	5-10 cm	12600	1.1	36.7	161	0.7	2.13	30900	35	163	775	49000	295	10400	646	1.7	10600	5.80	63	38	568
	10-20 cm	15500	2.3	33.9	259	0.9	2.21	32000	34	111	751	41700	329	9680	665	1.2	9300	4.50	86	40	886
2292358 (Back yard)	0-5 cm	16300	1.0	24.1	178	1	1.47	12900	28	83	407	38800	212	5820	472	1.0	5300	3.60	59	49	565
	0-5 cm	16200	1.2	26.1	206	0.9	1.73	13300	28	87	437	40600	257	5710	496	0.9	6010	3.80	63	48	654
	0-5 cm	15100	0.5	20.4	152	0.7	1.19	12400	25	73	349	30100	176	5430	407	0.8	4550	1.30	50	40	406
	5-10 cm	18500	1.8	35.3	212	1.2	1.77	16500	31	78	460	49400	221	6300	519	1.3	5470	2.40	79	56	754
	5-10 cm	15800	1.5	31.3	191	1.2	1.72	16000	26	67	393	58100	195	5550	553	1.5	4740	1.10	89	62	944
	5-10 cm	17500	0.7	29.8	192	0.9	1.33	15400	31	84	457	36800	249	6280	482	0.7	6250	0.80	60	44	511
	10-20 cm	17100	1.3	27.7	224	1.1	1.38	17800	26	57	366	43900	206	6000	464	1.0	4110	1.20	78	52	637
	10-20 cm	16800	0.9	27.3	175	1	1.28	17200	30	63	355	39100	168	5840	448	0.9	4390	1.50	74	48	572
	10-20 cm	18000	0.5	30.7	197	0.9	1.41	17600	33	78	455	37100	237	6470	455	0.8	5820	0.80	67	44	529
2292359 (Front yard)	0-5 cm	18200	<0.4	3.6	95	0.6	0.25	14600	22	13	31	19000	23	6570	422	0.3	162	<0.3	48	42	74
	5-10 cm	18800	<0.4	4.0	95	0.6	0.29	13300	32	12	29	20100	22	6100	472	0.3	117	<0.3	46	43	73
	10-20 cm	19600	<0.4	5.6	108	0.7	0.34	20900	24	18	68	23600	47	9150	495	0.5	632	<0.3	62	45	101
2292360 (Back yard)	0-5 cm	20800	<0.4	7.7	169	0.8	0.62	27100	25	25	116	25700	117	9670	518	0.5	1000	<0.3	67	44	175
	5-10 cm	32800	<0.4	11.3	224	1.3	0.62	38900	35	35	152	42800	117	14800	722	0.7	1550	<0.3	90	63	238
	10-20 cm	32500	0.7	23.4	377	1.4	1.18	25600	43	77	390	48500	292	12500	599	1.2	5210	0.70	86	67	491

Table 2: Results of Chemical Analysis (µg/g) of 2000 Residential Soil Samples, Port Colborne

Al = aluminum, Sb = antimony, As = arsenic, Ba = barium, Be = beryllium, Cd = cadmium, Ca = calcium, Cr = chromium, Co = cobalt, Cu = copper, Fe = iron, Pb = lead, Mg = magnesium, Mn = manganese, Mo = molybdenum, Ni = nickel, Se = selenium, Sr = strontium, V = vanadium, Zn = zinc. Data in bold and underlined exceed MOE Table A effects-based guideline. na - data not available

Site / Location	Soil Depth	Al	Sb	As	Ba	Be	Cd	Ca	Cr	Co	Cu	Fe	Pb	Mg	Mn	Mo	Ni	Se	Sr	V	Zn
2292361 (Front yard)	0-5 cm	20300	<0.4	5.6	121	0.7	0.42	20500	24	21	73	21300	37	9660	478	0.6	**528**	<0.3	73	44	111
	5-10 cm	23400	<0.4	6.8	148	0.8	0.45	31700	28	28	118	27100	64	14500	571	0.8	**1190**	<0.3	82	49	166
	10-20 cm	28400	<0.4	10.3	199	1.1	0.63	38800	34	**56**	231	32700	99	16900	541	1.1	**2540**	<0.3	90	58	200
2292362 (Back yard)	0-5 cm	24400	<0.4	7.0	159	0.9	0.38	26400	28	24	98	26400	85	11400	415	0.5	**954**	<0.3	65	50	155
	5-10 cm	35800	<0.4	9.9	242	**1.4**	0.47	34500	40	31	131	39600	111	15300	620	0.8	**1300**	<0.3	88	68	197
	10-20 cm	34900	<0.4	9.3	232	**1.4**	0.40	38100	37	31	150	36900	106	14900	561	0.7	**1320**	<0.3	95	66	197
2292363 (Front yard)	0-5 cm	15300	<0.4	3.7	102	0.5	0.42	13700	20	11	41	16800	19	5770	432	0.4	161	<0.3	51	34	80
	5-10 cm	21400	<0.4	5.7	158	0.7	0.49	19100	27	17	67	22900	39	9390	518	0.7	**454**	<0.3	77	46	113
	10-20 cm	29600	<0.4	7.5	203	1.1	0.56	34600	36	28	132	30600	78	16900	593	0.8	**1120**	<0.3	119	58	147
2292364 (Back yard)	0-5 cm	28000	0.5	9.8	214	1.1	0.72	29100	34	32	176	32100	154	12200	481	0.6	**1350**	<0.3	80	55	241
	5-10 cm	32000	<0.4	10.2	227	**1.3**	0.47	37600	35	31	146	35600	136	14900	519	0.6	**1530**	<0.3	91	62	209
	10-20 cm	31800	<0.4	11.9	272	**1.3**	0.56	39200	36	46	217	37000	**254**	15600	557	0.8	**2120**	<0.3	101	63	251
2292365 (Back yard)	0-5 cm	16000	5.3	**33.2**	447	**1.5**	5.08	33000	45	**175**	**720**	43400	**1130**	8140	819	2.2	**6930**	8.70	129	48	**1590**
	5-10 cm	20000	6.0	**47.7**	557	**2.2**	4.19	43500	47	**177**	**977**	72100	**1290**	7700	954	2.9	**12100**	6.30	192	46	**1440**
	10-20 cm	33100	<0.4	**32.3**	579	**4.6**	4.35	93400	50	**88**	**917**	47600	**877**	7390	1440	6.7	**8680**	1.30	393	33	**1390**
2292366 (Front yard)	0-5 cm	19400	<0.4	23.4	160	0.7	1.69	16400	40	**115**	**462**	43200	**213**	8830	669	5.2	**6870**	3.40	56	42	587
	5-10 cm	22200	<0.4	**41.2**	193	0.8	2.41	15200	48	**147**	**671**	57400	**245**	8460	804	5.9	**10500**	6.60	51	43	**836**
	10-20 cm	24400	<0.4	24.2	171	0.9	1.57	15100	40	**78**	**384**	39500	157	8620	654	4.8	**5380**	3.00	52	48	463
2292367 (Back yard)	0-5 cm	22400	<0.4	**26.7**	190	0.9	1.78	13800	37	**81**	**357**	35500	**207**	6480	671	4.4	**4240**	1.80	68	46	441
	5-10 cm	21200	<0.4	**30.0**	178	0.9	1.60	10500	36	**68**	**336**	36300	168	5950	606	4.4	**4610**	1.00	61	43	392
	10-20 cm	22700	<0.4	**33.4**	198	1	1.68	13200	38	**58**	**319**	36600	189	6420	582	4.7	**4180**	0.70	75	45	381
2292368 (Front yard)	0-5 cm	17300	<0.4	8.3	109	0.6	0.83	15200	25	32	153	20300	66	7120	409	4.0	**1470**	<0.3	54	36	190
	5-10 cm	17200	<0.4	9.0	123	0.6	0.99	16800	26	46	212	22200	100	7400	453	4.1	**1860**	<0.3	55	36	259
	10-20 cm	19100	<0.4	**30.3**	184	0.8	2.17	20900	38	**129**	745	46500	**252**	9270	680	5.4	**9120**	5.10	64	39	745
2292369 (Back yard)	0-5 cm	16200	<0.4	18.2	162	0.7	1.43	21900	31	**73**	**370**	30300	**213**	7350	520	4.7	**4310**	1.70	70	33	405
	5-10 cm	17200	<0.4	24.0	163	0.7	1.47	22600	33	**83**	**434**	34000	**205**	7990	538	4.7	**5430**	1.80	69	33	414
	10-20 cm	18500	<0.4	23.8	177	0.8	1.46	26900	31	**72**	**433**	33900	**206**	8070	542	4.7	**5100**	1.80	78	35	420

Table 2: Results of Chemical Analysis (µg/g) of 2000 Residential Soil Samples, Port Colborne

Al = aluminum, Sb = antimony, As = arsenic, Ba = barium, Be = beryllium, Cd = cadmium, Ca = calcium, Cr = chromium, Co = cobalt, Cu = copper, Fe = iron, Pb = lead, Mg = magnesium, Mn = manganese, Mo = molybdenum, Ni = nickel, Se = selenium, Sr = strontium, V = vanadium, Zn = zinc.   Data in bold and underlined exceed MOE Table A effects-based guideline.   na - data not available

Site / Location	Soil Depth	Al	Sb	As	Ba	Be	Cd	Ca	Cr	Co	Cu	Fe	Pb	Mg	Mn	Mo	Ni	Se	Sr	V	Zn
2292370 (Front yard)	0-5 cm	17700	<0.4	8.9	128	0.6	1.01	18700	27	42	176	24800	115	8290	505	4.4	1660	<0.3	59	35	239
	5-10 cm	17100	<0.4	10.8	131	0.6	1.09	16000	31	50	204	26500	135	7600	550	4.5	2060	<0.3	43	34	261
	10-20 cm	22500	<0.4	17.1	165	0.8	1.30	20100	39	56	276	36600	153	9070	644	4.7	3190	0.30	50	40	321
2292371 (Back yard)	0-5 cm	16300	<0.4	16.1	198	0.9	1.51	18200	28	58	335	27600	206	5990	548	4.5	3030	1.20	69	32	413
	5-10 cm	17100	<0.4	20.5	233	0.8	1.56	19800	31	66	411	31900	224	6500	588	4.6	3750	0.30	74	34	461
	10-20 cm	17400	<0.4	20.5	231	0.8	1.50	20900	34	61	454	31900	235	6270	619	4.8	3600	0.30	82	33	465
2292372 (Front yard)	0-5 cm	21700	<0.4	12.7	136	0.7	2.16	17700	35	51	214	27900	117	8140	444	4.8	2130	<0.3	60	41	274
	5-10 cm	20600	<0.4	14.8	154	0.7	2.86	19200	33	55	231	27800	145	8810	463	4.8	2280	0.50	64	39	328
	0-5 cm	20500	<0.4	12.9	129	0.6	2.32	16000	31	49	207	28300	109	7340	411	4.5	2270	<0.3	55	37	263
	5-10 cm	25700	<0.4	12.5	145	0.8	1.95	12500	35	50	202	30700	105	7140	451	4.4	2130	<0.3	51	45	253
	5-10 cm	22500	<0.4	13.9	148	0.8	3.94	14600	36	61	230	30200	139	7900	480	4.3	2380	<0.3	54	42	337
	10-20 cm	23100	<0.4	13.2	140	0.7	1.84	13000	33	47	201	29900	106	6370	440	4.2	2270	<0.3	51	41	258
	10-20 cm	23100	<0.4	16.4	150	0.8	2.19	16600	36	62	284	32800	136	7460	501	4.6	3360	<0.3	57	41	357
	10-20 cm	21600	<0.4	23.9	160	0.8	3.40	19600	38	90	423	41300	177	8880	567	5.3	5360	2.20	64	39	482
	10-20 cm	21000	<0.4	20.6	176	0.7	2.08	17900	43	73	339	36100	180	7010	485	4.9	4180	1.30	59	37	377
2292373 (Back yard)	0-5 cm	19200	<0.4	10.7	149	0.7	1.30	13200	30	46	197	26900	188	5190	408	4.3	2060	<0.3	48	38	287
	5-10 cm	18200	<0.4	9.1	154	0.8	1.17	17900	29	43	184	26100	162	6070	415	4.3	1890	<0.3	53	37	301
	0-5 cm	18300	<0.4	10.2	155	0.9	1.25	14700	31	47	204	26600	183	5890	426	4.2	1960	<0.3	53	38	345
	5-10 cm	20300	<0.4	11.7	174	1	1.27	11400	32	48	218	28400	207	5990	440	4.1	2200	<0.3	53	40	390
	5-10 cm	19200	<0.4	10.2	152	0.8	1.07	11900	30	40	169	25300	158	5720	418	3.9	1660	<0.3	46	39	289
	10-20 cm	20100	<0.4	11.5	171	0.9	1.23	10900	32	49	204	28300	188	5930	435	4.1	2170	<0.3	51	40	355
	10-20 cm	23500	<0.4	18.8	311	1.2	1.67	19200	41	65	376	41300	438	7440	534	5.0	4260	<0.3	78	43	559
	10-20 cm	19300	<0.4	13.8	203	1	1.28	26700	37	48	262	31300	236	7250	483	4.9	2780	<0.3	75	39	385
	10-20 cm	21900	<0.4	23.5	301	1.2	1.89	21400	49	69	443	38400	453	7830	516	5.1	4470	0.90	86	44	615
2292374 (Front yard)	0-5 cm	18400	<0.4	10.2	137	0.8	2.07	20700	31	55	206	30000	133	10300	485	4.6	2170	<0.3	59	38	323
	5-10 cm	20800	<0.4	16.9	165	1	2.83	24000	37	73	300	40000	171	11700	679	5.0	3440	1.10	68	43	441
	10-20 cm	21200	<0.4	17.0	169	1.1	2.38	27400	36	56	276	40800	156	12600	623	5.0	3190	<0.3	78	43	397

Part A - Soil Investigation: Tables

Table 2: Results of Chemical Analysis (µg/g) of 2000 Residential Soil Samples, Port Colborne

Al = aluminum, Sb = antimony, As = arsenic, Ba = barium, Be = beryllium, Cd = cadmium, Ca = calcium, Cr = chromium, Co = cobalt, Cu = copper, Fe = iron, Pb = lead, Mg = magnesium, Mn = manganese, Mo = molybdenum, Ni = nickel, Se = selenium, Sr = strontium, V = vanadium, Zn = zinc. Data in bold and underlined exceed MOE Table A effects-based guideline. na - data not available

Site / Location	Soil Depth	Al	Sb	As	Ba	Be	Cd	Ca	Cr	Co	Cu	Fe	Pb	Mg	Mn	Mo	Ni	Se	Sr	V	Zn
2292375 (Back yard)	0-5 cm	22700	<0.4	6.7	157	0.9	1.64	12800	32	42	153	27000	138	7080	522	4.1	1350	<0.3	48	44	278
	5-10 cm	24400	<0.4	7.1	161	1	1.69	10700	35	44	163	29600	130	6880	568	3.8	1440	<0.3	44	47	276
	10-20 cm	22800	<0.4	4.7	145	0.9	1.36	9950	33	33	121	28800	97	6540	602	3.9	1100	<0.3	38	45	225
2292376 (Front yard)	0-5 cm	20000	<0.4	13.1	174	0.9	1.44	22000	36	68	319	32500	329	10100	470	4.7	3340	0.90	69	41	403
	5-10 cm	25400	<0.4	17.4	212	1.1	1.63	24200	42	73	375	40000	188	11000	537	4.9	4050	1.10	77	49	452
	10-20 cm	30800	1.6	23.1	267	1.7	1.16	29300	40	52	303	42200	199	13000	533	1.2	2950	0.40	100	64	362
2292377 (Back yard)	0-5 cm	17200	1.5	14.6	190	1.1	1.56	45500	28	55	275	25900	174	23300	470	1.3	2300	1.00	111	40	380
2292378 (Back yard)	0-5 cm	16300	2.9	16.4	178	1	1.67	18200	26	50	252	22800	393	7550	343	0.7	2300	0.70	100	39	381
	5-10 cm	15300	3.7	17.1	186	1	1.69	19300	26	52	328	23800	490	7700	363	0.7	2290	0.90	96	38	395
	10-20 cm	17500	8.7	42.9	307	1.3	3.17	22000	40	117	694	43300	1140	8850	563	1.6	6670	3.80	130	48	938
2292379 (Front yard)	0-5 cm	11800	1.5	14.6	135	0.7	1.40	41400	33	130	640	22600	205	21400	514	1.3	3870	1.60	89	36	281
	5-10 cm	14800	2.2	31.6	176	1	2.51	31200	38	220	1120	33400	331	16000	613	1.8	6320	4.30	82	47	501
	10-20 cm	23400	1.5	28.4	226	1.4	2.30	31700	43	146	816	46600	246	14600	682	1.6	6060	1.00	93	54	470
2292380 (Back yard)	0-5 cm	29200	2	9.9	221	1.5	0.96	21300	42	41	163	35200	298	12900	605	1.0	1070	<0.3	149	61	256
	5-10 cm	30600	1.2	9.0	208	1.5	0.78	26100	46	36	132	34900	172	15100	612	1.1	869	<0.3	147	63	225
	10-20 cm	32800	<0.4	9.3	206	1.6	0.69	24400	47	34	113	36200	131	13300	616	0.9	768	<0.3	131	67	204
2292381 (Back yard)	0-5 cm	14900	2.8	10.9	297	0.8	2.84	54400	35	47	204	25500	594	23500	502	1.8	962	0.30	149	31	635
2292382 (Front yard)	0-5 cm	11900	<0.4	9.1	115	0.8	0.81	26700	21	31	162	22800	103	10900	476	5.1	1060	<0.3	79	25	179
2292382 (Back yard)	0-5 cm	21000	0.4	7.3	197	1.3	0.92	26300	32	28	113	30300	140	10400	594	4.6	666	<0.3	99	37	208
	5-10 cm	23800	0.5	5.7	219	1.5	0.89	26800	33	31	120	31900	160	10700	602	4.5	703	<0.3	100	41	224
	10-20 cm	24200	0.4	6.7	192	1.4	0.80	25600	34	28	108	30400	158	10200	541	4.7	620	<0.3	108	43	200
2292384 (Front yard)	0-5 cm	11200	<0.4	6.2	90	0.7	0.54	23000	18	15	73	16200	114	8530	322	4.1	563	<0.3	73	23	136
	5-10 cm	11100	<0.4	8.4	96	0.7	0.57	24900	17	16	78	17300	105	8640	328	4.2	626	<0.3	84	23	141
	10-20 cm	10800	<0.4	6.8	97	0.7	0.55	24700	17	17	74	16700	106	8120	314	4.1	702	<0.3	92	23	132
2292385 (Back yard)	0-5 cm	18000	<0.4	5.3	135	1	0.58	21800	25	20	69	22500	95	9030	448	4.0	502	<0.3	90	34	151
	5-10 cm	20800	0.4	5.9	144	1.1	0.57	23900	28	21	71	26400	103	9820	508	4.2	511	<0.3	94	37	146
	10-20 cm	21700	1.0	6.1	163	1.2	0.64	24000	31	23	84	27100	119	9830	477	4.4	576	<0.3	110	40	170

Table 2: Results of Chemical Analysis (μg/g) of 2000 Residential Soil Samples, Port Colborne

Al = aluminum, Sb = antimony, As = arsenic, Ba = barium, Be = beryllium, Cd = cadmium, Ca = calcium, Cr = chromium, Co = cobalt, Cu = copper, Fe = iron, Pb = lead, Mg = magnesium, Mn = manganese, Mo = molybdenum, Ni = nickel, Se = selenium, Sr = strontium, V = vanadium, Zn = zinc. Data in bold and underlined exceed MOE Table A effects-based guideline. na - data not available

Site / Location	Soil Depth	Al	Sb	As	Ba	Be	Cd	Ca	Cr	Co	Cu	Fe	Pb	Mg	Mn	Mo	Ni	Se	Sr	V	Zn
2292386 (Front yard)	0-5 cm	11400	<0.4	5.2	81	0.7	0.47	22800	17	13	52	15000	71	9150	275	3.9	400	<0.3	81	23	114
	5-10 cm	12000	<0.4	5.5	86	0.7	0.48	24800	17	14	54	15700	76	9460	317	4.1	465	<0.3	90	24	115
	10-20 cm	9710	<0.4	6.4	86	0.6	0.43	26200	15	13	48	16400	66	10300	371	4.0	435	<0.3	102	21	100
2292387 (Back yard)	0-5 cm	13600	<0.4	5.7	107	0.8	0.57	23100	21	17	64	19700	82	7780	378	4.1	489	<0.3	97	26	140
	5-10 cm	17700	<0.4	7.1	135	1.1	0.56	25800	28	18	64	22500	89	8850	414	4.0	454	<0.3	107	32	137
	10-20 cm	14400	<0.4	5.5	103	0.9	0.46	21500	20	15	50	19600	76	7360	349	3.7	418	<0.3	91	29	112
2292388 (Front yard)	0-5 cm	12300	<0.4	5.4	86	0.8	0.51	19900	18	14	53	16100	69	6930	331	3.8	478	<0.3	76	24	127
	5-10 cm	12300	<0.4	5.9	88	0.8	0.55	21300	18	15	56	17200	70	7120	321	3.8	507	<0.3	82	24	140
	10-20 cm	13100	<0.4	5.5	99	0.8	0.57	23800	19	15	66	17400	76	7030	338	3.9	523	<0.3	99	25	139
2292389 (Back yard)	0-5 cm	9470	<0.4	6.4	93	0.8	0.77	24500	15	18	79	18500	102	5300	442	3.8	851	<0.3	76	18	203
	5-10 cm	8940	<0.4	7.8	91	0.8	0.75	25500	15	16	75	16900	104	5480	397	3.7	707	<0.3	82	17	190
	10-20 cm	10600	<0.4	10.0	138	1.3	0.95	41700	17	22	110	20400	151	8030	487	4.4	1100	<0.3	138	17	234
2292390 (Front yard)	0-5 cm	10200	<0.4	5.0	79	0.6	0.46	22800	16	12	51	15300	49	6160	352	3.9	401	<0.3	71	20	109
	5-10 cm	11100	<0.4	5.7	84	0.7	0.52	22300	18	15	58	17200	59	6860	365	3.7	527	<0.3	73	23	130
	10-20 cm	11400	<0.4	6.7	87	0.8	0.53	24500	17	16	61	18300	66	7220	347	3.8	595	<0.3	86	23	129
2292391 (Back yard)	0-5 cm	11000	<0.4	7.8	121	1	1.02	28900	18	22	102	20600	174	6470	432	4.0	1030	<0.3	96	20	256
	5-10 cm	12100	<0.4	9.2	132	1	1.02	32000	18	22	108	20300	171	7050	478	4.4	986	<0.3	113	21	260
	10-20 cm	10300	<0.4	8.9	126	0.9	0.98	30000	18	26	112	21800	221	6600	470	4.5	1280	<0.3	104	20	263
2292392 (Front yard)	0-5 cm	7180	<0.4	14.6	155	0.7	1.85	43700	22	43	214	24200	278	20200	485	5.5	1910	1.30	149	17	442
	5-10 cm	7450	0.4	19.8	161	0.7	2.56	47300	26	60	299	35700	282	19300	644	5.8	3010	1.70	149	18	488
	10-20 cm	8780	<0.4	17.6	201	0.8	1.70	32400	20	34	215	25700	304	7830	511	4.7	2190	0.60	132	16	422
2292393 (Back yard)	0-5 cm	10400	<0.4	6.9	98	0.6	0.86	12900	18	26	111	22200	117	4550	491	3.8	1070	<0.3	41	25	285
	5-10 cm	8820	0.9	16.1	145	0.6	1.79	19700	24	48	242	32800	230	5480	596	4.4	2440	1.60	61	23	577
	10-20 cm	7140	1.0	23.8	268	0.6	2.32	27800	24	42	293	33100	383	6240	574	4.7	2420	1.20	88	15	910
2292394 (Front yard)	0-5 cm	10300	<0.4	14.2	120	0.7	1.51	18800	25	46	217	32700	196	7040	537	4.6	2420	1.20	63	24	370
	5-10 cm	13100	0.4	19.2	152	0.9	2.02	20700	31	54	284	37000	253	7500	662	4.7	2890	1.90	67	26	467
	10-20 cm	11500	<0.4	17.2	148	0.8	1.64	23300	25	43	260	33200	229	6910	563	4.5	2820	0.70	64	25	442

Table 2: Results of Chemical Analysis (µg/g) of 2000 Residential Soil Samples, Port Colborne

Al = aluminum, Sb = antimony, As = arsenic, Ba = barium, Be = beryllium, Cd = cadmium, Ca = calcium, Cr = chromium, Co = cobalt, Cu = copper, Fe = iron, Pb = lead, Mg = magnesium, Mn = manganese, Mo = molybdenum, Ni = nickel, Se = selenium, Sr = strontium, V = vanadium, Zn = zinc. Data in bold and underlined exceed MOE Table A effects-based guideline. na - data not available

Site / Location	Soil Depth	Al	Sb	As	Ba	Be	Cd	Ca	Cr	Co	Cu	Fe	Pb	Mg	Mn	Mo	Ni	Se	Sr	V	Zn
2292395 (Back yard)	0-5 cm	13400	<0.4	6.1	101	0.7	0.64	13900	18	18	70	18600	84	4830	491	3.8	**589**	<0.3	46	27	169
	5-10 cm	11300	<0.4	13.3	136	0.7	1.17	19700	22	35	164	25500	166	5810	611	4.2	**1400**	0.40	62	27	329
	10-20 cm	8770	0.7	19.2	188	0.7	1.62	27500	25	39	229	33700	**253**	6940	513	4.8	**2120**	0.70	75	22	491
2292396 (Front yard)	0-5 cm	18300	0.5	8.4	137	1	1.07	13500	28	32	141	28500	**299**	6470	493	4.1	**1270**	<0.3	43	37	269
	5-10 cm	18900	0.6	9.5	153	1	1.22	16600	29	36	162	29500	**311**	7390	514	4.4	**1510**	<0.3	47	39	298
	0-5 cm	19400	0.8	10.3	143	1	1.13	15400	30	36	164	32600	**332**	7010	551	4.3	**1700**	<0.3	49	38	295
	5-10 cm	22700	0.8	8.6	156	1.1	1.06	11900	32	34	145	31400	**283**	6890	529	3.9	**1420**	<0.3	41	43	266
	5-10 cm	26800	1.0	6.7	179	1.3	0.99	12200	35	30	127	32600	**261**	7580	591	4.0	**1130**	<0.3	103	49	240
	5-10 cm	21200	1.0	10.0	148	1	1.10	14600	31	37	178	33400	**349**	7220	581	4.3	**1690**	<0.3	46	40	295
	10-20 cm	15600	0.8	19.9	168	1	2.10	20800	36	**65**	**312**	47000	**666**	7570	657	5.0	**3690**	1.90	54	31	534
	10-20 cm	15500	1.0	18.9	161	0.9	1.79	28100	32	**56**	290	43900	**462**	8560	726	5.1	**3260**	0.50	63	29	483
	10-20 cm	19300	0.9	17.0	164	1	1.66	21800	35	**52**	270	42000	**580**	8060	663	4.8	**2910**	<0.3	56	35	449
2292397 (Back yard)	0-5 cm	9060	1.7	**27.1**	236	0.6	2.37	22900	25	46	240	31500	**448**	5120	524	4.4	**2630**	1.30	94	22	726
	5-10 cm	7640	0.8	25.0	183	0.6	2.56	24100	24	44	239	29200	**393**	5800	563	4.5	**2260**	1.90	93	20	556
	0-5 cm	10000	1.8	**30.1**	235	0.7	2.48	24500	29	47	262	29500	**510**	5630	572	4.7	**2380**	3.00	107	23	683
	5-10 cm	7060	1.5	**37.2**	238	0.6	2.48	31900	28	47	293	35400	**464**	6370	554	4.9	**3010**	1.10	116	17	752
	5-10 cm	7490	1.6	**41.9**	284	0.7	2.95	36000	27	46	**325**	32400	**549**	6560	580	5.0	**2840**	3.20	141	15	719
	5-10 cm	7320	2.9	**38.9**	248	0.7	2.61	31800	27	49	**309**	35800	**583**	6410	568	5.0	**3230**	2.40	119	17	685
	10-20 cm	6240	1.2	**29.4**	217	0.6	2.02	37500	23	34	250	27800	**408**	6450	484	4.7	**2220**	0.80	146	14	570
	10-20 cm	6070	1.2	**27.3**	225	0.5	2.05	35300	22	33	237	28200	**405**	6370	485	4.7	**2140**	1.90	131	14	589
	10-20 cm	5930	1.5	**29.3**	244	0.6	2.01	35900	25	37	249	31600	**454**	6520	480	4.7	**2550**	1.80	136	14	709
2292398 (Front yard)	0-5 cm*	15550	0.4	9.2	126	0.8	0.96	15100	24	33	158	28600	197	6805	481	4.1	**1575**	<0.3	54	33	260
	5-10 cm*	16300	0.5	12.4	135	0.9	1.12	16350	26	37	186	32100	**223**	6735	497	4.2	**1955**	0.33	54	32	294
	10-20 cm*	16850	0.3	14.0	149	0.9	1.19	24000	28	39	242	34350	**246**	8580	526	4.5	**2165**	0.38	69	33	385
2292399 (Back yard)	0-5 cm*	10045	<0.4	13.5	92	0.5	1.00	13450	19	24	117	19550	137	3910	331	3.5	**1130**	0.28	55	23	235
	5-10 cm*	10435	<0.4	16.7	100	0.5	1.09	16300	20	24	124	20550	143	4330	343	3.6	**1200**	<0.3	60	22	235
	10-20 cm*	7935	<0.4	24.1	148	0.5	1.44	25300	22	28	177	24250	199	5310	401	3.9	**1660**	0.66	99	17	311

Table 2: Results of Chemical Analysis (µg/g) of 2000 Residential Soil Samples, Port Colborne

Al = aluminum, Sb = antimony, As = arsenic, Ba = barium, Be = beryllium, Cd = cadmium, Ca = calcium, Cr = chromium, Co = cobalt, Cu = copper, Fe = iron, Pb = lead, Mg = magnesium, Mn = manganese, Mo = molybdenum, Ni = nickel, Se = selenium, Sr = strontium, V = vanadium, Zn = zinc. Data in bold and underlined exceed MOE Table A effects-based guideline. na - data not available

Site / Location	Soil Depth	Al	Sb	As	Ba	Be	Cd	Ca	Cr	Co	Cu	Fe	Pb	Mg	Mn	Mo	Ni	Se	Sr	V	Zn
2292400 (Front yard)	0-5 cm	17800	0.5	14.5	116	1	1.23	10100	27	36	177	28000	135	4170	581	3.7	**1880**	0.40	56	35	259
	5-10 cm	19200	0.4	17.7	134	1	1.33	11400	29	38	193	31200	156	4430	626	4.0	**2160**	0.80	67	36	286
	10-20 cm	14300	0.9	22.9	149	0.8	1.49	16800	27	45	246	35500	**208**	4420	528	4.2	**3190**	2.00	95	28	351
2292401 (Back yard)	0-5 cm	17400	1.0	10.3	131	0.9	1.05	13200	26	30	137	29900	182	4930	680	4.4	**1380**	<0.3	59	35	299
	5-10 cm	16700	1.0	10.1	127	0.9	1.03	12400	22	27	134	29800	190	5120	689	4.2	**1360**	<0.3	54	27	283
	10-20 cm	11700	0.6	11.5	165	0.8	1.09	21700	22	27	164	29600	**230**	5420	527	4.2	**1650**	0.40	93	27	362
2292402 (Front yard)	0-5 cm	20800	1.0	8.0	169	1	0.96	28300	31	24	99	29200	**279**	8640	536	4.7	**818**	<0.3	85	39	253
	5-10 cm	29000	0.8	4.9	198	**1.3**	0.79	27800	35	22	75	32300	**215**	9840	627	4.5	**552**	<0.3	88	50	202
	10-20 cm	29500	<0.4	6.2	174	**1.3**	0.76	24800	35	27	92	34400	114	9260	660	4.4	**742**	<0.3	79	51	166
2292403 (Back yard)	0-5 cm	18100	0.8	4.1	99	0.8	0.83	19200	22	14	57	19800	76	8870	587	4.2	191	<0.3	83	34	171
	5-10 cm	17500	<0.4	3.0	92	0.8	0.71	21400	22	13	47	19100	59	8860	575	4.1	170	<0.3	93	33	139
	10-20 cm	17600	0.7	2.6	89	0.7	0.56	23700	21	12	44	18500	48	8560	539	4.2	137	<0.3	114	33	118
2292404 (Front yard)	0-5 cm	23100	<0.4	4.3	140	1.1	0.60	46500	28	21	64	24900	68	15300	544	4.9	**295**	<0.3	109	40	148
	5-10 cm	23700	<0.4	6.1	145	1.2	0.78	47800	29	28	87	27600	98	15900	571	4.7	**509**	<0.3	119	42	182
	10-20 cm	14300	<0.4	10.6	109	0.8	1.10	28900	24	36	136	23800	148	9250	497	4.4	**952**	<0.3	90	30	260
2292405 (Back yard)	0-5 cm	16800	0.5	3.3	97	0.7	0.50	12000	20	11	41	19500	65	5700	387	3.5	170	<0.3	51	32	114
	5-10 cm	16300	0.4	6.5	126	0.7	0.75	19400	21	16	60	22300	121	7000	433	3.8	**432**	<0.3	65	31	195
	10-20 cm	6830	1.9	19.6	264	0.5	2.15	28800	23	32	219	31900	**417**	6190	550	4.3	**1660**	1.10	95	16	635
2292406 (Front yard)	0-5 cm	18300	0.5	3.1	93	0.8	0.52	12400	21	14	55	18200	53	5590	366	3.5	**313**	<0.3	69	35	102
	5-10 cm	22500	<0.4	5.9	132	1	0.69	21200	26	21	108	21400	80	7820	403	3.9	**639**	<0.3	106	42	140
	10-20 cm	24600	0.7	7.6	155	1.2	0.98	30200	30	23	122	25100	111	10000	448	4.5	**721**	<0.3	162	44	201
2292407 (Back yard)	0-5 cm	16300	<0.4	13.9	233	1.1	1.64	21000	30	32	187	29400	**258**	6520	536	4.2	**1400**	<0.3	115	33	465
	5-10 cm	16100	2.1	22.2	373	**1.3**	2.41	27800	40	48	**325**	43200	**394**	7130	740	5.2	**2580**	1.00	163	32	743
	10-20 cm	16400	3.1	**37.9**	451	**1.4**	3.25	32900	59	52	**392**	49200	**617**	6960	852	5.8	**3070**	1.80	215	30	**1000**
2292408 (Front yard)	0-5 cm	23200	<0.4	5.9	124	1.1	0.84	15900	34	25	95	29000	99	8800	534	4.2	**769**	<0.3	57	45	176
	5-10 cm	26400	<0.4	5.8	136	**1.3**	0.81	16900	37	25	94	30000	96	9470	552	4.1	**710**	<0.3	58	49	169
	10-20 cm	29200	<0.4	4.7	144	**1.3**	0.61	19800	37	20	63	30400	68	11000	561	4.2	**461**	<0.3	55	52	131

Table 2: Results of Chemical Analysis (µg/g) of 2000 Residential Soil Samples, Port Colborne

Al = aluminum, Sb = antimony, As = arsenic, Ba = barium, Be = beryllium, Cd = cadmium, Cr = chromium, Ca = calcium, Co = cobalt, Cu = copper, Fe = iron, Pb = lead, Mg = magnesium, Mn = manganese, Mo = molybdenum, Ni = nickel, Se = selenium, Sr = strontium, V = vanadium, Zn = zinc. Data in bold and underlined exceed MOE Table A effects-based guideline.   na - data not available

Site / Location	Soil Depth	Al	Sb	As	Ba	Be	Cd	Ca	Cr	Co	Cu	Fe	Pb	Mg	Mn	Mo	Ni	Se	Sr	V	Zn
2292409 (Back yard)	0-5 cm	19900	<0.4	12.2	173	1	1.62	15400	32	31	203	30300	**285**	6780	514	4.4	**1250**	<0.3	94	40	383
	5-10 cm	23600	0.4	15.6	210	1.2	1.76	15200	37	34	190	36300	**259**	6410	526	4.6	**1630**	<0.3	117	43	402
	10-20 cm	17000	1.3	**29.7**	699	1.1	2.82	23900	43	49	**1620**	47300	**534**	5480	628	5.5	**2800**	2.20	209	30	923
2292410 (Front yard)	0-5 cm	17800	<0.4	5.5	119	0.9	0.81	26100	26	19	95	22200	119	11300	497	4.1	**600**	<0.3	83	35	181
	5-10 cm*	18400	0.4	7.8	132	0.9	0.97	26900	30	18	86	24350	125	11000	578	3.1	**593**	0.51	86	37	207
	10-20 cm*	22850	0.5	11.9	190	1.2	0.93	25350	34	25	133	30375	**201**	10600	605	3.1	**1218**	0.55	136	45	246
2292411 (Back yard)	0-5 cm*	13650	1.8	8.6	99	0.5	1.07	26300	27	14	72	18825	91	10000	500	2.9	**361**	0.38	80	29	189
	5-10 cm*	12950	2	7	101	1	1.40	37950	30	13	83	20450	124	13775	562	3	**469**	0	83	28	242
	10-20 cm*	12975	2.3	7.5	130	0.6	1.69	47175	33	16	116	22325	**276**	17275	571	3.9	**618**	0.40	132	28	302
2292412 (Back yard)	0-5 cm	16700	<0.4	7.5	229	1	1.95	27800	30	45	226	23500	**307**	12000	420	4.4	**1540**	<0.3	87	46	493
	5-10 cm	17100	<0.4	8	223	1	2.07	29800	28	46	241	24700	**341**	12700	436	4	**1530**	<0.3	89	63	489
	10-20 cm*	15600	2.5	13.0	318	1	2.38	37300	31	50	326	25275	**446**	14775	463	3.7	**2318**	1.25	111	54	637
2292413 (Back yard)	0-5 cm	21600	<0.4	11.0	194	1	1.07	24200	35	49	210	24100	173	10200	463	4.6	**1290**	<0.3	63	41	354
	5-10 cm	25000	<0.4	11	223	1	1.18	18700	36	43	208	26900	190	8080	485	4	**1510**	<0.3	62	47	386
	10-20 cm*	23525	1.9	15.7	202	1	1.11	18025	35	43	217	27550	174	7343	540	3.0	**1863**	1.10	58	43	388
2292414 (Back yard)	0-5 cm	20300	1.0	21.8	285	1.2	2.00	21200	35	**83**	**427**	31100	**254**	8500	468	4.2	**3540**	1.40	92	42	636
	5-10 cm*	20700	0.8	20.3	282	1.1	1.92	17900	34	**82**	**426**	29900	**257**	7170	469	4.2	**3280**	1.40	77	42	638
	10-20 cm*	20150	2.5	22.3	288	1	1.78	16400	36	**95**	**438**	31750	**280**	6850	525	3.1	**3893**	2.03	70	43	**813**
2292415 (Front yard)	0-5 cm*	8523	1.2	3.9	82	0.4	0.26	42950	12	7	34	14125	14	6463	595	2.3	48	0.23	81	20	66
	5-10 cm*	11975	1.3	4.3	90	0.5	0.43	32325	18	16	57	16625	31	6193	562	2.2	272	0.38	69	26	97
	10-20 cm	20900	<0.4	5.3	134	1	0.93	21300	30	42	143	22800	89	6650	492	3.9	**1000**	<0.3	64	42	161
2292416 (Back yard)	0-5 cm	30300	<0.4	10.6	205	**1.5**	1.28	12900	42	**69**	265	29500	182	6520	441	3.9	**2210**	0.70	71	56	274
	5-10 cm	32500	<0.4	11.1	224	**1.7**	1.03	13700	40	**58**	251	33100	183	7740	464	3.7	**2430**	<0.3	68	59	259
	10-20 cm	29200	<0.4	19.9	255	**1.6**	1.35	18100	41	**61**	**345**	37600	**260**	7660	471	4.1	**3930**	0.80	87	54	404
2292417 (Front yard)	0-5 cm	19800	<0.4	7.6	133	1	0.85	25600	29	**67**	246	23500	169	14600	471	4.2	**2120**	<0.3	143	41	246
2292418 (Back yard)	0-5 cm	19400	1.3	17.5	254	**1.3**	1.96	24000	40	**93**	**396**	32100	**308**	9760	504	4.7	**4030**	2.20	95	42	699
	5-10 cm	22400	1.7	18.7	330	**1.6**	1.77	30800	49	**67**	**328**	32600	**334**	12300	492	5.5	**3470**	1.20	140	49	825
	10-20 cm	28200	<0.4	13.0	232	**1.5**	1.12	24600	41	**54**	254	33800	**204**	14100	496	4.5	**2510**	<0.3	90	53	341

Table 2: Results of Chemical Analysis (µg/g) of 2000 Residential Soil Samples, Port Colborne

Al = aluminum, Sb = antimony, As = arsenic, Ba = barium, Be = beryllium, Cd = cadmium, Ca = calcium, Cr = chromium, Co = cobalt, Cu = copper, Fe = iron, Pb = lead, Mg = magnesium, Mn = manganese, Mo = molybdenum, Ni = nickel, Se = selenium, Sr = strontium, V = vanadium, Zn = zinc. Data in bold and underlined exceed MOE Table A effects-based guideline. na - data not available

Site / Location	Soil Depth	Al	Sb	As	Ba	Be	Cd	Ca	Cr	Co	Cu	Fe	Pb	Mg	Mn	Mo	Ni	Se	Sr	V	Zn
2292419 (Front yard)	0-5 cm	15200	<0.4	12.0	167	0.8	1.64	15400	32	134	388	23500	324	6920	492	3.7	3750	2.80	47	37	400
	5-10 cm	16600	0.5	15.6	145	0.9	1.80	13000	31	122	421	25800	256	5940	469	3.7	4320	4.40	43	38	360
	10-20 cm	19500	<0.4	15.0	135	1	1.17	11700	30	74	329	30200	129	6050	588	3.7	4190	1.60	40	40	266
2292420 (Back yard)	0-5 cm	15000	0.5	12.5	148	0.8	2.17	16100	30	103	364	24400	176	6950	480	3.7	2990	2.70	102	32	289
	5-10 cm	15500	<0.4	14.6	132	0.9	2.25	14200	29	93	367	25300	144	6750	531	3.6	3210	2.20	84	32	269
	10-20 cm	16400	<0.4	15.0	157	0.9	1.11	19500	26	141	459	27100	151	7750	472	3.8	3660	1.40	131	32	240
2292421 (Front yard)	0-5 cm	22500	1.1	15.0	178	1.2	1.73	11900	42	151	514	32000	292	7090	738	4.1	5080	2.90	44	46	573
	5-10 cm	26100	<0.4	10.4	161	1.4	1.27	10800	35	79	288	30300	153	7960	838	3.7	2860	0.70	37	47	407
	10-20 cm	26600	0.7	15.2	172	1.4	1.17	15100	38	71	337	33700	176	9650	680	4.4	4210	2.10	42	46	370
2292422 (Back yard)	0-5 cm	16700	<0.4	5.0	103	0.8	0.79	15800	22	29	105	18400	101	7680	344	3.4	963	<0.3	45	33	150
	5-10 cm	17900	<0.4	5.9	111	0.9	0.85	17500	23	31	115	20300	111	8440	378	3.4	1070	<0.3	47	34	157
	10-20 cm	15700	<0.4	7.8	99	0.8	0.79	22900	21	29	120	20100	127	9230	389	3.7	1360	<0.3	59	31	162
2292423 (Front yard)	0-5 cm	19800	3.0	15.1	180	1.3	1.74	19300	36	90	394	32300	201	7010	492	4.4	4400	1.90	76	40	374
	5-10 cm	22100	2.1	13.0	178	1.4	1.25	17800	30	53	268	29700	160	6470	422	3.6	2740	0.90	76	40	277
	10-20 cm	25100	5.8	23.3	276	1.8	1.55	23700	40	63	354	39900	324	7200	465	4.5	4780	<0.3	125	42	408
2292424 (Front yard)	0-5 cm	16200	0.6	12.1	169	1.1	1.28	24200	31	68	301	26300	205	9480	476	4.2	3140	0.70	83	36	377
	5-10 cm	18500	1.3	18.2	207	1.5	1.47	24100	34	77	400	29300	246	9260	474	4.2	4060	1.30	124	38	514
	10-20 cm	18600	1.6	26.7	201	1.3	1.63	25600	34	82	454	33700	223	9650	428	4.4	5580	2.20	117	38	453
2292425 (Back yard)	0-5 cm	19800	<0.4	6.0	161	1.1	1.45	20800	28	44	149	23500	155	8750	482	3.6	1200	<0.3	71	37	257
	5-10 cm	25000	<0.4	5.3	184	1.3	0.99	22400	31	38	125	27600	130	10300	519	3.6	967	<0.3	74	43	221
	10-20 cm	20900	<0.4	7.7	141	1.1	0.88	24000	26	40	163	26800	93	10300	477	3.7	1550	<0.3	61	37	185
2292426 (Front yard)	0-5 cm	20000	<0.4	4.3	123	1	0.70	20700	27	32	106	22000	99	12700	456	3.9	799	<0.3	73	41	168
	5-10 cm	20200	<0.4	4.5	119	1.1	0.62	22500	27	30	94	22000	89	13300	478	3.9	687	<0.3	66	41	152
	10-20 cm	19800	<0.4	4.3	121	1	0.86	23200	27	27	103	23300	131	13900	554	4.1	894	<0.3	57	39	173
2292427 (Front yard)	0-5 cm	19700	<0.4	5.0	119	1	0.80	13900	26	34	143	21300	96	6960	357	3.7	1120	<0.3	55	40	196
	5-10 cm	24500	<0.4	6.4	143	1.2	0.86	11900	32	40	163	24700	110	6820	438	3.6	1320	<0.3	53	46	226
	10-20 cm	24500	<0.4	6.5	152	1.2	0.82	11900	31	42	185	25400	160	6890	422	3.7	1680	<0.3	50	44	219

Part A - Soil Investigation: Tables

Table 2: Results of Chemical Analysis (µg/g) of 2000 Residential Soil Samples, Port Colborne

Al = aluminum, Sb = antimony, As = arsenic, Ba = barium, Be = beryllium, Cd = cadmium, Ca = calcium, Cr = chromium, Co = cobalt, Cu = copper, Fe = iron, Pb = lead, Mg = magnesium, Mn = manganese, Mo = molybdenum, Ni = nickel, Se = selenium, Sr = strontium, V = vanadium, Zn = zinc. Data in bold and underlined exceed MOE Table A effects-based guideline. na - data not available

Site / Location	Soil Depth	Al	Sb	As	Ba	Be	Cd	Ca	Cr	Co	Cu	Fe	Pb	Mg	Mn	Mo	Ni	Se	Sr	V	Zn
2292428 (Back yard)	0-5 cm	22100	<0.4	7.9	201	1.2	2.65	14400	32	39	206	25400	154	6480	364	4.0	**1690**	<0.3	83	43	312
	5-10 cm	29100	<0.4	9.6	266	**1.6**	1.60	15800	41	49	256	30100	**206**	7360	431	4.1	**2000**	<0.3	101	51	400
	10-20 cm	29500	<0.4	14.1	330	**1.7**	1.85	13600	41	**56**	276	33000	189	7180	460	4.2	**2830**	<0.3	123	52	401
2292429 (Front yard)	0-5 cm	10300	<0.4	5.3	68	0.6	0.49	11800	17	30	124	16300	95	5630	402	3.4	**1080**	<0.3	34	29	143
	5-10 cm	10600	<0.4	3.4	61	0.6	0.44	9450	16	23	93	14800	73	4620	391	3.1	**719**	<0.3	30	28	105
	10-20 cm	11300	<0.4	8.6	102	0.7	0.77	13900	19	48	217	19700	134	6250	443	3.5	**2010**	0.50	39	30	225
2292430 (Back yard)	0-5 cm	16300	0.6	17.1	242	1.2	2.09	19300	34	**82**	**366**	30000	**623**	7230	500	4.5	**3360**	2.80	87	37	625
	5-10 cm	20600	1.5	**26.9**	352	**1.4**	2.56	21000	39	**91**	**516**	36700	**797**	7470	555	4.7	**5070**	2.80	103	43	742
	10-20 cm	23700	5.7	**28.5**	565	**1.8**	2.27	28900	49	**74**	**522**	37500	**1460**	8130	576	4.7	**4960**	1.40	209	47	**916**
2292431 (Front yard)	0-5 cm	16400	<0.4	16.0	182	1	1.36	20300	31	**79**	**414**	30100	**401**	7940	492	4.1	**3400**	1.60	70	41	639
	5-10 cm	16200	<0.4	19.7	155	1	1.38	19800	29	**84**	**442**	31600	**325**	8180	507	4.3	**4120**	2.50	65	41	533
	10-20 cm	15600	<0.4	21.2	168	1	1.21	19200	26	**62**	**419**	31100	**250**	6540	496	3.9	**3380**	2.00	76	39	403
2292432 (Back yard)	0-5 cm	19800	<0.4	17.3	244	1.2	1.91	16500	37	**74**	**408**	31200	**386**	6450	548	4.4	**3920**	1.20	78	42	592
	5-10 cm	22100	1.4	20.0	274	**1.4**	2.16	15000	39	**84**	**465**	34400	**531**	6190	635	4.3	**4730**	1.90	82	45	632
	10-20 cm	21900	1.1	24.7	292	**1.4**	1.90	16100	36	**75**	**468**	34700	**485**	6060	618	4.1	**4890**	2.20	91	44	571
2292433 (Front yard)	0-5 cm	19400	1.9	10.6	168	1.2	1.54	28400	33	**60**	299	23800	**274**	12300	439	4.6	**2290**	0.80	114	34	494
	0-5 cm	21700	1.7	8.8	174	**1.3**	1.52	23900	38	**60**	278	24000	**257**	9710	449	4.3	**2240**	<0.3	95	37	505
	0-5 cm	23200	3.2	8.9	196	**1.3**	1.69	24300	39	**62**	288	24100	**313**	10300	466	4.4	**2180**	0.30	97	41	605
	5-10 cm	22400	2.1	12.5	198	**1.4**	1.70	28600	41	**78**	**382**	26900	**348**	12600	483	4.7	**2930**	1.30	118	38	598
	5-10 cm	23200	2.4	11.8	203	**1.4**	1.74	24400	39	**81**	**360**	27800	**347**	10800	482	4.5	**3010**	<0.3	97	41	629
	5-10 cm	26200	4.6	15.0	215	**1.5**	2.14	27300	50	**120**	**489**	29800	**518**	12800	564	4.7	**3640**	1.40	104	49	**930**
	10-20 cm	29700	3.0	16.1	248	**1.8**	1.85	27900	44	**89**	**493**	35300	**358**	12200	519	4.6	**4900**	1.00	161	45	705
	10-20 cm	25400	2.9	22.4	231	**1.6**	2.27	27000	43	**132**	**597**	40500	**383**	11900	587	4.9	**6310**	2.00	110	43	**801**
	10-20 cm	28900	4.6	19.6	285	**1.8**	2.20	25400	47	**122**	**564**	38100	**459**	11600	555	4.8	**5430**	1.30	116	49	**839**

Table 2: Results of Chemical Analysis (µg/g) of 2000 Residential Soil Samples, Port Colborne

Al = aluminum, Sb = antimony, As = arsenic, Ba = barium, Be = beryllium, Cd = cadmium, Ca = calcium, Cr = chromium, Co = cobalt, Cu = copper, Fe = iron, Pb = lead, Mg = magnesium, Mn = manganese, Mo = molybdenum, Ni = nickel, Se = selenium, Sr = strontium, V = vanadium, Zn = zinc. Data in bold and underlined exceed MOE Table A effects-based guideline. na - data not available

Site / Location	Soil Depth	Al	Sb	As	Ba	Be	Cd	Ca	Cr	Co	Cu	Fe	Pb	Mg	Mn	Mo	Ni	Se	Sr	V	Zn
2292434 (Back yard)	0-5 cm	21900	0.8	11.0	164	**1.3**	1.05	10300	29	**65**	278	29400	**209**	4030	482	3.8	**2870**	1.00	61	38	358
	0-5 cm	21300	0.7	10.4	169	1.2	1.08	10300	27	**63**	261	28500	194	3910	458	3.7	**2700**	0.70	62	37	363
	0-5 cm	23300	0.5	10.9	178	**1.3**	1.12	10400	30	**70**	283	30300	**209**	4370	466	3.8	**3080**	0.70	62	43	365
	5-10 cm	23700	1.4	12.7	192	**1.5**	1.09	11100	32	**66**	**326**	34700	**219**	4160	547	3.9	**3530**	1.10	68	39	380
	5-10 cm	23300	0.9	12.6	192	**1.4**	1.19	11300	30	**72**	**312**	34800	**253**	4270	577	3.7	**3480**	0.60	69	38	461
	5-10 cm	26300	0.7	15.8	200	**1.5**	1.28	11700	33	**71**	**329**	34300	**226**	4680	542	3.9	**3510**	0.80	70	44	391
	10-20 cm	27800	0.6	11.1	258	**1.6**	0.97	15100	35	46	270	33600	**251**	5040	521	4.0	**2660**	<0.3	89	43	355
	10-20 cm	26900	0.4	8.8	194	**1.4**	0.83	10300	32	44	200	32200	172	4760	520	3.8	**2180**	<0.3	68	43	301
	10-20 cm	30600	0.6	15.6	321	**1.8**	1.26	13900	39	**59**	325	38900	**320**	5560	505	4.1	**3910**	0.30	106	50	434
2292435 (Back yard)	0-5 cm	25900	5.1	17.8	258	**1.4**	2.32	17300	38	**84**	**471**	33400	**315**	6870	549	4.2	**3590**	1.70	90	48	516
	5-10 cm	29500	1.2	22.5	238	**1.6**	1.95	17000	41	**98**	**571**	38900	**473**	7190	666	4.4	**4880**	1.20	93	52	459
	10-20 cm	31700	0.8	23.2	297	**1.8**	1.73	25000	58	**149**	**1230**	41400	**261**	7720	540	4.3	**6390**	1.60	149	48	503
2292436 (Back yard)	0-5 cm	13400	<0.4	7.1	125	0.7	1.50	16000	22	29	103	20200	**228**	7270	543	3.4	**809**	<0.3	62	27	213
2292437 (Front yard)	0-5 cm*	14100	1.6	7.1	87	0.7	0.77	15750	23	30	134	20800	84	7185	556	2.3	**984**	0.60	42	34	140
	5-10 cm*	14700	1.6	8.9	91	0.7	0.82	15300	24	33	147	21925	93	7015	621	2.2	**1085**	0.58	42	34	144
	10-20 cm*	14125	1.5	6.8	79	0.7	0.63	16250	21	21	96	21350	57	6175	625	2.1	**690**	0.48	42	32	102
2292438 (Back yard)	0-5 cm*	14775	1.5	8.8	90	0.8	0.82	19150	22	28	124	19450	123	6338	512	2.3	**1230**	0.73	52	31	204
	5-10 cm*	14900	1.9	10.9	121	0.8	0.83	18750	23	29	130	20500	127	6560	519	2.3	**1313**	0.73	52	33	214
	10-20 cm*	15425	1.9	12.2	135	0.9	0.76	20925	23	30	148	21950	142	7028	509	2.9	**1565**	0.68	61	34	219
2292439 (Front yard)	0-5 cm	21200	<0.4	10	135	1	0.97	13400	29	**56**	216	25400	106	8380	471	5	**2190**	2	42	44	216
	5-10 cm	24300	<0.4	10.0	150	**1.4**	0.89	15900	31	48	187	27700	95	9830	531	5.1	**1900**	<0.3	42	47	192
	10-20 cm*	25900	0.4	10.4	171	**1.3**	0.76	28350	34	39	184	29825	79	11800	601	2.8	**1683**	0.73	65	48	206
2292440 (Back yard)	0-5 cm	16300	<0.4	17.1	185	1.2	1.41	15600	25	40	202	20900	**202**	6300	401	5.0	**1930**	0.90	75	35	322
	5-10 cm*	15650	0.5	18.8	191	1	1.26	18075	25	40	219	20200	**223**	6223	403	2.5	**2098**	1.04	82	34	338
	10-20 cm*	18700	0.7	**27.6**	261	**1.3**	1.53	19625	30	48	233	23775	**272**	6490	459	2.6	**2853**	1.75	118	39	408

Table 2: Results of Chemical Analysis (µg/g) of 2000 Residential Soil Samples, Port Colborne

Al = aluminum, Sb = antimony, As = arsenic, Ba = barium, Be = beryllium, Cd = cadmium, Ca = calcium, Cr = chromium, Co = cobalt, Cu = copper, Fe = iron, Pb = lead, Mg = magnesium, Mn = manganese, Mo = molybdenum, Ni = nickel, Se = selenium, Sr = strontium, V = vanadium, Zn = zinc. Data in bold and underlined exceed MOE Table A effects-based guideline. na - data not available

Site / Location	Soil Depth	Al	Sb	As	Ba	Be	Cd	Ca	Cr	Co	Cu	Fe	Pb	Mg	Mn	Mo	Ni	Se	Sr	V	Zn
2292441 (Front yard)	0-5 cm	21900	<0.4	15.2	171	**1.4**	1.54	23600	33	**71**	**322**	23900	144	10300	452	5.8	**3050**	1.10	70	48	282
	0-5 cm	21200	<0.4	16.5	174	**1.4**	1.65	24400	35	**74**	**343**	24200	151	10600	430	5.7	**3240**	2.20	73	47	286
	0-5 cm	22100	<0.4	19.2	186	**1.4**	1.65	24000	33	**80**	**385**	24800	151	10200	424	5.6	**3950**	0.90	71	47	304
	5-10 cm	22800	<0.4	20	182	1	1.73	23400	33	**84**	**426**	27200	157	10300	467	6	**4480**	3	74	49	329
	5-10 cm	22300	<0.4	21.0	180	**1.4**	1.77	23800	34	**87**	**427**	28000	**201**	10100	485	5.6	**4580**	2.90	70	48	318
	5-10 cm	21100	<0.4	24	172	1	2.11	24300	35	**110**	**507**	29600	187	10800	472	6	**6330**	4	69	46	356
	10-20 cm	22700	1	24	182	1	0.92	26200	33	**78**	**456**	28400	146	9867	472	2	**5077**	3	77	47	314
	10-20 cm*	19733	0.6	24.4	179	1.1	1.05	25433	30	**82**	**462**	27567	170	9583	453	2.5	**5440**	3.00	71	43	346
	10-20 cm*	21925	0.5	22.8	176	1.2	1.06	24025	32	**82**	**460**	28650	148	9103	441	2.8	**5328**	1.70	72	45	302
2292442 (Back yard)	0-5 cm*	14900	0.5	13.6	122	0.9	1.14	14933	23	36	192	17500	118	6197	289	2.2	**1813**	1.77	61	33	264
	0-5 cm	13700	<0.4	13.0	107	0.9	1.12	13900	20	36	177	16400	100	5790	273	4.7	**1680**	1.10	52	31	230
	5-10 cm	14700	<0.4	12.2	122	1	1.21	14800	22	36	171	17900	102	6150	282	4.8	**1670**	0.90	59	34	231
	5-10 cm*	16200	0.5	16.0	134	1	1.14	15933	24	38	214	18333	136	6500	294	2.1	**2017**	1.63	68	35	271
	5-10 cm*	13775	1	15	118	1	0.98	16150	21	38	207	16825	117	6308	271	2	**2045**	1	58	31	263
	5-10 cm	15200	<0.4	14	127	1	1.32	14700	23	45	223	18800	114	6450	301	5	**2300**	2	61	35	245
	10-20 cm	13400	1	17	143	1	1.11	20567	22	32	205	17633	124	7067	281	2	**1990**	1	88	29	291
	10-20 cm*	10400	0.4	16.4	102	0.7	0.84	18700	18	36	237	15625	91	6108	238	2.3	**2145**	1.14	65	25	258
	10-20 cm*	13800	0.5	21.6	157	0.9	1.34	19950	23	37	233	18075	125	7093	278	2.6	**2198**	1.19	71	29	312
2292443 (Front yard)	0-5 cm	21800	<0.4	16.0	153	**1.3**	1.68	13700	34	**83**	356	27200	170	8090	593	5.6	**3670**	1.10	42	49	522
	5-10 cm	20300	<0.4	**29.7**	159	**1.3**	2.71	15300	38	**164**	**709**	37700	**218**	8930	712	5.9	**8900**	5.20	40	49	789
	10-20 cm*	19000	1	21	145	1	0.97	18600	29	**72**	**493**	27367	158	8257	551	2	**5387**	2	43	42	414
2292444 (Back yard)	0-5 cm	19700	<0.4	9.2	135	1.2	0.95	13500	29	32	148	22300	98	7970	483	4.9	**1240**	1.50	49	43	255
	5-10 cm	19800	<0.4	12.1	142	1.2	1.00	16300	29	35	162	23100	97	9250	490	5.1	**1400**	<0.3	51	42	255
	10-20 cm*	14033	0.7	17.6	159	0.9	0.78	20133	26	39	240	21933	**240**	7203	341	2.6	**2287**	0.95	76	36	329
2292445 (Front yard)	0-5 cm	28000	1.2	16.0	245	**1.7**	1.84	19700	154	**56**	**314**	27000	179	9040	447	5.5	**2910**	1.30	93	54	626
	5-10 cm	28600	1.7	18.6	270	**1.8**	2.09	19900	245	**63**	**359**	28300	187	9410	449	5.5	**3390**	0.90	90	55	**827**
	10-20 cm*	26475	1.1	19.1	242	**1.5**	1.42	20525	183	**52**	**323**	27100	167	8980	440	2.8	**3170**	1.59	89	50	673
2292446 (Back yard)	0-5 cm*	15250	0.4	8.2	136	1.2	0.88	17975	29	24	134	19875	120	8393	526	2.8	**1085**	0.63	121	34	339
	5-10 cm*	15300	0.4	7.8	127	**1.3**	0.87	19650	28	24	120	19925	110	8833	529	2.7	**1038**	0.58	135	34	319
	10-20 cm*	19800	1	16	243	3	1.14	20900	35	31	195	22067	**224**	8070	462	4	**1527**	1	273	45	451
2292447 (Front yard)	0-5 cm	20400	9.4	7.6	216	1.1	1.20	20000	32	43	179	25200	**524**	9330	539	4.5	**1620**	<0.3	63	41	311

Part A - Soil Investigation: Tables

Table 2: Results of Chemical Analysis (µg/g) of 2000 Residential Soil Samples, Port Colborne

Al = aluminum, Sb = antimony, As = arsenic, Ba = barium, Be = beryllium, Cd = cadmium, Ca = calcium, Cr = chromium, Co = cobalt, Cu = copper, Fe = iron, Pb = lead, Mg = magnesium, Mn = manganese, Mo = molybdenum, Ni = nickel, Se = selenium, Sr = strontium, V = vanadium, Zn = zinc.   Data in bold and underlined exceed MOE Table A effects-based guideline.   na - data not available

Site / Location	Soil Depth	Al	Sb	As	Ba	Be	Cd	Ca	Cr	Co	Cu	Fe	Pb	Mg	Mn	Mo	Ni	Se	Sr	V	Zn
	5-10 cm	21200	8.8	7.5	176	1.1	1.05	18300	31	40	157	25800	**360**	8730	460	4.2	**1430**	<0.3	56	41	246
	10-20 cm	17800	8.0	7.9	153	1	1.44	28000	27	35	153	26200	**278**	11100	479	4.4	**1570**	<0.3	65	35	222
2292448 (Back yard)	0-5 cm	26200	**23.6**	9.7	409	**1.4**	1.60	27600	40	43	225	32700	**1340**	9600	505	4.5	**2190**	<0.3	102	48	583
	5-10 cm	26300	**19.1**	8.5	329	**1.4**	1.45	23100	38	42	210	31900	**1010**	8650	463	4.5	**2000**	<0.3	96	48	490
	10-20 cm	25500	**18.7**	12.0	366	**1.4**	1.83	25600	41	49	**312**	35600	**1130**	9520	512	4.6	**2650**	<0.3	101	47	645
2292449 (Front yard)	0-5 cm*	16175	2.6	15.0	147	0.9	1.38	25775	30	43	224	30500	177	11000	532	3.5	**1975**	1.39	86	36	335
	5-10 cm*	19075	2.7	14.6	154	1	1.30	28500	31	38	188	32025	155	11900	550	3.4	**1790**	0.90	77	40	297
	10-20 cm*	19075	2.6	13.4	139	1	1.04	24850	29	32	159	31375	126	10250	524	3.4	**1610**	0.83	64	39	260
2292450 (Back yard)	0-5 cm*	9820	2.2	15.6	160	0.6	1.39	22350	23	28	175	23625	**227**	6805	417	2.6	**1470**	1.23	115	28	353
	5-10 cm	9610	6.9	16.0	138	0.7	1.35	21500	24	28	151	24100	**207**	6600	442	5.0	**1310**	0.55	105	29	316
	10-20 cm*	9593	2.4	17.8	149	0.6	1.26	23275	21	28	180	24400	**206**	6700	413	2.6	**1548**	0.87	120	26	319
2292451 (Front yard)	0-5 cm*	13750	1.7	7.1	87	0.7	1.00	14400	22	21	106	20150	95	5813	360	2.4	**909**	0.60	58	30	172
	5-10 cm*	13725	1.7	7.6	84	0.7	0.98	14525	22	22	112	20125	105	5760	351	2.3	**895**	0.60	58	30	166
	10-20 cm*	12375	1.9	13.3	104	0.6	1.43	14450	23	38	211	27350	162	4838	430	2.8	**1933**	1.13	64	27	295
2292452 (Front yard)	0-5 cm	19900	5.6	7.8	168	1	0.98	17400	32	23	100	22300	167	9180	428	5.0	**667**	<0.3	70	42	255
	5-10 cm*	19975	1.7	7.9	152	0.9	1.00	11750	28	21	93	22950	158	6735	427	2.4	**585**	0.58	58	41	235
	10-20 cm*	20000	1.8	6.9	152	0.9	0.96	10148	28	20	88	23725	162	6135	409	2.2	**607**	0.50	47	40	229
2292453 (Back yard)	0-5 cm*	12900	2.0	10.8	186	0.6	1.19	10875	23	18	130	18450	**309**	3833	445	2.6	**606**	0.58	76	34	343
	5-10 cm*	12775	1.4	9.1	182	0.6	1.10	10528	21	18	115	18850	**266**	3640	482	2.1	**601**	0.63	74	33	302
	10-20 cm*	12400	1.6	9.6	168	0.6	0.93	10650	20	18	160	18600	**242**	3463	433	2.6	**650**	0.55	72	32	287
2292454 (Front yard)	0-5 cm*	12725	1.4	10.8	111	0.6	1.01	16825	21	28	136	19575	166	7175	402	2.7	**1078**	0.73	66	29	249
	5-10 cm*	13750	1.3	9.0	90	0.5	0.86	11975	19	27	118	18875	129	5863	361	2.5	**982**	0.68	47	29	200
	10-20 cm*	14200	2.0	18.0	152	0.8	1.21	23225	25	45	227	30425	199	10825	571	3.6	**2350**	1.89	88	30	324
2292455 (Back yard)	0-5 cm*	12225	1.0	5.8	75	0.4	0.72	6853	18	17	73	14625	**208**	2713	236	2.3	**438**	0.60	41	26	237
	5-10 cm	13600	3.0	3.4	64	0.5	0.64	5040	17	16	57	16300	103	2480	239	2.9	**380**	<0.3	33	28	158
	10-20 cm	14200	2.9	4.9	77	0.6	0.66	5660	18	17	83	16700	106	2610	221	2.9	**558**	<0.3	45	27	148

Table 2: Results of Chemical Analysis (μg/g) of 2000 Residential Soil Samples, Port Colborne

Al = aluminum, Sb = antimony, As = arsenic, Ba = barium, Be = beryllium, Cd = cadmium, Ca = calcium, Cr = chromium, Co = cobalt, Cu = copper, Fe = iron, Pb = lead, Mg = magnesium, Mn = manganese, Mo = molybdenum, Ni = nickel, Se = selenium, Sr = strontium, V = vanadium, Zn = zinc. Data in bold and underlined exceed MOE Table A effects-based guideline. na - data not available

Site / Location	Soil Depth	Al	Sb	As	Ba	Be	Cd	Ca	Cr	Co	Cu	Fe	Pb	Mg	Mn	Mo	Ni	Se	Sr	V	Zn
2292456 (Front yard)	0-5 cm	11200	4.9	19.1	215	0.9	1.96	32700	33	49	293	28500	**462**	13100	513	5.1	**2280**	2.53	143	30	520
	5-10 cm	11700	5.0	**25.4**	291	1.1	2.27	32600	37	**56**	**371**	37200	**675**	10500	595	5.3	**3260**	2.91	183	31	629
	10-20 cm	12400	6.6	**31.7**	311	1.2	2.53	30000	40	**68**	**435**	48100	**662**	9930	672	5.4	**4830**	4.37	195	32	692
2292457 (Back yard)	0-5 cm	13100	3.1	5.0	66	0.5	0.81	8580	18	17	70	16300	104	3740	285	3.3	**487**	<0.3	46	29	176
	5-10 cm	13000	2.6	4.0	56	0.5	0.64	7130	17	15	59	15700	84	3540	255	3.2	**425**	<0.3	35	28	146
	10-20 cm	12300	3.1	5.7	78	0.6	0.75	8450	19	17	87	17600	175	3520	259	3.3	**665**	<0.3	48	26	182
2292458 (Front yard)	0-5 cm	22600	<0.4	5.5	134	1.1	0.94	11500	33	30	120	19900	93	7030	448	4.0	**709**	<0.3	48	49	161
	5-10 cm	22300	<0.4	4.6	127	1.1	0.80	11200	30	26	101	19400	76	6970	400	3.6	**601**	<0.3	46	47	138
	10-20 cm	22400	<0.4	5.7	124	1.1	0.74	12200	30	26	102	21500	71	7520	466	4.0	**696**	<0.3	49	46	142
2292459 (Back yard)	0-5 cm	15000	<0.4	6.2	90	0.7	0.98	6050	21	26	118	17900	101	3150	289	3.2	**864**	<0.3	27	33	212
	5-10 cm	14400	<0.4	4.2	71	0.7	0.67	4580	18	17	67	14500	61	2650	229	2.6	**442**	<0.3	22	30	132
	10-20 cm	17100	<0.4	12.7	137	1	1.57	11600	27	35	180	24200	149	4960	383	3.8	**1670**	<0.3	48	35	319
2292460 (Front yard)	0-5 cm	16100	<0.4	4.9	89	0.9	0.57	9700	23	18	91	21100	61	5600	520	3.5	**579**	<0.3	35	34	123
	5-10 cm	16500	<0.4	6.2	92	0.9	0.59	10200	24	20	98	22400	71	5790	546	3.6	**655**	<0.3	38	34	129
	10-20 cm	19000	<0.4	9.0	111	1.1	0.89	14000	30	31	178	27200	113	7280	511	4.0	**1310**	<0.3	52	39	192
2292461 (Back yard)	0-5 cm	17000	<0.4	8.5	105	1	0.91	13600	26	26	114	20700	74	6240	393	3.9	**1070**	<0.3	71	37	164
	5-10 cm	18200	<0.4	8.6	110	1.1	0.91	13200	26	26	113	21400	72	6130	377	3.7	**1020**	<0.3	75	38	160
	10-20 cm	21700	<0.4	8.0	122	1.2	0.91	11400	29	25	110	23800	76	6040	425	3.8	**1100**	<0.3	72	41	165
2292462 (Front yard)	0-5 cm	15700	<0.4	7.6	97	0.9	0.78	12100	25	22	95	21100	91	6740	435	4.2	**749**	<0.3	39	35	187
	5-10 cm	17300	<0.4	8.0	101	0.9	0.74	12500	25	23	102	22700	87	7590	459	3.9	**780**	<0.3	42	38	177
	10-20 cm	18500	<0.4	10.8	117	1	0.99	15400	28	30	144	26800	119	9160	469	4.2	**1250**	<0.3	53	39	234
2292463 (Back yard)	0-5 cm	14400	<0.4	9.0	92	0.8	1.01	12500	21	22	109	17900	77	4500	407	3.7	**903**	<0.3	59	33	174
	5-10 cm	13900	<0.4	9.5	91	0.8	0.93	11000	21	23	109	18000	71	4610	374	3.4	**987**	<0.3	57	33	161
	10-20 cm	15200	<0.4	8.1	91	0.8	0.78	9810	21	19	91	17900	53	4480	361	3.3	**790**	<0.3	54	33	143

Table 2: Results of Chemical Analysis (µg/g) of 2000 Residential Soil Samples, Port Colborne

Al = aluminum, Sb = antimony, As = arsenic, Ba = barium, Be = beryllium, Cd = cadmium, Ca = calcium, Cr = chromium, Co = cobalt, Cu = copper, Fe = iron, Pb = lead, Mg = magnesium, Mn = manganese, Mo = molybdenum, Ni = nickel, Se = selenium, Sr = strontium, V = vanadium, Zn = zinc. Data in bold and underlined exceed MOE Table A effects-based guideline. na - data not available

Site / Location	Soil Depth	Al	Sb	As	Ba	Be	Cd	Ca	Cr	Co	Cu	Fe	Pb	Mg	Mn	Mo	Ni	Se	Sr	V	Zn
2292464 (Front yard)	0-5 cm	19600	<0.4	8.3	125	1.1	0.99	15300	30	27	111	24200	91	8710	436	4.3	884	<0.3	69	42	216
	0-5 cm	24800	<0.4	8.5	141	1.3	1.01	12500	36	28	113	26900	89	7350	468	4.4	937	<0.3	60	53	212
	0-5 cm	24400	<0.4	8.9	144	1.3	1.05	12800	37	29	116	26100	104	7660	426	4.2	956	<0.3	61	53	254
	5-10 cm	22300	<0.4	8.6	133	1.2	0.99	13700	32	28	116	27100	88	8180	453	4.3	972	<0.3	65	45	210
	5-10 cm	28400	<0.4	7.7	154	1.4	0.97	11100	38	29	112	29900	86	7540	487	4.1	934	<0.3	61	58	204
	5-10 cm	27800	<0.4	7.1	152	1.3	0.97	10900	37	27	103	28500	86	7440	486	4.0	812	<0.3	61	56	219
	10-20 cm	21700	<0.4	11.0	138	1.2	1.01	13400	32	31	136	28500	96	7520	443	4.0	1240	<0.3	60	44	226
	10-20 cm	27900	<0.4	8.9	154	1.4	1.01	12000	38	30	121	30300	88	7780	468	4.2	1070	<0.3	60	57	207
	10-20 cm	26900	<0.4	10.3	155	1.4	1.03	11300	37	30	124	30000	97	7350	451	4.0	1150	<0.3	62	55	226
2292465 (Back yard)	0-5 cm	20100	<0.4	7.1	121	1.1	1.00	12800	29	21	93	20200	80	6060	356	3.9	618	<0.3	74	44	214
	0-5 cm	18900	<0.4	7.6	116	1	0.95	13200	28	20	86	19000	71	6200	327	3.8	577	<0.3	72	42	191
	5-10 cm	19700	<0.4	6.8	119	1	0.93	12800	29	21	88	19400	94	6090	338	3.8	604	<0.3	77	43	193
	5-10 cm	19400	<0.4	6.6	114	1	0.93	13400	27	21	90	19200	74	6140	360	3.8	638	0.30	71	42	180
	5-10 cm	20600	<0.4	7.3	125	1.1	0.96	14500	30	22	93	20600	76	6660	359	3.8	636	<0.3	81	45	201
	5-10 cm	20400	<0.4	7.3	123	1.1	0.98	13400	29	22	92	19800	76	6340	349	3.9	689	<0.3	84	45	190
	10-20 cm	19600	<0.4	7.8	115	1	0.91	14500	28	20	84	19800	68	6190	344	3.8	622	<0.3	79	43	174
	10-20 cm	20100	<0.4	8.1	125	1.1	0.94	15300	28	21	93	19600	73	6600	339	3.9	594	<0.3	85	42	193
	10-20 cm	21100	<0.4	8.2	125	1.1	0.96	14100	29	21	91	20500	74	6240	347	3.8	700	<0.3	90	44	189
2292466 (Front yard)	0-5 cm	16100	<0.4	5.4	88	0.8	0.60	12900	23	20	71	21600	64	6350	447	3.8	545	<0.3	38	37	140
	5-10 cm	17900	<0.4	5.7	97	0.9	0.64	9910	25	22	75	22800	65	5810	490	3.8	562	<0.3	30	41	138
	10-20 cm	18900	<0.4	5.0	95	0.9	0.55	7900	25	19	59	22600	52	5550	490	3.6	444	<0.3	25	40	116
2292467 (Back yard)	0-5 cm	22700	<0.4	4.7	155	1.2	0.52	21100	32	18	55	26200	161	9330	590	4.4	274	<0.3	77	48	158
	5-10 cm	24100	<0.4	6.0	174	1.3	0.59	23900	34	22	67	28600	143	10300	597	4.5	413	<0.3	91	50	188
	10-20 cm	18600	<0.4	8.3	172	1.1	0.83	20900	30	26	108	26700	197	9080	524	4.7	804	<0.3	93	41	250
2292468 (Front yard)	0-5 cm	16500	<0.4	9.4	118	1	0.91	17000	25	29	147	24800	119	7170	597	4.4	1160	<0.3	52	38	218
	5-10 cm	17400	<0.4	10.5	130	1.1	1.15	17400	28	33	168	26200	133	7410	646	4.6	1250	<0.3	57	41	246
	10-20 cm	14200	<0.4	7.3	85	0.8	0.79	10900	22	27	120	24900	90	5190	601	3.9	1030	<0.3	36	33	181

Table 2: Results of Chemical Analysis (µg/g) of 2000 Residential Soil Samples, Port Colborne

Al = aluminum, Sb = antimony, As = arsenic, Ba = barium, Be = beryllium, Cd = cadmium, Ca = calcium, Cr = chromium, Co = cobalt, Cu = copper, Fe = iron, Pb = lead, Mg = magnesium, Mn = manganese, Mo = molybdenum, Ni = nickel, Se = selenium, Sr = strontium, V = vanadium, Zn = zinc. Data in bold and underlined exceed MOE Table A effects-based guideline. na - data not available

Site / Location	Soil Depth	Al	Sb	As	Ba	Be	Cd	Ca	Cr	Co	Cu	Fe	Pb	Mg	Mn	Mo	Ni	Se	Sr	V	Zn
2292469 (Back yard)	0-5 cm	14100	<0.4	14.6	175	0.8	1.40	24100	31	24	158	21000	**271**	6940	487	4.7	**974**	<0.3	97	32	332
	5-10 cm	15200	<0.4	18.0	220	1.1	1.71	24400	32	30	217	26100	**389**	6930	567	4.4	**1360**	<0.3	108	35	396
	10-20 cm	14600	<0.4	14.0	167	0.9	1.13	14800	26	24	125	24500	**296**	5390	583	4.1	**979**	<0.3	73	32	270
2292470 (Front yard)	0-5 cm	13100	<0.4	7.7	133	0.8	1.19	24700	24	27	148	17700	**234**	9220	395	4.3	**775**	<0.3	77	32	243
	5-10 cm	11100	<0.4	6.4	118	0.7	0.89	20500	21	23	122	15600	192	7870	333	4.1	**720**	<0.3	62	30	203
	5-10 cm	12300	<0.4	7.9	121	0.8	1.27	23400	24	27	147	18600	**203**	9730	394	4.4	**840**	<0.3	74	32	235
	5-10 cm	13700	<0.4	7.6	134	0.8	1.04	22500	25	28	156	18000	**232**	7850	385	4.2	**805**	<0.3	69	34	236
	5-10 cm	14900	<0.4	8.8	150	0.9	1.28	23600	27	32	177	20000	**271**	8250	425	4.3	**996**	<0.3	74	36	267
	5-10 cm	14100	<0.4	9.3	138	0.9	1.18	23800	26	33	185	21900	**238**	8930	434	4.7	**1090**	<0.3	72	35	265
	10-20 cm	14600	<0.4	7.6	135	0.9	1.03	17000	27	33	161	20100	**228**	6660	431	3.8	**1040**	<0.3	52	34	237
	10-20 cm	16200	<0.4	8.9	167	1	1.24	17900	29	34	203	21400	**269**	6870	468	4.0	**1120**	<0.3	60	38	273
	10-20 cm	16400	<0.4	10.7	160	1	1.38	20600	31	38	208	23800	**258**	8080	504	4.6	**1300**	<0.3	66	39	294
2292471 (Back yard)	0-5 cm	15100	<0.4	6.5	140	1	1.17	18500	25	25	113	18600	**207**	6960	386	4.1	**712**	<0.3	64	38	221
	0-5 cm	15200	<0.4	7.4	132	0.9	1.23	17700	24	26	117	19000	188	6840	395	4.0	**725**	<0.3	62	37	202
	0-5 cm	16700	<0.4	8.3	143	1	1.57	19500	30	29	123	20600	205	7480	412	4.3	**734**	<0.3	68	39	227
	5-10 cm	16200	<0.4	7.3	137	1	1.13	18700	25	27	114	19600	188	6960	386	4.0	**726**	<0.3	65	38	202
	5-10 cm	16200	<0.4	6.9	132	1	1.19	17500	24	27	120	20300	176	6650	390	3.9	**768**	<0.3	61	39	186
	5-10 cm	17600	<0.4	8.2	143	1	1.45	19000	28	29	126	21600	184	7350	407	4.2	**825**	<0.3	69	42	212
	10-20 cm	19600	<0.4	7.5	143	1.1	1.22	15700	28	27	114	23400	191	6770	461	3.9	**814**	<0.3	58	43	187
	10-20 cm	18100	<0.4	7.1	136	1.1	1.14	16900	26	29	125	22500	161	6770	459	3.7	**908**	<0.3	58	41	177
	10-20 cm	21200	<0.4	8.9	164	1.2	1.50	20500	31	34	146	24900	191	8030	478	4.3	**966**	<0.3	72	47	223
2292472 (Back yard)	0-5 cm	16500	<0.4	3.7	97	0.8	0.49	15300	31	15	49	20000	61	6380	501	3.8	**291**	<0.3	40	31	116
	5-10 cm	19500	<0.4	3.1	98	0.9	0.38	23100	23	14	34	22700	50	8040	564	3.5	166	<0.3	46	35	86
	10-20 cm	21300	<0.4	5.5	127	1	0.54	28600	27	21	71	27000	73	8600	651	3.8	**446**	<0.3	66	39	155
2292473 (Front yard)	0-5 cm	19500	<0.4	5.2	130	0.9	0.92	11700	26	29	135	22600	140	6270	584	3.6	**906**	<0.3	45	37	282
	5-10 cm	22600	<0.4	4.5	128	1	0.82	9220	27	28	139	24500	120	5830	662	3.5	**765**	<0.3	41	40	208
	10-20 cm	19800	<0.4	7.0	127	0.9	0.99	12600	25	32	193	23300	140	5880	537	3.7	**1050**	<0.3	56	37	234

Table 2: Results of Chemical Analysis (μg/g) of 2000 Residential Soil Samples, Port Colborne

Al = aluminum, Sb = antimony, As = arsenic, Ba = barium, Be = beryllium, Cd = cadmium, Ca = calcium, Cr = chromium, Co = cobalt, Cu = copper, Fe = iron, Pb = lead, Mg = magnesium, Mn = manganese, Mo = molybdenum, Ni = nickel, Se = selenium, Sr = strontium, V = vanadium, Zn = zinc. Data in bold and underlined exceed MOE Table A effects-based guideline. na - data not available

Site / Location	Soil Depth	Al	Sb	As	Ba	Be	Cd	Ca	Cr	Co	Cu	Fe	Pb	Mg	Mn	Mo	Ni	Se	Sr	V	Zn
2292474 (Front yard)	0-5 cm	24400	<0.4	10.9	232	1.2	1.34	17400	32	44	225	30700	324	7700	450	3.7	1880	<0.3	56	42	407
	5-10 cm	24500	<0.4	15.6	264	1.3	1.50	19800	34	45	257	34600	365	8050	481	4.0	2290	<0.3	62	41	451
	10-20 cm	22100	0.4	18.2	267	1.2	1.45	26500	30	40	224	35700	413	7810	461	4.1	2440	0.53	74	38	504
2292475 (Back yard)	0-5 cm	10200	<0.4	1.6	75	0.4	0.44	9860	11	6	15	9890	33	3340	170	2.7	87	<0.3	23	20	87
	5-10 cm	10600	<0.4	2.0	174	0.5	0.42	10200	12	7	18	11200	37	3150	192	2.7	144	<0.3	24	22	82
	10-20 cm	16000	<0.4	3.6	956	0.8	0.46	20500	20	11	34	17300	86	5970	316	3.4	232	<0.3	49	30	113
2292476 (Front yard)	0-5 cm	20700	<0.4	4.9	136	0.9	0.80	23400	27	33	143	24000	183	9760	419	3.9	1020	<0.3	55	37	220
	5-10 cm	23900	<0.4	6.2	150	1.1	0.81	23300	27	32	145	25800	172	9490	417	3.8	1100	<0.3	56	40	218
	10-20 cm	19900	<0.4	6.7	130	1	0.69	27900	24	30	134	25000	138	10400	459	3.7	1100	<0.3	58	35	198
2292477 (Back yard)	0-5 cm	22300	5.7	12.7	246	1.2	1.54	13500	39	38	253	34800	847	5840	504	4.1	1910	0.30	59	40	565
	5-10 cm	21900	11.3	13.0	253	1.2	1.50	13300	37	36	223	34500	960	5890	496	4.0	1810	0.38	58	38	507
	10-20 cm	23200	9.6	16.3	372	1.3	1.76	17600	41	43	331	38700	1170	6440	502	4.3	2540	0.82	77	39	643
2292478 (Front yard)	0-5 cm	27000	<0.4	6.3	164	1.2	0.98	15000	32	38	173	25600	156	7240	371	4.0	1380	<0.3	58	45	240
	5-10 cm	29000	<0.4	6.8	173	1.2	0.99	13300	32	39	170	26800	169	7000	379	3.7	1410	<0.3	57	45	242
	10-20 cm	25900	<0.4	11.2	193	1.2	1.17	16400	34	47	259	31000	187	7200	469	4.2	2570	<0.3	57	41	310
2292479 (Back yard)	0-5 cm	17200	0.9	16.7	264	1.4	1.69	18500	29	50	295	28900	299	5280	418	4.0	2520	1.10	115	36	579
	5-10 cm	18900	0.5	21.0	378	1.9	1.84	24300	33	50	357	29000	387	5210	423	4.4	2970	0.95	199	37	750
	10-20 cm	14400	1.3	17.0	420	1.8	1.47	30300	28	41	377	26900	483	5090	372	4.4	2530	0.34	219	30	733
2292480 (Front yard)	0-5 cm	21400	<0.4	5.2	117	1	0.65	16800	25	24	94	22400	75	8500	378	3.7	785	<0.3	44	38	147
	5-10 cm	23300	<0.4	4.5	127	1.1	0.61	16500	27	24	91	23600	73	8790	404	3.7	739	<0.3	43	40	144
	10-20 cm	23000	<0.4	4.6	125	1	0.68	15600	26	28	107	24600	84	8490	409	3.7	956	<0.3	41	39	152
2292481 (Back yard)	0-5 cm	18000	<0.4	5.4	113	0.9	0.72	13400	23	20	83	18600	75	5940	257	3.1	677	<0.3	60	35	164
	5-10 cm	21700	<0.4	6.7	128	1.1	0.89	16600	28	24	100	21600	89	6740	296	3.4	793	<0.3	77	39	193
	10-20 cm	19100	<0.4	6.6	125	1	0.81	19800	25	23	104	20100	85	7080	288	3.5	811	<0.3	77	35	189

Table 2: Results of Chemical Analysis (µg/g) of 2000 Residential Soil Samples, Port Colborne

Al = aluminum, Sb = antimony, As = arsenic, Ba = barium, Be = beryllium, Cd = cadmium, Ca = calcium, Cr = chromium, Co = cobalt, Cu = copper, Fe = iron, Pb = lead, Mg = magnesium, Mn = manganese, Mo = molybdenum, Ni = nickel, Se = selenium, Sr = strontium, V = vanadium, Zn = zinc. Data in bold and underlined exceed MOE Table A effects-based guideline. na - data not available

Site / Location	Soil Depth	Al	Sb	As	Ba	Be	Cd	Ca	Cr	Co	Cu	Fe	Pb	Mg	Mn	Mo	Ni	Se	Sr	V	Zn
2292482 (Front yard)	0-5 cm	17000	<0.4	8.6	112	1	0.80	13700	24	49	181	23600	93	6400	440	3.5	**1700**	0.47	46	36	208
	0-5 cm	17300	<0.4	8.3	117	1	0.86	14100	26	50	184	24800	101	6700	465	3.7	**1800**	0.35	46	37	219
	0-5 cm	21900	<0.4	8.4	143	1.2	0.93	15400	30	48	182	25900	106	7540	495	4.0	**1580**	<0.3	53	49	238
	5-10 cm	24400	<0.4	8.5	146	**1.3**	0.88	14100	32	50	185	28200	92	7660	481	3.6	**1800**	<0.3	47	45	208
	5-10 cm	26400	<0.4	6.3	155	**1.4**	0.79	14200	32	43	161	27800	88	8090	493	3.9	**1390**	<0.3	58	51	210
	5-10 cm	32800	<0.4	4.8	183	**1.7**	0.70	14100	38	39	134	30800	82	9530	475	3.8	**1160**	<0.3	53	60	191
	10-20 cm	20200	<0.4	8.7	132	1.2	0.72	17700	26	38	150	27400	77	7370	500	3.6	**1520**	<0.3	48	39	180
	10-20 cm	27000	<0.4	5.4	156	**1.4**	0.67	16100	32	35	129	28700	80	8680	480	3.7	**1180**	<0.3	52	52	177
	10-20 cm	28600	<0.4	6.2	168	**1.5**	0.67	18000	32	38	141	28700	75	9090	496	3.9	**1200**	<0.3	56	54	177
2292483 (Back yard)	0-5 cm	25400	<0.4	7.8	162	**1.4**	0.79	14300	31	28	108	23000	87	6380	369	3.6	**977**	<0.3	65	46	186
	0-5 cm	24800	<0.4	6.9	164	**1.3**	0.77	13600	28	27	109	22900	96	6180	367	3.6	**930**	<0.3	63	45	187
	0-5 cm	25800	<0.4	7.4	167	**1.4**	0.81	14000	31	28	111	23900	89	6390	381	4.0	**929**	<0.3	64	47	188
	5-10 cm	27900	<0.4	8.9	197	**1.5**	0.77	14700	32	29	110	24400	86	6730	374	3.7	**963**	<0.3	68	49	187
	5-10 cm	26800	<0.4	6.8	168	**1.4**	0.80	14600	30	29	113	23900	89	6530	380	3.5	**975**	<0.3	65	48	191
	5-10 cm	26300	<0.4	7.9	164	**1.4**	0.76	12800	30	28	107	23700	83	6380	362	3.4	**942**	<0.3	61	48	183
	10-20 cm	28500	<0.4	9.6	183	**1.5**	0.79	16300	32	27	107	25200	95	6860	367	3.9	**998**	<0.3	72	49	187
	10-20 cm	24700	<0.4	8.0	159	**1.3**	0.68	14700	28	24	97	22300	75	6300	331	3.4	**834**	<0.3	61	44	169
	10-20 cm	27100	<0.4	9.6	171	**1.4**	0.76	15100	32	26	104	24400	82	6650	355	3.6	**930**	<0.3	69	48	178
2292484 (Front yard)	0-5 cm	19700	<0.4	8.7	122	1.1	0.87	17700	28	22	89	21500	134	9220	423	5.5	**629**	<0.3	62	43	182
	5-10 cm	21900	<0.4	6.1	130	1.2	0.83	14800	29	24	89	23600	125	8250	454	5.3	**648**	<0.3	61	47	179
	10-20 cm	20500	<0.4	6.0	115	1.1	0.80	13800	27	24	89	23100	114	7200	480	5.5	**738**	<0.3	61	43	163
2292485 (Back yard)	0-5 cm	15400	<0.4	17.8	181	0.9	1.93	22600	32	23	131	20500	**313**	9490	312	6.2	**765**	<0.3	89	36	375
	5-10 cm	17800	<0.4	15.1	202	**1.3**	2.20	24500	32	26	139	22000	**372**	8540	338	6.0	**871**	<0.3	95	40	390
	10-20 cm	28400	<0.4	11.2	228	**1.5**	1.81	25600	39	27	220	28900	**259**	11700	383	6.1	**703**	<0.3	104	56	316
2292486 (Front yard)	0-5 cm	14300	<0.4	9.3	160	1	1.20	32500	26	29	145	21000	**277**	14200	458	6.7	**1010**	<0.3	79	37	274
	5-10 cm	17300	<0.4	9.3	178	1.1	1.33	35700	28	31	151	23400	**271**	16300	490	6.7	**1010**	<0.3	80	42	279
	10-20 cm	17500	<0.4	10.9	185	1	1.04	45500	26	31	145	24300	**406**	20300	491	7.0	**1300**	<0.3	84	38	253

Table 2: Results of Chemical Analysis (µg/g) of 2000 Residential Soil Samples, Port Colborne

Al = aluminum, Sb = antimony, As = arsenic, Ba = barium, Be = beryllium, Cd = cadmium, Ca = calcium, Cr = chromium, Co = cobalt, Cu = copper, Fe = iron, Pb = lead, Mg = magnesium, Mn = manganese, Mo = molybdenum, Ni = nickel, Se = selenium, Sr = strontium, V = vanadium, Zn = zinc. Data in bold and underlined exceed MOE Table A effects-based guideline. na - data not available

Site / Location	Soil Depth	Al	Sb	As	Ba	Be	Cd	Ca	Cr	Co	Cu	Fe	Pb	Mg	Mn	Mo	Ni	Se	Sr	V	Zn
2292487 (Back yard)	0-5 cm	14300	<0.4	2.2	83	0.8	1.26	9400	21	17	58	18200	92	5700	630	4.9	207	<0.3	40	34	155
	5-10 cm	13500	<0.4	1.4	54	0.7	0.62	4650	17	12	34	18300	44	4210	542	4.1	99	<0.3	24	31	75
	10-20 cm	12900	<0.4	1.5	46	0.7	0.44	2880	15	10	20	17400	25	3630	587	3.4	46	<0.3	17	28	52
2292488 (Front yard)	0-5 cm	19500	<0.4	7.7	143	1.1	1.10	16000	28	28	129	22100	168	7320	374	5.7	970	<0.3	62	44	232
	5-10 cm	22600	<0.4	7.8	159	1.2	1.17	17300	30	30	130	24600	160	8220	411	5.7	968	<0.3	64	49	232
	10-20 cm	27100	<0.4	7.0	172	1.4	0.85	27700	33	30	130	29700	125	11200	466	6.1	917	<0.3	85	53	187
2292489 (Back yard)	0-5 cm	26300	<0.4	8.3	192	1.4	1.13	22600	35	24	104	26300	153	8940	492	5.7	644	<0.3	82	54	246
	5-10 cm	28700	<0.4	8.8	200	1.5	1.98	22600	36	23	89	29700	148	10600	471	6.0	586	<0.3	73	57	658
	10-20 cm	33900	<0.4	6.0	249	1.7	0.96	20600	41	27	95	32800	158	10400	541	5.8	637	<0.3	76	64	256
2292490 (Front yard)	0-5 cm	21900	<0.4	6.7	157	1.3	1.87	12700	41	32	134	32400	178	5870	521	5.8	953	<0.3	57	47	334
	5-10 cm	19100	<0.4	7.9	140	1.2	1.87	14600	38	31	140	30300	182	6060	502	5.8	926	<0.3	60	41	339
	0-5 cm	21400	<0.4	8.0	150	1.3	2.23	14400	38	30	132	29700	169	6160	562	5.9	921	<0.3	59	44	303
	5-10 cm	24200	<0.4	7.3	149	1.4	1.67	11900	43	31	130	30500	152	6060	497	5.8	971	<0.3	54	50	284
	5-10 cm	20100	<0.4	7.6	129	1.2	1.60	12000	36	29	125	28900	147	5680	467	5.5	918	<0.3	51	41	278
	5-10 cm	22000	<0.4	7.3	140	1.3	1.64	11900	37	29	128	28600	150	5780	491	5.8	907	<0.3	52	44	272
	10-20 cm	22200	<0.4	13.7	180	1.6	2.38	22700	43	36	220	48900	264	5950	736	7.2	1200	0.40	106	39	561
	10-20 cm	22200	<0.4	13.8	172	1.6	2.34	21400	40	34	174	37800	224	6050	671	6.4	1230	<0.3	93	42	436
	10-20 cm	20500	<0.4	12.3	167	1.5	2.11	20700	36	32	164	34600	227	5680	588	6.0	1250	<0.3	90	39	387
2292491 (Back yard)	0-5 cm	23200	<0.4	7.5	154	1.3	0.74	30200	30	21	79	26200	93	14600	523	6.2	484	<0.3	87	47	169
	0-5 cm	20000	<0.4	8.6	146	1.2	1.02	23900	28	22	92	25600	115	11500	484	6.0	617	<0.3	85	42	207
	0-5 cm	20200	<0.4	9.5	147	1.2	1.02	26000	28	23	98	24800	128	11900	469	5.8	677	<0.3	97	43	212
	5-10 cm	19800	<0.4	8.3	147	1.2	0.76	35900	26	21	84	25300	91	15300	536	6.6	541	<0.3	100	40	185
	5-10 cm	24700	<0.4	6.2	157	1.3	0.74	36000	31	21	83	28600	113	13900	503	5.8	553	<0.3	104	46	163
	5-10 cm	21700	<0.4	8.8	150	1.3	0.97	24900	29	23	91	26700	99	11100	512	6.1	648	<0.3	95	45	186
	10-20 cm	20600	<0.4	7.9	141	1.2	0.60	37000	26	18	68	25200	78	14300	494	6.5	396	<0.3	98	40	128
	10-20 cm	22900	<0.4	8.4	163	1.3	0.87	25100	30	24	94	28100	113	11600	583	6.0	662	<0.3	92	46	191
	10-20 cm	26600	<0.4	7.0	160	1.4	0.80	33200	33	21	77	30000	91	12500	504	5.7	529	<0.3	118	50	na

Table 2: Results of Chemical Analysis (µg/g) of 2000 Residential Soil Samples, Port Colborne

Al = aluminum, Sb = antimony, As = arsenic, Ba = barium, Be = beryllium, Cd = cadmium, Ca = calcium, Cr = chromium, Co = cobalt, Cu = copper, Fe = iron, Pb = lead, Mg = magnesium, Mn = manganese, Mo = molybdenum, Ni = nickel, Se = selenium, Sr = strontium, V = vanadium, Zn = zinc. Data in bold and underlined exceed MOE Table A effects-based guideline. na - data not available

Site / Location	Soil Depth	Al	Sb	As	Ba	Be	Cd	Ca	Cr	Co	Cu	Fe	Pb	Mg	Mn	Mo	Ni	Se	Sr	V	Zn
2292492 (Front yard)	0-5 cm	14100	<0.4	3.5	61	0.6	0.47	4800	15	11	38	13000	56	2390	160	3.8	**296**	<0.3	21	26	83
	5-10 cm	13400	<0.4	2.4	52	0.5	0.41	3760	13	10	30	11600	47	1970	137	3.3	**221**	<0.3	17	24	68
	10-20 cm	14900	<0.4	2.8	49	0.5	0.40	3090	13	9	25	12600	39	1890	131	3.2	192	<0.3	14	25	61
2292493 (Back yard)	0-5 cm	17400	<0.4	17.9	191	**1.3**	1.42	30200	27	27	160	22800	**219**	12400	354	6.4	**1080**	<0.3	180	42	267
	5-10 cm	14600	<0.4	13.9	149	1.1	1.20	23500	24	25	139	19900	**207**	10300	308	6.3	**1010**	<0.3	134	38	242
2292494 (Front yard)	0-5 cm	20500	<0.4	6.7	127	1.1	1.09	10200	29	23	96	23500	120	5910	495	4.9	**725**	<0.3	41	44	209
	5-10 cm	20400	<0.4	4.8	122	1.1	0.90	9670	27	22	87	23400	101	5720	526	5.0	**662**	<0.3	39	43	181
	10-20 cm	24000	<0.4	9.8	139	1.2	0.57	14000	29	23	101	27400	106	7280	492	1.7	**786**	<0.3	52	46	206
2292495 (Back yard)	0-5 cm	16900	0.4	11.8	114	0.8	0.68	8500	22	19	95	18800	116	3810	308	1.7	**637**	<0.3	54	36	212
	5-10 cm	17400	0.5	13.2	118	0.8	0.67	8190	22	20	97	19400	113	3860	309	1.4	**702**	<0.3	54	37	196
	10-20 cm	19000	<0.4	13.9	124	0.8	0.65	8020	23	19	87	19400	114	4060	311	1.4	**620**	<0.3	56	39	185
2292496 (Front yard)	0-5 cm	26000	0.8	13.5	177	1.2	1.22	13700	36	38	150	27300	**221**	7140	437	2.3	**1070**	<0.3	68	56	374
	5-10 cm	27700	0.7	14.5	197	**1.3**	1.32	12800	36	40	156	29200	**210**	7350	744	2.3	**1110**	<0.3	67	58	351
	10-20 cm	21900	0.6	**27.7**	153	1	1.36	15300	30	30	129	26500	**270**	6930	372	2.2	**1070**	<0.3	64	50	338
2292497 (Back yard)	0-5 cm	15300	1.3	14.4	200	0.8	1.51	21600	26	24	127	20100	**469**	8590	309	2.1	**1100**	<0.3	86	37	419
	5-10 cm	15400	3.1	17.3	298	0.8	1.51	24400	29	26	204	20400	1200	8050	339	2.0	**1770**	<0.3	94	37	556
	10-20 cm	20100	1.4	18.8	258	1.1	1.33	22300	30	25	145	23800	**385**	7840	329	2.3	**1180**	<0.3	126	44	407
2292498 (Front yard)	0-5 cm	27100	0.7	12.0	187	**1.3**	0.95	14800	40	36	161	27800	**221**	7780	495	2.3	**1180**	<0.3	58	55	289
	5-10 cm	24900	0.5	11.4	186	1.2	1.00	13100	38	32	146	26500	194	7020	452	2.0	**1170**	<0.3	52	51	248
	10-20 cm	25000	0.5	14.1	177	1.2	0.69	18700	35	34	166	29000	**245**	8000	477	2.1	**1600**	<0.3	66	50	268
2292499 (Back yard)	0-5 cm	30600	<0.4	19.3	189	**1.3**	1.20	12400	37	24	109	24300	123	6350	484	2.1	**640**	<0.3	100	58	220
	5-10 cm	30800	0.6	20.0	189	**1.3**	1.24	11400	36	23	106	24700	122	5960	496	2.1	**624**	<0.3	102	58	207
	10-20 cm	33000	0.7	21.1	198	**1.4**	1.23	13400	37	23	110	26500	126	6380	449	1.9	**653**	<0.3	117	60	200
2292500 (Front yard)	0-5 cm	31100	0.5	9.3	185	**1.3**	1.04	17000	39	32	137	24300	194	8270	379	2.1	**1060**	<0.3	62	58	238
	5-10 cm	32300	0.5	10.5	199	**1.3**	1.43	12500	40	33	143	24100	**210**	7790	371	2.0	**1060**	<0.3	59	59	245
	10-20 cm	33000	0.5	11.2	189	**1.3**	0.97	12800	38	30	135	26400	171	8170	419	2.0	**1220**	<0.3	63	58	214

Table 2: Results of Chemical Analysis (µg/g) of 2000 Residential Soil Samples, Port Colborne

Al = aluminum, Sb = antimony, As = arsenic, Ba = barium, Be = beryllium, Cd = cadmium, Ca = calcium, Cr = chromium, Co = cobalt, Cu = copper, Fe = iron, Pb = lead,
Mg = magnesium, Mn = manganese, Mo = molybdenum, Ni = nickel, Se = selenium, Sr = strontium, V = vanadium, Zn = zinc. Data in bold and underlined exceed MOE
Table A effects-based guideline. na - data not available

Site / Location	Soil Depth	Al	Sb	As	Ba	Be	Cd	Ca	Cr	Co	Cu	Fe	Pb	Mg	Mn	Mo	Ni	Se	Sr	V	Zn
2292501 (Back yard)	0-5 cm	37700	<0.4	8.4	225	**1.7**	1.13	14300	46	28	109	27000	124	8530	359	2.2	**642**	<0.3	87	68	227
	5-10 cm	40000	0.4	8.7	243	**1.7**	1.29	16500	46	26	100	29300	118	9320	395	2.1	**515**	<0.3	92	70	207
	10-20 cm	34900	0.5	7.5	237	**1.6**	0.88	21100	42	23	68	33200	93	10700	518	2.0	**340**	<0.3	89	62	196
2292502 (Front yard)	0-5 cm	25200	0.8	17.0	177	**1.3**	0.81	15200	36	36	177	28400	**204**	7630	409	2.3	**1450**	<0.3	78	51	340
	5-10 cm	27300	0.8	19.1	204	**1.4**	0.80	15000	38	38	177	31900	**210**	8350	467	2.1	**1620**	<0.3	79	54	342
	10-20 cm	25700	0.9	**26.7**	190	**1.3**	0.75	18300	35	39	207	31600	197	8300	456	2.4	**1980**	<0.3	90	51	300
2292503 (Back yard)	0-5 cm	19900	1.5	18.2	317	1.1	1.05	24800	36	35	173	28300	**259**	8080	410	2.7	**1390**	0.40	134	48	492
	5-10 cm	23000	1.8	**26.1**	380	**1.3**	1.36	24800	43	40	208	31700	**280**	8240	457	2.7	**1630**	0.40	160	54	575
	10-20 cm	23300	1.9	25.0	384	**1.4**	1.29	26300	40	36	215	31600	**268**	8030	454	2.8	**1480**	<0.3	178	54	540
2292504 (Back yard)	0-5 cm	20700	1.7	18.3	251	**1.3**	2.78	26600	40	**58**	269	32300	**330**	10200	576	3.1	**1970**	0.70	114	55	**992**
	5-10 cm	22400	1.6	18.6	263	**1.4**	1.98	25500	38	47	237	33900	**236**	9380	540	2.8	**2010**	<0.3	128	52	727
	10-20 cm	22800	1.7	17.7	295	**1.5**	1.13	30800	35	37	279	31500	**213**	10000	497	2.5	**1540**	<0.3	153	49	561
2292505 (Front yard)	0-5 cm	18600	0.4	12.1	197	0.9	0.95	29100	28	24	115	23100	152	9220	548	2.2	**801**	0.40	82	39	343
	5-10 cm	19800	0.7	15.7	198	1	1.10	23100	30	31	154	26200	183	9340	502	2.3	**1300**	<0.3	73	44	343
	10-20 cm	19600	1.2	14.8	194	1	0.84	22600	30	34	241	28300	**269**	9270	552	2.1	**1470**	0.50	74	45	327
2292506 (Back yard)	0-5 cm	26500	<0.4	8.1	195	**1.3**	1.42	19800	35	23	113	25200	138	8930	349	5.5	**683**	<0.3	90	50	296
	5-10 cm	22700	<0.4	9.7	188	1.1	0.75	44600	30	21	95	26200	117	19100	440	2.1	**560**	<0.3	131	45	228
	10-20 cm	28600	<0.4	8.1	204	**1.5**	1.52	27500	38	24	103	25800	113	13800	396	6.4	**641**	<0.3	107	52	280
2292507 (Front yard)	0-5 cm	18300	<0.4	10.0	152	1.1	1.41	23400	31	32	148	24900	**248**	10300	484	6.0	**1120**	<0.3	67	42	330
	5-10 cm	21500	<0.4	13.4	194	**1.3**	1.63	23400	37	39	196	29200	**302**	10700	526	6.4	**1640**	<0.3	73	45	369
	10-20 cm	21700	<0.4	14.5	183	**1.3**	1.58	23300	33	37	176	30300	**260**	10000	511	6.0	**1700**	<0.3	83	44	350
2292508 (Back yard)	0-5 cm	25500	<0.4	9.5	180	**1.4**	1.49	14200	36	25	102	26500	160	7600	430	5.6	**603**	<0.3	59	52	256
	5-10 cm	26800	<0.4	7.2	168	**1.4**	1.38	12300	35	25	93	27100	122	8030	432	5.3	**563**	<0.3	51	53	216
	10-20 cm	26900	<0.4	9.8	178	**1.5**	1.48	14000	36	23	99	26000	143	7920	404	5.4	**608**	<0.3	61	53	226
2292509 (Front yard)	0-5 cm	13200	<0.4	4.3	82	0.7	0.63	9870	21	15	51	16900	94	4810	392	4.6	**290**	<0.3	32	31	140
	5-10 cm	13700	<0.4	5.4	99	0.7	0.62	14500	20	16	54	17900	125	5350	410	5.0	**352**	0.80	40	32	150
	10-20 cm	15000	<0.4	5.9	111	0.8	0.78	12900	25	22	74	20900	126	5830	462	5.0	**578**	0.40	44	36	190

Table 2: Results of Chemical Analysis (µg/g) of 2000 Residential Soil Samples, Port Colborne

Al = aluminum, Sb = antimony, As = arsenic, Ba = barium, Be = beryllium, Cd = cadmium, Ca = calcium, Cr = chromium, Co = cobalt, Cu = copper, Fe = iron, Pb = lead, Mg = magnesium, Mn = manganese, Mo = molybdenum, Ni = nickel, Se = selenium, Sr = strontium, V = vanadium, Zn = zinc. Data in bold and underlined exceed MOE Table A effects-based guideline. na - data not available

Site / Location	Soil Depth	Al	Sb	As	Ba	Be	Cd	Ca	Cr	Co	Cu	Fe	Pb	Mg	Mn	Mo	Ni	Se	Sr	V	Zn
2292510 (Back yard)	0-5 cm	13700	<0.4	6.7	149	0.7	1.00	15400	23	17	70	15800	**260**	6430	329	5.2	**429**	<0.3	53	30	276
	5-10 cm	14100	<0.4	6.7	165	0.8	1.11	17500	24	18	75	17500	**313**	7060	380	5.4	**474**	<0.3	61	33	311
	10-20 cm	14200	<0.4	7.9	160	0.8	1.18	18200	24	18	86	17900	**320**	7110	360	5.8	**461**	<0.3	64	32	302
2292511 (Back yard)	0-5 cm	16000	<0.4	5.7	94	0.6	0.44	12400	23	16	49	20100	74	6480	405	5.6	**357**	<0.3	47	40	136
	5-10 cm	17500	<0.4	5.3	95	0.7	0.35	14800	24	17	48	21300	63	7230	455	5.6	**337**	<0.3	63	44	125
	10-20 cm	11800	<0.4	4.4	61	0.4	0.29	18500	20	17	49	20400	47	7200	415	5.5	**421**	0.30	60	42	141
2292512 (Front yard)	0-5 cm	16500	<0.4	8.9	132	0.9	0.89	28400	27	33	174	21500	**214**	12700	472	6.1	**1010**	<0.3	91	41	314
	5-10 cm	18500	<0.4	10.5	139	1	0.92	29700	28	36	211	23200	**277**	13000	490	6.6	**1120**	<0.3	96	44	416
	10-20 cm	22200	0.5	11.6	144	1.1	0.77	24300	30	33	153	27000	**247**	10500	573	7.4	**1090**	<0.3	88	47	297
2292513 (Back yard)	0-5 cm	15300	6.8	15.3	446	0.7	2.10	15100	28	25	**1100**	21500	1350	5380	534	6.8	847	<0.3	77	39	**1210**
	5-10 cm	18400	2.5	**25.7**	284	1.3	1.84	21000	34	38	277	30500	**352**	6310	539	8.4	1930	0.40	131	43	583
	10-20 cm	15900	1.5	15.3	187	0.7	1.13	14000	27	26	201	22800	**251**	5020	480	6.8	**990**	<0.3	69	38	370
2292514 (Front yard)	0-5 cm	17400	1.0	14.4	197	1.1	1.17	35400	32	**51**	281	26300	**290**	12800	686	8.0	**1590**	<0.3	130	46	409
	5-10 cm	16100	1.0	15.2	190	1.1	1.19	35000	29	49	**323**	26100	**302**	12400	753	8.2	**1600**	<0.3	173	45	396
	10-20 cm	15000	1.2	16.3	174	0.9	0.77	31800	30	47	255	31200	**269**	9950	769	8.3	**2160**	0.40	110	40	356
2292515 (Back yard)	0-5 cm	17100	3.5	17.2	511	**2.2**	1.43	62100	32	44	226	34500	**414**	11000	2030	9.7	**1950**	0.80	151	39	643
	5-10 cm	14600	4.5	21.3	534	**1.8**	1.55	52900	34	39	222	42100	**587**	7950	3880	11.4	**2050**	1.20	169	33	685
	10-20 cm	14400	3.2	20.0	397	**1.5**	1.57	45500	35	28	175	37200	**461**	7260	2500	10.2	**1330**	0.50	179	32	544
2292516 (Front yard)	0-5 cm	19000	<0.4	8.4	124	0.8	0.52	15100	26	22	100	21400	137	6390	381	5.9	**668**	<0.3	55	42	177
	5-10 cm	21400	<0.4	9.5	139	1	0.53	15400	29	26	120	22700	132	7020	399	6.4	**735**	<0.3	55	45	185
	10-20 cm	21300	<0.4	8.6	129	0.9	0.47	15500	28	28	132	23400	116	7030	440	6.9	**772**	<0.3	50	46	175
2292517 (Front yard)	0-5 cm	26100	0.7	11.0	170	1.2	0.55	14900	35	43	199	28900	197	8410	522	8.1	**1090**	<0.3	61	60	545
	5-10 cm	29700	<0.4	9.1	172	**1.3**	0.54	9960	38	32	128	30900	128	7980	554	7.9	**688**	<0.3	47	61	301
	10-20 cm	27800	1.4	12.0	256	**1.3**	0.62	16700	39	34	152	33100	**363**	9610	571	8.8	**1210**	<0.3	62	55	590
2292518 (Back yard)	0-5 cm	14900	<0.4	5.8	90	0.6	0.61	8930	20	16	62	16600	164	4030	324	5.4	**380**	<0.3	34	35	173
	5-10 cm	15100	<0.4	5.7	87	0.5	0.60	7720	20	16	59	16000	184	3510	296	5.3	**368**	<0.3	30	35	155
	10-20 cm	16900	<0.4	6.6	113	0.7	0.72	9260	21	16	61	17000	162	4390	302	5.4	**412**	<0.3	35	36	169

Table 2: Results of Chemical Analysis (µg/g) of 2000 Residential Soil Samples, Port Colborne

Al = aluminum, Sb = antimony, As = arsenic, Ba = barium, Be = beryllium, Cd = cadmium, Ca = calcium, Cr = chromium, Co = cobalt, Cu = copper, Fe = iron, Pb = lead, Mg = magnesium, Mn = manganese, Mo = molybdenum, Ni = nickel, Se = selenium, Sr = strontium, V = vanadium, Zn = zinc. Data in bold and underlined exceed MOE Table A effects-based guideline. na - data not available

Site / Location	Soil Depth	Al	Sb	As	Ba	Be	Cd	Ca	Cr	Co	Cu	Fe	Pb	Mg	Mn	Mo	Ni	Se	Sr	V	Zn
2292519 (Front yard)	0-5 cm	21400	<0.4	8.6	142	0.9	0.31	19300	28	35	193	22200	121	9310	418	6.6	1270	<0.3	102	48	189
	5-10 cm	23000	0.6	11.6	146	1.1	0.03	25400	30	60	356	21900	101	11000	351	6.9	2690	<0.3	116	49	161
	10-20 cm	23800	<0.4	10.0	166	1.1	0.17	23900	32	39	217	24000	126	10300	497	7.4	1420	<0.3	161	51	193
2292520 (Back yard)	0-5 cm	18500	<0.4	10.0	135	0.9	0.03	24800	25	41	254	22500	94	9250	397	6.4	1870	<0.3	100	40	174
	5-10 cm	18600	<0.4	10.2	133	0.9	0.03	27100	25	44	274	22800	90	10300	383	6.7	1990	<0.3	111	40	163
	10-20 cm	20800	<0.4	9.3	158	1	0.14	33400	27	31	198	25400	92	10700	496	7.2	1260	<0.3	152	43	188
2292521 (Front yard)	0-5 cm	11600	2.3	10.2	163	0.7	0.92	25100	22	25	141	19000	370	8240	444	6.0	713	<0.3	89	29	316
	5-10 cm	12300	1.1	12.0	174	0.7	0.93	25100	23	27	165	19500	444	7700	476	6.3	843	<0.3	90	31	316
	10-20 cm	11300	<0.4	10.2	114	0.6	0.49	25000	21	33	222	18400	221	9660	496	6.2	928	<0.3	72	30	197
2292522 (Back yard)	0-5 cm	17100	<0.4	6.4	115	0.9	0.50	23100	23	14	41	21400	60	8860	385	6.3	202	<0.3	96	37	130
	5-10 cm	29900	<0.4	7.9	188	1.4	0.42	29400	37	21	45	34600	68	12500	563	8.2	208	<0.3	144	57	136
	10-20 cm	33100	<0.4	8.2	197	1.5	0.29	35700	38	21	45	37600	73	12800	609	8.7	203	<0.3	166	58	135
2292523 (Front yard)	0-5 cm	19900	0.8	13.5	201	1.1	1.26	16100	34	44	248	23400	222	7220	385	6.6	1430	<0.3	86	45	358
	5-10 cm	25800	0.7	15.7	230	1.3	1.48	16000	40	51	319	25000	246	7650	406	7.0	1590	<0.3	99	51	365
	10-20 cm	24200	1.0	15.5	219	1.2	1.13	14500	36	36	208	27100	199	6780	387	7.1	1600	<0.3	105	44	305
2292524 (Back yard)	0-5 cm	20200	0.9	19.9	164	1.1	1.07	15300	30	30	170	25500	160	5550	379	6.5	1280	<0.3	147	43	347
	5-10 cm	19200	1.1	19.1	155	1.1	0.89	13800	28	29	158	24100	148	5320	347	6.0	1260	<0.3	135	41	304
	10-20 cm	18700	2.4	20.5	204	1.1	1.00	17200	31	32	212	29700	228	5980	367	6.8	1620	0.30	153	39	381
2292525 (Front yard)	0-5 cm	11000	1.1	11.1	173	0.8	1.29	29800	27	38	249	21400	244	11500	470	5.5	1110	<0.3	73	33	314
	5-10 cm	12500	1.3	16.5	203	0.9	1.86	38200	33	50	507	23700	315	14100	534	6.4	1690	0.60	92	34	351
	10-20 cm	11800	1.7	15.4	347	0.9	1.70	49900	31	47	393	24100	317	14300	492	6.4	1680	0.70	113	31	433
2292526 (Back yard)	0-5 cm	15800	<0.4	6.4	156	0.8	0.63	14700	26	15	70	19300	164	7020	368	5.1	310	<0.3	61	35	208
	5-10 cm	17200	<0.4	6.7	174	0.9	0.70	18700	29	17	74	20400	192	8370	384	5.0	318	<0.3	78	37	229
	10-20 cm	16200	0.9	7.6	212	0.9	1.10	25400	33	17	97	22700	252	10200	431	5.8	365	<0.3	106	37	306
2292527 (Front yard)	0-5 cm	10600	0.7	9.8	191	0.7	0.93	26800	20	26	153	19500	328	9420	337	5.9	972	0.30	92	32	283
	5-10 cm	10400	0.5	9.6	184	0.8	0.91	26600	19	24	141	18000	314	8690	340	5.1	816	<0.3	88	30	267
	10-20 cm	13200	1.4	13.7	287	0.9	1.23	36500	35	30	184	24300	458	9620	394	6.7	1260	0.50	124	34	379

Table 2: Results of Chemical Analysis (µg/g) of 2000 Residential Soil Samples, Port Colborne

Al = aluminum, Sb = antimony, As = arsenic, Ba = barium, Be = beryllium, Cd = cadmium, Ca = calcium, Cr = chromium, Co = cobalt, Cu = copper, Fe = iron, Pb = lead, Mg = magnesium, Mn = manganese, Mo = molybdenum, Ni = nickel, Se = selenium, Sr = strontium, V = vanadium, Zn = zinc. Data in bold and underlined exceed MOE Table A effects-based guideline. na - data not available

Site / Location	Soil Depth	Al	Sb	As	Ba	Be	Cd	Ca	Cr	Co	Cu	Fe	Pb	Mg	Mn	Mo	Ni	Se	Sr	V	Zn
2292528 (Back yard)	0-5 cm	14200	0.9	7.2	135	0.8	0.71	11800	51	21	94	18600	174	4950	307	5.3	**585**	<0.3	62	32	339
	5-10 cm	16500	0.6	7.0	134	0.9	0.64	11200	34	19	89	17900	168	4910	286	5.3	**536**	<0.3	62	33	247
	10-20 cm	17800	0.6	7.9	170	1.1	1.00	11000	25	17	81	17700	169	4700	315	5.5	**399**	<0.3	82	34	**1750**
2292529 (Front yard)	0-5 cm	12700	<0.4	5.5	100	0.6	0.52	7030	18	14	55	17100	156	3760	361	4.7	**273**	<0.3	31	29	185
	5-10 cm	14200	<0.4	5.8	109	0.6	0.57	7510	28	16	66	18200	174	4020	396	5.0	**349**	<0.3	36	32	193
	10-20 cm	13500	<0.4	9.4	152	0.7	0.76	12700	24	25	125	21900	**314**	5570	452	5.9	**784**	<0.3	88	35	316
2292530 (Back yard)	0-5 cm	13700	<0.4	7.3	110	0.7	0.62	8580	18	13	71	13400	99	3230	189	4.0	**355**	<0.3	53	29	148
	5-10 cm	16700	<0.4	9.9	150	0.9	0.76	10000	21	15	83	15400	139	3590	226	4.9	**482**	<0.3	64	34	184
	10-20 cm	18900	<0.4	13.8	170	1	0.84	13000	26	18	102	20300	177	4900	289	5.1	**641**	<0.3	74	39	234
2292531 (Front yard)	0-5 cm	17700	<0.4	9.7	168	1.1	0.79	15700	30	28	131	22800	**246**	6640	399	5.3	**687**	<0.3	66	45	266
	5-10 cm	17300	0.7	7.9	156	1	0.75	16900	26	27	136	22200	200	7000	404	5.7	**650**	<0.3	66	42	223
	0-5 cm	21600	<0.4	9.8	162	1.1	0.85	15900	27	31	163	21900	**246**	6820	376	6.2	**844**	<0.3	64	42	257
	5-10 cm	19900	0.5	9.2	186	1.2	0.75	15300	31	29	129	24600	**252**	7020	417	5.7	**656**	<0.3	67	49	259
	5-10 cm	21400	0.5	8.5	169	1.1	0.76	16600	29	31	145	23700	**210**	7340	452	6.4	**762**	<0.3	68	46	237
	10-20 cm	21500	<0.4	9.8	181	**1.3**	0.76	13800	31	31	141	23900	**223**	6740	372	6.3	**720**	<0.3	63	48	247
	10-20 cm	19900	0.7	7.9	212	1.2	0.51	20000	29	24	82	26700	**257**	7530	426	5.9	**470**	<0.3	69	47	227
	10-20 cm	21100	<0.4	8.4	205	1.2	0.82	20800	29	30	138	23000	**264**	7930	364	5.9	**772**	<0.3	75	44	245
	10-20 cm	21100	<0.4	8.3	190	**1.3**	0.76	13800	29	29	136	23300	**228**	6700	367	5.9	**685**	<0.3	63	46	242
2292532 (Back yard)	0-5 cm	11400	2.1	20.0	263	0.9	1.59	25100	27	30	220	24800	**442**	5550	432	6.4	**1400**	1.10	121	37	527
	0-5 cm	8830	2.1	14.6	194	0.7	1.07	19200	21	24	168	19600	**354**	4660	339	5.6	**1070**	0.70	88	30	399
	0-5 cm	9530	2.0	16.0	205	0.8	1.38	19400	26	25	167	21100	**386**	4730	349	6.0	**1030**	1.00	92	31	429
	5-10 cm	11700	2.4	22.9	287	0.9	1.42	24300	28	32	232	25800	**480**	5300	446	6.3	**1420**	1.30	113	37	573
	5-10 cm	10300	2.1	18.7	247	0.8	1.32	21900	24	26	202	21200	**409**	4630	378	6.4	**1170**	1.10	101	32	475
	5-10 cm	11700	2.5	22.7	288	0.9	1.57	24000	27	31	211	30200	**542**	5360	451	7.9	**1320**	1.60	107	36	540
	10-20 cm	9630	1.8	20.0	269	0.7	1.11	23600	23	23	189	22700	**406**	4950	361	5.6	**1030**	1.10	109	35	439
	10-20 cm	9490	2.0	20.0	360	0.8	1.19	23500	26	22	190	20900	**637**	4380	376	6.2	**1060**	1.20	111	31	452
	10-20 cm	8990	2.2	16.8	211	0.8	1.09	20100	20	23	157	20100	**343**	4440	347	6.0	**1110**	0.90	92	29	404

Part A - Soil Investigation: Tables

Table 2: Results of Chemical Analysis (µg/g) of 2000 Residential Soil Samples, Port Colborne

Al = aluminum, Sb = antimony, As = arsenic, Ba = barium, Be = beryllium, Cd = cadmium, Ca = calcium, Cr = chromium, Co = cobalt, Cu = copper, Fe = iron, Pb = lead, Mg = magnesium, Mn = manganese, Mo = molybdenum, Ni = nickel, Se = selenium, Sr = strontium, V = vanadium, Zn = zinc. Data in bold and underlined exceed MOE Table A effects-based guideline. na - data not available

Site / Location	Soil Depth	Al	Sb	As	Ba	Be	Cd	Ca	Cr	Co	Cu	Fe	Pb	Mg	Mn	Mo	Ni	Se	Sr	V	Zn
2292533 (Front yard)	0-5 cm	11700	0.4	10.6	132	0.8	0.65	26900	20	33	198	18600	183	9920	360	5.3	1150	<0.3	73	29	242
	5-10 cm	10900	0.4	11.2	129	0.8	0.58	27200	21	39	205	20700	189	9530	367	5.8	1540	0.40	68	30	264
	10-20 cm	12300	0.5	14.4	170	0.9	0.69	26600	22	39	219	21800	233	9490	385	6.2	1500	0.70	83	31	341
2292534 (Back yard)	0-5 cm	11000	0.8	18.5	256	0.9	1.14	21200	37	24	152	19200	303	5970	297	6.0	997	<0.3	102	31	451
	5-10 cm	15500	1.6	23.5	345	1.3	1.59	26000	30	30	201	23000	444	6890	365	6.6	1260	0.40	142	37	650
	10-20 cm	12000	0.9	18.9	232	1	1.19	24900	22	22	151	19000	302	5990	280	5.7	1020	0.30	124	30	502
2292535 (Front yard)	0-5 cm	15400	0.4	8.4	106	0.8	0.53	19900	25	26	157	19200	135	8270	357	5.6	877	<0.3	72	33	171
	5-10 cm	15300	<0.4	8.3	99	0.8	0.40	18400	23	23	130	19100	110	7200	343	5.8	812	<0.3	66	32	150
	10-20 cm	15100	<0.4	16.8	149	1	1.68	38600	33	71	524	26400	344	16000	542	5.6	2330	2.10	91	37	319
2292536 (Back yard)	0-5 cm	18600	<0.4	16.6	350	1.2	2.03	24000	42	33	211	25900	400	7320	379	4.5	1420	1.10	122	44	566
	5-10 cm	nd	nd	nd	nd	nd	nd	nd	nd	nd	nd		nd	nd	nd	nd	nd	nd	nd	nd	nd
	10-20 cm	16000	<0.4	10.8	156	1	1.22	24900	29	32	187	23200	225	10300	488	4.6	1010	<0.3	71	37	311
2292537 (Front yard)	0-5 cm	20600	<0.4	12.9	185	1.2	1.40	29000	31	34	197	27100	259	10900	545	4.7	1130	<0.3	82	43	382
	5-10 cm	20200	<0.4	13.6	195	1.2	1.40	29300	32	35	200	27100	267	11200	565	4.8	1160	<0.3	81	42	386
	10-20 cm	19000	<0.4	16.4	181	1.1	1.47	26300	31	37	219	27900	309	9920	541	4.7	1400	<0.3	87	41	364
2292538 (Back yard)	0-5 cm	20900	<0.4	17.1	224	1.3	2.51	20200	38	54	304	27700	525	7850	440	4.7	1770	0.90	125	50	603
	5-10 cm	22000	<0.4	17.6	236	1.3	2.28	16900	37	42	237	31000	438	7050	429	4.4	1770	0.50	107	44	527
	10-20 cm	20100	<0.4	16.4	240	1.2	1.56	22500	32	26	155	27600	467	7470	413	4.3	1020	<0.3	121	40	363
2292539 (Front yard)	0-5 cm	20700	<0.4	6	122	1	1.00	10700	29	23	83	26300	94	6330	481	5	666	<0.3	38	45	187
	5-10 cm	19700	<0.4	5.0	112	1	0.75	8140	26	20	67	23700	79	5540	467	4.7	544	<0.3	32	42	152
	10-20 cm	19300	<0.4	3.7	101	1	0.61	7260	25	16	48	22300	56	5430	437	4.4	388	<0.3	28	40	126
2292540 (Back yard)	0-5 cm	20500	<0.4	2.5	90	0.7	0.43	3180	25	9	15	17400	51	3310	165	3.3	110	<0.3	27	36	80
	5-10 cm	19700	<0.4	1.1	78	0.7	0.29	1920	23	7	4	15800	31	2880	137	2.8	35	<0.3	17	34	52
	10-20 cm	21800	<0.4	3.6	110	0.8	0.60	5120	28	14	43	19600	89	3890	230	4.2	282	<0.3	35	39	129
2292541 (Front yard)	0-5 cm	20000	<0.4	14.5	165	1	1.89	24700	33	82	337	34800	171	9600	604	4.4	4240	1.45	84	39	398
	5-10 cm	23000	<0.4	21.7	182	1.2	2.43	20000	38	115	477	45500	236	8730	781	4.6	5720	2.25	79	43	435
	10-20 cm	22900	0.6	24.3	173	1.1	2.15	18500	46	100	474	55200	176	8520	720	5.2	6630	2.45	75	41	518

Table 2: Results of Chemical Analysis (µg/g) of 2000 Residential Soil Samples, Port Colborne

Al = aluminum, Sb = antimony, As = arsenic, Ba = barium, Be = beryllium, Cd = cadmium, Ca = calcium, Cr = chromium, Co = cobalt, Cu = copper, Fe = iron, Pb = lead, Mg = magnesium, Mn = manganese, Mo = molybdenum, Ni = nickel, Se = selenium, Sr = strontium, V = vanadium, Zn = zinc. Data in bold and underlined exceed MOE Table A effects-based guideline. na - data not available

Site / Location	Soil Depth	Al	Sb	As	Ba	Be	Cd	Ca	Cr	Co	Cu	Fe	Pb	Mg	Mn	Mo	Ni	Se	Sr	V	Zn
2292542 (Back yard)	0-5 cm	18600	<0.4	8.8	144	0.8	1.22	18700	27	26	137	22400	190	7620	503	3.7	1020	<0.3	58	37	275
	5-10 cm	19400	<0.4	12.5	167	0.9	1.42	22600	29	28	175	23300	272	8800	469	3.7	1200	<0.3	66	39	325
	10-20 cm	18600	1.4	20.3	195	1	1.96	19800	36	64	339	37600	345	7310	511	4.4	3480	1.27	69	37	486
2292543 (Front yard)	0-5 cm	20500	<0.4	6.2	127	1	0.82	17300	26	36	138	22600	102	9910	474	3.7	1440	<0.3	55	41	179
	5-10 cm	21600	<0.4	6.8	128	1.1	0.80	18200	27	32	127	23400	92	10300	516	3.9	1240	<0.3	53	42	164
	10-20 cm	24600	<0.4	6.8	150	1.2	0.81	24800	29	33	151	26100	109	13200	549	4.1	1410	<0.3	71	46	179
2292544 (Back yard)	0-5 cm	20300	<0.4	7.5	122	1	0.96	8050	29	35	142	26100	164	6060	486	3.4	1340	<0.3	36	42	268
	5-10 cm	22400	<0.4	6.7	127	1.1	0.95	11100	31	35	146	28100	151	7810	497	3.7	1350	<0.3	39	44	258
	10-20 cm	20700	1.5	16.2	261	1.1	1.91	15500	47	61	326	39600	406	8040	566	4.2	3520	0.92	61	40	673
2292545 (Front yard)	0-5 cm	15900	<0.4	14.0	166	0.9	1.24	23800	26	46	203	27100	219	10800	517	4.3	2410	0.55	166	32	406
	5-10 cm	17700	<0.4	12.7	164	1	1.12	22900	26	40	177	25600	194	11300	467	4.0	1910	<0.3	157	35	346
	0-5 cm	17600	<0.4	13.8	181	1	1.20	24200	28	41	184	26700	216	11300	519	4.3	2060	<0.3	351	35	397
	5-10 cm	19200	<0.4	15.7	184	1	1.35	25700	30	52	229	30900	271	11600	588	4.3	2790	0.52	189	38	456
	5-10 cm	17800	<0.4	17.6	183	1.1	1.39	26100	30	51	239	29300	243	11800	530	4.3	2790	<0.3	200	35	454
	5-10 cm	19700	<0.4	13.4	178	1.1	1.21	23600	29	40	185	28200	197	11300	545	4.2	2010	<0.3	198	39	375
	10-20 cm	14500	0.8	27.2	271	0.9	2.22	22900	32	86	403	38000	598	8280	595	4.5	5370	2.71	208	31	901
	10-20 cm	14600	0.9	24.2	265	0.9	2.15	26200	34	83	387	36600	515	9570	602	4.7	4700	2.26	247	32	793
	10-20 cm	15900	<0.4	23.3	235	1	1.81	24200	31	75	344	34700	412	8730	601	4.5	4190	2.21	247	33	699
2292546 (Back yard)	0-5 cm	13600	<0.4	29.2	151	0.7	1.96	17200	25	61	310	32700	190	4900	517	4.0	3900	2.10	91	26	499
	0-5 cm	13500	0.5	30.4	156	0.7	2.06	17700	26	70	352	37600	208	4930	582	4.1	4790	2.47	94	27	522
	0-5 cm	14100	0.5	29.8	160	0.8	2.02	17900	28	66	336	35400	252	4870	547	4.4	4320	2.65	96	28	526
	5-10 cm	13600	<0.4	31.8	151	0.7	2.03	17700	28	68	333	36300	196	5020	542	4.2	4560	2.37	88	27	502
	5-10 cm	13100	<0.4	32.8	153	0.7	2.06	17000	26	68	346	35800	198	4570	547	4.3	4600	2.59	100	26	507
	5-10 cm	12100	0.4	30.7	145	0.6	1.92	15700	24	63	323	32900	206	4160	514	3.9	4160	2.38	80	24	479
	10-20 cm	14200	0.7	37.6	170	0.8	2.31	18900	29	74	370	39900	217	4890	601	4.3	5060	3.11	102	27	553
	10-20 cm	14400	0.4	45.3	188	0.8	2.65	21500	30	74	428	37800	259	4860	626	4.4	4740	4.16	123	28	627
	10-20 cm	13200	1.1	43.8	186	0.8	2.56	20000	32	89	459	42800	246	4600	634	4.6	6590	4.26	111	26	621

Table 2: Results of Chemical Analysis (µg/g) of 2000 Residential Soil Samples, Port Colborne

Al = aluminum, Sb = antimony, As = arsenic, Ba = barium, Be = beryllium, Cd = cadmium, Ca = calcium, Cr = chromium, Co = cobalt, Cu = copper, Fe = iron, Pb = lead, Mg = magnesium, Mn = manganese, Mo = molybdenum, Ni = nickel, Se = selenium, Sr = strontium, V = vanadium, Zn = zinc. Data in bold and underlined exceed MOE Table A effects-based guideline. na - data not available

Site / Location	Soil Depth	Al	Sb	As	Ba	Be	Cd	Ca	Cr	Co	Cu	Fe	Pb	Mg	Mn	Mo	Ni	Se	Sr	V	Zn
2292547 (Front yard)	0-5 cm	16500	<0.4	21.1	146	1	1.74	16800	36	82	379	39600	239	7030	550	5.1	5090	4.09	58	35	486
	5-10 cm	17300	<0.4	24.6	141	1	1.73	19600	33	79	372	41600	182	8670	577	4.5	5420	3.46	56	36	458
	10-20 cm	17300	<0.4	22.8	137	1	1.74	19300	32	81	372	42800	181	8570	573	4.6	5530	2.86	56	37	454
2292548 (Back yard)	0-5 cm	13100	1.7	31.9	267	1	2.68	19600	36	112	548	47000	431	5550	631	4.8	7360	4.85	102	32	872
	5-10 cm	13500	1.8	41.9	301	1	3.47	22300	42	109	645	53500	437	5580	719	5.3	7580	4.70	111	31	1090
	10-20 cm	11700	2.4	24.9	283	0.9	2.08	20700	29	54	373	34500	339	4800	453	4.2	3730	2.97	111	27	727
2292549 (Front yard)	0-5 cm	34500	<0.4	14.1	196	1.6	1.46	11300	41	59	292	26900	94	7940	343	3.9	2790	<0.3	74	59	271
	5-10 cm	36700	<0.4	15.2	214	1.8	1.55	9480	44	57	295	29200	100	7150	361	3.8	2790	<0.3	72	63	239
	10-20 cm	20700	<0.4	15.4	158	1	1.54	17700	32	63	287	34400	224	6200	487	4.2	3530	0.73	75	39	364
2292550 (Back yard)	0-5 cm	16400	<0.4	13.3	256	1.2	1.41	18000	42	39	264	28800	228	4620	465	4.3	2030	<0.3	133	33	503
	5-10 cm	19100	0.8	12.9	268	1.3	1.50	16400	45	43	247	29200	245	4750	466	4.3	2190	<0.3	132	39	502
	10-20 cm	16100	1.0	22.5	408	1.9	2.21	22300	44	78	485	44000	429	5030	564	5.3	5000	2.13	232	38	870
2292551 (Front yard)	0-5 cm	15500	<0.4	11.0	126	0.8	1.24	15600	26	49	202	28200	138	5630	458	4.0	2400	1.25	78	31	304
	5-10 cm	18700	<0.4	12.2	147	0.9	1.30	15800	28	54	267	31800	152	5350	454	4.1	2610	1.27	92	36	321
	10-20 cm	18100	<0.4	12.4	148	0.9	1.21	14800	28	51	237	35200	135	5450	466	3.9	2920	0.95	88	34	320
2292552 (Back yard)	0-5 cm	13600	<0.4	9.0	147	0.7	0.96	10600	23	32	145	24100	144	4230	464	3.5	1560	<0.3	39	28	320
	5-10 cm	13400	<0.4	8.9	133	0.7	0.90	10200	24	32	141	23600	130	4170	447	3.5	1530	<0.3	36	28	308
	10-20 cm	15300	<0.4	13.8	187	0.8	1.48	15500	32	52	269	32300	215	5470	481	4.0	2870	1.18	59	31	505
2292553 (Front yard)	0-5 cm	14200	3.0	14.4	140	0.8	1.68	17900	28	73	289	30600	206	5760	493	4.3	3370	1.88	71	32	429
	5-10 cm	12300	4.3	24.0	153	0.8	1.89	18300	26	85	382	36500	211	5090	579	4.3	4980	3.47	76	27	476
	10-20 cm	12700	7.2	35.7	176	0.9	2.36	19400	28	84	471	40800	341	5290	650	4.6	5860	4.15	85	25	593
2292554 (Back yard)	0-5 cm	14200	<0.4	28.6	112	0.7	1.65	10200	27	86	398	36800	121	3480	490	3.8	4940	3.74	88	30	341
	5-10 cm	15200	<0.4	27.8	116	0.8	1.43	9990	29	60	322	38200	106	3540	445	3.7	4180	2.58	98	27	327
	10-20 cm	14900	<0.4	13.1	93	0.6	0.79	7710	22	27	162	19600	55	3470	230	3.0	1650	<0.3	73	28	155
2292555 (Back yard)	0-5 cm	14300	1.5	21.5	255	1	2.17	19900	34	68	408	36700	362	5310	526	4.2	4410	2.38	124	29	578
	5-10 cm	14600	2.8	25.4	274	1.1	2.40	20100	35	78	462	42700	336	5420	575	4.3	5280	4.73	137	28	648
	10-20 cm	14500	1.2	25.3	287	1.1	2.12	21200	33	65	420	38000	800	5170	522	4.2	4640	2.00	151	28	609

Table 2: Results of Chemical Analysis (µg/g) of 2000 Residential Soil Samples, Port Colborne

Al = aluminum, Sb = antimony, As = arsenic, Ba = barium, Be = beryllium, Cd = cadmium, Ca = calcium, Cr = chromium, Co = cobalt, Cu = copper, Fe = iron, Pb = lead, Mg = magnesium, Mn = manganese, Mo = molybdenum, Ni = nickel, Se = selenium, Sr = strontium, V = vanadium, Zn = zinc. Data in bold and underlined exceed MOE Table A effects-based guideline. na - data not available

Site / Location	Soil Depth	Al	Sb	As	Ba	Be	Cd	Ca	Cr	Co	Cu	Fe	Pb	Mg	Mn	Mo	Ni	Se	Sr	V	Zn
2292556 (Front yard)	0-5 cm	14400	<0.4	6.7	97	0.7	0.66	15300	19	21	84	17400	66	7090	480	3.6	720	<0.3	50	30	130
	5-10 cm	17500	<0.4	8.4	116	0.9	0.69	12200	21	24	99	19300	82	6580	464	3.4	851	<0.3	46	34	133
	10-20 cm	18100	<0.4	5.6	123	0.9	0.62	17100	23	19	70	20700	150	7690	483	3.7	528	<0.3	49	35	130
2292557 (Back yard)	0-5 cm	10700	<0.4	8.0	83	0.5	0.65	16900	17	23	112	16800	53	4040	386	3.5	1040	<0.3	54	22	141
	5-10 cm	12000	<0.4	11.4	81	0.6	0.81	8710	20	27	133	18400	65	3470	341	2.9	1270	<0.3	43	24	156
	10-20 cm	11800	<0.4	18.5	95	0.6	1.06	8880	20	34	190	21200	82	3170	364	3.0	1680	0.43	56	24	210
2292558 (Front yard)	0-5 cm	8810	<0.4	10.6	89	0.5	1.01	19000	18	30	133	18900	110	6280	392	3.6	1410	<0.3	63	21	230
	0-5 cm	9650	<0.4	11.0	102	0.6	1.13	19000	18	36	162	21000	134	6660	441	3.8	1670	0.47	57	22	280
	0-5 cm	9070	<0.4	10.5	91	0.5	1.25	18400	18	32	143	19200	119	6650	415	3.9	1490	0.45	51	20	283
	5-10 cm	9230	<0.4	13.7	98	0.6	1.31	17400	20	41	189	22500	158	6450	422	3.9	2060	0.56	62	21	299
	5-10 cm	10100	<0.4	14.7	107	0.6	1.30	18000	19	44	200	24900	152	6310	459	3.8	2320	0.57	66	23	310
	5-10 cm	9680	<0.4	13.5	100	0.6	1.26	17300	19	42	198	23200	148	6580	413	4.0	2170	0.54	52	22	291
	10-20 cm	8860	0.7	18.1	129	0.7	1.64	21900	27	53	254	29900	224	6470	471	4.3	2680	1.48	99	20	370
	10-20 cm	8700	<0.4	17.5	137	0.7	1.70	22600	21	61	276	33100	286	7090	493	4.5	3440	1.53	105	20	377
	10-20 cm	10000	<0.4	18.8	146	0.8	1.73	23900	23	57	273	31600	280	7750	504	4.3	2810	1.54	90	23	396
2292559 (Back yard)	0-5 cm	11800	<0.4	9.5	90	0.6	1.35	15200	20	24	113	17600	118	5440	289	3.5	983	<0.3	64	26	213
	0-5 cm	12800	<0.4	7.7	107	0.7	1.33	19100	21	24	122	17600	280	7920	343	3.8	878	<0.3	75	25	239
	0-5 cm	12700	<0.4	8.4	96	0.7	1.29	17900	23	23	106	17400	119	7220	294	3.6	994	<0.3	68	27	220
	5-10 cm	12700	<0.4	14.8	95	0.6	1.37	15900	23	25	115	18200	122	5870	316	3.6	948	<0.3	76	27	208
	5-10 cm	12800	<0.4	6.8	105	0.6	1.22	21600	22	22	116	17600	138	8960	313	3.9	780	<0.3	86	27	207
	5-10 cm	12500	<0.4	7.0	89	0.6	0.89	14100	48	20	142	15100	88	6080	263	3.5	751	<0.3	86	28	421
	10-20 cm	11200	0.4	29.3	150	0.6	3.07	12500	27	54	274	27400	428	4410	427	4.1	2610	1.60	100	24	429
	10-20 cm	13800	<0.4	18.1	139	0.8	1.81	15300	24	40	198	27900	193	5700	470	3.8	1970	<0.3	100	27	348
	10-20 cm	12300	<0.4	15.4	125	0.7	1.51	15000	30	39	200	25400	146	4620	369	3.7	2060	<0.3	102	26	438
2292560 (Front yard)	0-5 cm	8900	<0.4	12.0	95	0.6	1.19	24800	20	36	159	28200	192	7430	505	4.7	1980	<0.3	61	20	311
	5-10 cm	9490	1.4	23.6	123	0.7	2.25	24900	29	71	333	48800	316	7610	753	4.8	4820	2.00	69	20	586
	10-20 cm	7420	<0.4	10.8	77	0.5	0.85	17700	15	25	153	21000	133	5320	377	3.2	1920	<0.3	56	19	225

Table 2: Results of Chemical Analysis (µg/g) of 2000 Residential Soil Samples, Port Colborne

Al = aluminum, Sb = antimony, As = arsenic, Ba = barium, Be = beryllium, Cd = cadmium, Ca = calcium, Cr = chromium, Co = cobalt, Cu = copper, Fe = iron, Pb = lead, Mg = magnesium, Mn = manganese, Mo = molybdenum, Ni = nickel, Se = selenium, Sr = strontium, V = vanadium, Zn = zinc. Data in bold and underlined exceed MOE Table A effects-based guideline. na - data not available

Site / Location	Soil Depth	Al	Sb	As	Ba	Be	Cd	Ca	Cr	Co	Cu	Fe	Pb	Mg	Mn	Mo	Ni	Se	Sr	V	Zn
2292561 (Front yard)	0-5 cm	8090	<0.4	9.2	96	0.5	1.80	23300	29	41	190	29700	225	9100	471	3.7	2150	0.50	51	22	410
	5-10 cm	7200	1.1	17.5	103	0.6	2.06	25800	38	61	278	48000	256	8350	652	4.3	3350	1.40	60	20	647
	10-20 cm	6850	<0.4	17.9	93	0.6	1.82	24400	30	47	235	38300	212	7300	531	3.9	3130	0.80	54	17	466
2292562 (Back yard)	0-5 cm	10400	1.3	18.5	164	0.8	2.39	18900	30	70	334	39600	364	6620	545	4.2	4020	2.30	64	22	710
	5-10 cm	8670	1.2	22.0	157	0.7	2.26	20700	29	60	335	38900	319	6340	529	4.1	3240	2.50	62	18	691
	10-20 cm	6620	0.8	20.3	135	0.6	1.76	21500	21	47	249	32800	290	5610	439	4.0	2920	1.80	64	14	551
2292563 (Front yard)	0-5 cm	22500	<0.4	10.6	152	1.2	1.58	22400	45	50	210	25700	173	10400	433	5.1	1620	<0.3	62	50	359
	5-10 cm	23800	<0.4	12.5	157	1.2	1.60	18600	48	63	277	29100	183	8290	490	4.7	2320	0.50	54	50	357
	10-20 cm	23000	0.8	31.7	207	1.3	2.60	22500	45	112	598	41000	263	8400	540	4.9	7590	5.30	72	42	662
2292564 (Back yard)	0-5 cm	23200	1.4	19.1	203	1.3	2.30	17700	38	43	282	30200	274	6800	413	4.2	2030	0.50	85	47	578
	5-10 cm	25200	3.3	29.3	323	1.6	2.27	19900	42	50	485	34600	446	6530	346	4.3	3210	0.70	119	47	635
	10-20 cm	25500	1.3	28.6	266	1.6	2.06	21600	40	41	359	30200	258	6560	297	3.9	2780	0.70	133	46	512
2292565 (Front yard)	0-5 cm	23900	<0.4	12.7	190	1.3	1.80	22800	35	42	437	26000	244	9410	392	4.2	1900	<0.3	86	47	336
	5-10 cm	26500	<0.4	15.0	202	1.5	1.76	24700	36	43	244	28300	226	10300	410	4.1	2140	0.40	99	50	306
	10-20 cm	26100	<0.4	14.5	202	1.5	1.53	24300	35	35	215	27200	203	8660	372	5.0	1930	<0.3	115	48	273
2292566 (Back yard)	0-5 cm	18800	7.6	19.2	405	1.5	2.17	34500	40	52	306	28000	717	11200	495	4.4	2290	1.10	347	41	727
	5-10 cm	22100	13.5	25.2	470	1.7	2.17	32300	45	50	393	33600	925	9970	465	4.4	2880	0.50	180	41	779
	10-20 cm	22300	13.6	27.2	530	1.6	2.44	27100	68	46	372	32000	1100	7930	440	4.9	2720	0.50	173	42	853
2292567 (Front yard)	0-5 cm	21800	1.5	7.9	155	1.2	1.03	18600	33	31	153	24700	221	8390	415	3.5	1260	<0.3	63	44	280
	0-5 cm	20500	<0.4	7.2	146	1.1	0.99	19200	29	28	137	24300	126	8280	377	3.6	1190	<0.3	69	40	292
	0-5 cm	22400	<0.4	8.9	158	1.2	1.04	18400	32	30	149	26800	128	8380	412	3.7	1270	<0.3	66	43	275
	5-10 cm	23700	<0.4	11.6	156	1.3	1.21	21400	33	40	199	29200	150	9600	450	3.7	1920	<0.3	66	46	301
	5-10 cm	20500	<0.4	9.0	145	1.2	1.03	20800	32	31	167	31900	157	8390	408	3.7	1980	<0.3	69	39	263
	5-10 cm	21200	<0.4	8.6	154	1.2	1.07	19900	32	34	175	32500	133	7870	391	3.5	2150	<0.3	75	41	294
	10-20 cm	24100	<0.4	12.9	163	1.4	1.19	20700	33	40	229	44200	138	8910	404	3.9	3060	<0.3	74	44	264
	10-20 cm	22000	<0.4	8.6	148	1.2	0.91	25100	30	30	152	32800	103	9970	427	3.7	2340	<0.3	80	40	227
	10-20 cm	27600	<0.4	8.2	174	1.4	1.01	21700	35	33	173	36100	121	9180	445	3.6	1960	<0.3	68	48	229

Table 2: Results of Chemical Analysis (µg/g) of 2000 Residential Soil Samples, Port Colborne

Al = aluminum, Sb = antimony, As = arsenic, Ba = barium, Be = beryllium, Cd = cadmium, Ca = calcium, Cr = chromium, Co = cobalt, Cu = copper, Fe = iron, Pb = lead, Mg = magnesium, Mn = manganese, Mo = molybdenum, Ni = nickel, Se = selenium, Sr = strontium, V = vanadium, Zn = zinc.   Data in bold and underlined exceed MOE Table A effects-based guideline.   na - data not available

Site / Location	Soil Depth	Al	Sb	As	Ba	Be	Cd	Ca	Cr	Co	Cu	Fe	Pb	Mg	Mn	Mo	Ni	Se	Sr	V	Zn
2292568 (Back yard)	0-5 cm	20300	<0.4	9.8	217	1.2	1.47	21200	35	33	166	25700	**237**	7980	473	4.0	**1340**	<0.3	107	41	514
	0-5 cm	19000	<0.4	8.4	203	1.2	1.41	19700	34	32	158	25200	**298**	7840	408	3.8	**1350**	<0.3	96	40	498
	0-5 cm	20400	<0.4	8.3	208	1.2	1.47	20600	35	32	157	25400	**230**	8060	409	3.7	**1320**	<0.3	97	41	503
	5-10 cm	19500	<0.4	9.2	195	1.2	1.44	22200	33	33	189	25100	**229**	8300	393	3.7	**1440**	<0.3	102	40	489
	5-10 cm	20400	<0.4	9.3	219	**1.4**	1.43	20200	36	35	178	25700	**252**	8050	412	4.4	**1410**	<0.3	114	45	551
	5-10 cm	20900	<0.4	8.5	199	1.1	1.48	21800	33	32	155	26800	**242**	8110	404	3.7	**1420**	<0.3	95	39	463
	10-20 cm	19800	0.6	8.6	191	1.2	1.31	18300	33	32	165	25300	**223**	7030	370	3.6	**1440**	<0.3	89	40	440
	10-20 cm	20600	0.9	9.7	217	**1.3**	1.50	18100	35	35	178	26200	**280**	7320	391	3.7	**1540**	<0.3	104	41	511
	10-20 cm	21900	<0.4	10.4	216	1.2	1.49	20900	34	34	182	27700	**272**	7810	391	3.7	**1730**	<0.3	96	39	480
2292569 (Front yard)	0-5 cm	30700	<0.4	8.4	159	**1.4**	1.08	15500	36	32	141	30900	173	8710	437	3.5	**1370**	<0.3	71	49	259
	5-10 cm	32700	<0.4	7.5	166	**1.5**	1.01	20600	37	32	137	33900	152	10000	449	3.6	**1380**	<0.3	82	50	232
	10-20 cm	29600	<0.4	11.0	168	**1.4**	1.15	28800	34	34	172	33400	172	10700	461	3.6	**1870**	<0.3	98	45	244
2292570 (Back yard)	0-5 cm	nd	nd	nd	nd	nd	nd	nd	nd	nd	nd	nd	nd	nd	nd	nd	nd	nd	nd	nd	nd
	5-10 cm	21100	<0.4	7.8	136	1	0.91	27300	26	28	126	28600	159	9320	473	4.4	**1200**	<0.3	70	36	245
	10-20 cm	20300	<0.4	9.9	145	1	0.98	33300	27	28	141	29000	168	9290	411	3.8	**1490**	<0.3	79	34	245
2292571 (Front yard)	0-5 cm	19000	0.5	14.8	222	1	2.05	33400	31	50	242	29800	**334**	13800	449	4.1	**2520**	8.00	127	36	578
	5-10 cm	18900	2.3	18.9	180	1	2.33	44300	29	**55**	286	33900	**362**	17500	473	4.4	**3120**	9.00	138	34	565
	10-20 cm	25900	<0.4	13.0	186	**1.3**	1.42	44500	31	31	176	32600	190	15300	585	4.1	**1660**	<0.3	197	41	341
2292572 (Front yard)	0-5 cm	19900	<0.4	12.6	223	1.2	1.90	24700	33	46	247	25900	**342**	10600	484	4.3	**1910**	<0.3	70	41	542
	5-10 cm	23600	<0.4	19.6	274	**1.4**	2.10	26200	36	**52**	294	30700	**341**	10300	488	4.3	**2880**	<0.3	125	45	522
	10-20 cm	24200	<0.4	11.5	206	**1.4**	1.21	35500	32	30	185	26700	**222**	12700	468	4.2	**1450**	<0.3	87	44	334
2292573 (Back yard)	0-5 cm	18300	7.9	7.9	123	0.9	0.99	14100	27	21	124	20300	165	6670	476	3.5	563	<0.3	62	36	246
	5-10 cm	17200	**34.5**	14.1	176	1	1.80	24600	29	36	**353**	26000	**554**	9550	443	4.4	**1400**	<0.3	89	36	471
	10-20 cm	22600	91.1	27.1	326	**1.4**	2.59	24700	41	46	**625**	31900	**782**	9520	428	4.9	**2370**	<0.3	124	43	**822**
2292574 (Front yard)	0-5 cm	26800	<0.4	13.5	192	**1.4**	1.92	15300	37	49	221	31600	**239**	7950	821	4.1	**2080**	<0.3	52	52	397
	5-10 cm	30400	<0.4	15.3	243	**1.6**	1.92	13600	41	**51**	248	36300	**236**	8160	731	3.9	**2450**	<0.3	51	57	400
	10-20 cm	25400	<0.4	8.6	195	**1.4**	1.40	24400	31	26	198	28800	**219**	7540	588	4.0	**1180**	<0.3	67	46	443

Table 2: Results of Chemical Analysis (μg/g) of 2000 Residential Soil Samples, Port Colborne

Al = aluminum, Sb = antimony, As = arsenic, Ba = barium, Be = beryllium, Cd = cadmium, Ca = calcium, Cr = chromium, Co = cobalt, Cu = copper, Fe = iron, Pb = lead, Mg = magnesium, Mn = manganese, Mo = molybdenum, Ni = nickel, Se = selenium, Sr = strontium, V = vanadium, Zn = zinc. Data in bold and underlined exceed MOE Table A effects-based guideline. na - data not available

Site / Location	Soil Depth	Al	Sb	As	Ba	Be	Cd	Ca	Cr	Co	Cu	Fe	Pb	Mg	Mn	Mo	Ni	Se	Sr	V	Zn
2292575 (Front yard)	0-5 cm	15600	<0.4	15.3	187	0.9	1.41	22900	28	65	273	22200	505	8760	478	4.4	2770	<0.3	53	36	565
	5-10 cm	19400	<0.4	23.8	206	1.1	1.72	18100	33	86	617	28000	428	8080	494	4.2	4550	1.20	49	41	607
	10-20 cm	21800	<0.4	15.7	183	1.3	1.09	24300	31	48	245	24500	302	8630	498	3.9	2860	<0.3	62	41	362
2292576 (Back yard)	0-5 cm	14500	<0.4	18.3	177	1	1.36	18000	24	40	220	20300	270	6080	382	3.8	1950	0.40	66	32	356
	5-10 cm	13500	<0.4	18.5	164	0.9	1.20	20700	22	34	192	20100	239	6230	355	3.8	1790	<0.3	69	30	299
	10-20 cm	13000	<0.4	12.9	136	0.8	0.97	21600	18	20	116	15400	196	6590	293	3.7	883	<0.3	74	28	216
2292577 (Front yard)	0-5 cm	20500	<0.4	12.4	186	1.2	1.51	19800	30	54	278	25800	289	9100	445	4.2	2490	<0.3	75	42	365
	5-10 cm	18500	<0.4	9.3	168	1.1	1.29	19200	27	48	233	23600	248	8840	397	4.3	2160	<0.3	69	39	316
	0-5 cm	20100		12.1	194	1.2	1.50	20200	31	55	284	25600	283	9260	436	4.3	2530	<0.3	72	43	377
	5-10 cm	20600	<0.4	20.4	285	1.4	2.04	29500	40	74	504	30300	491	11000	472	4.7	4870	0.60	97	40	588
	5-10 cm	19700	<0.4	17.6	241	1.4	1.77	28200	30	63	381	28000	392	10700	489	4.4	3780	<0.3	107	39	481
	5-10 cm	21000	<0.4	15.8	253	1.4	1.59	27200	30	56	332	26700	383	10100	448	4.2	3020	<0.3	97	41	441
	10-20 cm	22600	0.6	19.0	283	1.4	1.81	30900	32	64	446	33800	456	10100	481	4.5	4770	<0.3	102	41	465
	10-20 cm	20800	<0.4	28.0	323	1.5	2.26	30200	33	82	531	35200	485	9250	502	4.6	5990	1.20	114	38	684
	10-20 cm	20600	<0.4	27.4	327	1.3	2.51	39000	32	80	589	34300	570	10800	524	4.7	6060	1.10	117	37	901
2292578 (Back yard)	0-5 cm	20500	0.6	12.9	344	1.2	1.84	19100	35	43	282	25900	493	6840	390	4.1	1910	<0.3	92	42	683
	0-5 cm	17300	0.7	11.3	285	1	1.69	17000	39	39	437	23700	519	6150	351	4.0	1740	<0.3	81	37	596
	0-5 cm	15600	0.7	12.2	244	1	2.09	17400	29	40	238	23200	384	6410	351	4.1	1830	0.30	73	35	514
	5-10 cm	20000	1.7	16.5	547	1.3	2.31	24800	41	48	441	32200	1430	7750	431	4.4	2660	<0.3	113	38	960
	5-10 cm	18300	1.7	19.0	624	1.1	2.43	25000	44	50	567	30600	661	7370	443	4.5	2690	<0.3	116	35	1100
	5-10 cm	16600	1.1	16.4	532	1.1	2.15	25000	39	46	351	27200	634	8060	399	4.4	2580	<0.3	103	33	864
	10-20 cm	15300	4.2	17.9	502	1.2	1.93	25300	35	36	350	26900	860	6150	317	4.3	2360	<0.3	128	29	869
	10-20 cm	14700	1.1	15.3	600	1	1.84	24500	33	35	327	25700	686	6240	345	4.1	2230	<0.3	105	29	846
	10-20 cm	12900	1.9	20.4	430	0.9	2.27	25600	37	40	513	25700	746	6440	335	4.3	2720	0.30	108	28	812
2292579 (Front yard)	0-5 cm	12000	<0.4	1.2	50	0.6	0.32	6090	19	12	37	17300	37	4330	580	2.7	139	<0.3	20	25	69
	5-10 cm	12100	<0.4	1.9	42	0.6	0.21	5420	15	10	25	18100	20	4070	655	2.4	60	<0.3	18	24	47
	10-20 cm	11300	<0.4	2.1	41	0.6	0.20	11800	15	9	27	32200	20	5550	1140	3.3	64	<0.3	24	24	59

Table 2: Results of Chemical Analysis (µg/g) of 2000 Residential Soil Samples, Port Colborne

Al = aluminum, Sb = antimony, As = arsenic, Ba = barium, Be = beryllium, Cd = cadmium, Ca = calcium, Cr = chromium, Co = cobalt, Cu = copper, Fe = iron, Pb = lead, Mg = magnesium, Mn = manganese, Mo = molybdenum, Ni = nickel, Se = selenium, Sr = strontium, V = vanadium, Zn = zinc. Data in bold and underlined exceed MOE Table A effects-based guideline. na - data not available

Site / Location	Soil Depth	Al	Sb	As	Ba	Be	Cd	Ca	Cr	Co	Cu	Fe	Pb	Mg	Mn	Mo	Ni	Se	Sr	V	Zn
2292580 (Back yard)	0-5 cm	33500	<0.4	15.9	254	1.1	2.48	24900	37	47	250	45800	**338**	7720	386	4.8	**3300**	<0.3	90	37	494
	5-10 cm	37000	<0.4	20.5	287	1.2	2.20	46700	39	47	261	49500	**565**	7940	397	4.3	**3610**	<0.3	114	39	521
	10-20 cm	33600	0.8	21.6	289	1.1	2.12	55000	39	39	241	45700	**707**	8080	385	4.3	**3110**	<0.3	125	34	463
2292581 (Front yard)	0-5 cm	30900	<0.4	21.4	130	0.8	1.32	11100	24	42	212	40400	149	5620	467	3.8	**3220**	<0.3	39	33	282
	5-10 cm	32600	<0.4	21.0	128	0.9	1.39	11800	23	40	186	42900	136	5820	894	3.5	**3250**	<0.3	42	33	256
	10-20 cm	38900	<0.4	19.2	145	1.1	1.21	45300	27	38	196	50200	129	9790	1170	4.0	**3000**	<0.3	62	38	235
2292582 (Back yard)	0-5 cm	14500	<0.4	13.9	90	0.6	1.09	5680	20	22	85	32800	83	2980	421	3.0	755	<0.3	29	29	211
	5-10 cm	14400	<0.4	12.2	89	0.6	1.09	5660	20	21	84	33600	84	2970	434	3.1	**722**	<0.3	29	29	212
	10-20 cm	13900	<0.4	18.9	157	0.8	1.44	10700	22	31	149	27000	151	4100	718	3.5	**1480**	<0.3	54	29	427
2292583 (Front yard)	0-5 cm	22500	<0.4	4.5	107	1	0.91	9070	26	33	109	23200	68	5260	371	4.0	**864**	<0.3	40	43	256
	5-10 cm	17200	<0.4	4.9	98	0.9	0.78	7650	24	34	140	22100	66	4410	356	3.3	**1140**	<0.3	42	39	197
	10-20 cm	25200	<0.4	7.0	130	1.1	0.88	16300	28	37	154	28900	65	8720	481	3.7	**1600**	<0.3	54	46	218
2292584 (Front yard)	0-5 cm	21000	<0.4	7.8	154	1	1.51	25000	32	**86**	287	25600	120	12300	500	4.1	**3000**	<0.3	61	41	217
	5-10 cm	23600	<0.4	7.9	165	1.1	2.19	26600	31	**88**	**305**	27500	107	13500	528	4.1	**2770**	<0.3	53	44	214
	10-20 cm	28000	<0.4	30.5	213	**1.4**	1.80	28800	41	**151**	**653**	54500	150	13700	670	4.9	**10400**	1.50	60	49	415
2292585 (Back yard)	0-5 cm	15700	<0.4	5.7	111	0.8	1.00	22700	29	39	191	21000	100	8710	347	4.5	**1820**	<0.3	81	30	214
	5-10 cm	18500	<0.4	7.8	128	1	1.11	27200	25	49	236	24000	113	11500	400	4.2	**2100**	<0.3	88	36	250
	10-20 cm	20900	<0.4	12.8	142	1	1.14	35500	28	**64**	**489**	29600	152	17500	494	4.4	**4030**	<0.3	88	39	283
2292587 (Front yard)	0-5 cm	38300	<0.4	1.8	305	**1.7**	1.87	36000	54	47	153	35900	**371**	14700	661	4.3	**1240**	<0.3	118	64	416
	5-10 cm	40500	<0.4	6.8	514	**1.8**	3.78	37900	53	**109**	**809**	40100	**552**	16300	681	4.9	**3470**	<0.3	133	69	**931**
	10-20 cm	41200	<0.4	7.1	345	**1.8**	1.73	51800	49	**82**	**415**	41400	**216**	19800	646	4.7	**2950**	<0.3	431	69	415
2292588 (Back yard)	0-5 cm	34000	<0.4	1.4	193	**1.5**	0.57	33900	38	30	103	31700	98	16400	576	4.3	**873**	<0.3	101	57	160
	5-10 cm	37300	<0.4	2.5	205	**1.6**	0.54	34400	39	29	91	34900	118	15800	746	4.1	675	<0.3	114	61	195
	10-20 cm	45800	<0.4	0.3	278	**2**	0.75	40800	67	27	60	40200	103	17300	788	4.5	373	<0.3	120	73	146
2292589 (Front yard)	0-5 cm	30100	<0.4	3.0	201	**1.3**	0.61	52500	34	35	96	29600	104	21600	574	4.5	**910**	<0.3	144	52	175
	5-10 cm	36700	<0.4	0.6	202	**1.6**	0.43	53600	39	20	54	33800	49	21000	585	4.1	**201**	<0.3	126	60	156
	10-20 cm	34200	<0.4	1.3	228	**1.5**	0.65	37900	38	48	129	38600	97	18400	806	4.1	**1540**	<0.3	102	57	161

Table 2: Results of Chemical Analysis (µg/g) of 2000 Residential Soil Samples, Port Colborne

Al = aluminum, Sb = antimony, As = arsenic, Ba = barium, Be = beryllium, Cd = cadmium, Cr = chromium, Co = cobalt, Cu = copper, Fe = iron, Pb = lead, Mg = magnesium, Mn = manganese, Mo = molybdenum, Ni = nickel, Se = selenium, Sr = strontium, V = vanadium, Zn = zinc. Data in bold and underlined exceed MOE Table A effects-based guideline. na - data not available

Site / Location	Soil Depth	Al	Sb	As	Ba	Be	Cd	Ca	Cr	Co	Cu	Fe	Pb	Mg	Mn	Mo	Ni	Se	Sr	V	Zn
2292590 (Back yard)	0-5 cm	29800	<0.4	2.0	176	1.3	0.46	45500	32	21	69	29500	55	21900	516	4.4	357	<0.3	94	50	117
	5-10 cm	34400	<0.4	1.6	192	1.5	0.75	35800	38	22	108	31600	61	19200	452	4.1	380	<0.3	84	56	111
	10-20 cm	31900	<0.4	5.0	188	1.3	0.58	33000	37	32	134	29600	116	18600	487	4.1	1310	<0.3	75	52	159
2292591 (Front yard)	0-5 cm	16600	<0.4	17.8	220	1	2.25	27600	37	222	646	34300	237	13400	598	4.9	9760	3.90	63	41	747
	5-10 cm	29000	<0.4	16.1	272	1.4	1.76	35100	41	136	504	41100	203	15200	618	4.7	6640	0.30	83	55	580
	10-20 cm*	19975	1.4	27.1	204	1	1.55	26300	35	144	658	47625	222	10825	559	2.2	11825	2.08	63	48	473
2292592 (Back yard)	0-5 cm	33900	<0.4	11.5	353	1.6	1.28	24700	44	85	339	44100	250	13000	773	4.7	4740	<0.3	95	60	431
	5-10 cm	38400	<0.4	13.3	319	1.9	1.24	14900	47	80	329	43000	249	10700	461	4.4	5350	<0.3	98	67	353
	10-20 cm	47300	<0.4	8.3	294	2.1	0.58	17600	52	49	193	50800	94	14400	840	4.0	2610	<0.3	85	75	194
2292593 (Front yard)	0-5 cm	26400	<0.4	15.3	340	1.3	2.02	18800	41	144	452	42400	238	9330	641	4.4	6430	0.80	65	54	539
	5-10 cm	26700	<0.4	12.9	284	1.3	1.66	23100	40	159	428	42600	274	9990	710	4.3	5860	1.00	74	51	514
	0-5 cm	29700	<0.4	11	268	1	1.34	21000	41	129	325	40800	262	10200	636	4	4390	<0.3	72	57	418
	5-10 cm	26900	<0.4	15.5	349	1.3	1.55	22600	41	87	393	40900	231	9960	603	4.8	5110	<0.3	84	52	497
	5-10 cm	24700	<0.4	24.1	449	1.2	2.95	24800	50	146	722	55000	420	9700	716	5.3	9030	2.60	82	49	936
	5-10 cm	30500	<0.4	14.6	370	1.4	2.34	22800	47	82	398	42600	326	11200	600	4.3	4340	<0.3	71	57	648
	10-20 cm*	23800	4.5	45.2	517	1.4	35.33	23313	50	159	890	56050	771	8583	712	3.0	12350	4.78	105	51	945
	10-20 cm	26400	<0.4	27.2	512	1.4	3.31	22700	52	121	653	48600	553	9040	624	5.3	8530	2.20	102	51	960
	10-20 cm	39600	<0.4	5	290	2	1.17	29500	47	38	144	39500	170	13600	720	4	1420	<0.3	87	66	300
2292594 (Back yard)	0-5 cm	15000	<0.4	23.8	452	1	2.62	21500	42	172	640	38600	603	7530	542	4.8	8470	6.40	93	37	1020
	0-5 cm	15800	<0.4	26.0	440	1.2	2.56	21300	43	163	645	39700	614	6790	539	5.1	7940	5.00	121	38	1060
	0-5 cm*	15025	1.6	30.5	323	1	1.54	20350	37	177	632	39575	427	6530	590	2.5	8473	5.13	98	39	769
	5-10 cm*	15675	2.1	43.0	417	1	1.73	22350	40	144	776	47450	674	7795	545	2.7	10950	6.25	96	37	1230
	5-10 cm	18000	<0.4	35.6	464	1.3	3.33	21300	44	171	823	52100	750	6570	561	5.2	13400	5.80	127	37	1160
	5-10 cm*	18325	1.0	36.0	375	1.1	1.91	19900	41	139	772	48800	582	7550	622	3.4	10650	5.61	93	39	981
	10-20 cm*	14675	1.5	29.7	329	1	1.94	22925	39	86	526	45975	549	6853	498	4.1	6855	3.78	107	34	1075
	10-20 cm*	17775	1.1	26.6	342	1	1.61	21975	36	86	629	40450	512	6600	466	3.3	7448	3.76	112	38	849
	10-20 cm*	17950	0.9	32.9	348	1.1	1.60	22125	37	102	605	44075	546	7010	532	3.5	8540	4.00	115	37	866

Table 2: Results of Chemical Analysis (μg/g) of 2000 Residential Soil Samples, Port Colborne

Al = aluminum, Sb = antimony, As = arsenic, Ba = barium, Be = beryllium, Cd = cadmium, Ca = calcium, Cr = chromium, Co = cobalt, Cu = copper, Fe = iron, Pb = lead, Mg = magnesium, Mn = manganese, Mo = molybdenum, Ni = nickel, Se = selenium, Sr = strontium, V = vanadium, Zn = zinc. Data in bold and underlined exceed MOE Table A effects-based guideline. na - data not available

Site / Location	Soil Depth	Al	Sb	As	Ba	Be	Cd	Ca	Cr	Co	Cu	Fe	Pb	Mg	Mn	Mo	Ni	Se	Sr	V	Zn
2292595 (Front yard)	0-5 cm*	13200	0.4	6.3	85	0.6	0.47	7518	18	33	103	17975	78	4195	417	2.1	1213	0.66	31	31	150
	5-10 cm	14900	<0.4	10.4	174	0.9	0.86	13700	25	54	233	23300	199	5710	435	3.8	2730	<0.3	64	34	300
	10-20 cm	12500	<0.4	12.0	143	0.8	0.71	23000	25	50	262	21500	167	6890	353	3.9	3170	0.60	72	29	283
2292596 (Back yard)	0-5 cm	14600	<0.4	2.9	93	0.8	0.24	26800	18	52	66	16300	37	10500	373	3.8	550	<0.3	75	33	135
	5-10 cm	15400	<0.4	2.3	94	0.9	0.23	30200	18	37	61	16700	37	11300	367	3.7	547	<0.3	84	34	104
	10-20 cm	14100	<0.4	4	94	1	0.24	34000	19	60	68	16100	39	12000	406	4	675	<0.3	96	34	100
2292597 (Front yard)	0-5 cm	19000	<0.4	13.5	235	1.2	1.67	19900	40	63	301	33100	409	6720	508	4.6	3460	0.60	95	37	539
	5-10 cm	19800	1.0	16.9	245	1.4	1.78	21500	40	64	328	33900	368	6860	540	4.5	3590	0.70	106	38	530
	10-20 cm	16800	<0.4	24.2	256	1.5	2.14	22200	39	85	428	42000	363	6220	566	5.3	5940	2.43	143	35	587
2292598 (Back yard)	0-5 cm	12300	0.8	9.7	138	0.7	1.72	14300	45	46	180	21500	210	4920	303	3.9	2190	0.40	60	26	323
	5-10 cm	12900	1.7	13.6	158	0.8	1.86	17700	44	51	207	20600	364	5330	326	4.1	2440	0.64	67	27	336
	10-20 cm	12500	0.7	16.3	213	1.2	1.79	18800	36	56	257	22300	289	5160	335	4.4	3000	0.70	145	28	400
2292599 (Front yard)	0-5 cm*	13800	<0.4	8.1	87	0.6	0.99	11800	21	43	156	22500	111	5730	406	3.6	2140	<0.3	34	28	245
	5-10 cm*	13850	0.4	10.6	79	0.5	0.78	9158	20	40	151	20825	93	4440	367	2.3	1963	0.60	28	29	214
	10-20 cm	14600	<0.4	10.9	77	0.7	1.04	8890	23	48	212	25400	98	4340	403	3.5	2990	0.30	29	27	235
2292600 (Back yard)	0-5 cm	13300	<0.4	7.8	75	0.5	0.96	6900	25	41	138	17600	118	3250	258	3.1	1750	<0.3	33	27	268
	5-10 cm	13600	<0.4	3.8	48	0.5	0.56	3970	16	21	67	15000	58	2400	200	2.6	837	0.30	24	26	129
	10-20 cm	14100	6.4	6.7	71	0.6	1.10	4930	20	36	135	20800	101	2770	273	3.0	1540	<0.3	24	26	205
2292601 (Front yard)	0-5 cm	4580	<0.4	1.3	22	0.2	0.18	18000	8	5	8	8820	8	5380	240	3.2	48	<0.3	34	16	30
	5-10 cm	4880	<0.4	2.7	19	0.3	0.18	19400	9	5	6	10100	6	5480	236	3.3	38	<0.3	37	19	23
	10-20 cm	32100	0.7		134	1.2	0.18	6520	40	16	8	27100	35	8050	220	3.3	38	<0.3	73	63	74
2292602 (Back yard)	0-5 cm	5970	<0.4	4.1	61	0.3	0.71	19100	13	30	99	12400	74	5960	286	3.6	1040	<0.3	60	20	195
	5-10 cm	12900	0.7	22.5	304	0.8	2.82	19100	31	85	486	37100	334	6430	465	4.5	6240	4.90	81	30	990
	10-20 cm	18600	0.7	28.3	230	1.1	2.81	32000	33	80	2720	41000	355	8820	474	5.1	7410	3.78	113	36	1210
2292603 (Front yard)	0-5 cm	24700	<0.4	12.2	191	1.2	1.53	16100	32	58	264	27700	146	7860	430	4.5	2860	0.69	72	45	310
	5-10 cm	26600	<0.4	11.2	185	1.3	1.41	13900	33	50	236	27300	115	7570	405	4.3	2560	<0.3	68	47	260
	10-20 cm	18000	<0.4	30.3	221	1.1	2.23	15700	38	101	612	48500	206	6740	682	5.0	7920	3.90	69	34	546

Table 2: Results of Chemical Analysis (µg/g) of 2000 Residential Soil Samples, Port Colborne

Al = aluminum, Sb = antimony, As = arsenic, Ba = barium, Be = beryllium, Cd = cadmium, Ca = calcium, Cr = chromium, Co = cobalt, Cu = copper, Fe = iron, Pb = lead, Mg = magnesium, Mn = manganese, Mo = molybdenum, Ni = nickel, Se = selenium, Sr = strontium, V = vanadium, Zn = zinc.   Data in bold and underlined exceed MOE Table A effects-based guideline. na - data not available

Site / Location	Soil Depth	Al	Sb	As	Ba	Be	Cd	Ca	Cr	Co	Cu	Fe	Pb	Mg	Mn	Mo	Ni	Se	Sr	V	Zn
2292604 (Back yard)	0-5 cm	5630	<0.4	4.1	39	0.3	0.43	20000	11	13	39	11500	43	6580	249	3.7	419	<0.3	54	21	82
	5-10 cm	6580	0.4	9.5	95	0.5	0.96	17800	14	30	136	14800	163	5230	280	4.0	1500	0.56	81	20	232
	10-20 cm	17400	6.8	41.2	533	1.8	4.31	19700	49	153	791	60200	1800	5700	722	6.2	10300	5.75	190	38	1350
2292605 (Front yard)	0-5 cm	16700	<0.4	20.2	232	1	2.53	35500	35	90	491	37200	389	16200	622	5.7	5700	2.80	124	34	667
	0-5 cm	18100	<0.4	18.1	254	1.1	2.71	28400	35	81	444	35700	327	12800	599	5.4	4890	2.66	114	36	620
	0-5 cm	15500	<0.4	20.9	206	0.9	2.83	26300	34	88	502	37400	351	11900	593	5.2	5780	3.11	95	32	662
	5-10 cm	21500	<0.4	34.1	235	1.2	3.33	22700	44	138	739	57400	372	10200	799	6.7	9570	4.01	89	40	766
	5-10 cm	21200	<0.4	25.5	240	1.3	2.58	24600	37	113	610	48100	313	10700	718	5.3	7820	2.90	91	40	740
	5-10 cm	19300	<0.4	33.2	218	1.2	3.22	20800	40	145	799	58000	376	9520	818	5.4	9520	4.98	85	36	844
	10-20 cm	18000	<0.4	40.8	265	1.2	3.37	24900	38	134	888	59400	392	9040	836	6.0	10800	5.03	90	32	794
	10-20 cm	18000	<0.4	31.9	216	1.2	2.74	24300	35	113	716	48000	306	9540	719	5.3	8160	3.47	89	36	758
	10-20 cm	18100	<0.4	28.0	212	1.1	3.04	22700	34	107	654	48200	333	9670	725	5.3	8230	3.81	94	35	686
2292606 (Back yard)	0-5 cm	19000	<0.4	16.7	250	1.1	1.92	25700	34	88	391	36500	284	10200	1280	5.8	4230	2.20	134	36	519
	0-5 cm	20000	0.5	18.8	194	1.2	1.84	13700	35	96	392	35400	196	6800	680	4.7	4710	2.76	91	43	433
	0-5 cm	21400	0.8	22.3	241	1.3	3.01	14700	39	101	447	36800	269	6230	702	5.0	4910	3.42	114	43	537
	5-10 cm	22300	3.1	22.0	307	1.2	2.54	13500	46	96	465	45200	408	6480	671	5.1	5700	2.79	82	42	666
	5-10 cm	26500	0.5	24.5	224	1.4	1.97	14300	43	100	504	48900	220	7660	750	4.8	6450	1.98	78	45	548
	5-10 cm	23100	1.2	24.8	253	1.4	2.33	13700	43	113	554	50200	241	6500	964	5.1	7110	2.51	103	42	604
	10-20 cm	22400	1.9	29.0	252	1.3	2.38	16400	46	113	559	53900	320	7430	766	5.4	7830	3.72	87	41	689
	10-20 cm	27700	0.6	33.4	267	1.5	2.17	19600	49	99	541	53700	252	9160	847	5.1	7690	3.00	99	47	583
	10-20 cm	23300	0.4	32.1	271	1.5	2.24	17500	46	113	567	55500	309	7560	836	5.6	8190	3.76	120	42	599
2292608 (Back yard)	0-5 cm	13500	<0.4	19.0	131	0.8	1.88	15100	27	90	339	35100	185	5410	628	4.6	4140	2.66	60	27	450
	5-10 cm	12600	0.6	24.7	119	0.7	1.93	16000	30	90	378	42900	88	5180	715	4.5	4780	2.11	56	25	442
	10-20 cm	12900	0.7	20.5	102	0.7	1.73	16800	25	73	338	35500	157	5370	591	4.6	4560	1.34	56	26	364
2292609 (Back yard)	0-5 cm	22900	<0.4	2.9	125	1	0.54	7170	30	23	68	22900	58	5840	415	3.4	670	<0.3	66	41	140
	5-10 cm	28800	<0.4	1.1	143	1.2	0.43	5680	33	20	44	26600	47	6690	433	3.1	392	<0.3	37	50	106
	10-20 cm	36200	<0.4	1.9	177	1.5	0.47	6640	42	23	47	33700	54	8330	500	3.7	390	<0.3	52	61	124

Table 2: Results of Chemical Analysis (μg/g) of 2000 Residential Soil Samples, Port Colborne

Al = aluminum, Sb = antimony, As = arsenic, Ba = barium, Be = beryllium, Cd = cadmium, Ca = calcium, Cr = chromium, Co = cobalt, Cu = copper, Fe = iron, Pb = lead, Mg = magnesium, Mn = manganese, Mo = molybdenum, Ni = nickel, Se = selenium, Sr = strontium, V = vanadium, Zn = zinc. Data in bold and underlined exceed MOE Table A effects-based guideline. na - data not available

Site / Location	Soil Depth	Al	Sb	As	Ba	Be	Cd	Ca	Cr	Co	Cu	Fe	Pb	Mg	Mn	Mo	Ni	Se	Sr	V	Zn
2292610 (Front yard)	0-5 cm	14800	<0.4	6.0	100	0.5	0.51	6390	17	23	69	16300	73	3380	241	6.3	**685**	<0.3	25	25	152
	5-10 cm	14700	<0.4	5.5	94	0.4	0.44	5250	17	22	62	14500	61	3050	237	6.2	**708**	<0.3	21	25	123
	10-20 cm	16600	0.4	11.0	135	0.6	0.76	8280	22	36	156	21800	122	4630	390	8.2	**1490**	<0.3	30	30	217
2292611 (Back yard)	0-5 cm	16400	0.5	9.9	146	0.6	0.90	8910	22	30	110	22700	123	4920	482	8.3	**808**	<0.3	33	30	232
	5-10 cm	17700	<0.4	11.0	161	0.7	0.86	10100	24	32	109	23400	129	5500	491	8.8	**820**	<0.3	36	32	246
	10-20 cm	18600	1.2	13.2	180	0.8	1.29	14900	30	40	153	27700	190	6590	487	10.3	**1190**	<0.3	51	35	341
2292612 (Front yard)	0-5 cm	17100	0.7	12.2	141	0.8	0.75	23400	26	47	144	27200	137	12100	547	10.6	**1380**	<0.3	57	35	233
	5-10 cm	19300	0.9	12.5	150	1	0.79	28600	29	48	150	29700	137	15800	626	11.8	**1370**	<0.3	66	37	238
	10-20 cm	19300	0.8	12.0	132	0.9	0.73	29100	29	47	144	31500	101	15200	678	11.7	**1470**	<0.3	60	36	203
2292613 (Back yard)	0-5 cm	20700	0.9	10.5	158	1	0.98	21100	30	40	123	27400	118	11400	556	11.0	**1030**	<0.3	54	39	222
	5-10 cm	21700	0.9	9.8	159	1	0.95	28600	29	40	115	28200	112	15200	593	11.6	**936**	<0.3	61	40	210
	10-20 cm	23500	0.5	9.6	150	1	0.78	33200	31	39	100	29800	79	16300	630	12.1	**835**	<0.3	66	42	166
2292614 (Side yard)	0-5 cm	17500	1.4	17.4	131	0.9	0.87	19900	27	**56**	210	30600	116	8560	512	10.6	**2270**	1.00	59	34	218
	5-10 cm	21700	1.1	17.8	151	1	0.77	24500	33	**57**	202	32600	108	10100	548	11.8	**2080**	<0.3	64	41	211
	10-20 cm	21300	1.5	17.8	154	1	0.96	20600	30	**57**	210	34000	121	8720	599	11.6	**2190**	0.80	67	39	216
2292615 (Front yard)	0-5 cm	13600	<0.4	7.6	95	0.6	3.13	16900	25	25	58	18800	105	7920	488	8.1	**430**	<0.3	51	31	140
	0-5 cm	13500	<0.4	5.8	95	0.5	0.94	19600	21	24	56	17200	83	8780	434	7.9	**418**	<0.3	50	30	124
	0-5 cm	16500	1.1	6.9	125	0.7	1.33	18300	26	29	67	21500	97	9370	501	9.2	**503**	<0.3	62	35	159
	5-10 cm	14000	<0.4	5.6	98	0.6	0.78	20200	21	25	49	19300	73	8550	491	8.4	**330**	<0.3	55	32	119
	5-10 cm	10700	<0.4	3.6	63	0.4	0.43	18900	17	19	37	14500	55	9010	316	7.0	**277**	<0.3	45	28	85
	5-20 cm	13500	<0.4	6.3	98	0.6	0.84	17500	21	25	60	19200	83	8460	471	8.2	**412**	<0.3	69	31	133
	10-20 cm	19900	0.5	7.6	139	0.9	0.75	22700	28	31	60	25100	75	9160	621	10.3	**406**	<0.3	79	40	136
	10-20 cm	17500	<0.4	7.1	123	0.8	0.61	22500	26	28	60	22200	93	10400	474	9.7	**402**	<0.3	78	38	147
	10-20 cm	22900	0.5	7.1	147	1	0.83	15400	31	33	60	27200	77	9950	679	10.8	**412**	<0.3	64	45	133

Table 2: Results of Chemical Analysis (µg/g) of 2000 Residential Soil Samples, Port Colborne

Al = aluminum, Sb = antimony, As = arsenic, Ba = barium, Be = beryllium, Cd = cadmium, Ca = calcium, Cr = chromium, Co = cobalt, Cu = copper, Fe = iron, Pb = lead, Mg = magnesium, Mn = manganese, Mo = molybdenum, Ni = nickel, Se = selenium, Sr = strontium, V = vanadium, Zn = zinc. Data in bold and underlined exceed MOE Table A effects-based guideline. na - data not available

Site / Location	Soil Depth	Al	Sb	As	Ba	Be	Cd	Ca	Cr	Co	Cu	Fe	Pb	Mg	Mn	Mo	Ni	Se	Sr	V	Zn
2292616 (Back yard)	0-5 cm	11000	<0.4	6.5	76	0.4	1.26	13700	18	22	59	15800	103	6280	467	7.0	352	<0.3	36	26	134
	0-5 cm	13300	<0.4	7.7	96	0.5	0.77	12000	20	25	68	17900	119	6300	521	7.7	396	<0.3	40	29	164
	0-5 cm	12600	0.4	7.6	79	0.4	0.81	13400	18	24	67	16500	111	6530	481	7.2	418	<0.3	37	27	162
	5-10 cm	9880	<0.4	6.9	78	0.4	0.88	20500	15	23	72	15500	116	7740	361	7.1	446	<0.3	51	25	161
	5-10 cm	11600	<0.4	6.3	86	0.4	0.91	15200	16	22	61	16400	145	7250	460	7.2	351	<0.3	41	26	175
	5-10 cm	11100	0.4	6.6	74	0.4	1.26	15600	15	24	68	15600	123	7180	394	7.1	426	<0.3	40	25	282
	10-20 cm	11300	0.6	7.6	98	0.4	0.87	22700	18	27	88	18500	140	9150	379	8.0	582	<0.3	66	29	192
	10-20 cm	12600	<0.4	6.9	113	0.5	1.03	19800	18	26	76	17400	131	8690	423	7.9	479	<0.3	71	27	246
	10-20 cm	8070	<0.4	5.2	56	0.3	1.39	16800	13	21	54	13600	95	7390	271	6.5	364	<0.3	38	23	408
2292617 (Front yard)	0-5 cm	16900	1.4	16.4	177	0.9	1.43	24200	31	57	238	29700	316	10400	615	11.7	1920	0.60	84	34	361
	5-10 cm	17900	<0.4	15.3	158	1.1	1.72	19200	36	50	259	33700	251	8440	646	4.7	2630	1.40	70	36	401
	10-20 cm	19800	<0.4	15.7	160	1.2	1.56	19200	32	46	251	32900	196	8930	638	4.6	2430	0.90	65	37	316
2292618 (Front yard)	0-5 cm	13900	<0.4	2.4	83	0.7	0.66	26700	24	13	43	19100	64	10100	487	4.4	107	<0.3	60	28	131
	5-10 cm	15700	<0.4	2.3	94	0.8	0.56	29500	32	16	42	21500	67	10000	567	4.7	101	<0.3	67	31	117
	10-20 cm	na	na	na	na	na	na	na	na	na	na	na	na	na	na	na	na	na	na	na	na
2292619 (Back yard)	0-5 cm	13500	<0.4	2.7	79	0.7	0.72	27200	23	14	47	18700	69	10300	519	4.3	196	<0.3	56	27	132
	5-10 cm	13900	<0.4	4.1	82	0.7	0.69	30800	21	12	37	19000	51	11100	514	4.3	97	<0.3	61	29	112
	10-20 cm	na	na	na	na	na	na	na	na	na	na	na	na	na	na	na	na	na	na	na	na
2292620 (Front yard)	0-5 cm	10100	<0.4	5.8	149	0.6	1.16	18400	25	22	81	21300	254	7560	582	4.2	985	<0.3	42	25	267
	5-10 cm	10900	0.4	4.8	167	0.6	1.07	14400	26	18	64	21200	324	6590	605	3.8	591	<0.3	33	25	233
	10-20 cm	10900	3.6	15.9	389	0.9	2.36	24000	43	64	290	43100	769	9070	680	5.3	5940	0.80	82	24	591
2292621 (Back yard)	0-5 cm	15900	<0.4	4.1	87	0.8	0.47	20000	22	14	45	18000	82	10100	362	3.9	298	<0.3	35	32	135
	5-10 cm	17700	<0.4	2.4	96	0.8	0.44	24400	23	13	38	19000	77	11200	377	4.1	228	<0.3	36	34	127
	10-20 cm	20500	<0.4	2.3	98	1	0.39	17900	25	12	31	21900	40	8760	440	3.7	184	<0.3	31	39	100
2292622 (Front yard)	0-5 cm	18400	0.7	3.8	111	1	0.65	19500	26	19	77	21900	115	10600	443	4.3	450	<0.3	42	34	173
	5-10 cm	19400	<0.4	4.3	117	1	0.58	21100	28	18	61	22200	92	11600	544	4.1	367	<0.3	40	36	145
2292623 (Back yard)	0-5 cm	15400	<0.4	3.4	90	0.8	0.58	15400	22	16	53	18300	97	7800	349	3.8	341	<0.3	33	31	163
	5-10 cm	15400	<0.4	4.4	96	0.8	0.66	16700	22	16	55	18400	122	7960	348	3.8	381		35	32	186
	10-20 cm	9150	<0.4	2.5	55	0.5	0.39	22900	14	10	31	12700	96	7340	266	3.8	199	<0.3	35	22	119

Table 2: Results of Chemical Analysis (µg/g) of 2000 Residential Soil Samples, Port Colborne

Al = aluminum, Sb = antimony, As = arsenic, Ba = barium, Be = beryllium, Cd = cadmium, Ca = calcium, Cr = chromium, Co = cobalt, Cu = copper, Fe = iron, Pb = lead, Mg = magnesium, Mn = manganese, Mo = molybdenum, Ni = nickel, Se = selenium, Sr = strontium, V = vanadium, Zn = zinc.   Data in bold and underlined exceed MOE Table A effects-based guideline. na - data not available

Site / Location	Soil Depth	Al	Sb	As	Ba	Be	Cd	Ca	Cr	Co	Cu	Fe	Pb	Mg	Mn	Mo	Ni	Se	Sr	V	Zn
2292624 (Front yard)	0-5 cm	23000	6.1	22.0	208	1.3	2.13	27600	37	70	383	41400	332	9140	687	5.2	4100	2.40	85	40	427
	5-10 cm	25200	5.8	22.9	220	1.4	2.01	30000	34	61	347	41900	286	9260	680	4.8	4010	2.30	89	42	377
	10-20 cm	21900	6.0	19.9	213	1.3	1.86	32800	28	53	334	37800	279	8220	651	4.9	3800	2.10	102	37	359
2292625 (Back yard)	0-5 cm	29100	5.1	18.6	234	1.3	2.18	26400	38	41	190	34700	289	12300	626	4.8	1750	<0.3	133	49	330
	5-10 cm	31000	5.6	17.1	230	1.3	1.35	29600	39	38	221	35900	302	13600	687	4.7	1580	<0.3	146	51	287
	10-20 cm	32700	6.2	12.0	249	1.5	1.48	29500	39	44	197	37000	212	13200	617	5.0	1730	<0.3	160	53	299
2292626 (Side yard)	0-5 cm	21600	5.2	12.9	174	1	1.16	17700	29	37	162	26400	197	8470	403	4.1	1610	0.40	77	38	222
	5-10 cm	27300	5.5	14.9	213	1.2	1.36	21400	35	47	203	33500	254	10200	488	4.7	2090	0.40	95	46	273
	10-20 cm	24700	5.5	20.6	218	1.2	1.60	24200	34	48	218	31100	290	10000	440	4.5	2190	0.90	126	43	299
2292627 (Front yard)	0-5 cm	21600	5.6	15.9	189	1	1.44	20500	33	76	296	32300	263	9080	470	4.8	2850	1.10	82	39	332
	5-10 cm	21900	5.5	20.5	172	1.1	1.51	19400	30	62	275	31200	179	7280	416	4.4	2890	2.40	84	39	262
	10-20 cm	30400	5.9	18.9	205	1.4	1.60	21000	38	64	302	40100	160	8760	540	4.7	3480	1.60	92	52	278
2292628 (Back yard)	0-5 cm	22100	4.7	13.2	178	1.1	1.30	25500	35	54	237	31200	198	11200	554	4.8	2400	0.90	108	42	325
	5-10 cm	31600	5.0	7.9	192	1.3	0.83	15100	39	36	144	31600	124	9300	561	4.4	1450	<0.3	73	53	238
	10-20 cm	22700	6.5	10.1	149	1	0.85	25800	36	35	180	29900	146	10200	954	5.1	1870	0.60	73	38	342
2292629 (Front yard)	0-5 cm	17500	4.6	28.2	200	1.1	2.47	19000	35	193	676	43900	291	7370	766	5.1	7870	8.60	81	38	592
	0-5 cm	13300	5.4	36.1	204	0.9	2.91	16200	43	209	780	65800	373	5960	865	5.9	10600	10.00	64	32	794
	0-5 cm	12000	6.2	30.8	195	0.9	2.73	16500	36	181	674	53300	407	5810	758	5.2	9460	9.90	65	30	733
	5-10 cm	16500	5.7	42.6	197	1	3.03	14100	37	150	734	55800	345	5610	760	4.9	9860	8.50	57	31	721
	5-10 cm	17600	6.5	57.8	254	1.2	4.30	17300	53	262	1100	83300	459	6350	1050	6.4	15500	19.40	71	31	1150
	5-10 cm	15700	6.1	44.6	253	1.1	3.57	15700	47	218	949	71000	421	5770	876	5.9	13100	14.20	66	32	934
	10-20 cm	19300	5.5	29.4	172	1.1	2.03	15700	29	75	450	38300	211	5670	572	4.2	5720	4.30	60	40	399
	10-20 cm	13600	4.5	11.0	101	0.8	0.85	11200	22	34	182	29700	92	4930	353	3.4	2600	0.90	37	41	188
	10-20 cm	10700	4.3	18.2	109	0.7	1.52	12200	22	62	304	33900	185	4990	441	4.0	4330	3.80	39	31	348

Table 2: Results of Chemical Analysis (µg/g) of 2000 Residential Soil Samples, Port Colborne

Al = aluminum, Sb = antimony, As = arsenic, Ba = barium, Be = beryllium, Cd = cadmium, Ca = calcium, Cr = chromium, Co = cobalt, Cu = copper, Fe = iron, Pb = lead, Mg = magnesium, Mn = manganese, Mo = molybdenum, Ni = nickel, Se = selenium, Sr = strontium, V = vanadium, Zn = zinc. Data in bold and underlined exceed MOE Table A effects-based guideline. na - data not available

Site / Location	Soil Depth	Al	Sb	As	Ba	Be	Cd	Ca	Cr	Co	Cu	Fe	Pb	Mg	Mn	Mo	Ni	Se	Sr	V	Zn
2292630 (Back yard)	0-5 cm	14600	4.5	10.1	141	0.8	1.33	20900	23	36	163	24800	156	6850	457	4.2	**1650**	0.80	63	29	349
	0-5 cm	10300	4.5	8.2	121	0.6	1.16	22400	21	32	129	21500	188	7510	334	4.0	**1410**	0.80	59	24	322
	0-5 cm	13300	6.0	10.9	164	0.8	1.66	22300	25	39	172	23200	**244**	7870	401	4.3	**1580**	1.00	75	27	447
	5-10 cm	15900	6.0	17.1	264	1	1.92	24900	28	44	257	31900	**351**	7540	443	4.6	**2750**	2.70	87	30	615
	5-10 cm	15700	7.5	22.4	295	1.1	2.78	27700	33	**61**	**402**	38500	**448**	8190	533	4.8	**3780**	2.40	117	31	751
	5-10 cm	13600	6.1	14.8	182	0.9	1.89	26200	27	**51**	246	32000	**298**	8220	435	4.0	**2720**	1.90	79	30	492
	10-20 cm	19600	6.1	23.1	219	1.2	2.11	31200	31	44	274	35300	**252**	10100	526	4.7	**2800**	1.80	101	37	492
	10-20 cm	17400	8.3	21.9	281	1.2	2.12	29400	32	**55**	**337**	39800	**612**	8150	545	4.8	**3830**	1.80	126	32	625
	10-20 cm	19300	6.6	23.7	383	**1.6**	2.35	31800	34	48	**358**	37700	**498**	8320	548	4.8	**3200**	1.50	165	37	692
2292631 (Front yard)	0-5 cm	20800	4.0	5.4	88	0.7	0.52	4960	23	22	70	21100	63	3970	311	3.0	**914**	<0.3	24	35	123
	5-10 cm	20800	3.5	5.7	89	0.7	0.56	5840	23	22	75	22000	63	4280	329	3.3	**927**	<0.3	23	36	125
	10-20 cm	23100	4.4	13.0	137	1	0.96	12000	30	36	164	28800	113	6120	423	4.0	**2120**	1.10	38	40	197
2292632 (Back yard)	0-5 cm	37700	4.6	2.9	171	1.3	0.64	8990	39	21	67	27200	84	6940	306	3.6	**545**	<0.3	51	55	152
	5-10 cm	27800	<0.4	8.7	174	1.2	0.98	11900	37	32	118	28900	125	7080	379	3.8	**1140**	<0.3	55	51	228
	10-20 cm	20000	5.3	17.7	164	0.9	1.47	15900	30	45	200	32100	200	6840	487	4.1	**2290**	0.60	59	37	295
2292633 (Front yard)	0-5 cm	26300	4.4	8.5	187	1.2	1.12	14600	33	36	160	28600	190	6580	451	4.1	**1710**	<0.3	59	45	272
	5-10 cm	28200	5.0	10.4	225	**1.3**	1.26	15900	35	41	190	32300	**226**	7470	529	4.2	**2170**	<0.3	63	47	307
	10-20 cm	28800	5.7	11.7	220	**1.3**	1.23	31300	34	39	212	35400	**202**	10500	587	4.7	**2450**	<0.3	85	47	305
2292634 (Back yard)	0-5 cm	28000	4.6	8.3	182	1.2	1.24	12100	35	40	170	30900	**276**	6490	558	4.0	**1740**	<0.3	45	48	304
	5-10 cm	27600	5.3	6.2	165	1.2	0.95	10400	32	33	139	28500	150	6570	539	3.7	**1450**	<0.3	38	46	224
	10-20 cm	30000	5.7	9.5	208	**1.4**	1.09	23100	41	40	208	34400	190	10500	5620	5.1	**2040**	<0.3	60	53	288
2292635 (Front yard)	0-5 cm	30000	5.5	8.0	180	**1.3**	1.14	14000	35	41	172	34200	132	7780	525	4.1	**1990**	<0.3	53	50	240
	5-10 cm	30900	5.1	8.9	187	**1.3**	1.06	13500	35	38	172	34300	134	7910	548	4.2	**2030**	<0.3	52	51	221
	10-20 cm	31200	5.6	11.1	212	**1.4**	1.12	22600	39	44	225	38900	160	9880	597	4.8	**2790**	<0.3	65	51	260
2292636 (Front yard)	0-5 cm	19000	5.2	13.8	151	0.8	1.17	23800	27	45	225	31400	198	10800	490	4.6	**3030**	0.80	58	34	321
	5-10 cm	17800	5.2	15.2	143	0.8	1.15	27300	26	46	229	32600	192	11600	540	4.6	**3140**	1.10	61	32	302
	10-20 cm	17700	5	17	151	1	1.32	35600	27	**51**	259	35000	181	14100	604	5	**3590**	1	68	32	321
2292637 (Front yard)	0-5 cm	21600	5.1	14.5	188	1	1.64	16700	35	**63**	267	39100	**248**	8270	607	4.5	**3830**	1.30	56	38	418
	5-10 cm	20400	6.6	**27.1**	203	1	2.16	16300	37	**85**	**385**	49500	**302**	7780	688	4.7	**6420**	3.6	49	34	521
	10-20 cm	20300	6.1	19.4	186	1	1.58	14900	31	**54**	254	35900	**223**	7010	562	4.3	**3650**	1.40	47	37	341

Table 2: Results of Chemical Analysis (µg/g) of 2000 Residential Soil Samples, Port Colborne

Al = aluminum, Sb = antimony, As = arsenic, Ba = barium, Be = beryllium, Cd = cadmium, Ca = calcium, Cr = chromium, Co = cobalt, Cu = copper, Fe = iron, Pb = lead, Mg = magnesium, Mn = manganese, Mo = molybdenum, Ni = nickel, Se = selenium, Sr = strontium, V = vanadium, Zn = zinc.   Data in bold and underlined exceed MOE Table A effects-based guideline. na - data not available

Site / Location	Soil Depth	Al	Sb	As	Ba	Be	Cd	Ca	Cr	Co	Cu	Fe	Pb	Mg	Mn	Mo	Ni	Se	Sr	V	Zn
2292638 (Back yard)	0-5 cm	17500	5.4	12.3	175	0.9	1.14	19800	31	47	205	35700	**263**	8210	521	4.5	**2850**	0.80	66	36	352
	5-10 cm	24100	8	15	232	1	1.22	21900	36	50	287	39500	**428**	9310	598	5	**2810**	0	81	44	374
	10-20 cm	24300	6.8	13.0	272	1.2	1.14	21500	36	43	207	37600	**406**	8510	491	4.4	**2630**	0.40	89	43	404
2292640 (Back yard)	0-5 cm	15300	<0.4	1.7	65	0.6	0.72	4990	17	17	51	14700	53	3120	226	2.7	**420**	<0.3	19	30	113
	5-10 cm	15700	<0.4	3.0	63	0.6	0.55	4380	16	17	47	14600	48	3050	224	2.6	**429**	<0.3	18	30	99
	10-20 cm	17800	<0.4	5.2	89	0.8	0.69	7890	22	25	85	17900	79	4710	341	3.3	**779**	<0.3	28	35	139
<T		1000	1	1	5	2.5	1	500	5	2.5	5	1000	10	250	25	2.5	2.5	1	5	5	25
≤W		200	0.2	0.2	1	0.5	0.2	100	1	0.5	1	200	2	50	5	0.5	0.5	0.2	1	1	5

<T = a measurable trace amount; interpret with caution

≤W = no measurable response; less than reported value

Table 3: Results of Chemical Analysis (µg/g) of 2001 Residential Soil Samples, Port Colborne

Al = aluminum, Sb = antimony, As = arsenic, Ba = barium, Be = beryllium, Cd = cadmium, Ca = calcium, Cr = chromium, Co = cobalt,
Cu = copper, Fe = iron, Pb = lead, Mg = magnesium, Mn = manganese, Mo = molybdenum, Ni = nickel, Se = selenium, Sr = strontium,
V = vanadium, Zn = zinc. Data in bold and underlined exceed MOE Table A effects-based guideline. na - data not available

Site/Location	Soil Depth	Al	Sb	As	Ba	Be	Cd	Ca	Cr	Co	Cu	Fe	Pb	Mg	Mn	Mo	Ni	Se	Sr	V	Zn
2292702 (vacant lot)	0-5 cm	19000	0.5	13	170	1	0.3	33000	40	25	120	28000	180	13000	530	1.2	990	1.3	100	40	200
	0-5 cm	20000	1.5	14	300	1	1	32000	36	23	120	28000	1600	13000	550	1.3	740	1.2	100	40	690
	5-10 cm	21000	0.6	12	170	1.1	0.3	36000	32	26	120	29000	110	13000	560	0.9	1000	1.6	120	42	160
	5-10 cm	21000	0.7	12	170	1.1	0.4	35000	29	24	97	28000	130	12000	540	0.6	880	1.4	100	42	170
	10-20 cm	22000	0.5	13	160	1.1	0.1	32000	42	27	100	30000	76	11000	510	0.6	1100	1.8	110	43	140
	10-20 cm	22000	0.5	11	160	1.1	0.3	35000	29	22	83	29000	70	12000	510	0.25	820	1.5	100	44	120
2292703 (front yard)	0-5 cm	13000	0.3	4.1	80	0.6	0.5	11000	18	8.7	23	14000	24	6000	300	0.25	46	0.3	50	29	67
	0-5 cm	12000	0.1	4	77	0.6	0.3	12000	17	7.4	23	14000	26	6100	290	0.25	49	0.4	50	28	67
	5-10 cm	14000	0.1	5.2	93	0.6	0.3	43000	19	8.4	21	18000	18	17000	430	0.25	31	0.1	100	32	62
	5-10 cm	14000	0.1	4.4	90	0.6	0.4	28000	20	8.1	19	18000	22	12000	400	0.8	31	0.1	77	32	60
	10-20 cm	18000	0.1	6.3	120	0.8	0.4	40000	23	11	21	24000	20	17000	560	0.25	27	0.1	130	40	66
	10-20 cm	16000	0.1	6	110	0.8	0.1	46000	21	9.7	20	22000	17	18000	470	0.25	27	0.1	130	36	61
2292704 (front yard)	0-5 cm	12000	0.1	3.4	69	0.5	0.1	8300	16	6.4	21	13000	21	4800	270	0.25	46	0.3	52	27	65
	0-5 cm	11000	0.1	3.2	70	0.5	0.4	10000	16	7	24	13000	21	5300	280	0.25	53	0.3	58	26	74
	5-10 cm	12000	0.1	3.7	79	0.5	0.3	28000	16	6.7	23	15000	21	12000	330	0.25	37	0.1	100	29	60
	5-10 cm	12000	0.1	3.6	74	0.5	0.5	25000	16	7.2	18	14000	21	10000	320	0.25	35	0.3	92	27	58
	10-20 cm	11000	0.1	4.3	93	0.5	0.1	64000	16	7.9	20	16000	17	24000	440	0.25	25	0.1	180	28	63
	10-20 cm	11000	0.1	4.1	92	0.5	0.1	67000	16	7.7	19	16000	11	25000	450	0.25	19	0.1	170	27	60
2292705 (front yard)	0-5 cm	12000	0.1	3.7	74	0.5	0.3	11000	17	7.8	26	14000	27	5400	280	0.6	50	0.3	45	27	74
	0-5 cm	12000	0.3	3.5	72	0.5	0.6	13000	17	7.6	27	14000	31	5900	280	0.25	46	0.3	51	28	76
	5-10 cm	12000	0.1	3.8	73	0.5	0.1	14000	16	7.3	19	14000	24	6600	280	0.25	35	0.3	60	26	57
	5-10 cm	13000	0.3	3.7	75	0.5	0.6	18000	17	7.3	18	14000	24	8300	300	0.25	34	0.3	67	28	56
	10-20 cm	11000	0.1	4.6	90	0.5	0.1	59000	16	7.9	20	17000	85	22000	440	0.25	21	0.1	140	28	60
	10-20 cm	12000	0.3	5.1	92	0.6	0.1	60000	17	7.9	19	17000	20	22000	440	0.25	22	0.1	160	29	60
2292706 (front yard)	0-5 cm	15000	1	10	140	0.8	1.2	20000	57	47	230	24000	150	9800	470	3.4	1400	1.8	54	40	440
	0-5 cm	14000	1.1	11	150	0.8	1.4	22000	37	42	220	23000	170	10000	460	3.3	1300	1.7	53	36	360
	5-10 cm	15000	1.3	12	140	0.9	1.4	22000	53	45	320	25000	130	10000	460	2.7	1700	1.8	52	38	320
	5-10 cm	16000	1	11	170	0.9	1.3	23000	33	42	250	27000	130	9900	500	2.2	1600	1.7	62	38	320
	10-20 cm	18000	1	15	210	1.1	0.9	26000	31	37	230	26000	120	12000	450	1.8	1600	2	67	41	300
	10-20 cm	15000	2.9	17	200	0.9	0.8	30000	25	37	390	26000	130	10000	440	0.25	2100	2.1	66	34	330
2292707 (back yard)	0-5 cm	9900	1.7	9.4	100	0.6	1.1	21000	22	31	160	20000	130	8900	360	1.1	1300	1.3	52	31	310
	0-5 cm	9500	0.9	6.7	82	0.5	0.9	18000	19	19	93	16000	93	7100	310	1.1	710	1	42	26	250
	5-10 cm	12000	1.6	12	120	0.7	1.2	24000	29	35	180	22000	140	9500	410	0.7	1400	1.5	66	33	320
	5-10 cm	11000	4.4	12	130	0.7	1.1	26000	22	30	170	20000	140	9300	380	0.25	1200	1.6	61	30	300
	10-20 cm	11000	2.3	18	190	0.8	1	28000	27	49	400	24000	180	9600	410	0.9	2700	2.7	100	32	400
	10-20 cm	11000	2.6	18	160	0.7	1.1	25000	25	52	340	24000	180	8800	390	1.6	2500	2.7	75	30	420

Soil Investigation and Human Health Risk Assessment for the Rodney Street Community, Port Colborne: March 2002

Table 3: Results of Chemical Analysis (µg/g) of 2001 Residential Soil Samples, Port Colborne

Al = aluminum, Sb = antimony, As = arsenic, Ba = barium, Be = beryllium, Cd = cadmium, Ca = calcium, Cr = chromium, Co = cobalt,
Cu = copper, Fe = iron, Pb = lead, Mg = magnesium, Mn = manganese, Mo = molybdenum, Ni = nickel, Se = selenium, Sr = strontium,
V = vanadium, Zn = zinc. Data in bold and underlined exceed MOE Table A effects-based guideline. na - data not available

Site/Location	Soil Depth	Al	Sb	As	Ba	Be	Cd	Ca	Cr	Co	Cu	Fe	Pb	Mg	Mn	Mo	Ni	Se	Sr	V	Zn
2292708 (front yard)	0-5 cm	9300	1.2	17	140	0.6	0.5	20000	22	36	180	25000	220	6400	480	0.8	1900	2.6	80	25	360
	0-5 cm	8900	1.7	13	130	0.5	0.6	19000	21	35	170	26000	190	6000	470	1	1800	2.3	73	25	330
	5-10 cm	9100	1.4	19	140	0.6	0.3	19000	21	36	180	26000	200	5600	460	1.2	1900	2.6	72	25	340
	5-10 cm	9400	1.6	14	150	0.6	0.6	19000	21	38	190	27000	210	5800	500	0.7	2000	2.5	79	26	360
	10-20 cm	8100	1.4	19	140	0.5	0.4	20000	18	33	170	25000	190	5800	420	0.6	1900	2.4	68	23	330
	10-20 cm	10000	1.5	15	180	0.6	0.6	22000	23	46	230	31000	240	6400	540	0.25	2500	2.3	84	27	420
2292709 (front yard)	0-5 cm	10000	1.1	14	100	0.6	0.4	24000	50	26	140	17000	170	8800	320	0.8	1400	2	61	26	270
	0-5 cm	9600	1	14	94	0.5	0.6	24000	60	23	130	16000	150	9000	320	0.7	1200	1.8	57	24	250
	5-10 cm	11000	1.2	16	100	0.6	0.6	23000	51	28	140	18000	170	8100	340	0.25	1500	1.9	51	28	260
	5-10 cm	11000	1	16	100	0.6	0.4	24000	56	29	170	18000	180	8600	330	0.25	1600	2	54	28	270
	10-20 cm	12000	0.7	11	110	0.6	0.8	19000	35	22	100	20000	150	6900	390	0.6	940	1.4	61	30	200
	10-20 cm	10000	0.7	13	95	0.6	0.6	20000	27	21	110	18000	140	7500	370	0.6	920	2	55	26	180
2292710 (back yard)	0-5 cm	12000	0.8	13	110	0.6	0.6	26000	46	20	100	17000	120	9400	300	0.6	980	1.4	63	27	220
	0-5 cm	12000	0.8	13	110	0.6	0.5	27000	42	20	100	17000	130	10000	310	0.7	980	1.4	65	29	230
	5-10 cm	14000	1.1	14	120	0.7	0.3	32000	58	22	120	20000	180	13000	350	0.25	1200	1.5	68	32	230
	5-10 cm	14000	0.7	16	120	0.7	0.3	32000	49	21	110	19000	130	13000	340	1	960	1.7	72	33	210
	10-20 cm	18000	0.5	10	130	0.8	0.4	29000	30	14	47	23000	74	12000	380	0.25	330	0.8	69	38	110
	10-20 cm	15000	0.4	9.8	100	0.7	0.1	36000	28	12	51	18000	71	16000	340	0.25	350	0.9	91	31	120
2292711 (side yard)	0-5 cm	19000	1.1	17	140	1	0.1	14000	27	77	320	21000	99	6900	410	0.25	3100	3.3	58	42	200
	0-5 cm	21000	1.1	19	150	1.1	1	14000	31	83	360	23000	110	7000	510	0.25	3500	3.4	63	43	220
	5-10 cm	26000	1	19	180	1.3	0.1	16000	33	62	300	24000	78	8900	380	0.25	3100	2.8	69	53	190
	5-10 cm	23000	1.1	22	160	1.3	0.7	14000	31	80	390	24000	140	7200	390	0.25	4200	4.2	71	47	200
	10-20 cm	21000	1.1	15	180	1.2	0.7	30000	41	63	250	27000	110	12000	600	2.9	2500	2.2	90	44	210
	10-20 cm	24000	1.4	23	190	1.3	0.6	26000	48	82	410	32000	150	12000	580	0.25	4500	3.3	96	50	260
2292712 (side yard)	0-5 cm	17000	3.2	15	140	0.9	0.4	14000	29	50	210	30000	140	7600	440	0.8	2500	2.6	60	40	270
	0-5 cm	17000	1.4	18	170	0.9	0.6	16000	41	62	270	34000	200	7400	500	1.1	3000	3.5	62	40	410
	5-10 cm	21000	1.6	22	180	1.2	0.5	15000	35	64	300	41000	180	7900	580	0.6	3600	3.6	67	48	360
	5-10 cm	20000	1.3	21	180	1.1	0.7	14000	36	60	280	38000	180	7700	620	0.7	3400	3.2	58	45	560
	10-20 cm	21000	1.3	20	170	1.1	0.6	13000	33	42	210	36000	120	7500	570	0.7	2400	3	55	44	240
	10-20 cm	22000	1.3	19	230	1.1	0.6	15000	38	44	330	35000	160	7800	520	0.7	2300	2.6	59	46	290
2292712 (front yard)	0-5 cm	7200	1.8	21	100	0.7	0.6	28000	24	120	420	52000	280	7500	820	1.5	4200	3.2	46	27	460
	0-5 cm	6400	1.7	21	100	0.7	0.8	30000	21	100	330	37000	340	7500	710	0.9	3300	3.2	46	25	420
	5-10 cm	6300	2.9	51	110	0.6	0.1	22000	35	180	680	110000	260	5800	890	2.2	8600	5.3	39	28	720
	5-10 cm	6700	1.6	28	93	0.6	0.3	25000	24	98	570	67000	260	6600	780	1.8	5800	2.9	43	29	570
	10-20 cm	3700	1	21	35	0.5	0.1	28000	9	24	200	22000	83	7400	340	0.25	1800	1.8	40	23	180
	10-20 cm	3500	0.4	8.3	30	0.5	0.1	29000	10	15	120	17000	58	7200	260	0.25	1100	0.8	39	23	100

Table 3: Results of Chemical Analysis (µg/g) of 2001 Residential Soil Samples, Port Colborne

Al = aluminum, Sb = antimony, As = arsenic, Ba = barium, Be = beryllium, Cd = cadmium, Ca = calcium, Cr = chromium, Co = cobalt,
Cu = copper, Fe = iron, Pb = lead, Mg = magnesium, Mn = manganese, Mo = molybdenum, Ni = nickel, Se = selenium, Sr = strontium,
V = vanadium, Zn = zinc. Data in bold and underlined exceed MOE Table A effects-based guideline. na - data not available

Site/Location	Soil Depth	Al	Sb	As	Ba	Be	Cd	Ca	Cr	Co	Cu	Fe	Pb	Mg	Mn	Mo	Ni	Se	Sr	V	Zn
2292713 (side yard)	0-5 cm	2600	0.8	17	29	0.5	0.1	40000	10	16	74	13000	41	8800	250	0.8	650	1.5	50	10	130
	0-5 cm	2900	0.5	10	31	0.5	0.4	41000	10	18	81	14000	60	8800	270	0.25	720	1.1	51	12	140
	5-10 cm	2800	1.1	21	26	0.5	0.1	42000	9	20	97	18000	54	8600	280	0.6	890	5.5	51	14	150
	5-10 cm	3500	0.8	16	40	0.5	0.1	45000	13	34	160	28000	86	8200	380	0.6	1600	1.8	53	16	220
	10-20 cm	2800	0.6	13	27	0.5	0.1	39000	9	20	92	19000	46	8100	290	0.6	890	1.2	47	18	130
	10-20 cm	3600	1.6	19	44	0.5	0.3	40000	13	37	200	29000	96	7700	400	0.9	1900	1.9	48	19	240
2292714 (back yard)	0-5 cm	3200	1.2	22	47	0.5	0.6	36000	12	34	130	26000	69	8200	400	1.2	1200	2.9	54	15	240
	0-5 cm	2900	1.4	18	60	0.5	0.3	36000	12	26	100	22000	160	8400	340	0.25	930	1.8	50	15	230
	5-10 cm	2800	0.9	21	46	0.5	0.4	36000	12	33	130	28000	66	8200	370	0.6	1200	2.4	49	16	220
	5-10 cm	2500	0.8	14	37	0.5	0.3	35000	9	25	100	24000	77	8000	310	0.6	1000	1.4	47	17	160
	10-20 cm	2200	0.7	16	24	0.5	0.1	34000	7	16	65	16000	27	7600	270	0.25	590	1.4	44	14	130
	10-20 cm	2300	0.6	12	19	0.5	0.1	33000	7	15	63	16000	23	7800	260	0.25	580	1.2	43	14	110
2292714 (back yard)	0-5 cm	5600	2.5	48	120	0.7	0.7	34000	37	110	380	67000	150	7000	930	2.3	3500	4.4	60	20	650
	0-5 cm	4000	1.6	41	86	0.5	0.7	34000	18	81	280	40000	120	7500	660	1.3	2700	4.8	53	16	430
	5-10 cm	4500	2.2	66	150	0.6	0.7	29000	33	160	530	120000	220	6500	950	3	6400	5.7	51	22	830
	5-10 cm	5400	2.1	58	160	0.8	1.5	30000	38	190	670	140000	240	6000	1200	3.9	8000	7.4	58	24	950
	10-20 cm	5200	2.1	69	260	0.5	1.3	26000	33	110	580	100000	280	6200	1000	2.2	6100	5.2	51	24	930
	10-20 cm	4600	2.8	49	130	0.5	1	30000	26	86	420	74000	180	6800	770	2.5	4400	4.7	51	21	680
2292721 (fill area)	0-5 cm	14000	0.5	6.2	85	0.6	0.6	31000	21	10	25	21000	33	9600	580	0.5	65	0.2	64	32	75
	0-5 cm	14000	0.4	7.8	90	0.7	0.4	29000	20	13	31	21000	29	9500	600	0.5	180	0.3	58	32	70
	10-20 cm	14000	0.8	11	100	0.7	0.7	34000	23	30	120	27000	60	10000	640	0.6	1400	1.4	68	33	130
	0-5 cm	14000	0.4	9.2	100	0.7	0.4	31000	21	15	44	22000	56	9800	570	0.5	310	0.3	63	33	110
	5-10 cm	14000	0.4	10	100	0.7	0.6	26000	23	22	70	23000	65	8800	650	0.7	620	0.8	62	34	140
	10-20 cm	14000	0.7	10	100	0.7	0.3	28000	24	33	110	24000	95	9500	580	0.5	1200	1.5	61	35	150
2292721 (west of fill area)	0-5 cm	11000	0.9	12	100	0.6	0.9	15000	18	50	160	20000	170	7300	400	0.5	1600	2	46	29	300
	5-10 cm	15000	2.1	11	110	0.7	0.7	13000	23	33	120	23000	120	6300	420	0.8	1200	1.5	43	34	180
	10-20 cm	15000	1.3	14	150	0.9	0.7	23000	37	50	210	28000	180	8300	420	0.9	2300	2.6	76	37	310
	0-5 cm	8600	1	9.8	77	0.5	0.7	19000	17	56	160	18000	200	9000	420	0.5	1800	2.2	44	25	280
	5-10 cm	12000	0.8	12	87	0.6	0.2	12000	18	37	130	20000	160	5900	400	0.6	1400	1.6	37	30	180
2024902 (north side)	0-5 cm	11000	2.1	11	140	0.6	0.9	32000	24	31	130	18000	130	16000	340	1.4	910	1.6	120	27	300
	0-5 cm	12000	2	11	170	0.7	0.9	31000	24	34	140	20000	180	16000	370	1.4	940	1.4	130	30	320
2024901 (south side)	0-5 cm	17000	1.4	15	110	0.9	1.3	18000	25	40	180	20000	81	8700	350	1.2	1400	1.8	75	35	220
	0-5 cm	18000	1.1	16	120	1	1.2	18000	27	42	180	21000	93	8600	360	1	1400	2.2	76	37	240
	5-10 cm	18000	1.4	20	110	1	1	18000	26	43	190	21000	86	8700	360	0.5	1500	2.8	72	37	230
	5-10 cm	17000	1.3	19	110	1	0.9	15000	26	41	190	21000	89	7600	350	1.4	1500	1.7	64	36	230

Table 3: Results of Chemical Analysis (µg/g) of 2001 Residential Soil Samples, Port Colborne

Al = aluminum, Sb = antimony, As = arsenic, Ba = barium, Be = beryllium, Cd = cadmium, Ca = calcium, Cr = chromium, Co = cobalt, Cu = copper, Fe = iron, Pb = lead, Mg = magnesium, Mn = manganese, Mo = molybdenum, Ni = nickel, Se = selenium, Sr = strontium, V = vanadium, Zn = zinc. Data in bold and underlined exceed MOE Table A effects-based guideline. na - data not available

Site/Location	Soil Depth	Al	Sb	As	Ba	Be	Cd	Ca	Cr	Co	Cu	Fe	Pb	Mg	Mn	Mo	Ni	Se	Sr	V	Zn
2022803 (east of drive)	0-5 cm	6200	1.3	14	66	0.5	0.2	19000	19	48	170	24000	160	9200	400	1	2100	2.1	63	22	250
	0-5 cm	6600	1.4	15	79	0.5	0.9	21000	22	62	220	29000	220	10000	500	1.8	2700	2.3	79	25	310
	5-10 cm	6800	1.2	16	64	0.5	0.9	25000	20	57	200	25000	150	12000	460	1.4	2300	2.5	66	24	230
	5-10 cm	7100	1.1	16	53	0.5	1.1	21000	18	56	200	24000	160	10000	460	1.4	2000	2.3	56	25	240
	10-15 cm	6000	0.4	8	52	0.5	0.4	20000	13	13	64	13000	43	7900	230	0.9	600	0.7	90	18	100
	10-15 cm	7000	1.1	16	53	0.5	0.9	24000	22	65	220	29000	190	12000	530	1.2	2500	2.1	57	27	270
2022804 (east yard)	0-5 cm	8700	1.4	17	120	0.5	0.8	13000	28	62	230	33000	320	5000	530	1.1	2700	2	60	27	400
	0-5 cm	12000	0.6	11	110	0.5	0.7	7700	20	25	89	23000	110	4200	570	0.6	880	1.2	27	29	210
	5-10 cm	7300	2.6	40	170	0.6	1.1	16000	40	140	510	67000	540	4700	880	2	6600	5.9	64	34	800
	5-10 cm	7600	2.1	30	170	0.6	1.2	16000	36	110	410	55000	420	5400	790	3.9	5400	4.2	58	32	670
	10-20 cm	6300	2.9	60	200	0.6	0.9	17000	42	140	600	70000	450	3600	860	3	8100	5	83	31	920
	10-20 cm	6600	2.5	49	180	0.6	1.6	16000	48	140	560	70000	420	3900	890	3.7	7200	5.8	73	32	860
2022805 (south back yard)	0-5 cm	12000	0.5	11	90	0.6	0.5	17000	19	28	91	21000	90	7700	430	1	910	1.5	72	28	160
	0-5 cm	10000	0.4	8.7	75	0.5	0.4	14000	16	21	63	18000	82	6300	390	1	580	1	55	26	140
	5-10 cm	14000	0.8	12	130	0.8	0.9	27000	21	37	130	24000	210	11000	440	0.6	1100	1.4	94	31	230
	5-10 cm	12000	0.9	13	130	0.7	0.8	23000	20	34	140	26000	160	8600	470	1.1	1200	1.3	110	31	250
	10-15 cm	12000	0.6	9.6	96	0.6	0.6	22000	18	28	94	21000	100	8100	400	0.7	800	1	73	28	160
2022806 (west back yard)	0-5 cm	8900	2.7	73	160	0.8	1.9	15000	55	200	840	100000	720	5900	1300	4.9	11000	6.9	56	41	1300
	0-5 cm	10000	0.4	8.8	87	0.5	0.7	10000	16	21	72	20000	88	5100	450	0.5	660	0.9	32	26	160
	5-10 cm	9900	2.1	33	160	0.7	2	14000	34	110	410	55000	420	5100	800	2.1	4900	3.9	49	37	890
	5-10 cm	11000	0.8	13	110	0.6	0.6	13000	21	55	180	27000	180	5200	530	1.2	1900	2	41	31	340
	10-20 cm	6600	2.9	83	190	0.6	1.6	14000	43	130	660	86000	620	4000	1000	4.2	8100	4.8	56	31	1200
	10-20 cm	8300	1.9	30	170	0.6	1	17000	25	72	360	42000	440	5500	690	1.9	4100	2.7	66	30	560
2022807 (west side yard)	0-5 cm	11000	1	15	94	0.6	0.7	9600	24	43	170	30000	190	5000	610	1.6	1800	1.7	30	30	320
	0-5 cm	11000	0.6	11	91	0.5	0.5	8400	20	34	120	26000	160	4600	620	1.4	1300	1.4	25	29	230
	5-10 cm	8200	1.1	16	110	0.5	0.6	12000	22	56	190	31000	240	4500	480	1.2	2300	2.1	42	26	360
	5-10 cm	8800	3	91	160	0.9	1.1	14000	59	210	850	120000	670	5300	1500	4.8	10000	6.5	52	44	1400
	10-20 cm	8900	2.7	66	160	0.8	1	15000	44	160	770	94000	550	4900	1100	3.1	10000	4.9	62	36	1200
	10-20 cm	8700	2.4	85	150	0.8	0.7	13000	53	160	780	110000	560	4700	1300	4.5	11000	3.8	54	38	1400
<T		1000	1	1	5	2.5	1	500	5	2.5	5	1000	10	250	25	2.5	2.5	1	5	5	25
≤W		200	0.2	0.2	1	0.5	0.2	100	1	0.5	1	200	2	50	5	0.5	0.5	0.2	1	1	5

<T = a measurable trace amount; interpret with caution

≤W = no measurable response; less than reported value

Table 4: Results of Chemical Analysis (μg/g) of 2000 Trench Soil Samples, Port Colborne

Al = aluminum, Sb = antimony, As = arsenic, Ba = barium, Be = beryllium, Cd = cadmium, Ca = calcium, Cr = chromium, Co = cobalt,
Cu = copper, Fe = iron, Pb = lead, Mg = magnesium, Mn = manganese, Mo = molybdenum, Ni = nickel, Se = selenium, Sr = strontium,
V = vanadium, Zn = zinc. Data underlined and in bold exceed MOE Table A effects-based guideline. <T = a measurable trace amount; interpret with caution
≤W = no measurable response; less than reported value

Site/Location	Soil Depth	Al	Sb	As	Ba	Be	Cd	Ca	Cr	Co	Cu	Fe	Pb	Mg	Mn	Mo	Ni	Se	Sr	V	Zn
2292641 Baseball park, SE corner of Davis & Rodney Sts., near 2nd base	30 - 35 cm	25500	1.6	8.5	222	**1.3**	0.2	53100	32	29	182	27900	76	20400	455	2.3	**1500**	0.3	246	49	156
	30 - 35 cm	19700	1.3	9.3	160	1.0	0.2	55700	25	29	191	24400	77	22900	499	2.2	**1430**	0.3	282	41	154
	60 - 65 cm	24600	1.3	8.7	240	**1.4**	0.2	74000	27	29	188	27000	82	23900	668	2.3	**1740**	0.3	292	45	132
	60 - 65 cm	24300	2.0	9.0	205	**1.3**	0.2	67700	34	29	158	29400	77	22800	568	2.6	**1540**	0.3	215	49	137
	100 - 105 cm	12100	2.5	**33**	85	0.4	0.2	16100	24	**89**	**524**	38400	127	4660	377	2.5	**6680**	2.5	68	31	341
	100 - 105 cm	24100	1.6	13	179	**1.2**	0.2	48100	30	**51**	**440**	29600	86	18300	517	2.4	**3020**	0.3	190	49	166
2292642 Baseball park, SE corner of Davis and Rodney Sts., centre of outfield	30 - 35 cm	22400	1.4	9.9	207	**1.3**	0.2	70200	28	27	148	26600	67	29700	408	2.5	**1500**	0.3	336	48	128
	30 - 35 cm	22500	1.1	8.2	320	1.4	0.2	84500	32	23	123	24200	63	31200	519	2.5	**1290**	0.3	460	44	107
	60 - 65 cm	22700	2.1	11	224	**1.4**	0.2	66200	28	27	135	29800	71	24600	601	2.6	**1430**	0.3	263	47	119
	60 - 65 cm	18500	0.8	4.2	95	0.5	0.2	6320	25	11	37	16200	24	4440	198	1.1	**304**	0.3	53	40	63
	100 - 105 cm	20500	1.6	16	224	**1.4**	0.2	62600	25	32	207	29600	99	23800	574	2.6	**2100**	0.3	226	43	166
	100 - 105 cm	10700	0.5	14	73	0.3	0.2	11100	16	22	266	21400	29	4700	246	1.4	**2210**	0.3	54	31	127
2292643 North shoulder of Rodney Street, between Mitchell & Davis Sts.	30 - 35 cm	12000	3.8	**37**	118	1.0	0.2	97600	38	177	**749**	53700	183	39100	690	3.5	**9730**	4.6	171	41	436
	30 - 35 cm	9020	2.9	**31**	149	0.8	0.2	106000	30	176	**699**	46900	148	49200	597	3.2	**8900**	4.8	130	36	334
	60 - 65 cm	12900	7.2	**43**	309	2.1	3.3	18900	31	25	81	168000	247	4050	1920	4.6	**204**	0.3	124	76	**3190**
	60 - 65 cm	27000	6.5	19	525	**4.2**	2.3	84600	21	23	52	137000	150	10700	1400	2.7	**384**	0.3	323	73	**3710**
	100 - 105 cm	20700	1.1	6.4	116	0.6	0.3	14600	25	11	48	17500	34	4840	191	1.6	**374**	0.3	82	36	113
	100 - 105 cm	23900	0.8	2.5	121	0.7	0.2	5420	28	11	5	17100	28	5500	189	1.0	66	0.3	40	42	81
2292644 NW corner of vacant lot south of Rodney Street between Welland and Fares Sts.	30 - 35 cm	2520	0.2	6.9	19	0.5	0.2	27800	6	9	35	12500	25	7180	163	1.3	**364**	0.3	42	14	81
	30 - 35 cm	3170	1.3	16	38	0.5	0.3	29800	16	30	158	33700	69	7340	316	2.2	**1650**	0.5	52	16	293
	60 - 65 cm	2700	0.2	2.6	24	0.5	0.2	30600	5	4	12	7780	18	7870	156	1.2	102	0.3	63	13	34
	60 - 65 cm	3440	1.2	13	32	0.5	0.3	30400	17	24	115	31200	58	7940	333	2.4	**1310**	0.3	47	23	218
	100 - 105 cm	2720	0.2	3.2	34	0.5	0.4	21600	6	4	10	6460	5	1260	233	3.0	97	0.3	117	23	9
	100 - 105 cm	2870	0.2	4.3	47	0.5	0.3	25700	7	6	21	6930	5	1430	279	5.2	**210**	2.5	145	22	28
2292645 NE corner of vacant lot south of Rodney Street between Welland & Fares Sts.	30 - 35 cm	3340	0.2	7.5	29	0.5	0.4	30700	7	6	48	14300	21	7030	222	5.3	**289**	0.3	53	16	89
	30 - 35 cm	3780	0.2	**27**	68	0.5	1.9	29100	12	49	275	41300	123	6660	725	6.4	**3320**	0.9	56	9	409
	60 - 65 cm	2720	0.2	3.7	16	0.5	0.2	30100	7	4	16	8740	8	7590	161	5.4	92	0.3	48	17	32
	60 - 65 cm	2480	0.2	2.3	12	0.5	0.2	14900	5	3	1	6100	5	6250	112	4.6	15	0.3	28	13	13
	100 - 105 cm	1420	0.2	5.0	26	0.5	0.3	21800	4	5	18	6490	7	2090	152	4.1	185	0.3	87	6	35
	100 - 105 cm	1440	0.2	3.9	24	0.5	0.2	22700	4	4	12	5710	3	2100	133	4.0	123	0.5	98	6	24
2292647 West trench in park east of Welland Street & north of Nickel Street	30 - 35 cm	20400	0.2	7.9	153	1.2	1.0	24900	40	25	107	37300	138	9100	623	7.0	**516**	0.3	75	38	289
	30 - 35 cm	7960	0.2	14	117	0.8	1.5	40900	54	30	167	56300	124	8780	1290	9.3	**975**	0.3	97	13	499
	60 - 65 cm	12600	9.4	17	307	**2.5**	5.9	40300	122	**52**	141		**479**	3770	4050	12.8	**558**	1.4	157	1.0	**3410**
	60 - 65 cm	10300	8.0	13	282	**2.1**	6.2	30500	94	**53**	151		**520**	3200	3990	10.6	**640**	0.8	126	1.0	**3680**

Table 4: Results of Chemical Analysis (µg/g) of 2000 Trench Soil Samples, Port Colborne

Al = aluminum, Sb = antimony, As = arsenic, Ba = barium, Be = beryllium, Cd = cadmium, Ca = calcium, Cr = chromium, Co = cobalt,
Cu = copper, Fe = iron, Pb = lead, Mg = magnesium, Mn = manganese, Mo = molybdenum, Ni = nickel, Se = selenium, Sr = strontium,
V = vanadium, Zn = zinc. Data underlined and in bold exceed MOE Table A effects-based guideline. <T = a measurable trace amount; interpret with caution

≤W = no measurable response; less than reported value

Site/Location	Soil Depth	Al	Sb	As	Ba	Be	Cd	Ca	Cr	Co	Cu	Fe	Pb	Mg	Mn	Mo	Ni	Se	Sr	V	Zn
	100 - 105 cm	7070	0.2	15	121	0.9	1.2	37100	13	13	124	20900	97	2500	343	7.2	739	0.3	469	22	173
	100 - 105 cm	4980	0.2	13	100	0.6	1.0	26100	9	12	86	14500	104	1780	247	6.4	727	0.3	302	18	120
2292647	30 - 35 cm	8740	10.5	32	168	1.1	3.5	53400	110	47	134	15100	335	11500	3300	10.7	613	0.3	224	34	1870
East trench in park	30 - 35 cm	13100	0.8	9.7	49	0.7	0.5	122000	28	15	59	18100	34	70400	227	3.3	301	0.3	73	33	59
west of Welland	60 - 65 cm	10400	9.3	28	198	1.4	4.4	54100	101	47	132	147000	344	12800	3610	9.3	578	0.3	199	34	1860
Street & north of	60 - 65 cm	10400	6.7	21	195	1.4	2.4	50100	56	46	128	129000	234	11000	2750	5.2	852	0.3	189	34	1380
Nickel Street	100 - 105 cm	16600	0.7	3.6	69	0.5	0.3	10600	19	10	17	17200	34	5600	226	1.5	97	0.3	26	33	76
	100 - 105 cm	15400	0.4	3.5	70	0.5	0.3	11600	19	11	17	17400	42	6020	242	1.5	107	0.3	27	33	82
2292648	0-5 cm	16600	0.8	3.2	72	0.5	0.3	14500	19	10	16	18200	38	6860	254	1.6	89	0.3	31	34	77
West berm in park	0-5 cm	15900	0.6	4.0	75	0.5	0.3	15500	18	10	16	17700	43	7020	312	1.5	90	0.3	31	33	74
west of Welland	5-10 cm	17200	1.4	4.4	84	0.6	0.3	27000	21	11	16	20900	39	12000	367	1.7	92	0.3	45	35	79
Street & north of	5-10 cm	20300	1.1	4.6	109	0.7	0.3	33200	23	13	16	23200	37	14100	479	1.7	89	0.3	54	40	85
Nickel Street	10-15 cm	24100	1.4	5.3	126	0.9	0.4	30800	29	14	19	29000	41	13300	482	1.8	105	0.3	65	46	130
	10-15 cm	16400	1.0	3.8	93	0.6	0.3	32700	22	12	17	21100	44	11900	374	1.8	107	0.3	54	34	100
2292649	0-5 cm	14400	0.2	1.7	71	0.6	0.4	11600	19	11	22	17300	55	5630	257	4.9	108	0.3	28	30	96
East berm in park	0-5 cm	14100	0.2	2.7	67	0.6	0.4	11200	17	11	21	16300	53	5550	248	4.9	115	0.3	24	30	87
west of Welland	5-10 cm	16200	0.2	3.1	79	0.7	0.4	14700	20	11	18	19100	45	7130	304	5.5	102	0.3	32	32	87
Street & north of	5-10 cm	14100	0.2	3.7	81	0.7	0.5	18100	20	11	21	18200	60	7590	307	5.5	117	0.3	34	27	105
Nickel Street	10-15 cm	26800	0.2	2.4	173	1.3	0.4	30800	33	16	23	29200	106	15200	514	6.3	98	0.3	63	49	118
	10-15 cm	18400	0.2	4.7	124	0.9	0.5	35800	25	14	30	23700	110	14000	496	6.4	135	0.3	63	36	133
	15-20 cm	19000	0.2	2.8	124	0.9	0.5	33200	26	15	30	24600	87	13500	447	6.3	143	0.3	66	38	131
	15-20 cm	25100	0.2	2.9	157	1.2	0.4	33200	32	17	22	30100	61	15100	585	6.4	95	0.3	69	46	105
<T		1000	1	1	5	2.5	1	500	5	2.5	5	1000	10	250	25	2.5	2.5	1	5	5	25
≤W		200	0.2	0.2	1	0.5	0.2	100	1	0.5	1	200	2	50	5	0.5	0.5	0.2	1	1	5

Table 5: Summary of the 2000 - 2001 Residential Soil Data (0 - 5 cm Depth)

Element	Minimum	Maximum	Mean	10th Percentile	20th Percentile	30th Percentile	40th Percentile	50th Percentile (Median)	60th Percentile	70th Percentile	80th Percentile	90th Percentile
Aluminum	2600	38300	16432	8822	11000	13200	14900	16300	17760	19620	20980	23900
Antimony	0.1	23.6	2.0	0.4	0.5	0.7	0.9	1.1	1.4	1.8	2.9	5.2
Arsenic	1	350	14	5	7	8	10	11	13	15	17	21
Barium	22	511	153	83	97	111	125	143	158	175	192	234
Beryllium	0.2	2.2	0.9	0.6	0.7	0.7	0.8	0.9	1.0	1.1	1.2	1.3
Cadmium	0.0	5.1	1.2	0.5	0.6	0.8	0.9	1.1	1.2	1.4	1.6	2.0
Calcium	3180	91000	19169	10079	12900	14100	15600	17900	19400	21520	24400	28600
Chromium	8	154	29	18	21	24	26	28	30	32	35	39
Cobalt	5	222	48	19	24	28	32	37	43	50	65	85
Copper	8	1100	210	69	100	124	148	170	205	242	301	393
Iron	8820	100000	26252	17600	19555	21300	23000	24800	26300	28520	30600	35940
Lead	8	1600	202	74	95	117	140	167	193	224	276	357
Magnesium	1400	31900	7711	4290	5600	6150	6580	7140	7780	8710	9648	11200
Manganese	160	2030	473	323	374	408	436	463	482	505	547	613
Molybdenum	0.3	11.7	3.6	0.9	1.4	3.0	3.7	3.9	4.2	4.4	4.8	5.7
Nickel	46	11000	2038	467	735	974	1230	1540	1886	2280	3024	4304
Selenium	0.2	10.0	2.2	0.4	0.7	1.0	1.2	1.7	2.2	2.8	3.5	4.9
Strontium	19	690	73	39	46	53	59	63	70	78	90	110
Vanadium	10	68	36	24	27	30	33	35	38	41	43	48
Zinc	30	1590	335	141	187	220	255	290	330	380	449	590

Data are μg/g dry weight
N.B. Does not include the 2000 trench samples

Table 6: Summary of the 2000 - 2001 Residential Soil Data (5 - 10 cm Depth)

Element	Concentration (µg/g)											
	Minimum	Maximum	Mean	10th Percentile	20th Percentile	30th Percentile	40th Percentile	50th Percentile (Median)	60th Percentile	70th Percentile	80th Percentile	90th Percentile
Aluminum	2500	40500	17803	8800	11840	13728	15700	17750	19500	21200	23260	26800
Antimony	0.1	34.5	2.3	0.4	0.5	0.8	1.0	1.3	1.6	2.1	2.9	5.6
Arsenic	1	110	16	6	7	9	11	13	15	18	22	30
Barium	19	624	167	88	100	123	138	152	168	186	210	265
Beryllium	0.3	2.2	1.0	0.6	0.7	0.8	0.9	1.0	1.1	1.2	1.3	1.4
Cadmium	0.0	4.3	1.2	0.4	0.6	0.8	0.9	1.1	1.2	1.5	1.8	2.2
Calcium	1920	77000	19921	10000	12500	14500	16000	18038	21000	23590	26000	30800
Chromium	9	245	30	19	23	26	28	30	32	34	37	41
Cobalt	5	262	53	19	24	29	34	40	48	56	71	97
Copper	4	1120	250	67	101	130	160	191	234	295	374	492
Iron	10100	140000	30895	18170	21000	23600	25800	27650	29700	32000	35760	45000
Lead	6	1430	214	72	95	120	140	170	200	242	311	408
Magnesium	1400	21000	7859	4379	5600	6300	6735	7405	8032	8785	10000	11830
Manganese	137	3880	514	345	390	429	457	481	512	548	597	688
Molybdenum	0.3	11.8	3.7	1.0	1.8	3.0	3.7	3.9	4.2	4.4	4.8	5.8
Nickel	31	17000	2601	485	755	1061	1368	1735	2238	2889	3792	5706
Selenium	0.1	19.4	2.6	0.4	0.6	1.0	1.4	1.9	2.5	2.9	4.3	5.8
Strontium	16	590	75	38	46	54	61	67	74	84	96	116
Vanadium	14	70	38	25	28	32	34	37	40	42	46	51
Zinc	23	1440	369	140	186	220	252	296	356	432	529	687

Data are µg/g dry weight
N.B. Does not include the 2000 trench samples

Table 7: Summary of the 2000 - 2001 Residential Soil Data (10 - 20 cm Depth)

Element	Concentration (µg/g)											
	Minimum	Maximum	Mean	10th Percentile	20th Percentile	30th Percentile	40th Percentile	50th Percentile (Median)	60th Percentile	70th Percentile	80th Percentile	90th Percentile
Aluminum	2200	47300	17818	7932	11360	13818	15600	18000	19800	21345	23320	27150
Antimony	0.1	91.1	2.3	0.4	0.6	0.7	0.8	1.1	1.4	1.9	2.7	5.6
Arsenic	0	130	18	7	8	11	13	16	18	20	26	32
Barium	19	956	185	95	119	135	150	165	182	202	235	287
Beryllium	0.3	4.6	1.0	0.6	0.7	0.8	0.9	1.0	1.1	1.2	1.3	1.5
Cadmium	0.1	35.3	1.3	0.4	0.6	0.8	0.9	1.1	1.2	1.5	1.8	2.2
Calcium	2880	93400	22555	12000	14980	17000	19245	21150	23245	25315	29000	34610
Chromium	7	183	31	20	23	26	28	30	32	34	37	41
Cobalt	8	214	51	20	26	31	35	40	48	56	72	100
Copper	8	2720	280	83	116	151	186	220	258	330	420	551
Iron	12000	110000	32024	19600	22960	25293	27100	29750	32230	35200	39020	47000
Lead	11	1800	236	76	97	128	160	188	221	257	318	454
Magnesium	990	25000	8143	4742	5600	6291	6846	7405	8100	9040	10000	12230
Manganese	131	5620	532	347	404	440	470	494	526	566	617	691
Molybdenum	0.2	12.1	3.7	0.9	1.9	2.7	3.5	3.8	4.1	4.5	4.9	5.8
Nickel	19	14000	2892	594	940	1237	1638	2163	2657	3290	4652	6301
Selenium	0.1	8.8	2.3	0.5	0.7	1.0	1.4	1.8	2.2	3.0	3.9	5.1
Strontium	14	431	87	43	54	61	68	75	87	96	110	136
Vanadium	12	75	37	25	28	32	35	37	39	42	45	51
Zinc	52	1750	392	140	188	227	283	330	385	451	570	721

Data are µg/g dry weight
N.B. Does not include the 2000 trench samples

Table 8: Summary of the 2000 - 2001 Residential Soil Data, All Depths Combined

Element	Concentration (μg/g)											
	Minimum	Maximum	Mean	10th Percentile	20th Percentile	30th Percentile	40th Percentile	50th Percentile (Median)	60th Percentile	70th Percentile	80th Percentile	90th Percentile
Aluminum	2200	47300	17346	8700	11300	13500	15300	17300	19030	20600	22520	26300
Antimony	0.1	91.1	2.2	0.4	0.5	0.7	0.9	1.2	1.5	1.9	2.9	5.5
Arsenic	0	350	16	6	8	9	11	13	15	18	21	29
Barium	19	956	169	88	101	122	139	154	169	188	211	266
Beryllium	0.2	4.6	1.0	0.6	0.7	0.8	0.9	1.0	1.1	1.2	1.3	1.4
Cadmium	0.0	35.3	1.2	0.4	0.6	0.8	0.9	1.1	1.2	1.5	1.7	2.2
Calcium	1920	93400	20543	10620	13300	15000	17000	19000	21200	23900	26300	32000
Chromium	7	245	30	19	22	25	27	29	31	33	36	40
Cobalt	5	262	51	20	24	30	34	39	47	55	70	95
Copper	4	2720	246	70	106	134	161	196	230	284	360	471
Iron	8820	140000	29706	18030	20900	23020	25200	27000	29200	31900	35500	42600
Lead	6	1800	217	74	96	120	148	173	202	244	299	406
Magnesium	990	31900	7903	4500	5600	6220	6726	7320	8000	8800	9950	11900
Manganese	131	5620	506	340	389	425	453	478	500	540	588	671
Molybdenum	0.2	12.1	3.7	0.9	1.7	2.8	3.6	3.9	4.2	4.4	4.8	5.8
Nickel	19	17000	2508	518	800	1070	1400	1730	2200	2810	3784	5592
Selenium	0.1	19.4	2.4	0.4	0.7	1.0	1.3	1.8	2.3	2.9	3.9	5.2
Strontium	14	690	78	40	49	56	62	68	76	87	99	121
Vanadium	10	75	37	25	28	31	34	37	39	42	45	50
Zinc	23	1750	365	140	187	223	260	305	356	418	520	665

Data are μg/g dry weight
N.B. Does not include the 2000 trench samples

Table 9: Samples from Port Colborne Submitted for pH in Distilled Water

Sample ID	Soil pH (Run 1)	Soil pH (Run 2)	Soil pH (Mean)	Soil depth (cm)	Site / Location
800	7.18	7.18	7.18	5	2292547
805	7.11	7.09	7.1	293	2292548
824	7.11	7.05	7.08	5	2292555
854	7.22	7.27	7.25	5	2292561
858	7.42	7.37	7.4	130	2292562
946	7.48	7.52	7.5	293	2292583
947	7.03	6.99	7.01	5	2292640
952	7.14	7.19	7.17	5	2292484
956	7.19	7.21	7.2	130	2292485
1019	7.42	7.37	7.39	293	2292502
1020	7.13	7.13	7.13	5	2292503
1059	7.01	7.03	7.02	5	2292516
1134	7.26	7.26	7.26	5	2292537
1139	7.29	7.32	7.31	293	2292538
1330	7.36	7.37	7.36	5	2292584
1334	7.25	7.35	7.3	130	2292585
1373	7.56	7.61	7.58	130	2292595
1375	7.51	7.51	7.51	5	2292596
1379	7.29	7.21	7.25	130	2292597
1381	7.09	7.1	7.09	5	2292598
3341	7.38	7.37	7.37	5	2292410
3346	7.61	7.7	7.65	293	2292411
3814	7.22	7.3	7.26	130	2292376
3816	6.89	6.88	6.88	5	2292377
3834	7.66	7.65	7.65	293	Rodney/Fares St
3892	7.11	7.09	7.1	5	2292473
5182	7.25	7.28	7.26	5	2292327
5187	7.76	7.75	7.75	293	2292328
5252	6.98	6.99	6.98	130	2292445
5313	7.58	7.63	7.6	293	2292449
5315	6.95	6.85	6.9	130	2292450
5361	7.23	7.24	7.23	130	2292321
5363	7.21	7.26	7.23	5	2292322
5390	7.28	7.31	7.29	5	2292470
5404	7.52	7.61	7.57	293	2292471
5407	7.36	7.38	7.37	293	2292471
		Overall Mean	7.23		

Table 10: Nickel Speciation Results for Selected Rodney Street Community Soil Samples

Sample Number	MOE[1] Total Nickel (μg/g)	Lakefield Research Limited					St Marys[7] Total Nickel (μg/g)	SSRL[2] Nickel Oxide (% of Total Nickel)
		Total Nickel (μg/g)	Soluble Nickel (% of Total Nickel)	Nickel Sulfide (% of Total Nickel)	Nickel Metal (% of Total Nickel)	Nickel Oxide (% of Total Nickel)		
Processed Samples[5]								
772	6,630	6,000	0.32	7.02	7.98	84.6	NS	NS
799	6,590	5,300	0.35	8.88	7.03	83.8	NS	NS
1332	10,400	10,300	0.57	6.65	5.11	87.6	NS	NS
1348	9,760	7,900	0.33	9.46	14.10	76.1	NS	NS
1408	10,800	10,600	0.26	6.35	38.70	54.6	13,305	94.0[3]
1415	6,450	6,200	0.28	6.30	6.16	87.3	NS	NS
3740	9,300	7,900	0.65	5.96	5.96	87.4	9,573	84.7[4]
3768	6,930	6,000	0.29	10.50	10.60	78.6	NS	NS
3770	8,680	7,400	0.36	6.02	5.93	87.7	NS	NS
3821	6,670	6,600	0.29	9.50	11.60	78.6	NS	NS
Mean	**8,221**	**7,420**	**0.37**	**7.66**	**11.30**	**80.6**	**11,440**	**89.3**
Bulk Samples (not processed)[6]								
772	NS	7,900	0.44	8.40	5.75	85.4	NS	NS
799	NS	3,500	0.77	16.50	5.77	76.9	NS	NS
1332	NS	8,300	0.27	15.10	3.70	81.0	NS	NS
1348	NS	7,900	0.27	18.90	16.20	64.7	NS	NS
1408	NS	12,200	0.32	7.62	4.01	88.0	NS	NS
1415	NS	9,300	0.27	10.80	5.97	83.0	NS	NS
3740	NS	7,700	0.68	11.80	5.64	81.9	NS	NS
3768	NS	6,700	0.34	12.10	7.34	80.2	NS	NS
3770	NS	5,000	0.49	14.50	3.93	81.1	NS	NS
3821	NS	6,300	0.39	12.60	4.52	82.5	NS	NS
Mean	**NS**	**7,800**	**0.42**	**12.80**	**6.28**	**80.5**	**NS**	**NS**

1. MOE Laboratory Services Branch **total nickel** by ICP-MS.
2. Stanford Synchrotron Radiation Laboratory **nickel oxide** by XAFS (X-Ray Absorption Fine Structure).
3. Mean of 3 replicate analysis of the same sample (R1= 100%, R2=87%, R3=95%).
4. Mean of 3 replicate analysis of the same sample (R1=91%, R2=87%, R3=76%).
5. Dried, ground, and sieved, following MOE standard protocol for processing soil.
6. Dried, but not processed.
7. St. Marys University was contracted to conduct nickel speciation tests, they used the SSRL laboratory.
NS=No Sample.

Table 11: Metal and Arsenic Concentration of Inco Port Colborne 1978 Refinery Dust

Dust Origin	Dust Composition (%)							
	Arsenic	Cobalt	Copper	Iron	Lead	Nickel	Sulfur	Zinc
Tumblast Stack Dust - two major phases identified, a magnesium-silicon alloy (50% nickel) and a nickel-magnesium alloy (80% nickel). - minor nickel-silicon-magnesium iron phase.	NL	0.24	0.14	2.62	NL	42.9	<0.20	NL
Cobalt Multiclone Stack Dust - major phase is cobalt sulfate hydrate. - minor phases are cobaltous oxide and iron hydrogen sulfate hydrate.	NL	28.7	<0.02	4.7	NL	0.53	17.7	NL
Cottrell Precipitator Dust - most prevalent compound is nickel oxide. - lead sulfate is also a major phase. - nickel sulfate and copper sulfate are minor phases.	0.38	0.66	7.6	0.61	10.48	38.7	7.06	0.14
Submerged Combustion Evaporator Stack - an aqueous slurry, when dried the only compound detected was nickel oxide.	NL	NL	0.04	0.23	NL	72.2	0.76	NL
Based on dust samples provided to MOE by Inco from the Port Colborne refinery (MOE 1978). NL=Not listed.								

Table 12: Number of Properties in the Rodney Street Community Where the Maximum Soil Value on the Property Exceeds MOE Generic Effects-based Soil Guidelines Before and After the 25 Nickel-contaminated Properties are Remediated

Chemical	Guideline[1]	Number of Properties Exceeding Guideline Before and After Soil Nickel Remediation[2]	
		Before	After
Nickel	8,000[5]	25	0
Copper	225	131	107
Cobalt	40	141	117
Arsenic	20	91	67
Zinc	600	73	50
Beryllium	1.2	93	82
Antimony	13	3	3
Cadmium	12	1	0
Lead	200	145	123
Lead	400[3]	59	47
Lead	1,000[4]	11	9

1 - MOE Table B, non-potable groundwater, residential/parkland landuse, fine-textured soil (except nickel).
2 - 25 properties remediated to meet the 8,000 µg/g nickel health risk-based soil intervention level.
3 - bare soil to which children have regular access.
4 - sodded/covered area to which children have regular access.
5 - soil nickel intervention level

Table 13: Estimated Total Emissions of Particulate Matter, Iron and Nickel from Inco and Algoma over the Operating Life of the Facilities

Contaminant	Algoma Emissions (tonnes)	Inco Emissions (tonnes)
Particulate Matter	25,225	30,037
Iron	8,954	111
Nickel	N/A	18,375

N/A - data not available
Source: JWEL, 2001c

Table 14: Mean Soil Concentration and Upper and Lower 95% Confidence Intervals by Sample Depth from 2000-2001 Data

Element	0-5 cm Depth			5-10 cm Depth			10-20 cm Depth		
	Lower 95% CI	Mean	Upper 95% CI	Lower 95% CI	Mean	Upper 95% CI	Lower 95% CI	Mean	Upper 95% CI
Aluminum	15,895	16,432	16,969	17,183	17,803	18,423	17,167	17,818	18,470
Antimony	1.64	1.99	2.34	1.82	2.29	2.76	1.53	2.33	3.12
Arsenic	12.0	13.6	15.1	15.1	16.3	17.5	17.2	18.5	19.7
Barium	147	153	159	160	167	175	177	185	194
Beryllium	0.89	0.91	0.94	0.96	0.99	1.02	1.00	1.03	1.07
Cadmium	1.12	1.17	1.23	1.15	1.21	1.28	1.12	1.28	1.44
Calcium	18,373	19,169	19,965	19,102	19,921	20,739	21,648	22,555	23,462
Chromium	27.78	28.75	29.73	29.36	30.50	31.64	29.67	30.65	31.64
Cobalt	44.6	47.6	50.6	49.0	52.6	56.2	48.4	51.4	54.4
Copper	198	210	223	233	250	267	260	280	300
Iron	25,440	26,252	27,063	29,486	30,895	32,304	30948	32,024	33,100
Lead	188	202	216	199	214	229	219	236	253
Magnesium	7,430	7,711	7,992	7,589	7,859	8,128	7,840	8,143	8,446
Manganese	460	473	486	494	514	534	506	532	558
Molybdenum	3.5	3.6	3.8	3.5	3.7	3.9	3.5	3.7	3.8
Nickel	1,884	2,038	2,192	2,370	2,601	2,831	2672	2,892	3,112
Selenium	2.01	2.24	2.48	2.29	2.59	2.89	2.10	2.30	2.50
Strontium	68.37	72.80	77.22	71.17	75.20	79.23	82.45	86.59	90.73
Vanadium	34.88	35.70	36.52	36.76	37.66	38.56	36.38	37.30	38.21
Zinc	318	335	352	347	369	390	370	392	414

Statistics calculated using Microsoft Excel 97 SR-2 software

Part A - Soil Investigation: Tables

Table 15: Results of Pearson Products Correlation Test on the Soil Data from all Residential Properties, All Depths Combined

	Aluminum	Antimony	Arsenic	Barium	Beryllium	Cadmium	Calcium	Chromium	Cobalt	Copper	Iron	Lead	Magnesium	Manganese	Molybdenum	Nickel	Selenium	Strontium	Vanadium	Zinc
Aluminum	1.000																			
Antimony	0.029	1.000																		
Arsenic	-0.205	0.155	1.000																	
Barium	0.383	0.215	0.197	1.000																
Beryllium	0.804	0.097	-0.030	0.615	1.000															
Cadmium	0.176	0.139	0.122	0.456	0.302	1.000														
Calcium	0.043	0.071	0.000	0.266	0.193	0.086	1.000													
Chromium	0.440	0.115	0.351	0.473	0.515	0.263	0.089	1.000												
Cobalt	-0.052	0.098	0.598	0.309	0.124	0.270	0.023	0.377	1.000											
Copper	-0.049	0.167	0.590	0.463	0.183	0.339	0.093	0.388	0.852	1.000										
Iron	-0.017	0.142	0.636	0.305	0.140	0.179	0.087	0.395	0.760	0.698	1.000									
Lead	0.006	0.361	0.306	0.724	0.289	0.414	0.202	0.318	0.380	0.511	0.377	1.000								
Magnesium	0.378	0.027	-0.176	0.162	0.324	0.063	0.706	0.180	-0.035	-0.044	-0.045	0.005	1.000							
Manganese	0.064	0.093	0.307	0.237	0.205	0.117	0.206	0.247	0.395	0.354	0.541	0.214	0.103	1.000						
Molybdenum	0.297	0.050	-0.105	0.251	0.372	0.237	0.075	0.186	-0.024	0.011	0.024	0.165	0.149	0.158	1.000					
Nickel	-0.103	0.112	0.629	0.340	0.102	0.293	0.039	0.346	0.929	0.878	0.798	0.426	-0.098	0.397	-0.045	1.000				
Selenium	-0.398	0.109	0.574	0.084	-0.218	0.087	0.007	0.145	0.729	0.655	0.661	0.265	-0.196	0.302	-0.256	0.764	1.000			
Strontium	0.097	0.127	0.068	0.439	0.331	0.183	0.636	0.193	0.064	0.173	0.113	0.334	0.313	0.111	0.112	0.094	0.045	1.000		
Vanadium	0.896	0.022	-0.077	0.397	0.764	0.150	0.014	0.495	0.093	0.066	0.103	0.053	0.373	0.117	0.204	0.010	-0.247	0.098	1.000	
Zinc	-0.094	0.225	0.541	0.653	0.229	0.416	0.139	0.438	0.688	0.780	0.677	0.753	-0.084	0.360	0.092	0.733	0.571	0.298	0.021	1.000

Number of observations: 1504 or 1505 (except antimony with 1314)

Soil Investigation and Human Health Risk Assessment for the Rodney Street Community, Port Colborne: March 2002

Table 15.1: Results of Pearson Products Correlation Test on the Soil Data from all Residential Properties (0 - 5 cm Depth)

	Aluminum	Antimony	Arsenic	Barium	Beryllium	Cadmium	Calcium	Chromium	Cobalt	Copper	Iron	Lead	Magnesium	Manganese	Molybdenum	Nickel	Selenium	Strontium	Vanadium	Zinc
Aluminum	1.000																			
Antimony	0.062	1.000																		
Arsenic	-0.147	0.164	1.000																	
Barium	0.457	0.298	0.138	1.000																
Beryllium	0.829	0.120	-0.024	0.656	1.000															
Cadmium	0.309	0.220	0.052	0.646	0.472	1.000														
Calcium	-0.034	0.166	0.016	0.275	0.136	0.176	1.000													
Chromium	0.413	0.156	0.528	0.492	0.499	0.357	0.114	1.000												
Cobalt	-0.014	0.146	0.447	0.388	0.161	0.374	0.068	0.407	1.000											
Copper	-0.021	0.218	0.498	0.525	0.195	0.465	0.094	0.465	0.891	1.000										
Iron	0.060	0.232	0.485	0.358	0.203	0.281	0.098	0.452	0.758	0.737	1.000									
Lead	0.070	0.463	0.183	0.740	0.281	0.535	0.236	0.317	0.365	0.546	0.375	1.000								
Magnesium	0.291	0.117	-0.106	0.262	0.337	0.213	0.721	0.180	0.049	0.032	0.026	0.154	1.000							
Manganese	0.061	0.187	0.322	0.355	0.269	0.246	0.262	0.328	0.547	0.519	0.709	0.300	0.179	1.000						
Molybdenum	0.352	0.059	-0.087	0.277	0.453	0.390	0.078	0.178	-0.014	0.035	0.046	0.135	0.196	0.181	1.000					
Nickel	-0.070	0.160	0.449	0.379	0.108	0.376	0.037	0.377	0.949	0.901	0.806	0.380	-0.032	0.533	-0.031	1.000				
Selenium	-0.390	0.176	0.396	0.117	-0.209	0.097	0.039	0.154	0.709	0.670	0.650	0.259	-0.146	0.401	-0.282	0.766	1.000			
Strontium	0.055	0.160	0.032	0.343	0.207	0.233	0.661	0.154	0.042	0.112	0.088	0.249	0.307	0.097	0.066	0.057	0.050	1.000		
Vanadium	0.903	0.060	-0.031	0.530	0.835	0.318	-0.014	0.501	0.158	0.158	0.191	0.170	0.300	0.180	0.291	0.079	-0.217	0.077	1.000	
Zinc	-0.019	0.283	0.369	0.732	0.253	0.625	0.194	0.459	0.669	0.806	0.651	0.778	0.066	0.471	0.074	0.706	0.557	0.240	0.146	1

Number of observations: 507 (except antimony with 446 and zinc with 463)

Table 15.2: Results of Pearson Products Correlation Test on the Soil Data from all Residential Properties (5 - 10 cm Depth)

	Aluminum	Antimony	Arsenic	Barium	Beryllium	Cadmium	Calcium	Chromium	Cobalt	Copper	Iron	Lead	Magnesium	Manganese	Molybdenum	Nickel	Selenium	Strontium	Vanadium	Zinc
Aluminum	1.000																			
Antimony	-0.001	1.000																		
Arsenic	-0.278	0.182	1.000																	
Barium	0.406	0.228	0.232	1.000																
Beryllium	0.822	0.102	-0.056	0.648	1.000															
Cadmium	0.263	0.232	0.196	0.632	0.464	1.000														
Calcium	0.016	0.112	0.017	0.272	0.167	0.135	1.000													
Chromium	0.387	0.110	0.266	0.458	0.495	0.359	0.077	1.000												
Cobalt	-0.082	0.139	0.734	0.332	0.122	0.368	0.031	0.366	1.000											
Copper	-0.085	0.196	0.741	0.477	0.174	0.474	0.089	0.400	0.945	1.000										
Iron	-0.113	0.179	0.780	0.263	0.076	0.179	0.086	0.348	0.792	0.754	1.000									
Lead	-0.008	0.422	0.404	0.749	0.294	0.547	0.224	0.298	0.420	0.563	0.371	1.000								
Magnesium	0.415	0.026	-0.201	0.195	0.393	0.134	0.702	0.187	-0.049	-0.043	-0.080	-0.002	1.000							
Manganese	-0.018	0.152	0.453	0.296	0.186	0.190	0.229	0.245	0.478	0.456	0.611	0.283	0.081	1.000						
Molybdenum	0.265	0.068	-0.070	0.287	0.381	0.396	0.101	0.181	0.010	0.049	0.031	0.190	0.174	0.213	1.000					
Nickel	-0.148	0.151	0.774	0.346	0.070	0.353	0.033	0.346	0.942	0.939	0.812	0.461	-0.116	0.474	0.004	1.000				
Selenium	-0.406	0.177	0.717	0.076	-0.221	0.122	0.038	0.164	0.747	0.722	0.696	0.286	-0.192	0.385	-0.179	0.797	1.000			
Strontium	0.065	0.178	0.075	0.407	0.280	0.226	0.680	0.136	0.047	0.140	0.082	0.306	0.313	0.133	0.105	0.076	0.045	1.000		
Vanadium	0.894	-0.011	-0.118	0.423	0.803	0.208	0.020	0.446	0.071	0.060	0.034	0.044	0.413	0.071	0.185	-0.021	-0.274	0.089	1.000	
Zinc	-0.118	0.261	0.688	0.666	0.217	0.576	0.149	0.455	0.749	0.842	0.713	0.746	-0.080	0.467	0.127	0.786	0.612	0.263	0.020	1

Number of observations: 498 (except antimony with 438 and zinc with 467)

Table 15.3: Results of Pearson Products Correlation Test on the Soil Data from all Residential Properties (10 – 20 cm Depth)

	Aluminum	Antimony	Arsenic	Barium	Beryllium	Cadmium	Calcium	Chromium	Cobalt	Copper	Iron	Lead	Magnesium	Manganese	Molybdenum	Nickel	Selenium	Strontium	Vanadium	Zinc
Aluminum	1.000																			
Antimony	0.027	1.000																		
Arsenic	-0.253	0.139	1.000																	
Barium	0.312	0.190	0.195	1.000																
Beryllium	0.772	0.085	-0.072	0.553	1.000															
Cadmium	0.118	0.106	0.155	0.379	0.220	1.000														
Calcium	0.099	0.016	-0.092	0.219	0.218	0.043	1.000													
Chromium	0.516	0.117	0.219	0.484	0.549	0.237	0.059	1.000												
Cobalt	-0.068	0.071	0.674	0.238	0.082	0.242	-0.038	0.353	1.000											
Copper	-0.066	0.141	0.592	0.401	0.147	0.283	0.054	0.333	0.785	1.000										
Iron	-0.003	0.101	0.698	0.290	0.115	0.189	0.024	0.419	0.757	0.633	1.000									
Lead	-0.041	0.328	0.352	0.696	0.274	0.369	0.138	0.338	0.360	0.452	0.394	1.000								
Magnesium	0.414	-0.007	-0.272	0.059	0.254	-0.012	0.699	0.168	-0.107	-0.113	-0.092	-0.115	1.000							
Manganese	0.108	0.048	0.219	0.135	0.173	0.070	0.151	0.215	0.272	0.217	0.427	0.128	0.078	1.000						
Molybdenum	0.286	0.043	-0.168	0.217	0.314	0.153	0.055	0.200	-0.075	-0.034	-0.002	0.171	0.086	0.117	1.000					
Nickel	-0.119	0.078	0.706	0.284	0.079	0.283	-0.005	0.317	0.937	0.816	0.770	0.417	-0.152	0.275	-0.108	1.000				
Selenium	-0.425	0.056	0.672	0.056	-0.258	0.084	-0.075	0.098	0.723	0.611	0.640	0.249	-0.263	0.193	-0.332	0.745	1.000			
Strontium	0.149	0.096	0.064	0.528	0.465	0.178	0.561	0.288	0.100	0.218	0.134	0.424	0.311	0.091	0.169	0.106	0.025	1.000		
Vanadium	0.893	0.024	-0.128	0.289	0.682	0.088	0.012	0.541	0.047	-0.003	0.097	-0.037	0.399	0.113	0.146	-0.032	-0.264	0.115	1.000	
Zinc	-0.145	0.204	0.604	0.592	0.196	0.337	0.063	0.398	0.645	0.718	0.663	0.741	-0.216	0.232	0.073	0.692	0.541	0.369	-0.086	1

Number of observations: 500 (except antimony with 430 and zinc with 499)

Soil Investigation and Human Health Risk Assessment for the Rodney Street Community, Port Colborne: March 2002

Table 16: Grouping of Chemical Elements Based on Their Relative Statistical (r) Association with Nickel, Iron and Lead

Group*	Chemical Elements from Table 20 Ranked by Coefficient of Correlation (r), all Depths Combined									
	r 0.099	r 0.199	r 0.299	r 0.399	r 0.499	r 0.599	r 0.699	r 0.799	r 0.899	r 0.999
Inco Nickel Group	vanadium (0.010) calcium (0.039) strontium (0.094)	beryllium (0.102) antimony (0.112)	cadmium (0.293)	barium (0.340) chromium (0.346) manganese (0.397)	lead (0.426)		arsenic (0.629)	zinc (0.733) selenium (0.764) iron (0.798)	copper (0.878)	cobalt (0.929)
Algoma Iron Group	molybdenum (0.024) calcium (0.087)	vanadium (0.103) strontium (0.113) beryllium (0.140) antimony (0.142) cadmium (0.179)		barium (0.305) lead (0.377) chromium (0.395)		manganese (0.541)	arsenic (0.636) selenium (0.661) zinc (0.677) copper (0.698)	cobalt (0.760) nickel (0.798)		
Urban Domestic Residential Lead Group	magnesium (0.005) vanadium (0.053)	molybdenum (0.165)	calcium (0.202) manganese (0.214) selenium (0.265) beryllium (0.289)	arsenic (0.306) chromium (0.318) strontium (0.334) antimony (0.361) iron (0.377) cobalt (0.380)	cadmium (0.414) nickel (0.426)	copper (0.511)		barium (0.724) zinc (0.753)		

* In the Nickel row all r values are relative to nickel, in the Iron row all r values are relative to iron, in the Lead row, all r values are relative to lead.

Table 17: Ratios of Nickel, Copper and Cobalt from Three Areas in Port Colborne Compared to Various Ontario Background Values

Area	Mean Soil Metal Concentration (µg/g)			Metal Soil Ratios	
	Ni	Cu	Co	Ni:Cu	Ni:Co
NE of Inco[1]	1809	182	32	9.9:1	56:1
Rodney St. Community[2]	2545	250	50	10.1:1	51:1
Trench Samples[3]	1401	148	32	9.5:1	44:1
Background Table F[4] OTR_{98}[5] Mean[5]	43 38 14	85 41 17	21 16 8	0.5:1 0.9:1 0.8:1	2.0:1 2.4:1 1.8:1

1 - mean of 8 1998/1999 MOE sample sites to the NE of Inco in the maximum deposition zone.
2 - mean of all 2000 and 2001 Rodney Street Residential Community samples.
3 - mean of all trench samples, excluding the park berms.
4 - MOE Table F (MOE 1997a).
5 - Ontario Typical Range 98th percentile and mean (MOE 1993c)

Table 18: Soil Metal Levels Observed Around Selected Industries in Ontario

Location and Type of Source	Soil Concentration in μg/g*						
	Iron	Nickel	Copper	Cobalt	Arsenic	Lead	Zinc
Welland: specialty steel manufacturer[1]	24500	139	32	11	8.5	67	179
Hamilton: steel manufacturer[2]	25500	32	28	NA	4	49	118
Wawa: Iron sintering[3]	95000	13	35	6.8	273	58	NA
Ottawa: Iron foundry[4]	34000	32	32	NA	NA	40	131
Cambridge: Iron Foundry[5]	15200	13	21	6	NA	67	183
Sudbury: Nickel and copper mining, smelting, refining[6]	80000	2300	2800	788	510	1000	270
Port Colborne: 90th percentile from the Rodney Street community	42280	5588	471	91	29	406	NA

* MOE investigations, data from selected sites believed to be directly impacted by the source.
1. Atlas Specialty Steel, 1993 and 1995 (MOE 1994, 1997b)
2. Stelco, 1976
3. Algoma Ore Division, 1998 (MOE 1999a)
4. Ivaco, 1985 and 1987 (MOE 1986, 1990)
5. Crowe Foundry, 1992 (MOE 1993b)
6. Inco/Falconbridge, 1997 and 2000 (MOE 2001b)
NA data not available or not evaluated

Table 19: Comparison of Soil Lead Levels in the Rodney Street Community with Other Ontario Urban Communities (all data are µg/g, dry weight)

Study/Source	Mean	Median	Range	90th Percentile
All MOE Rodney Street data, excluding trenches (2000 & 2001)	217	173	6 - 1,800	406
MOE Rodney Street data after remediation[1], excluding trenches	208	167	6 - 1,600	383
MOE data from Toronto lead study area[2] - residential properties and curbside samples (1973)	509	429	18 - 3,180	930
MOE data from Toronto lead study area[2] - residential properties only	421	364	18 - 1,450	693
MOE data from Toronto-wide soil study[3] - suburban and urban, residential properties and parks (1991)	202	130	15 - 3,150	325
MOE data from Toronto soil/paint study[4] - less than 3 m from wall, paint lead levels 1,100 µg/g - 310,000 µg/g (1993)	354	NA	180 - 890	NA
MOE data from Toronto soil/paint study[4] - more than 5 m from wall	75	NA	30 - 100	NA
Integrated across the property (Oakland, 1995)[5]	897*	880	56 - 88,176	NA
Integrated across the property (Los Angles, 1995)[5]	188*	190	30 - 1973	NA
Integrated across the property (Sacramento, 1995)[5]	234*	229	26 - 2,664	NA
Dripline near the wall (urban USA, 2001)[6]	324	40	NA	633
Midyard of property (urban USA, 2001)[6]	105	28	NA	243
Property with house paint containing 90,000 µg/g lead - integrated across property (New Orleans)[7]	258	NA	22 - 730	NA
Property with house paint containing 90,000 µg/g lead - less than (approx) 1 m from house (New Orleans)[7]	730	NA	NA	NA
Property with house paint containing 90,000 µg/g lead - more than (approx) 1 m from house (New Orleans)[7]	100	NA	22 - 220	NA
Property with house paint containing 130,000 µg/g lead - integrated across property (New Orleans)[7]	2,130	NA	360 - 3,900	NA
Property with house paint containing 130,000 µg/g lead - less than (approx) 1 m from house (new Orleans)[7]	3,900	NA	NA	NA
Property with house paint containing 130,000 µg/g lead - more than (approx) 1 m from house (new Orleans)[7]	360	NA	NA	NA

* - Geometric mean
1 - After remediation of 25 properties with soil nickel levels greater than 8,000 µg/g
2 - MOE 1974
3 - MOE 191-1991
4 - MOE 1995
5 - Sutton et al 1995
6 - U.S. Department of Housing and Urban development, 2001
7 - Mielke et al 2001

Table 20: Mean and Median Lead Concentrations in Soil in the Rodney Street Community, Properties Summarized by House Age

House Age*	Soil Metal Concentration (µg/g dry weight)		Number of Houses	% of Houses
	Mean Lead	Median Lead		
Before 1920	260	207	73	40.1
Between 1920 and 1949	195	160	99	54.4
After 1950	125	102	10	5.5

* based on Port Colborne municipal tax records, age not available for all homes

Table 21: Calculated Soil Nickel Concentrations in the Rodney Street Community Based on a Range of Estimated Nickel Deposition Rates from Inco Emissions*

	Nickel Deposition Rate (g/m²)**			
	5,000	10,000	15,000	20,000
Soil Depth (cm)	Soil Nickel Concentration (µg/g)			
0 - 5	66,667	133,333	200,000	266,667
0 - 10	33,333	66,667	100,000	133,333
0 - 15	22,222	44,444	66,667	88,889
0 - 20	16,667	33,333	50,000	66,667
0 - 25	13,333	26,667	40,000	53,333
0 - 30	11,111	22,222	33,333	44,444

* assumes: bulk density of 1 m³ of soil is 1,500 kg, all the nickel stays in the selected soil depth (no wash-out or leaching).
** Source: JWEL (2001c), estimated deposition over the operating life of the refinery.

Table 22: Comparison of Soil Arsenic Levels in the Rodney Street Community of Port Colborne with Other Ontario communities Where Arsenic-related Health Studies Have Been Conducted (all data are µg/g dry weight)*

Town	Mean	Median	Range	90th Percentile
Port Colborne: Rodney St. community, all 2000 & 2001 data (excluding trenches) n = approx. 2,000	16	13	<1 - 350	30
Port Colborne: Rodney St. community, 2000 & 2001 data (excluding trenches) after remediation of 25 properties exceeding nickel intervention level n = approx. 1,750	13	11	<1 - 62	22
Port Hope: all townsite data[3]	20	nc	2 - 234	nc
Deloro: all townsite residential data (excluding mine site)[1] n = approx. 450	111	78	2 - 605	225
Wawa: all townsite residential data (excluding Algoma Ore Division property and fume kill zone)[2] n = approx. 400	45	20	2 - 1,045	81
1 - MOE 1999b 2 - MOE 1999a 3 - MOE 1991 nc - not calculated * Health studies in Deloro and Wawa and health risk assessment in Port Hope concluded no measurable increase in health risk (see Part B Main Document, Section 5.7)				

Part A
Soil Investigation:
Soil Contour Maps and Figures

Table of Contents
Soil Investigation: Soil Contour Maps and Figures

Figure 1: Estimated Amount of Nickel Emitted from the Port Colborne Inco Refinery from 1918 to 2000 (Source: JWEL, 2001c)

Figure 2: Soil Sampling Pattern on Residential Properties in the Rodney Street Community

How a Residential Property is Sampled

Note: For demonstration purposes only. The actual pattern and location of the nine core samples taken may have varied from property to property according to site specific conditions.

A = 3 composite samples
B = 3 composite samples

TOTAL of 6 composites

9 Core Samples

0-5 cm + + + + + + + + + = Composite Sample

5-10 cm + + + + + + + + + = Composite Sample

10-20 cm + + + + + + + + + = Composite Sample
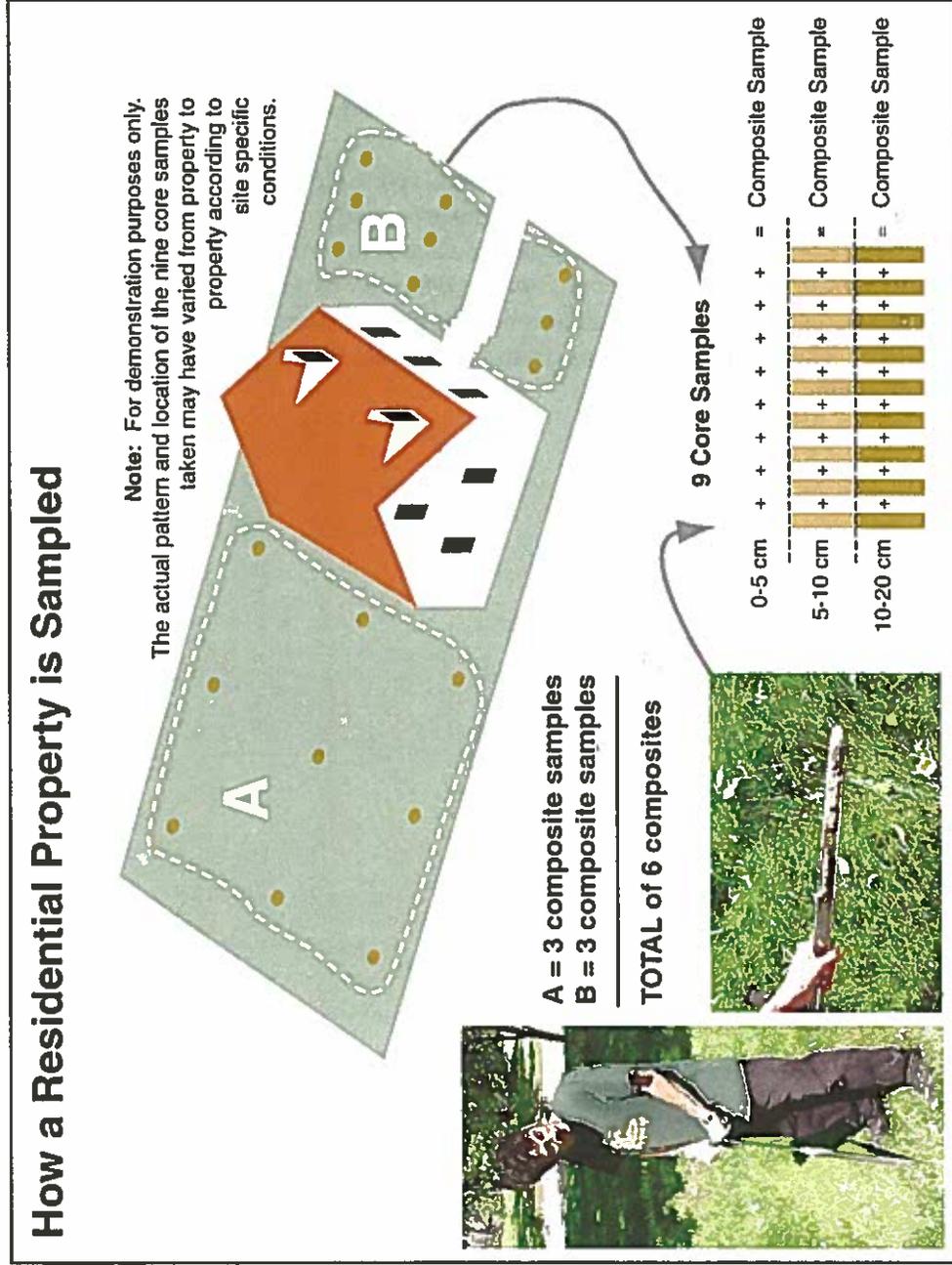
11.0 Regional Soil Arsenic and Selenium Contour Map

The MOE provided Jacques Whitford Environmental Ltd (JWEL) with archived surface soil samples (0-5 cm) collected by the MOE for the 1999 MOE Port Colborne regional soil investigation report. The MOE had not analyzed these samples for arsenic or selenium, as neither chemical had been found in previous MOE investigations to consistently exceed the MOE generic effects-based soil guidelines. JWEL analyzed the samples for arsenic and selenium as part of their Contaminants of Concern study for the Port Colborne CBRA.

The MOE used the JWEL soil data from the archived samples and produced Figure 2: Arsenic in 0-5 cm soil depth in the regional Port Colborne area, and Figure 3: Selenium in 0-5 cm soil depth in the regional Port Colborne area. The arsenic and selenium regional soil contour maps were prepared using Surfer (*Surfer Version 7.00 for Windows* by Golden Software Inc.) with the default options, and the same coordinates and number of lines as in the 2000 MOE report (Kuja et al., 2000b). The stations shown are from the original report, and not all of them had arsenic and selenium data.

Soil Investigation and Human Health Risk Assessment for the Rodney Street Community, Port Colborne: March 2002

Figure 3: Arsenic in 0-5 cm Soil Depth in the Regional Port Colborne Area

Part A - Soil Investigation: Soil Contour Maps and Figures

Selenium in Surface (0-5 cm) Soil
Port Colborne

- 1 to 1.99 ug/g Selenium
- 2 to 2.99 ug/g Selenium
- 3 ug/g and greater Selenium

- s Surface (0-5 cm), 1999
- g Surface (0-5 cm), 1998
- δ Surface (0-5 cm), Historical Surveys
- u Soil Profile (0-5, 5-10, 10-15 cm), 1998

LAKE ERIE

Figure 4: Selenium in 0-5 cm Soil Depth in the Regional Port Colborne Area

Part A - Soil Investigation: Soil Contour Maps and Figures
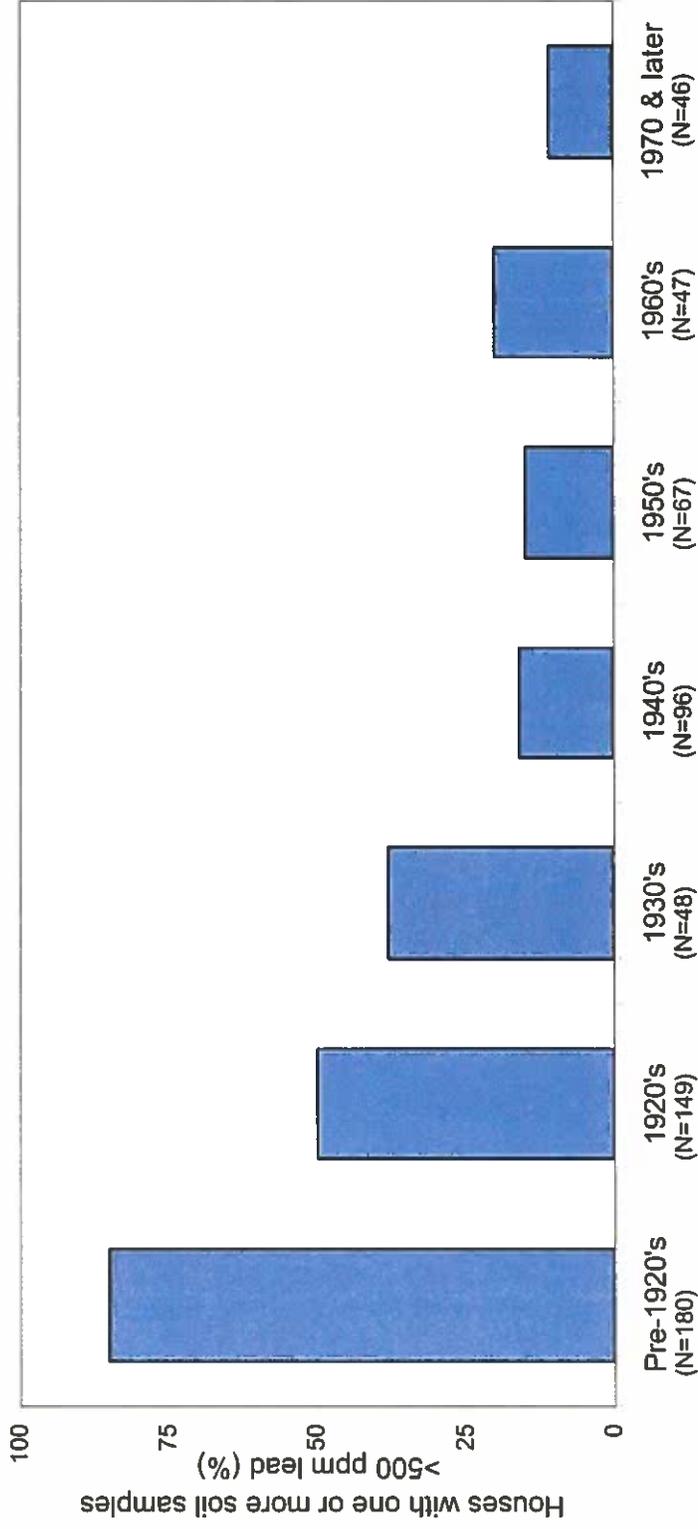
Figure 5: Relationship Between House Age and the Occurrence of Soil Lead Levels Greater than 500 ppm. (adapted from: Sutton et al., 1995)

11.1 Methodology for Producing Surfer/ArcView Soil Contaminant Contour Maps
11.1.1 Software Utilized

Two software packages were used to generate the maps. The data analysis and creation of the concentration contours was done using *Surfer Version 7.00 for Windows* by Golden Software Inc. The output from Surfer was imported into *ArcView GIS Version 3.1* by Environmental Systems Research Institute, Inc., and combined with base maps, roads and properties, to produce the final maps. The base map data was obtained from the City of Port Colborne, Public Works Department.

11.1.2 Data Used

For the contour maps produced in this report, all sampling stations collected south of Louis Street in 2000 as part of the intensive sampling of the Rodney Street community or as individual complaint investigations, were used to generate the contours.

11.1.3 Mapping Process

The process involved in creating the maps was to analyze the data and create the desired contours using the Surfer program. The individual contours were exported from Surfer as ESRI Shape files. The polygon portion of the Shape files were imported into ArcView GIS and modified to remove polygon holes created by the export process. The resulting polygons were combined with the street base maps, and the station locations were imported from the Phytotoxicology Information Management System (PIMS). Layouts were then created to include a legend, labels, scale and compass, and printed for the report.

11.1.4 Surfer

For all data sets, a Krigging gridding method was used and the search option was set to use all data. For all contours, smoothing was set at high. All co-ordinates were in Universal Trans Mercator (UTM) Easting and Northing. Where duplicate or triplicate samples existed for a sampling location the program was set to use the average of the results.

11.1.5 Surfer Settings

Grid Line Geometry				
	Minimum	**Maximum**	**Spacing**	**# of Lines**
X Axis (Easting)	643185 m	643527 m	3 m	115
Y Axis (Northing)	4748834 m	4749383 m	3 m	184
Duplicates - averaged Matrix Smoothing - none				

11.2 Arc View
11.2.1 Station Map

A base map was created by importing the Autocad DXF files provided by the City of Port Colborne, Works Department and converting to Arcview Shape files. Only the road, rail lines, property boundaries and building foot prints layers were turned on. To this was added all of the stations sampled in 2000 by importing the station co-ordinates and related information from the PIMS database.

11.2.2 Contour Maps

The street layer of the station map was used as the underlying map for all contour maps. Property boundaries, building foot prints and street numbers were not included. The polygons for each contour interval were imported into Arcview as individual shape files from Surfer. Any polygon holes were removed and combined with the other contour intervals. Grey scales were used to differentiate contours for printing purposes.

11.2.3 Final Maps

A separate ArcView layout was produced for each of the maps and consisted of a base map, contour polygons, scale, compass and legend. Sampling stations were included on the contour polygons maps. These layouts were imported into the report.
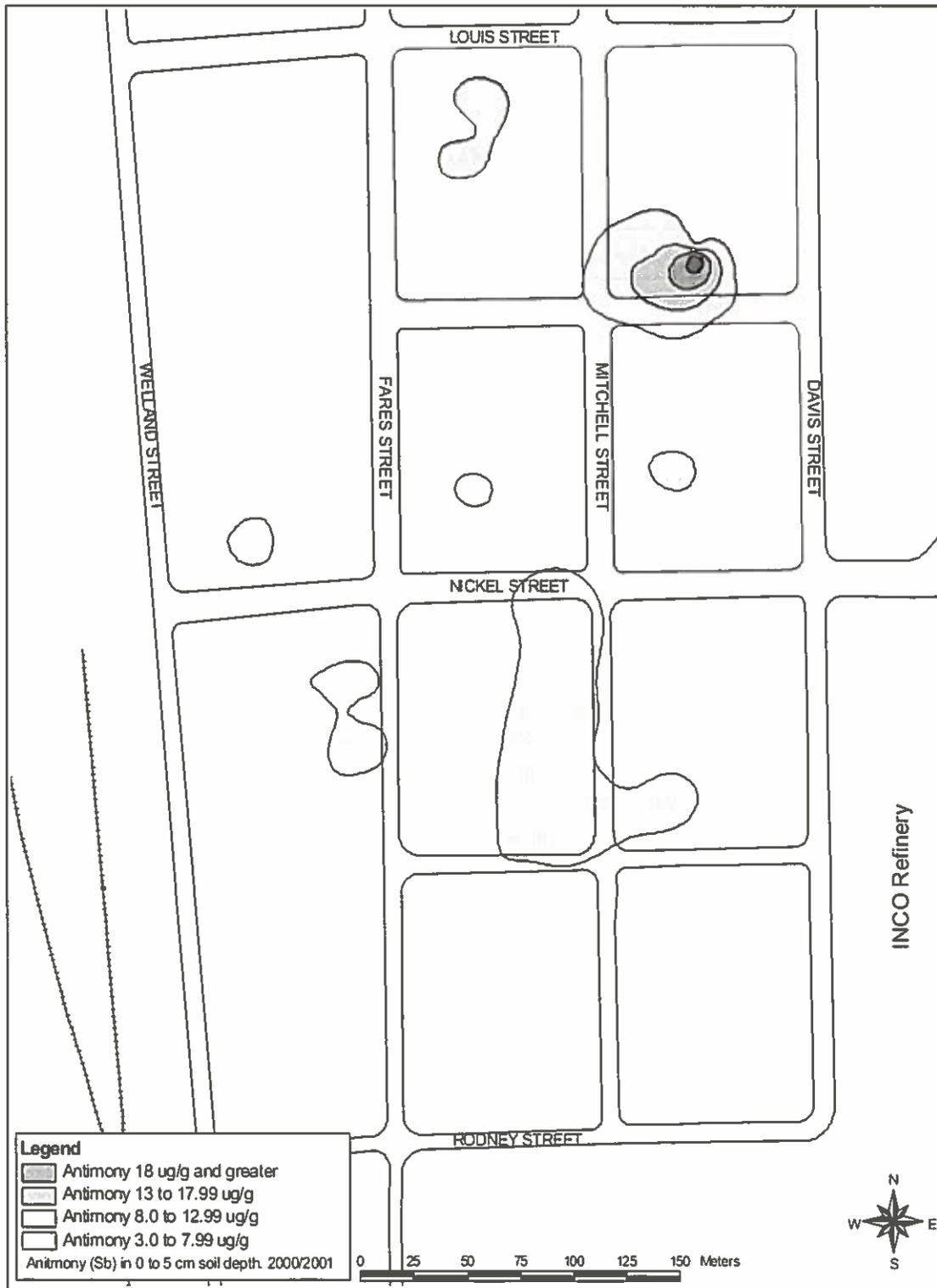
Map 1: Antimony in soil (0 to 5 cm) in the Rodney St. Community, 2000 & 2001.
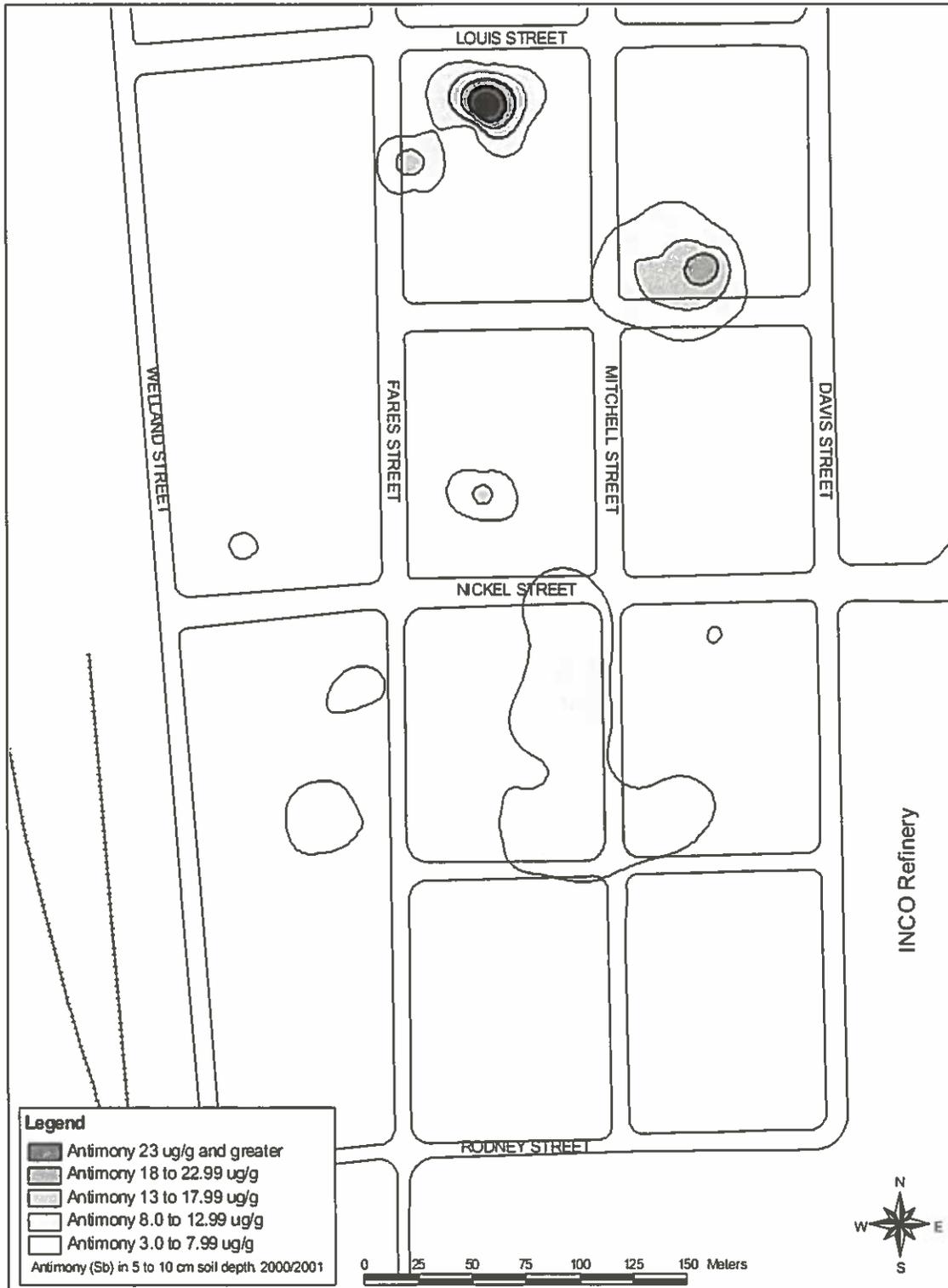
Map 2: Antimony in soil (5 to 10 cm) in the Rodney St. Community, 2000 & 2001.

Legend
- Antimony 23 ug/g and greater
- Antimony 18 to 22.99 ug/g
- Antimony 13 to 17.99 ug/g
- Antimony 8.0 to 12.99 ug/g
- Antimony 3.0 to 7.99 ug/g

Antimony (Sb) in 5 to 10 cm soil depth 2000/2001
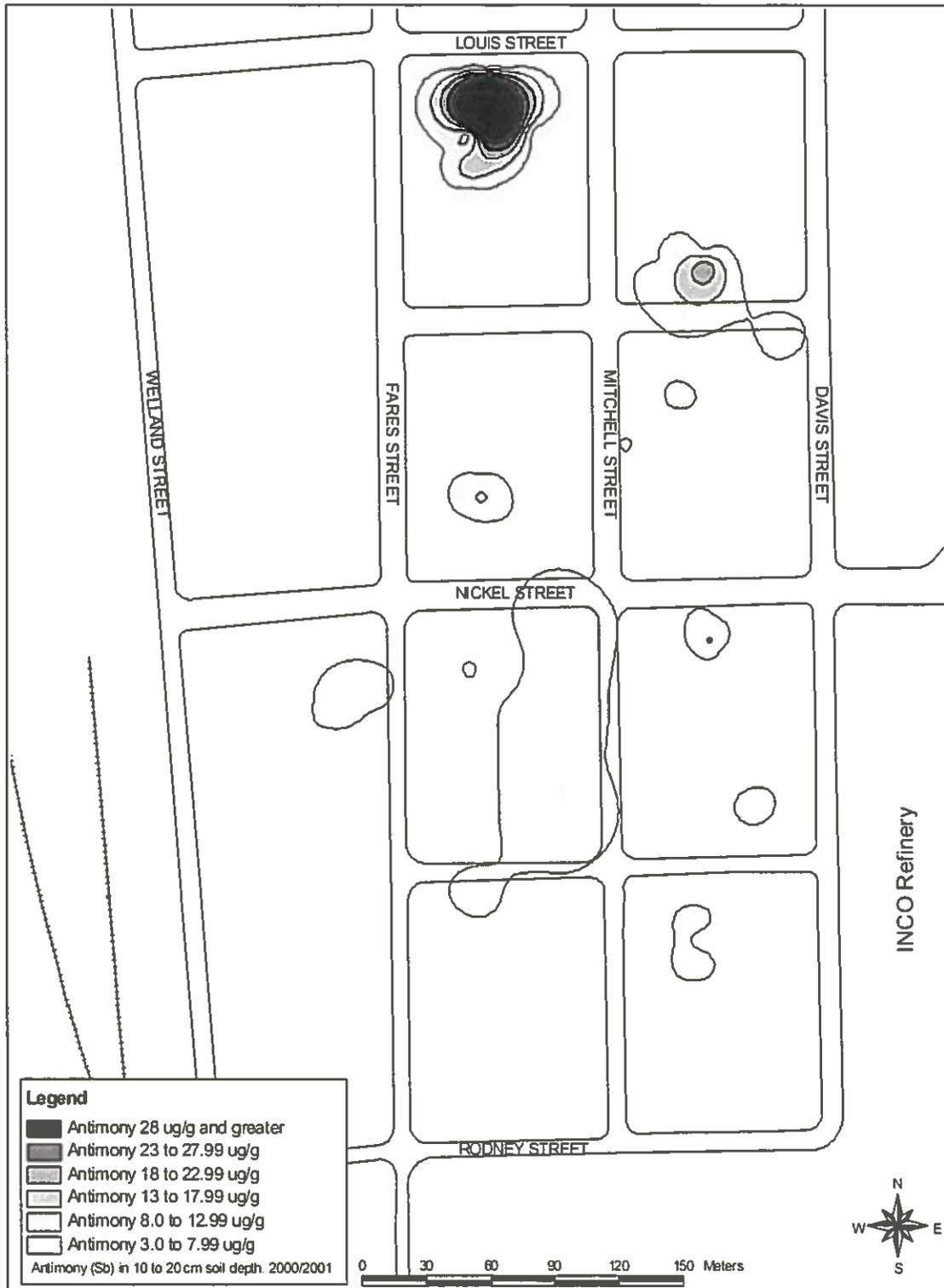
Map 3: Antimony in soil (10 to 20 cm) in the Rodney St. Community, 2000 & 2001.
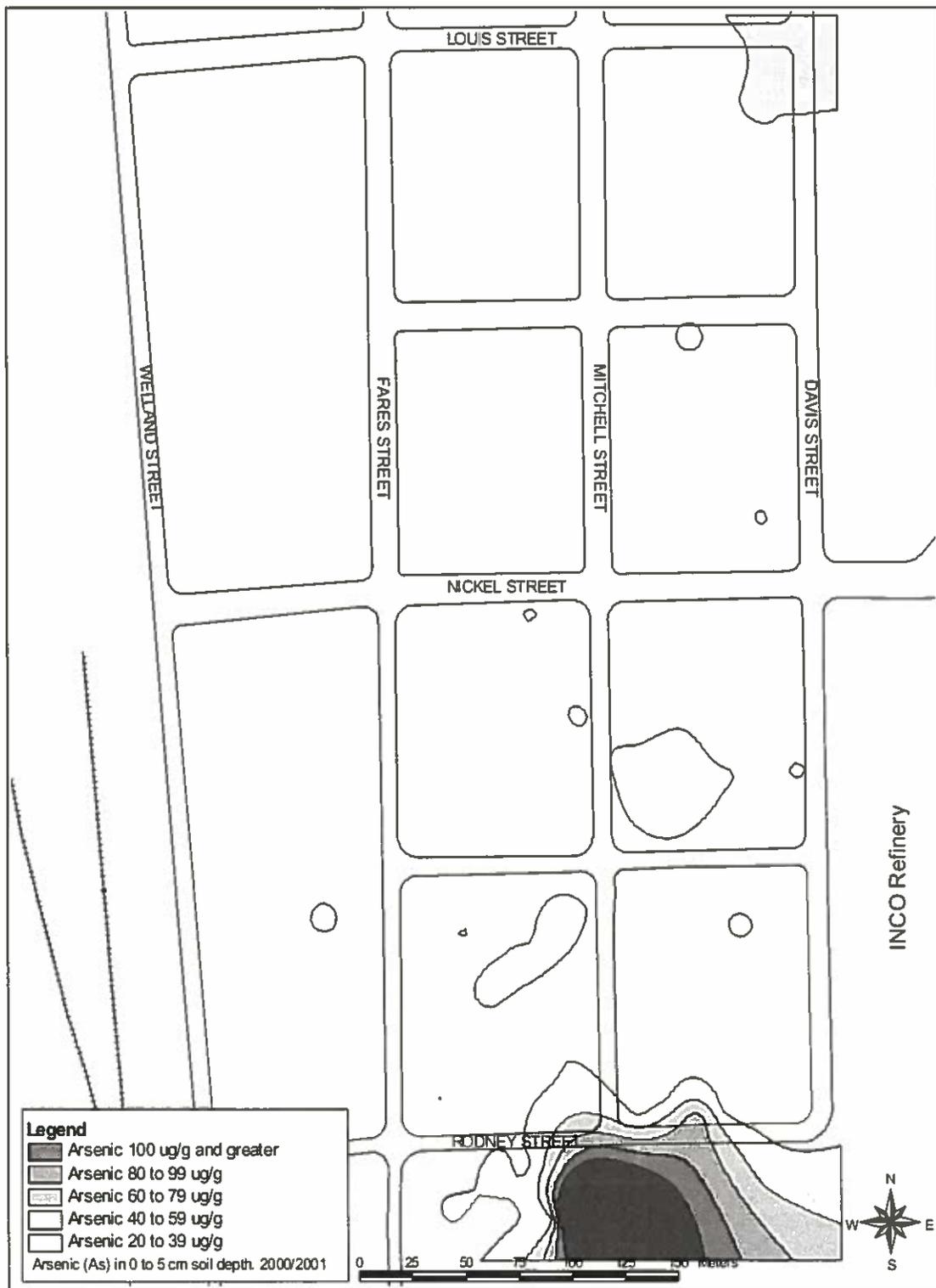
Map 4: Arsenic in soil (0 to 5 cm) in the Rodney St. Community, 2000 & 2001.
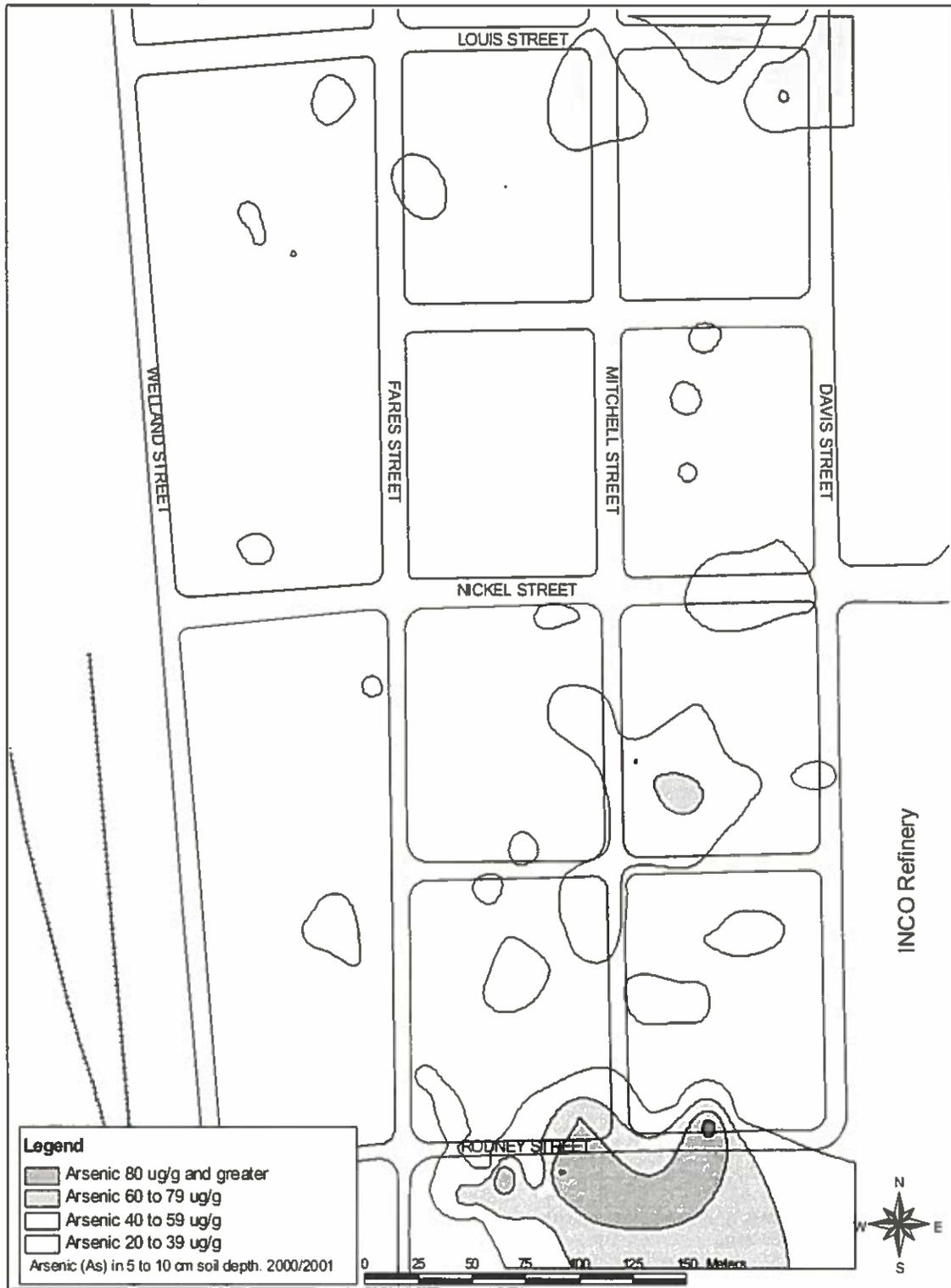
Map 5: Arsenic in soil (5 to 10 cm) in the Rodney St. Community, 2000 & 2001.

Legend
Arsenic 100 ug/g and greater
Arsenic 80 to 99 ug/g
Arsenic 60 to 79 ug/g
Arsenic 40 to 59 ug/g
Arsenic 20 to 39 ug/g
Arsenic (As) in 10 to 20 cm soil depth. 2000/2001
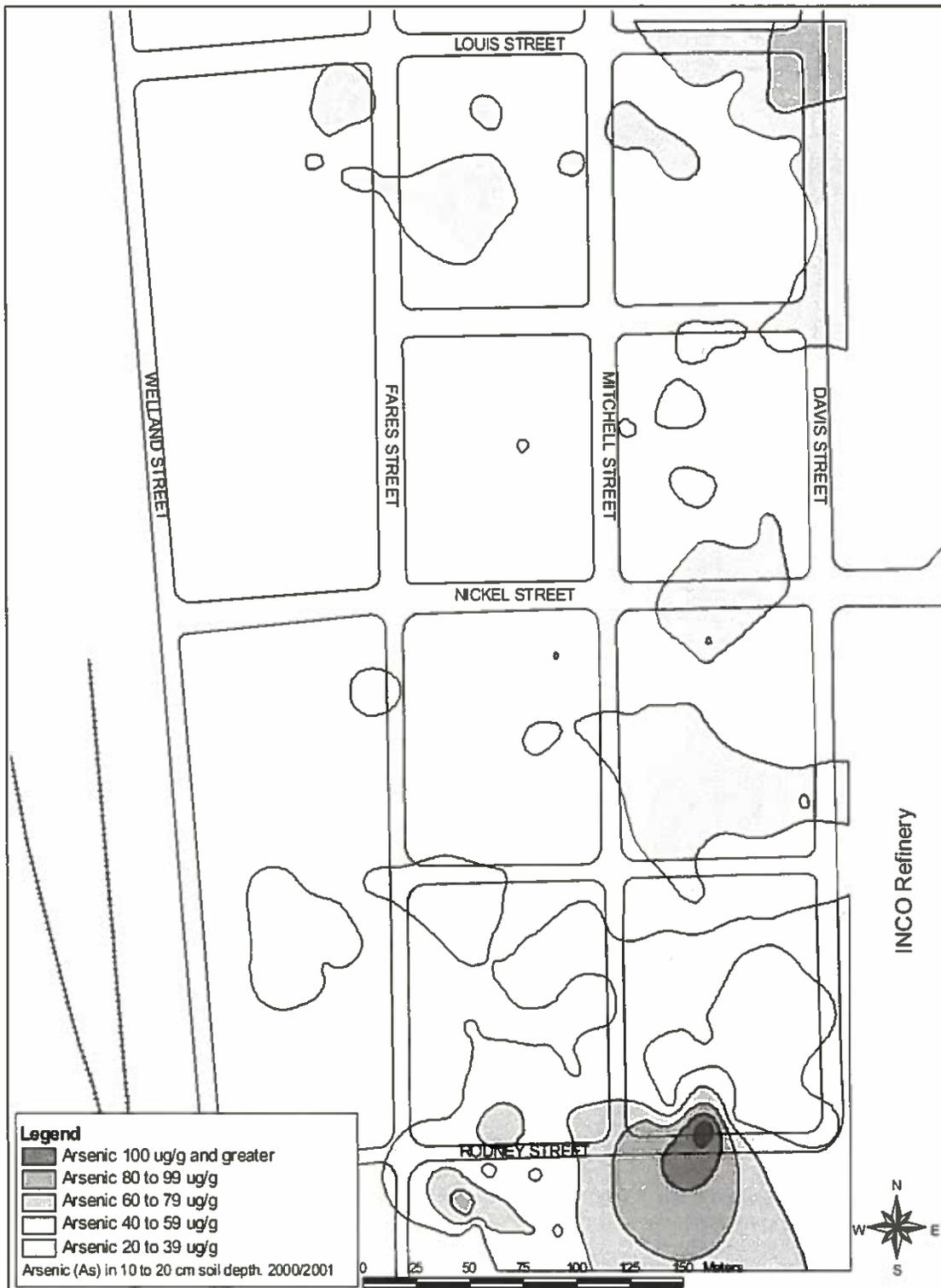
Map 6: Arsenic in soil (10 to 20 cm) in the Rodney St. Community, 2000 & 2001.

Map 7: Beryllium in soil (0 to 5 cm) in the Rodney St. Community, 2000 & 2001.
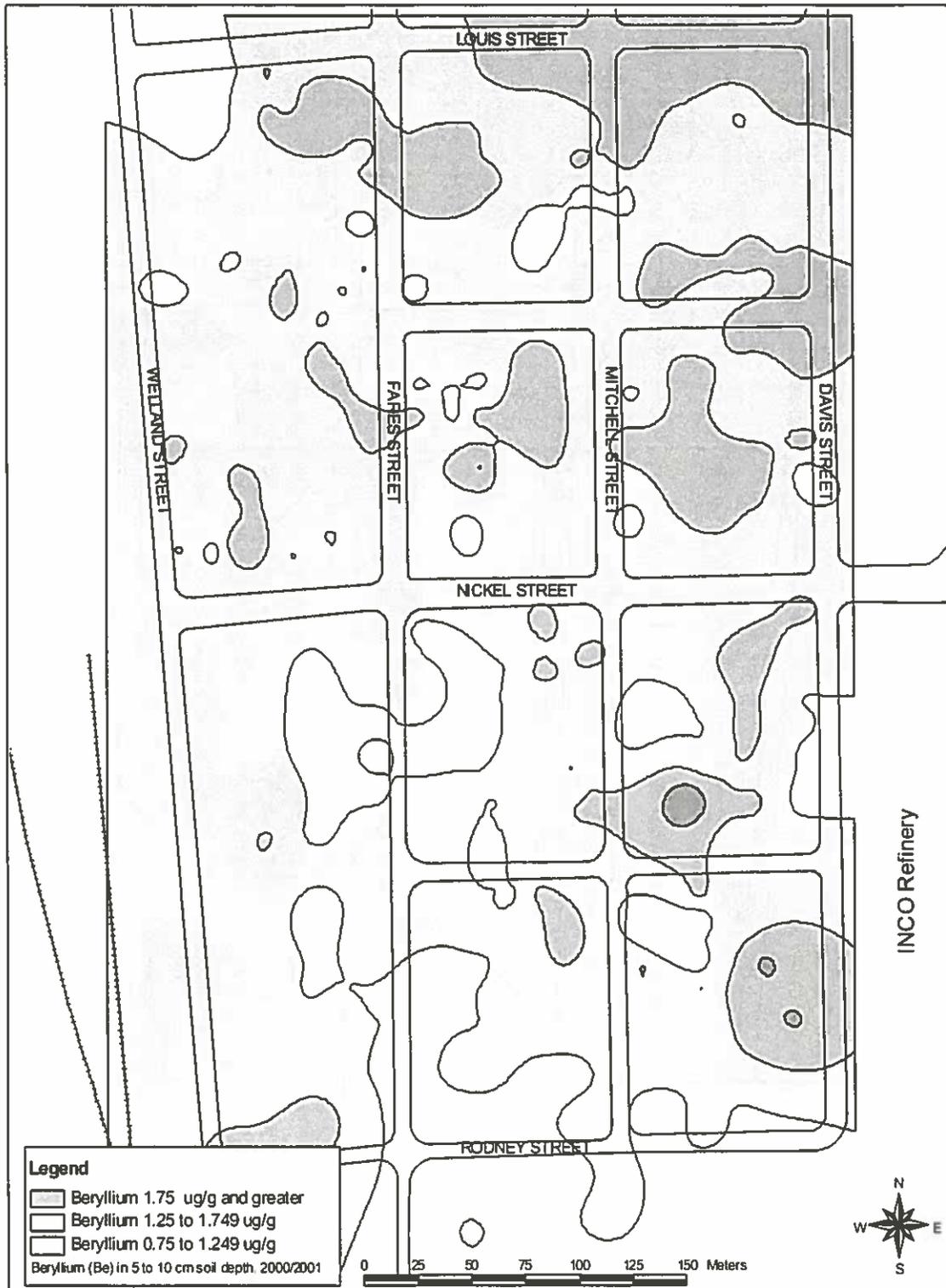
Map 8: Beryllium in soil (5 to 10 cm) in the Rodney St. Community, 2000 & 2001.

Map 9: Beryllium in soil (10 to 20 cm) in the Rodney St. Community, 2000 & 2001.

Map 10: Cadmium in soil (0 to 5 cm) in the Rodney St. Community, 2000 & 2001.
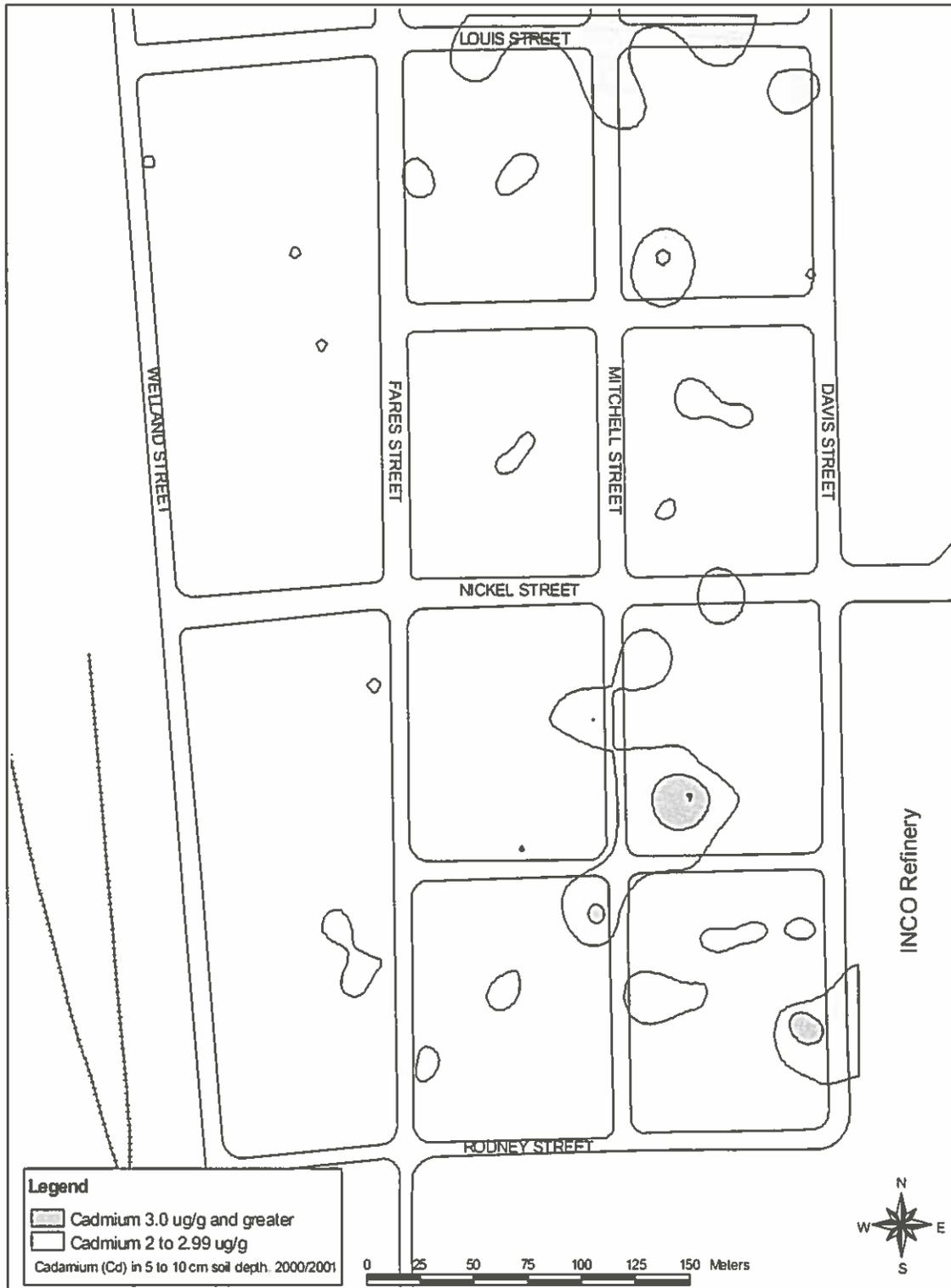
Map 11: Cadmium in soil (5 to 10 cm) in the Rodney St. Community, 2000 & 2001.
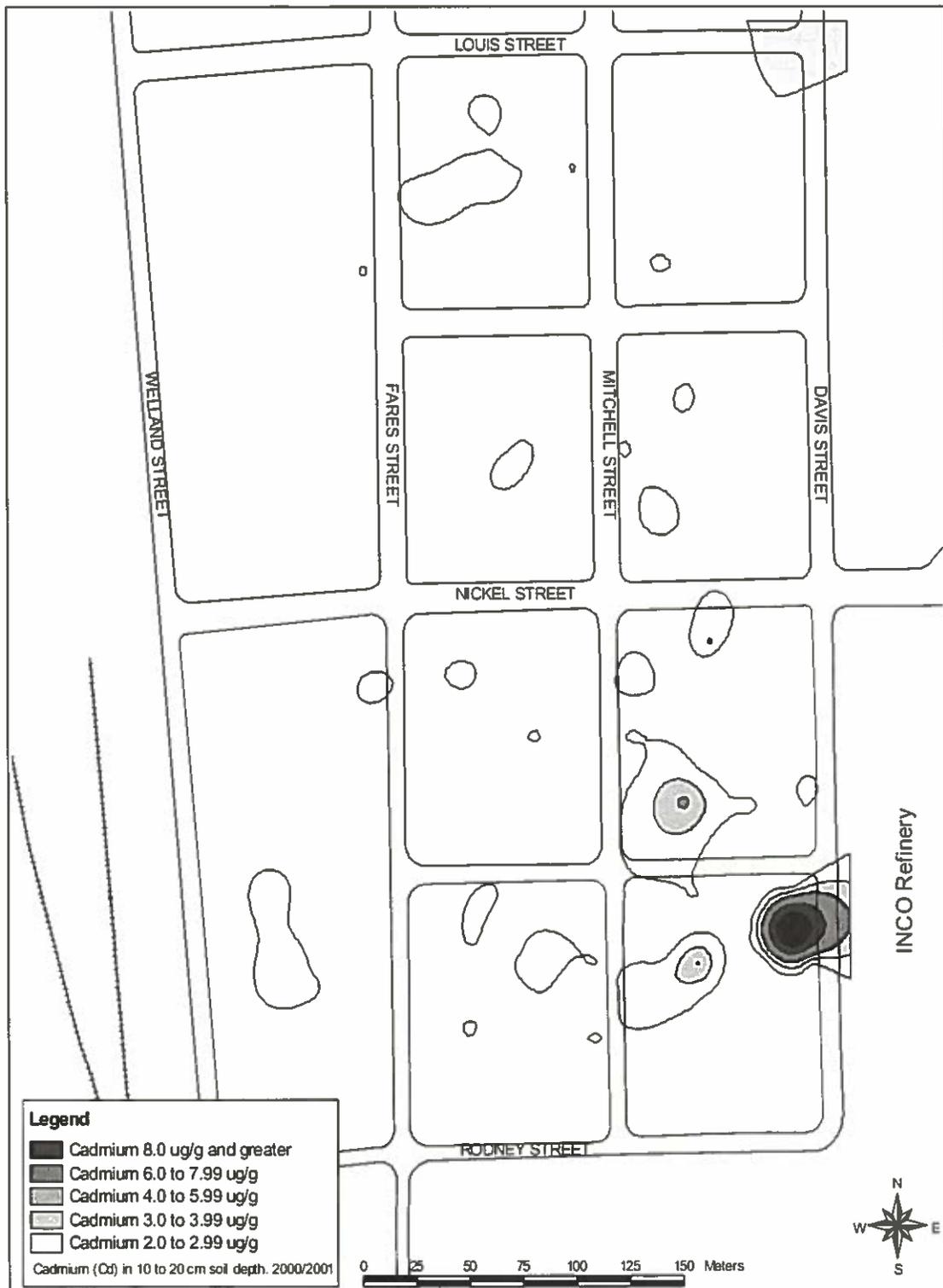
Map 12: Cadmium in soil (10 to 20 cm) in the Rodney St. Community, 2000 & 2001.
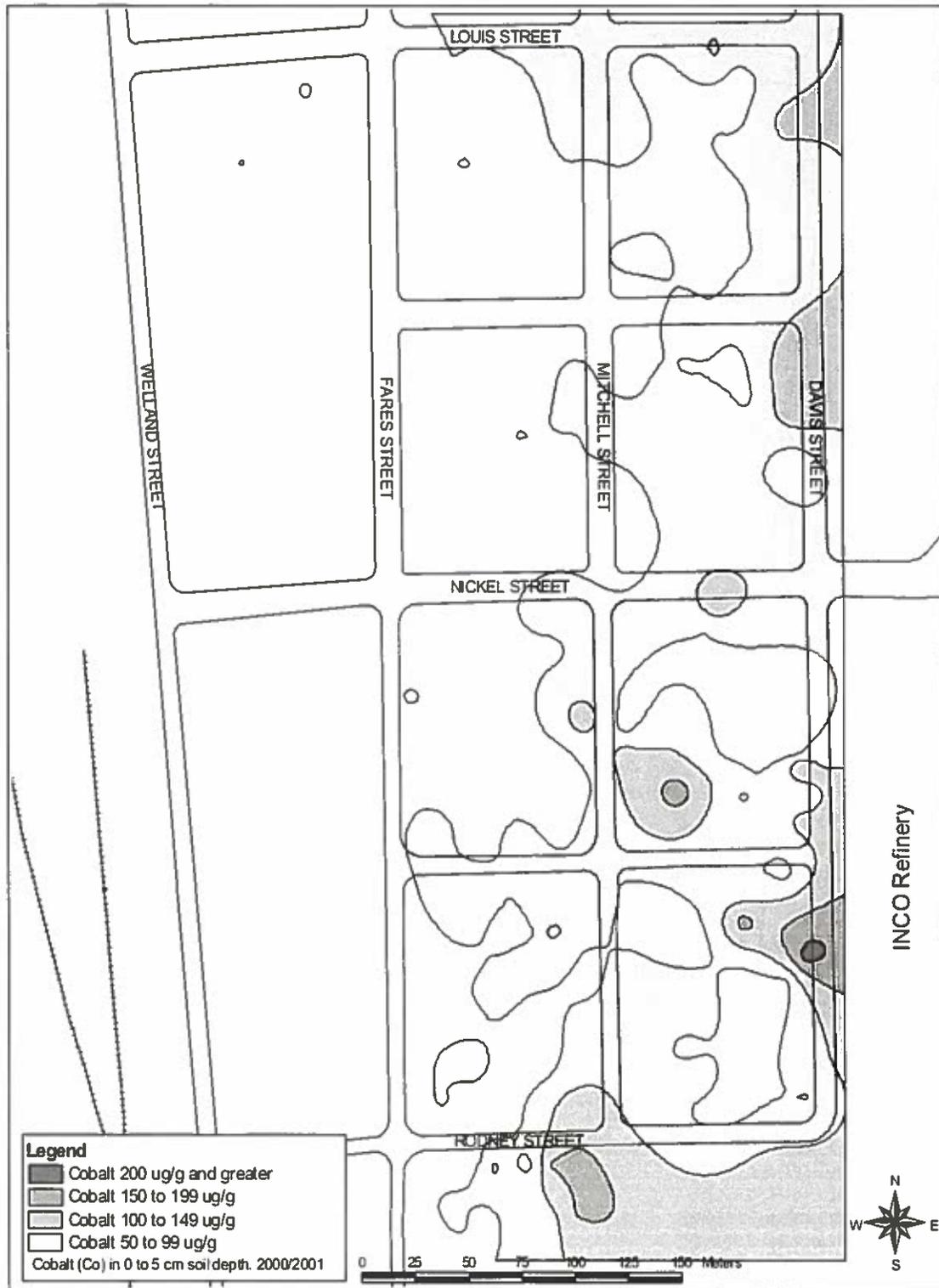
Map 13: Cobalt in soil (0 to 5 cm) in the Rodney St. Community, 2000 & 2001.
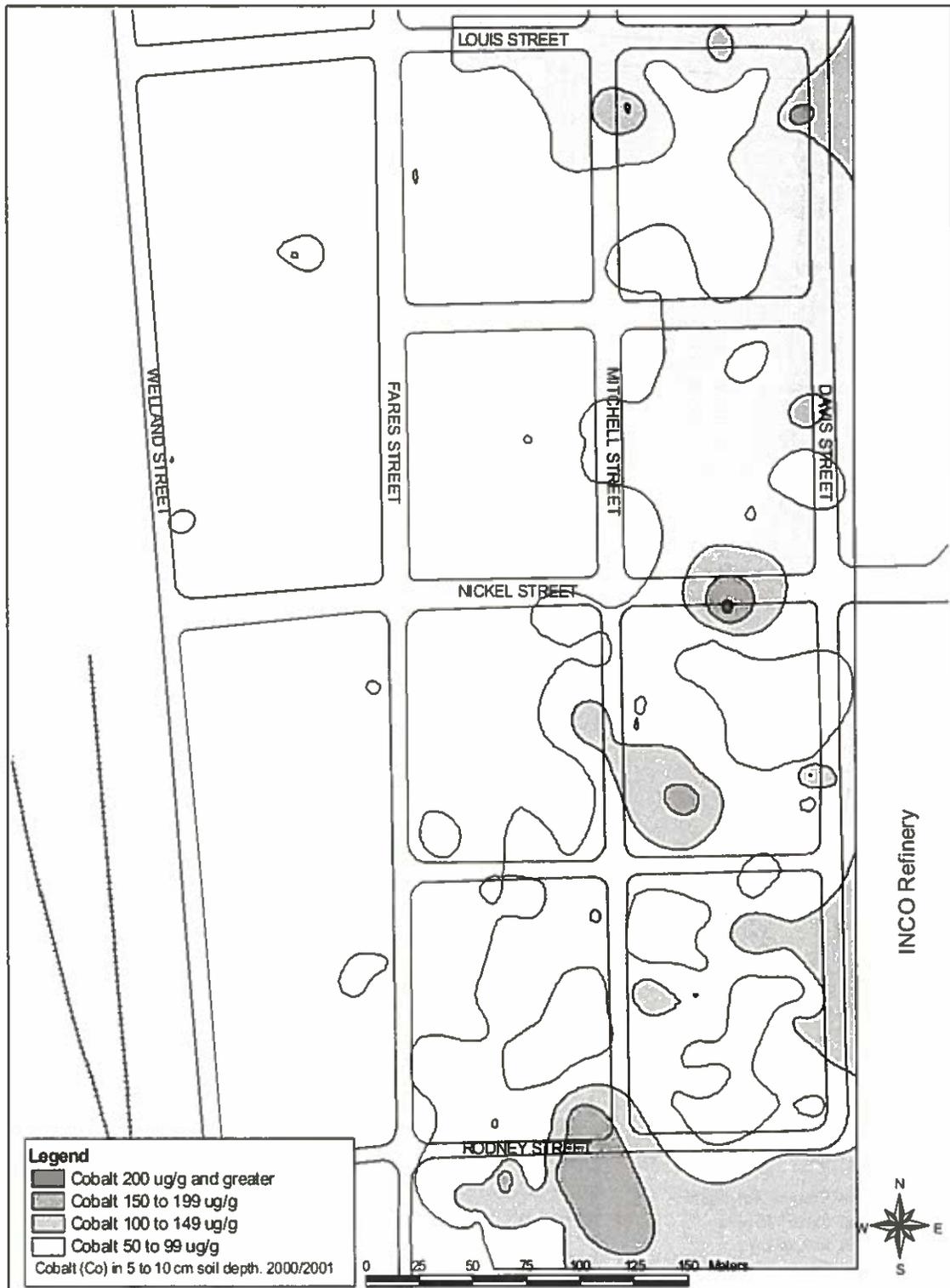
Map 14: Cobalt in soil (5 to 10 cm) in the Rodney St. Community, 2000 & 2001.
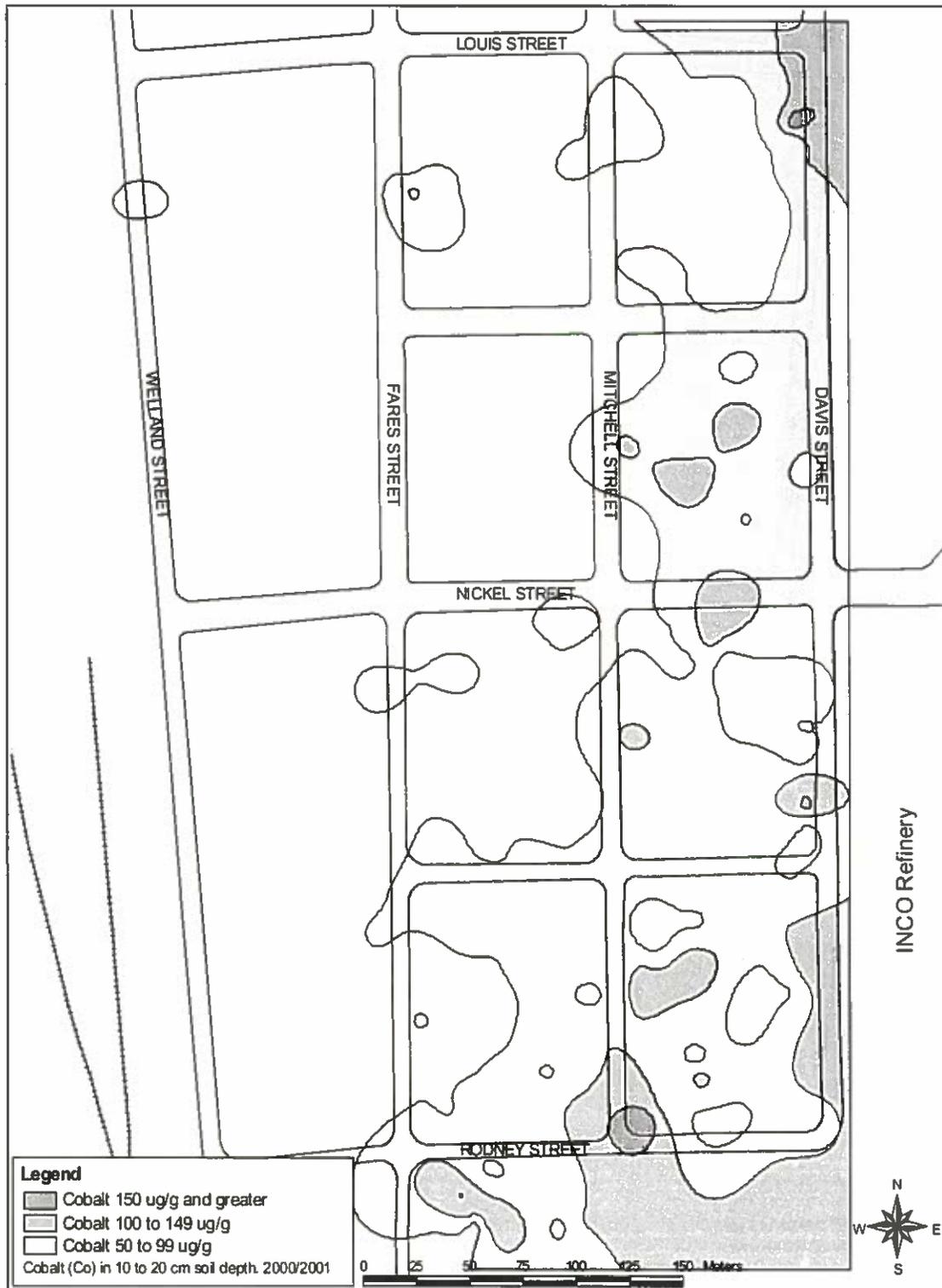
Map 15: Cobalt in soil (10 to 20 cm) in the Rodney St. Community, 2000 & 2001.
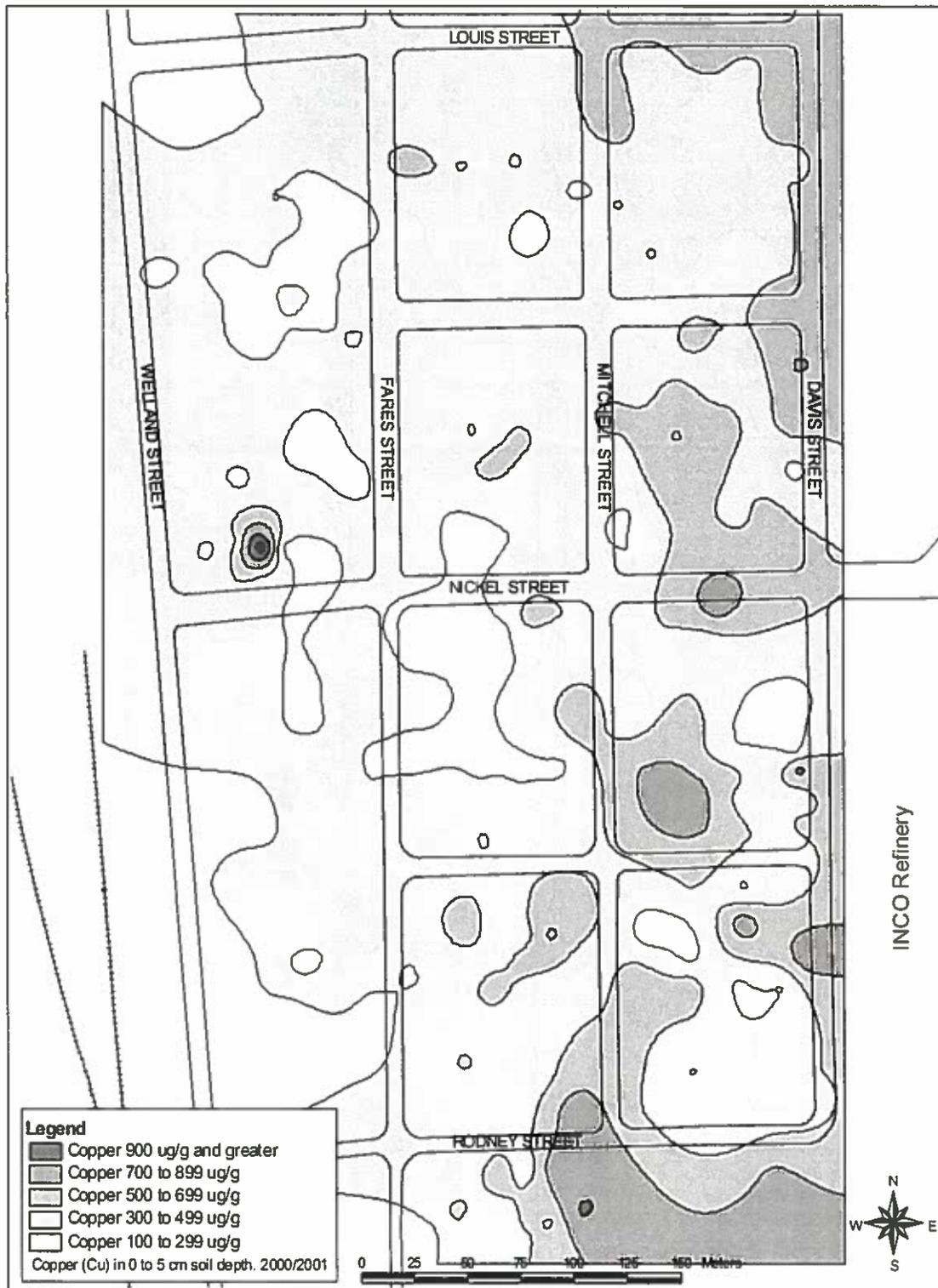
Map 16: Copper in soil (0 to 5 cm) in the Rodney St. Community, 2000 & 2001.

Map 17: Copper in soil (5 to 10 cm) in the Rodney St. Community, 2000 & 2001.

Map 18: Copper in soil (10 to 20 cm) in the Rodney St. Community, 2000 & 2001.
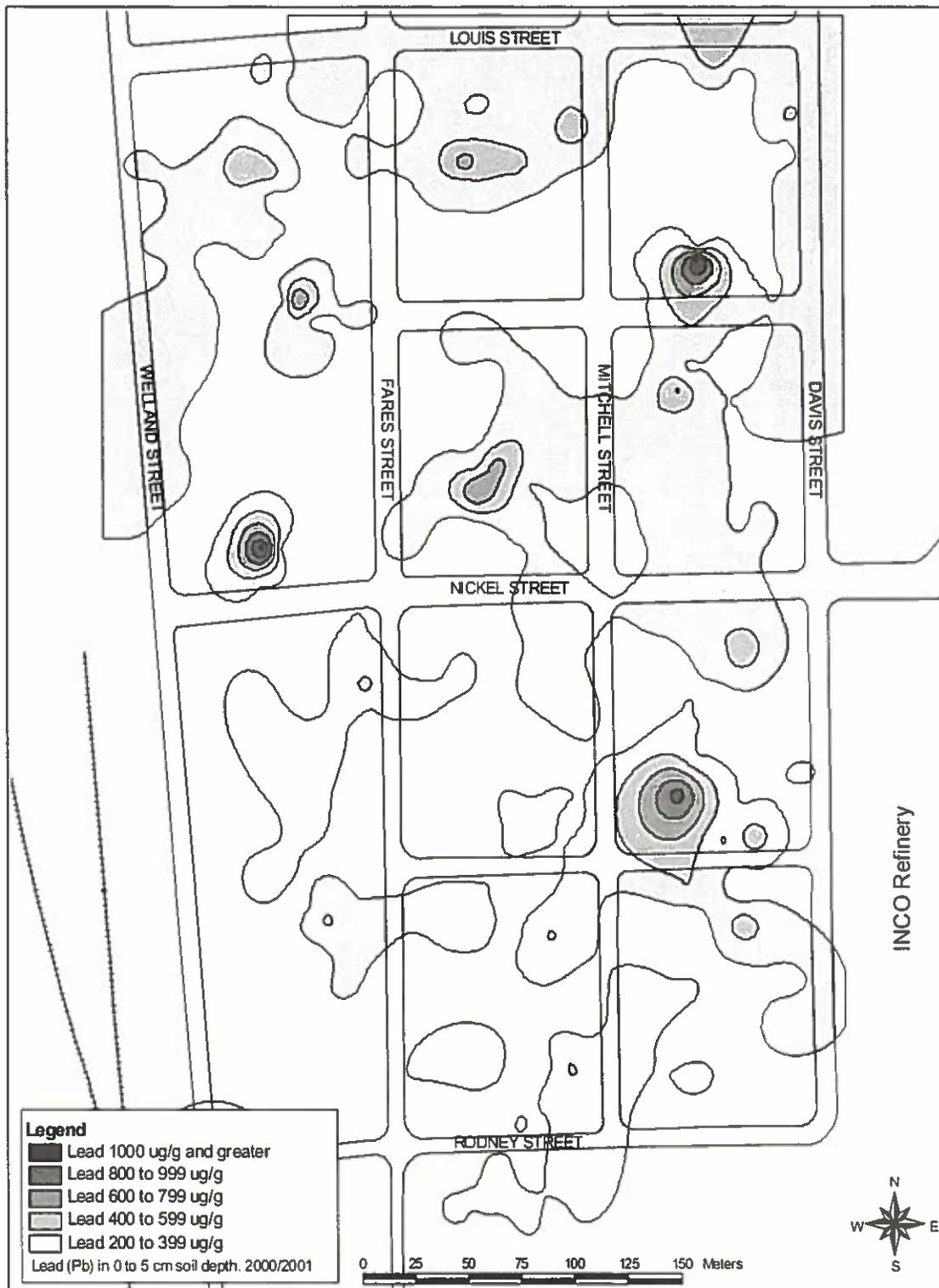
Map 19: Lead in soil (0 to 5 cm) in the Rodney St. Community, 2000 & 2001.
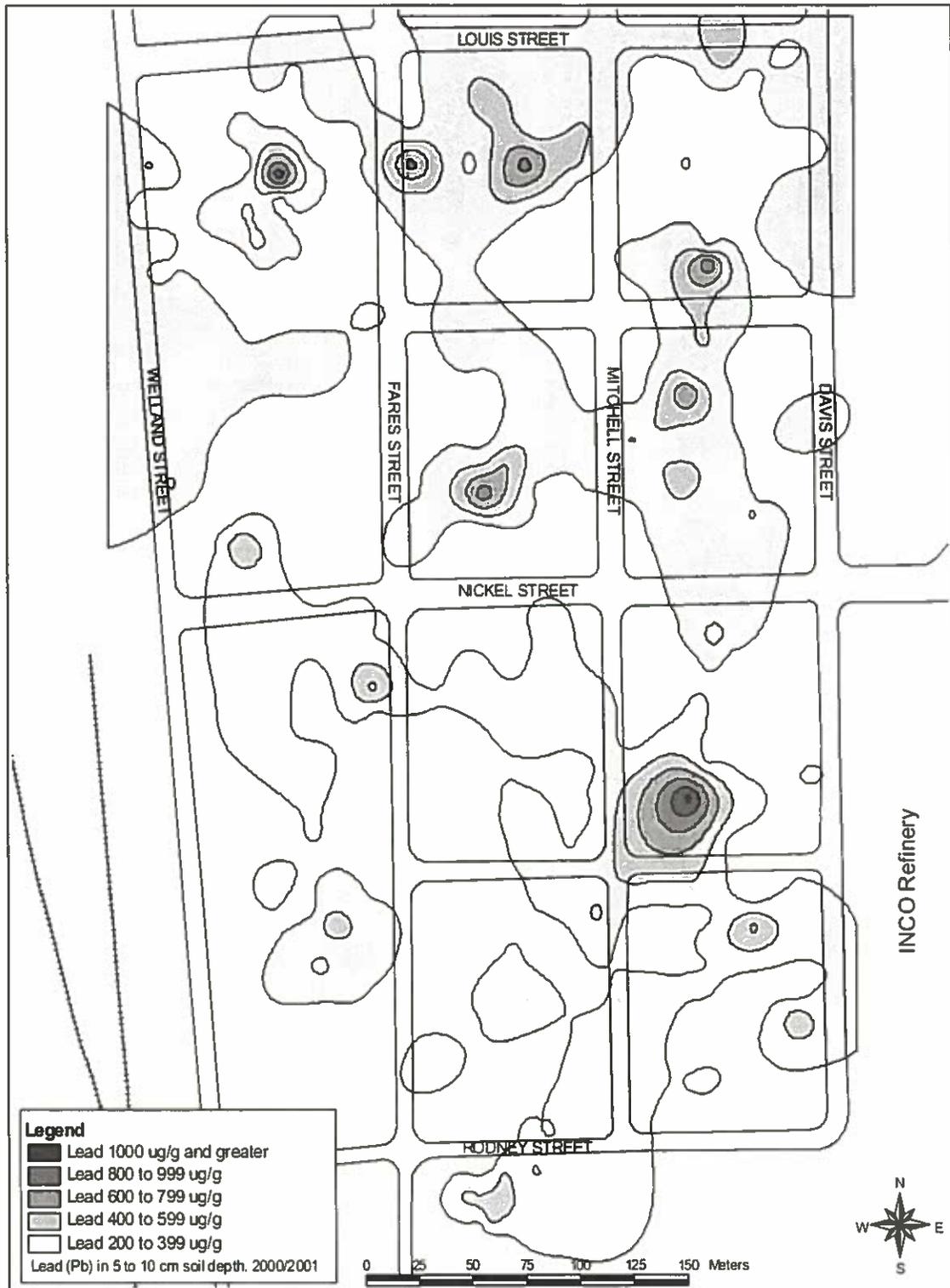
Map 20: Lead in soil (5 to 10 cm) in the Rodney St. Community, 2000 & 2001.

Map 21: Lead in soil (10 to 20 cm) in the Rodney St. Community, 2000 & 2001.

Map 22: Nickel in soil (0 to 5 cm) in the Rodney St. Community, 2000 & 2001.

Map 23: Nickel in soil (5 to 10 cm) in the Rodney St. Community, 2000 & 2001.
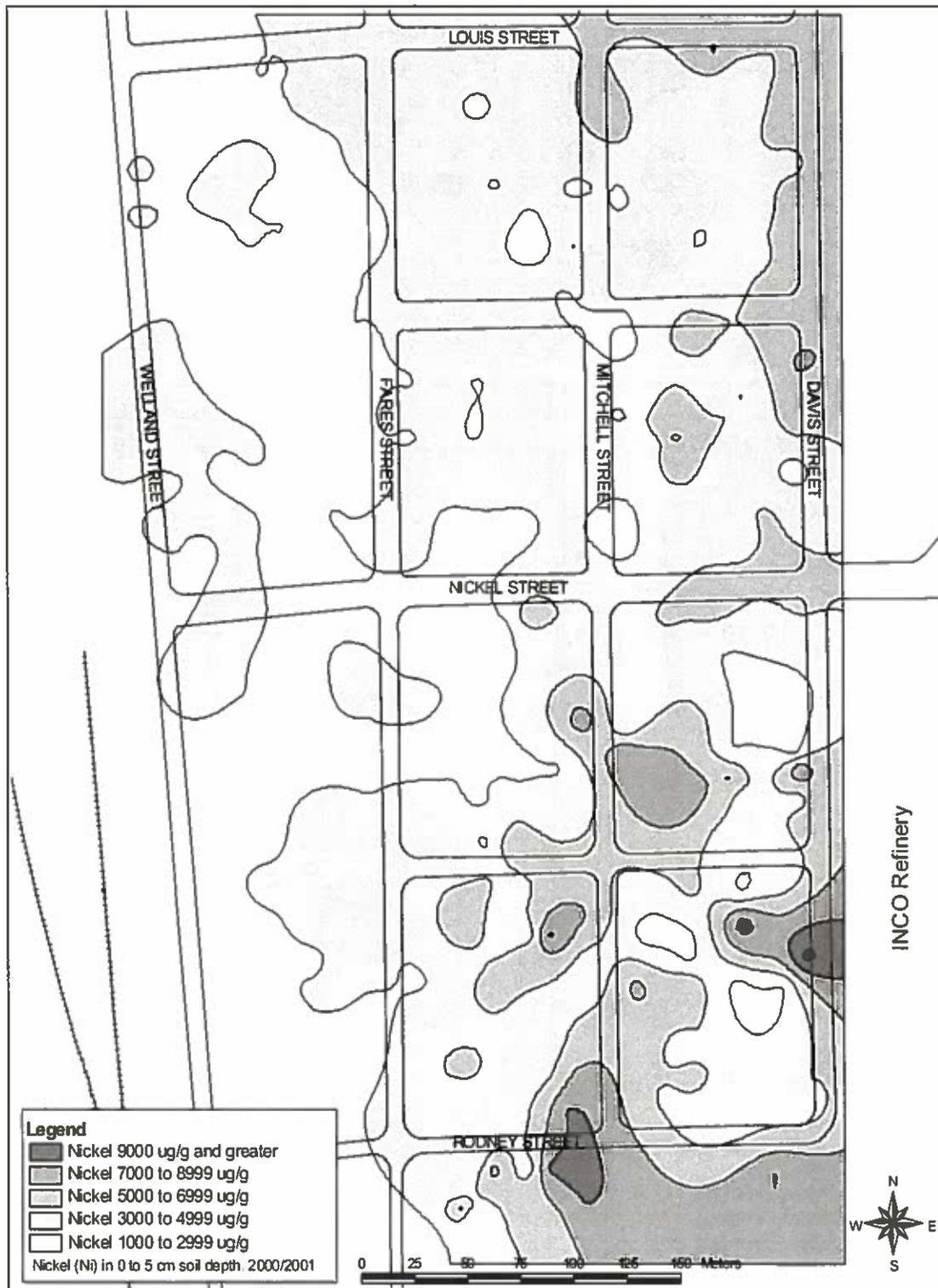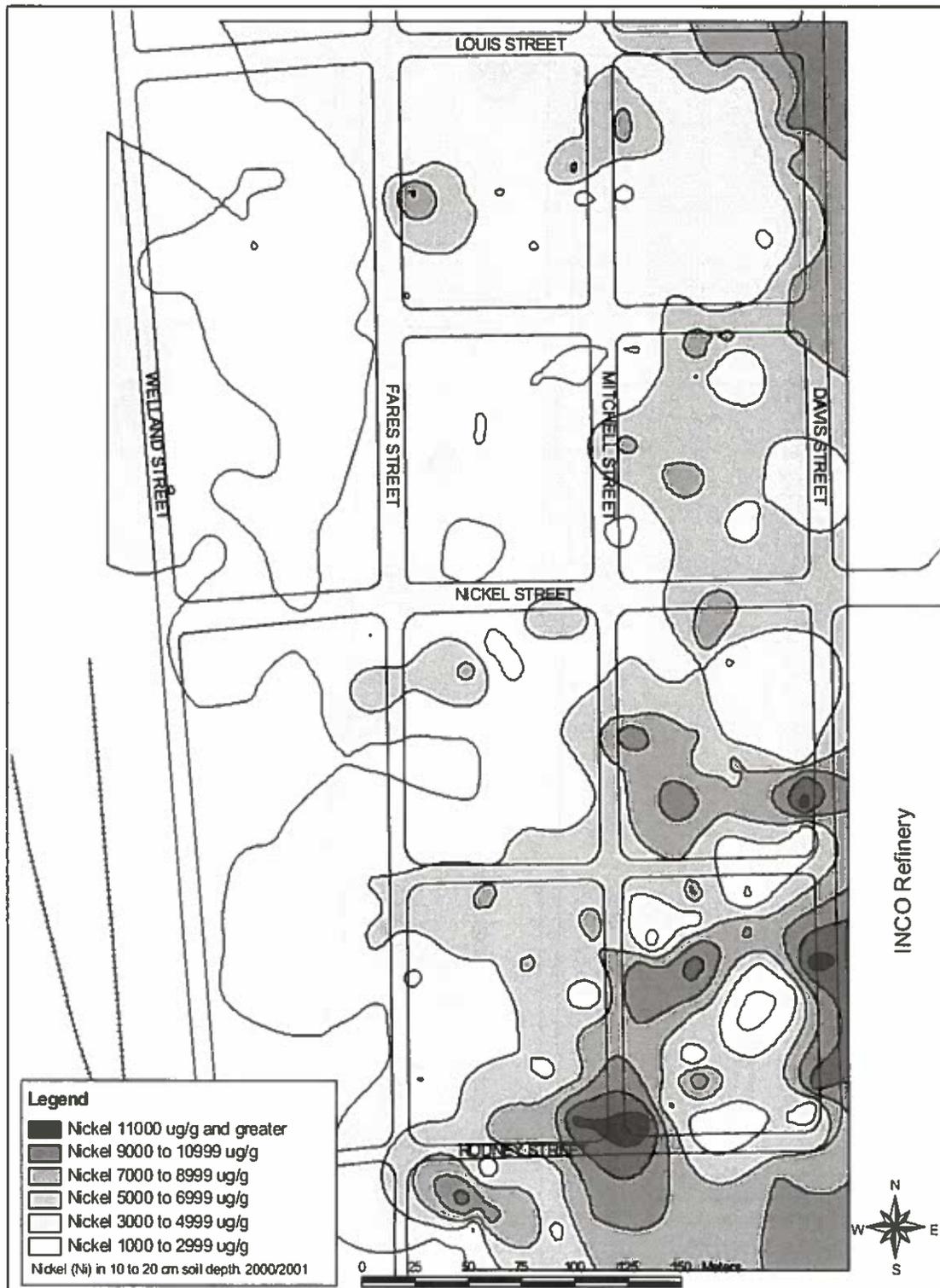
Map 24: Nickel in soil (10 to 20 cm) in the Rodney St. Community, 2000 & 2001.
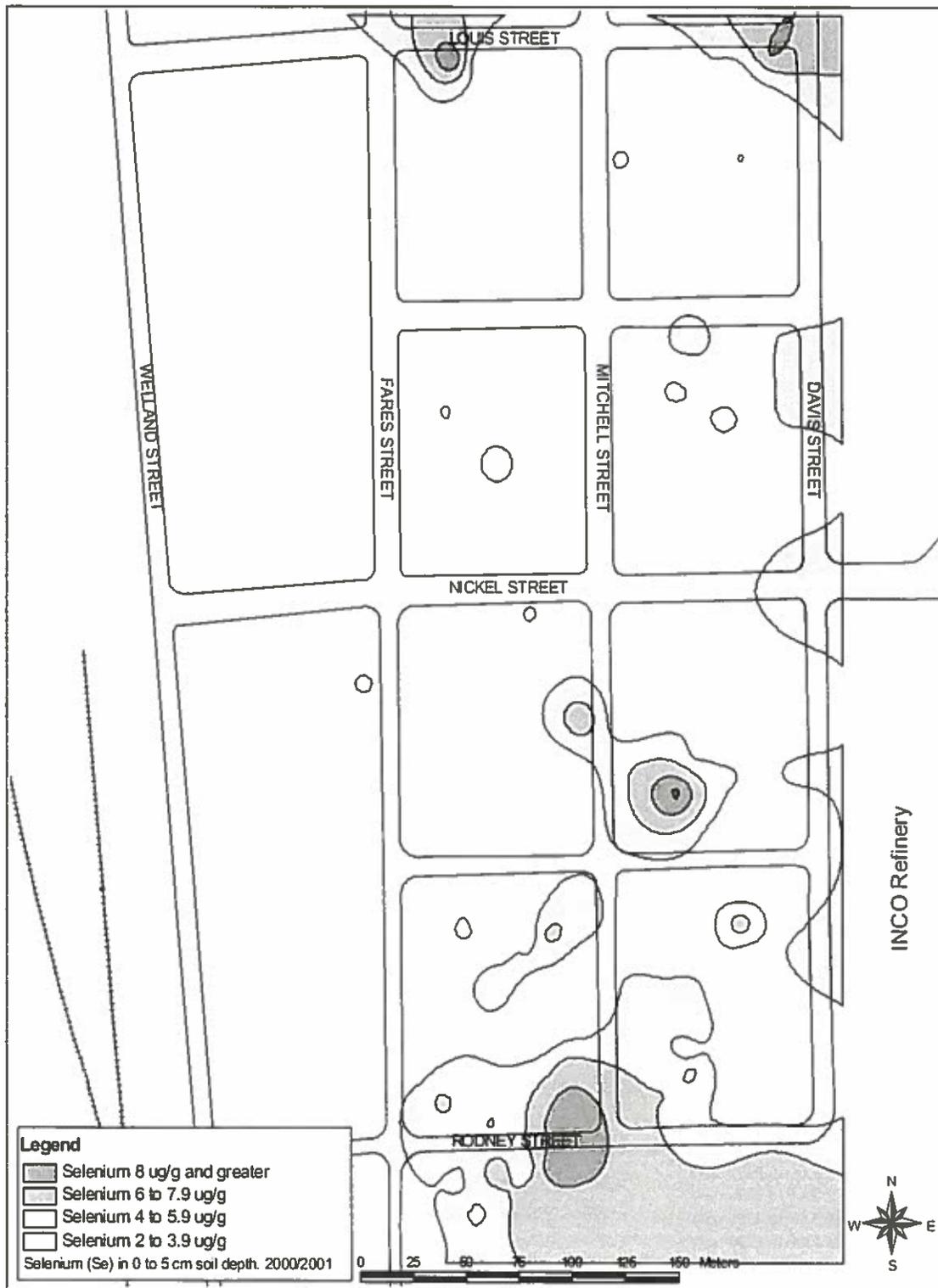
Map 25: Selenium in soil (0 to 5 cm) in the Rodney St. Community, 2000 & 2001.
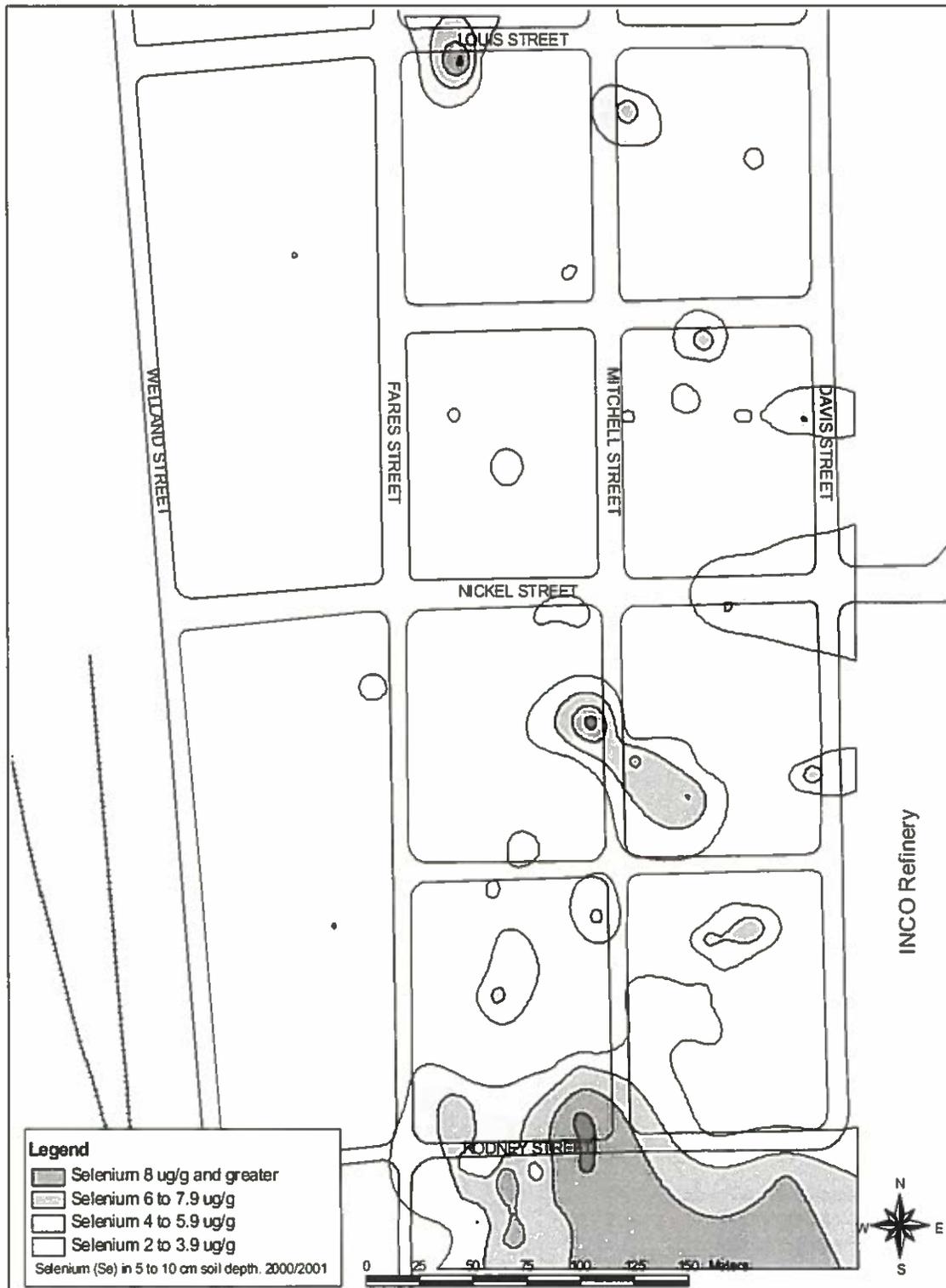
Map 26: Selenium in soil (5 to 10 cm) in the Rodney St. Community, 2000 & 2001.
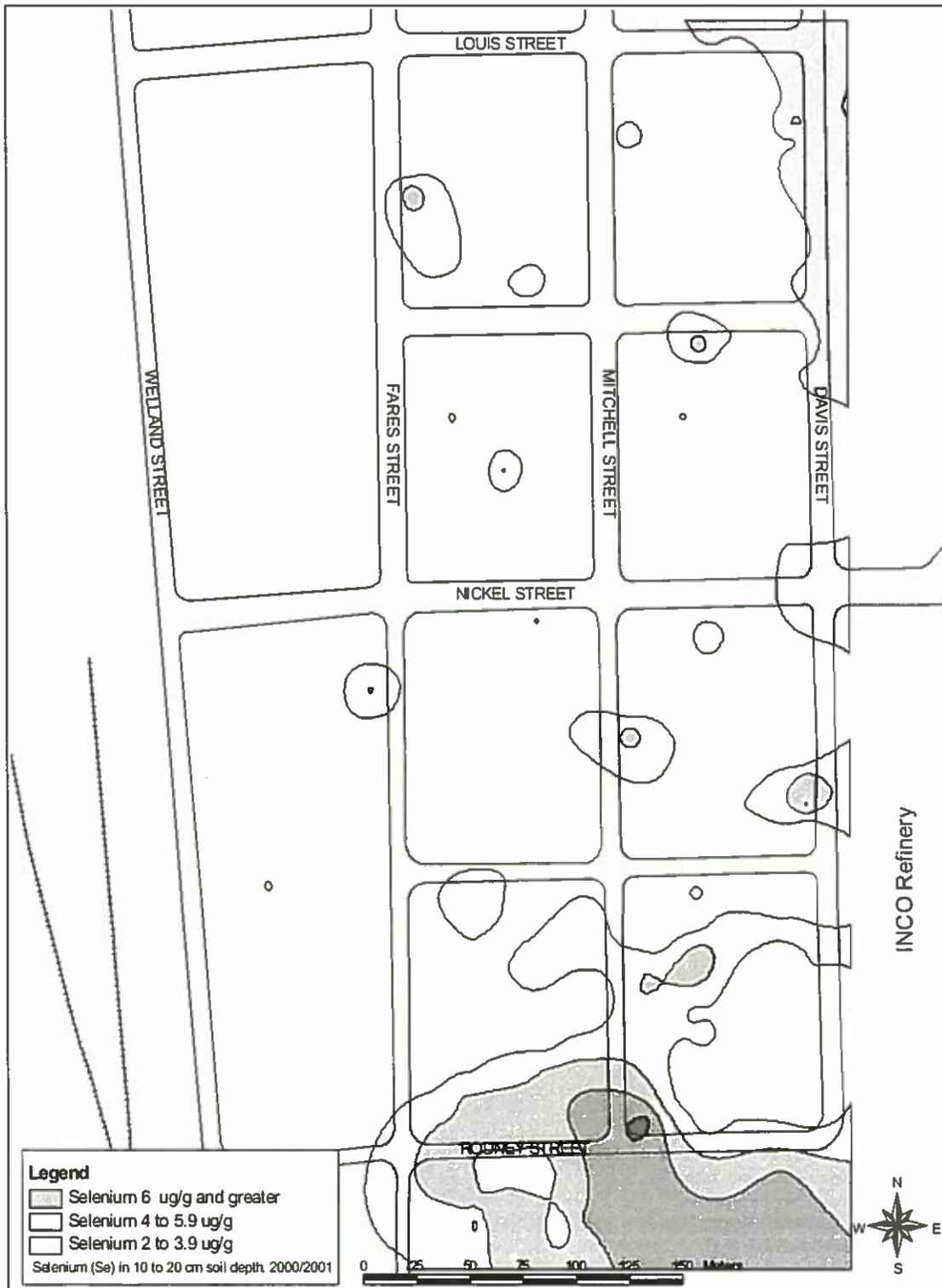
Map 27: Selenium in soil (10 to 20 cm) in the Rodney St. Community, 2000 & 2001.
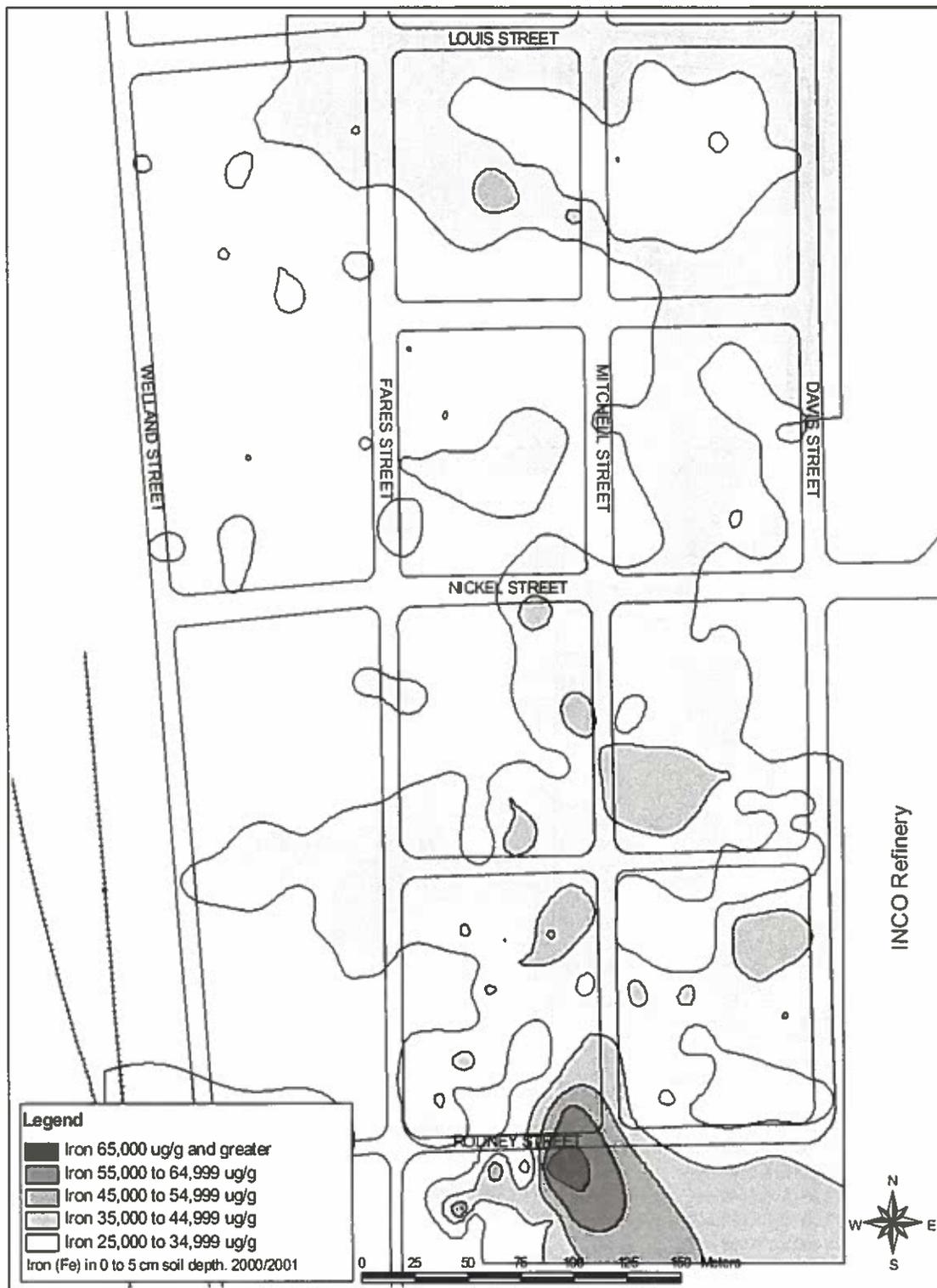
Map 28: Iron in soil (0 to 5 cm) in the Rodney St. Community, 2000 & 2001.

Map 29: Iron in soil (5 to 10 cm) in the Rodney St. Community, 2000 & 2001.

Map 30: Iron in soil (10 to 20 cm) in the Rodney St. Community, 2000 & 2001.

Part B

Human Health Risk Assessment

Main Document

Table of Contents
Human Health Risk Assessment: Main Document

List of Tables
Human Health Risk Assessment: Main Document

1.0 Introduction

This document is part of a combined Ontario Ministry of the Environment (MOE) Standards Development Branch soil investigation (Part A) and health-based risk assessment report (Part B). The health-based risk assessment is designed to answer community health concerns raised by the discovery of elevated levels of nickel and other metals below the normal surface soil sampling depth (0 - 5 cm) on a Rodney Street property in June 2000. The soil metal concentrations were higher than previously measured (Part A, Section 3.3).

While this study is health-based, it is not a community health study. This health-based risk assessment is directed at assessing exposure based on environmental sampling to selected metals in Rodney Street properties to evaluate whether health-based exposure limits are exceeded and whether there is an exposure level (or soil concentration) that warrants further actions (including soil remediation) to reduce exposure to identified soil metal concentrations. The approach presented uses current soil levels based on the most recent soil monitoring information as shown in the accompanying soil investigation (Part A, Section 6.0). Other exposure parameters include levels of metals in outdoor air, drinking water, supermarket food and home grown backyard vegetables and fruits. This approach takes a snapshot of existing exposures. Historic exposures are only addressed to the degree that present environmental concentrations are related to past industrial activities and past usage of the properties by their owners. This information is used to estimate the exposure of Rodney Street community residents without taking measurements from people directly. By contrast, a health study can involve taking blood and urine samples (under medical supervision) to measure human exposures directly. Such procedures are not performed by the MOE. It should be noted that an Inco-sponsored health study (the Ventana Community-Wide Health Assessment Project (CHAP)) is ongoing in the Port Colborne area as part of the Community Based Risk Assessment (CBRA).

For historical reasons and the proximity of the Inco metal refinery, the primary focus of the investigation was directed at the widespread and elevated levels of nickel in the community. Initially this study was targeted at performing a detailed human health risk assessment (HHRA) for this metal. However, as information on other metals became available, a need to assess the potential for health risks due to these other metals was indicated, and a detailed HHRA for each of these other metals was performed. The other metals were initially selected for further study on the basis that their soil concentrations exceeded the residential soil quality criteria (Table A) of the ministry's *Guideline for Use at Contaminated Sites in Ontario* (MOEE, 1997). Exceedance of the Table A guidelines does not necessarily imply that exposure constitutes an undue risk to health because the Table A guidelines are generic and several are based on ecotoxicological effects. Health based Table A guidelines incorporate an adequate margin of safety and are set well below any concentration where health effects might occur. Refer to Section 4.0 of Part A for a more detailed discussion of the derivation and application of these guidelines.

Because of the extensive knowledge of risks related to arsenic and lead in soil, particularly

through similar and more detailed risk assessments and health studies in other Ontario towns and cities, a careful analysis comparing levels, conditions and risk in these other situations to levels and conditions in the Rodney Street community allowed meaningful insight into the question of possible increased risk. Additionally, for both lead and arsenic, a weight of evidence approach with consideration of various factors from the most recent scientific and regulatory literature including recent assessments conducted in other Ontario communities, are used to support derivation of appropriate risk management strategies.

1.1 Purpose and Scope

A human health risk assessment of the elevated metal(s) concentrations found in the soil in the Rodney Street community of Port Colborne, Ontario was conducted by the Ontario Ministry of the Environment and released on March 30, 2001. On May 2, 2001, a letter was sent to the residents of the Rodney Street community advising them that the ministry was reassessing its March 30th report. This reassessment was due to the discovery of a calculation error in one of the test methods used by the ministry in establishing the intervention level. In addition, the ministry received a number of comments with respect to the ministry being either too stringent or not stringent enough in some of the values it used and the approach it took in developing the intervention level for nickel.

In response to these developments, the ministry initiated new studies to verify the bioaccessibility of metals in the Rodney Street community soils, and to obtain more information on the speciation of nickel in these soils. This new information and the comments received resulted in a revised report that was released as a draft document for public consultation in October 2001. The ministry considered all comments received through the public consultation process, and submitted a revised draft for further peer review by an international expert panel prior to public release of this final report (see Appendix 8).

MOE adopted a risk assessment framework to evaluate the environmental and human health risks of metals in Rodney Street community soils. The risk assessment paradigm which has dominated the regulatory decision making processes for the past two decades is the one promulgated in the 1983 US National Research Council (NRC) document, "Risk Assessment in the Federal Government: Managing the Process" (NRC, 1983a). This risk assessment paradigm and local variations on its theme have been adopted worldwide. As well as U.S. agencies, it is used by Canada and the Provinces, WHO (World Health Organization) and jurisdictions in most countries. Detailed methods for interpreting toxicological information and the various exposure pathways are extensive and constantly being updated as the science evolves.

The intent of the Rodney Street community risk assessment was two-fold. One objective was to assess the potential for health risks from current exposure pathways (soil, air, drinking water and food). The second objective was to use pathway-specific information to develop soil intervention levels to protect against any immediate or future health risk for this community.

The human health risk assessment makes use of environmental monitoring data and recent toxicological information to estimate exposures and potential health effects. Current toxicological information is examined to determine the types of health effects which have been reported following exposure to each of these metals (hazard identification), and to identify the levels of exposure at which the reported effects were manifested (dose-response assessment). It also makes use of multi-pathway modeling to estimate the total exposure to each of these metals which are likely to occur (exposure assessment). It then combines the toxicological and exposure information to estimate the potential health effects which may occur (risk characterization). Each of these components, hazard identification, exposure assessment, dose-response assessment and risk characterization has been described in detail in previous health risk assessment reports which have evaluated potential health risks associated with exposures to various metals in the soils in Port Colborne (MOE, 1998) and other locations in Ontario (MOE, 1991; 1999; 2001a; MOEE, 1995).

The human health risk assessment includes the following components:

- *A multimedia approach*, which considers total exposure from all environmental media, was chosen to characterize the risk. The approach recognizes that contaminants are present simultaneously in food, air, water, consumer products, soil and/or dust.

- *The exposure pathways of concern*, which include inhalation and incidental ingestion of soil particles derived from backyard soils, dermal contact with the soil and ingestion of backyard produce. In addition, exposures to supermarket food, ambient air and drinking water are estimated. The exposure model estimates daily intakes from all exposure pathways for different age classes (infant, toddler, child, teen and adult). Food basket data included recent Canadian Market Basket Survey information and backyard vegetable data collected from both the Rodney Street community and the Port Colborne area.

- *Important receptors,* which include, infants, toddlers, children, teens and adults. Toddlers (aged seven months to less than five years) represent the most important receptor due to their increased exposures to soil and hand-to-mouth behavior compared with other receptor age groups.

- *Assessment of the bioaccessibility of nickel (and other metals) from soil*. The use of an *in vitro* simulated gastrointestinal extraction method (bioaccessibility) for nickel (and other metals) in soil was investigated using both simulated stomach acid leach and bioaccessibility test data.

- *Dermal exposure to nickel (and other metals)*. The dermal exposure pathway was examined as an intake pathway, however, even though contact dermatitis (for nickel) is a relatively common occurrence (approximately 10%) in the general population (i.e., due to contact with coinage, jewelry and stainless steel objects), oral and dermal exposure limits

for this endpoint have not been developed by other regulatory agencies.

- *Toxicological assessment of nickel (and other metals)*. The dose-response assessment, assessed cancer and non-cancer exposure limits based on nickel species characterization (other metals were not speciated).

- *Development of health-based site-specific intervention levels*. An important output of the risk assessment process are intervention levels. These are tools for evaluating and making decisions for cleaning up contaminated soils.

Another paradigm for evaluating the association between human health risks and exposures to chemicals and other substances or disease causing agents is epidemiology. Well documented epidemiological data has a clear advantage over animal studies, since concerns about extrapolating from animals to man do not arise. The epidemiology paradigm is based on a number of criteria for establishing causality, these include:

    a) the strength of association as measured by the relative risk;
    b) the consistency of the association;
    c) the temporal association between cause and effect;
    d) a dose-response relationship; and
    e) specificity of association.

Human epidemiological studies have their own set of limitations related to proving that there is a causal association between chemical exposure and effects in the population. As described in section Appendix 2-9.2.3.2, the inhalation unit risk factors for estimating lung cancer risk from inhaling nickel compounds are based on epidemiological studies of nickel refinery workers.

2.0 Identifying Metals of Concern

An extensive sampling program has been carried out for the homes in the Rodney Street community in Port Colborne (Part A, Section 5.1). The monitoring program identified ten metals that are present in the soil at levels that exceed the current Ministry of the Environment guidelines for medium/fine textured soil in a residential community (MOEE, 1997). The range of reported concentrations for each of the ten metals are listed in Table 2-1. The respective MOE Table A criteria are also listed. Because this assessment focuses on human health, metal levels were also compared to the human health specific screening values originally developed for the MOE Table A which are listed in the *Rationale Document* (MOEE, 1996) which is one of three supporting documents for the MOE *Guideline* (MOEE, 1997). The data in Table 2-1 shows that for seven of the ten metals including; antimony, arsenic, beryllium, cadmium, copper, lead and nickel, the highest reported concentrations exceed both the MOE Table A criteria and their respective human health screening value. For the remaining three metals, cobalt, selenium and zinc, the maximum levels reported in Rodney Street community soil are below their respective

human health based criteria. Based on this, cobalt, selenium and zinc would not be expected to be human health concerns for the residents of the Rodney Street community. However, the previous risk assessment undertaken by the MOE included cobalt as a metal of concern. Therefore, cobalt has been carried through the current assessment of exposure and risk. Selenium and zinc have not been carried through to the detailed risk assessment because the screening assessment has shown that these metals are not present in soil in sufficient quantity to warrant further assessment. Based on the screening of metals shown in Table 2-1, eight metals have been identified for inclusion in the detailed assessment of exposure and risk for the Rodney Street community.

For two of the eight metals; arsenic and lead, the MOE has undertaken detailed assessments of exposure and health risk in communities similar to the Rodney Street community (MOE, 1991; 1999; 2001a; MOEE, 1995). The results of these previous assessments and our review of the Rodney Street community (see sections 5.7 Arsenic and 5.8 Lead) have been used to develop management strategies for arsenic and lead in the Rodney Street community of Port Colborne. These strategies are presented in the Recommendations and Conclusions section of the report (Section 7.0). Therefore, the human health risk assessment has focused on the remaining six metals. The detailed exposure assessment used the highest reported concentration of each metal in the soil from the Rodney Street community. The metals carried through to the risk assessment, and the soil concentrations used in the assessment are summarized in Table 2-2.

Because nickel is the major focus of this assessment, a review of default or generic soil guidelines for nickel in a residential context was undertaken. It is difficult to comment on the use and derivation of guidelines developed in other jurisdictions due to a lack of documentation. However, it is apparent that for the most part these guidelines are "triggers" or screening values requiring either action or further assessment, and are not generally soil intervention levels. This tiered approach is similar to the procedure outline in the MOE's guidelines for contaminated sites (MOEE, 1997).

Table 2-1: Summary of Soil Data for the Rodney Street Community

Metal	Concentration in Soil (µg/g)[1]				MOE Cleanup Criteria[2] (µg/g)	
	Minimum	Median	Average	Maximum	Guideline Criterion	Human Health Screening Value
Antimony	0.10	1.20	2.20	91.1	13	13
Arsenic	<0.6	13	16	350	25	-
Beryllium	0.2	1.0	1.0	4.6	1.2	0.37
Cadmium	<0.14	1.1	1.2	35.3	12	14
Cobalt	5	39	51	262	50	2,700
Copper	4	196	246	2,720	300	1,100
Lead	6	173	217	1,800	200	200
Nickel	19.0	1,730	2,508	17,000	200	310
Selenium	0.10	1.80	2.4	19.4	10	320
Zinc	23	305	365	1,750	800	16,000

1: (0-20 cm; based on approximately 2000 sample points).

2: Table A/B criteria for metals in residential/parkland soil for medium/fine textured soil.

Table 2-2: Metals Considered in the Exposure Assessment

Metal	Soil Concentration (µg/g)
Antimony	91.1
Beryllium	4.6
Cadmium	35.3
Cobalt	262
Copper	2,720
Nickel	17,000

3.0 Toxicity Assessment

The screening of chemicals in the soil in the Rodney Street community identified eight metals of potential concern (Section 2.0). The objectives of the toxicity assessment are:

- S   to provide the reader with a brief understanding of the toxicological effects that have been reported to be associated with exposure to the chemicals of concern;
- S   to identify whether each metal of concern is considered to have carcinogenic or non-carcinogenic effects; and
- S   to identify suitable exposure limits against which exposures can be compared to provide estimates of potential health risks.

The toxicological profiles are *not* intended to:

- S   be exhaustive examinations of all the toxicological information available for each metal;
- S   be used to develop exposure limits for exposure routes where no exposure limits are available; or
- S   critically review and/or modify currently existing exposure limits set by Agencies such as the US EPA, WHO, Health Canada, etc.

This toxicity assessment outlines the toxicological effects that have been reported to be associated with inhalation, ingestion and dermal contact exposures to antimony, arsenic, beryllium, cadmium, cobalt, copper, lead and nickel, and identify whether each metal should be considered as a carcinogen or a non-carcinogen based on the exposure pathway. The type of exposure limit selected is dependent upon whether a compound is considered to have non-carcinogenic or carcinogenic effects. However, in some cases, e.g., lead and cobalt, cancer potency factors are not available. The types of exposure limits associated with both types of compounds are discussed below (Tables 3-1 and 3-2).

The relevance of toxicological endpoints derived from animal studies to humans is a challenging task from a risk assessment perspective. The primary difficulty is that the contaminant levels the study animals are typically exposed to are much higher than exposures faced by human populations. The effects of these high exposures are assessed by a variety of histopathological methods.

In most cases, the molecular mechanisms causing the metal related adverse effects are not well understood, and in many cases, there is the uncertainty that the mechanisms leading to an adverse effect may differ between metal species as well as between the route of exposure. In addition, many animal studies are based on a metal species that is convenient to administer but may not be the one to which human populations are exposed.

The finding of adverse non-cancer effects in animals is then related to human populations by the use of uncertainty factors. Uncertainty factors provide a safety margin in the extrapolation of the estimates of adverse effects when the mechanisms leading to adverse effects are not well understood (discussed in section Appendix 2-1).

In the case of carcinogenic compounds, that may act by damaging DNA, which are generally considered to work through a non-threshold mechanism which means that an adverse effect is assumed to have the potential to occur at any level of exposure. Any exposure to a carcinogen is considered to be associated with some level of risk. At very low doses, the probability that an adverse effect (cancer) will occur is extremely small. The probability of developing cancer increases as the dose increases. Cancer potency factors are developed by extrapolating from animal test data or cancer incidences in exposed human populations. Incremental increases in life-time cancer risk are estimated by comparing the established potency for each compound with the calculated chronic daily intakes (CDI) for that compound. In addition, potential life-time cancer risks from inhaling airborne metals can be assessed by comparing the annual average air concentration with inhalation unit risk factors from various agencies (Table 3.2).

The toxicological profiles also examine the effect that the route of exposure has on the toxicological activity of each compound. For some compounds, the route by which the compound enters the body can have a marked effect on the toxicological effects that occur. In cases where the toxicological effects of a chemical differ between the routes of exposure, it is necessary to assess inhalation and ingestion exposures independently. For example, exposure to beryllium, cadmium and nickel by inhalation may be carcinogenic, but the data suggests they are not proven to be carcinogenic if the exposure is via ingestion. However, arsenic may be carcinogenic following either inhalation or ingestion exposure. Therefore, where route-specific exposure limits are available, the toxicological profiles will provide both. In cases where exposure limits are available for a single route of exposure, the toxicological profiles will not develop exposure limits by route-to-route extrapolation. Although complex route-to-route extrapolation is undertaken in some situations, it is typically discouraged by the US EPA and similar regulatory agencies because it requires detailed knowledge of pharmacokinetic and pharmacodynamic factors and extensive modeling. All of which are beyond the scope of the current assessment.

The selection of exposure limits (cancer and non-cancer) requires consideration of a number of important attributes of the available values. The framework and methods for developing exposure limits are well described by all major agencies.

Evaluation of any study used to estimate human health risk based on either toxicity testing in laboratory animals or an epidemiological study of exposed humans, utilizes extensive scientific criteria to determine the reliability of the testing protocols used. Other factors, such as the health of the animals during the test, the appropriate determination of toxicity endpoints, the statistical significance of any dose-response relationships found, and the occurrence of any confounding

factors that affect the results are also considered. Epidemiological studies have a different set of rules to determine causal association between disease endpoints and human exposure patterns.

In terms of animal testing studies, the age of the study is not necessarily as important as the following of recognized and approved protocols for treating and exposing the animals to the substance being tested and identifying the resulting adverse effects. The statistical reliability of the data demonstrating the presence or absence of adverse health endpoints or any apparent dose-response relationship arising from the study are an important factor. More recent testing information generally benefits from more recent developments in toxicology and adds to the weight of evidence used to develop exposure limits.

Adoption of a particular animal testing (or epidemiology) study by regulatory agencies as supporting documentation for their exposure limits, is an important criterion for evaluation and selection of relevant toxicological information. Regulatory agencies then use such criteria to select studies to support the development of exposure limits, e.g. reference dose (RfD), tolerable daily intake (TDI), etc. In addition, using the dose-response (or comparable extrapolation techniques from epidemiology studies), agencies apply uncertainty factors (UFs) for threshold, non-cancer endpoints, or mathematical low dose extrapolation methods for non-threshold, cancer endpoints.

Expert judgement is required in both the application of uncertainty factors to the identified threshold value and the extrapolation to low dose estimates. While RfD or risk-based concentrations can be generated using all the appropriate risk assessment protocols and principles, the resulting exposure limits may not be immediately achievable. This may be due to background concentrations, intakes from specific pathways, technical feasibility or other reasons, though none of these reasons should necessarily preclude the use of well documented and reviewed exposure limits. It should be noted that just because one exposure limit is lower than another does not necessarily imply that the limit provides additional safety, since most exposure limits incorporate adequate margins of safety. Also, how an exposure limit is applied to reduce risk can have implications for the ultimate "safety" of the value. Consequently, while exposure limits from different agencies using current, reliable toxicological information may vary, any claims as to their relative safety vis-a-vis each other requires an assessment of how the exposure limits are applied.

A summary of the information contained in Appendix 2 is provided in Table 3-1 and Table 3-2. For each of the six metals selected for detailed risk assessment, the selected reference dose, toxicological end point and reference to the appropriate section of Appendix 2 is provided. These selected exposure limits have been used in conjunction with the exposure estimates (Section 4.0) to characterize potential risks (Section 5.0) associated with exposures to each of the metals in residential soil in the Rodney Street community.

Table 3-1: Selected Exposure Limits and Toxicological End Points for Non-Carcinogenic Effects

Assessed Compound	Route	Non-Cancer End Points		Appendix Reference
		R/D/R/C[1]	End Point	
Antimony	Oral	0.4 µg/kg-day	decreased longevity and altered blood chemistry in rats	A2-2
	Inhalation	0.2 µg/m³	pulmonary toxicity in rats	
Beryllium	Oral	2 µg/kg-day	intestinal lesions in dogs	A2-4
	Inhalation	0.02 µg/m³	beryllium sensitization in human populations	
Cadmium	Oral	1 µg/kg-day	kidney damage in humans	A2-5
	Inhalation	NC[2]	NC[2]	
Cobalt	Oral	20 µg/kg-day	kidney effects in renally compromised patients	A2-6
	Inhalation	0.03 µg/m³	squamous metaplasia in rodent larynx	
Copper	Oral	140 µg/kg-day	liver damage	A2-7
	Inhalation	2.4 µg/m³	inhalation chronic reference exposure limit	
Nickel	Oral	20 µg/kg-day	reproductive effects and decreased body and organ weights in rats	A2-9
	Inhalation	NC	NC	

1. R/D/R/C - reference dose/reference concentration.

2. NC - exposure limit not considered for use in this human health risk assessment.

Table 3-2: Selected Cancer Potency Values for Contaminants of Concern

Compound	Route	Cancer End Points			Appendix Reference
		UR[1]	SF[2]	End Point	
Beryllium	Oral	NA[3]		NA[3]	A2-4
	Inhalation	2.4×10^{-3} $(\mu g/m^3)^{-1}$		lung cancer in humans	
Cadmium	Oral	NA		NA	A2-5
	Inhalation	1.8×10^{-3} $(\mu g/m^3)^{-1}$		lung cancer in cadmium workers	
Nickel	Oral	NA		NA	A2-9
	Inhalation	2.4×10^{-4} $(\mu g/m^3)^{-1}$; 3.8×10^{-4} $(\mu g/m^3)^{-1}$ and TC_{05} $(40 \mu g/m^3)$ *		lung cancer in nickel refinery workers	

1. UR = Unit Risk = risk per ($\mu g/L$) oral or ($\mu g/m^3$) in air.
2. SF = Cancer slope factor = risk per dose body weight i.e., per ($\mu g/kg$-day).
3. NA = No value available or not considered carcinogenic via oral exposure.
* TC_{05} = Tumourigenic Concentration (05) = concentration in air associated with 5% increase in incidence or mortality due to tumors.

4.0 Exposure Assessment

The current assessment has been undertaken to provide interested/concerned parties with estimates of the metal exposures that could be experienced by people living in the Rodney Street community. Like all residents of Ontario, people living in the Rodney Street community are exposed to metals from a number of sources including, processed food, drinking water and air. In addition to these general exposures that are common to the population of Ontario, the residents of the Rodney Street community can be exposed to metals in dust, soil and in home grown produce. A detailed assessment was undertaken for people living in the Rodney Street community to develop estimates of the total daily exposure experienced by people of all ages. Specific details of exposure assessment methods are found in Appendices 3 to 7.

Two exposure areas not addressed in detail are occupational exposures and the indoor environment.

The MOE's mandate is to protect the environment and human health as it is related to the environment. MOE does not directly address workplace exposures. Worker safety and exposures in the workplace are regulated by the MOL (Ministry of Labour) and other groups.

The indoor setting is a complex, enclosed environment which is subject to many activities, voluntary and otherwise. In addition to impacts from the outdoor environment, a complex array of substances are released from cooking, heating, consumer products, furnishings and pets. Smoking can have a significant impact on indoor air quality. Certain hobbies and home renovation / decoration can also release contaminants into the home environment.

Jurisdictionally, various agencies have a role related to:

- whether the indoors is a workplace or not (this includes schools, daycare facilities and mixed residential / commercial situations);

- public health issues;

- home construction and consumer goods; and

- whether industrial emissions, discharges or other sources of contamination impinge on the indoor environment.

In this particular assessment, the MOE focused on estimating indoor exposures that may result from outdoor air and soil from the surrounding property entering a home. Enforcement of its regulations and guidelines to protect outdoor air quality, drinking water quality and soil quality (the province only has limited jurisdiction over food quality and consumer products) is the main way that MOE protects environmental quality and human health.

The on-going Community Based Risk Assessment (CBRA) process will be examining the indoor exposure issue for Port Colborne.

4.1 Receptor Identification
4.1.1 Identification of Potential Receptors

The Rodney Street community in Port Colborne is a residential neighborhood. The properties are municipally serviced with domestic water that is not derived from groundwater in the area. People living in the homes in the Rodney Street community will be exposed to the metals present in the soil, but not to any metals that may be present in the groundwater in the area. Because this is a residential community, anybody living in the area can be expected to come into contact with metals present in the soil in the neighborhood. A list of age groups who can be expected to be exposed to metals in the soil is provided in Table 4-1.

Table 4-1: Potential Human Receptors in the Rodney Street Community

Potential Receptor	Activity Assumptions
Infant (0 - 6 months of age)	Assumed to be present on residential property for up to 23 hours per day every day over each phase of a 70 year lifetime. Ingested soil/dust is assumed to originate from the area with the highest reported level of each metal in soil in the Rodney Street community.
Toddler (7 months - <5 years)	Levels of indoor dust were assumed to be proportional to outdoor soil on a mass concentration basis. The ratio of soil intake to dust intake is not proportional to the ratio of the number of waking hours that a child spends outdoors versus indoors. Children spend only 15 to 30% of their waking hours playing outdoors but are assumed to be more likely to ingest soil while outdoors (45% of the child's soil/dust intake is assumed to occur outdoors).
Child (5 - <12 years)	Soil/dust assumed to adhere to skin every day of the year.
Teen (12 - <20 years)	All backyard produce consumed assumed to contain the 95th percentile concentration of all metal levels found and/or predicted. All drinking water assumed to contain highest concentration found in the municipal drinking water treatment and distribution system.
Adult (20+ years)	All inhaled air is assumed to contain the average of reported annual average levels of each metal measured in Port Colborne or other similar sites in Ontario.

4.1.2 Identifying Exposure Pathways

Residents in the Rodney Street community can be exposed to the metals in the soil by one of three different routes including; inhalation, ingestion and dermal contact. There are several factors that can contribute to the exposures experienced by each of these routes. For example, the ingestion of soil and the consumption of backyard produce would contribute to ingestion exposures, while skin contact with soil would contribute to dermal contact exposures. Each of these possibilities, known as exposure pathways, contribute to the total daily exposures experienced by residents. The potential exposure pathways that could contribute to these exposures are listed in Table 4-2, along with the rationale for their inclusion in the assessment. Table 4-2 also identifies exposure pathways that have not been considered and provides rationale for their exclusion from the process. In order to estimate any potential risks associated with exposure to the metals in the soil, the contribution that each included pathway makes to the total daily exposure must be assessed.

Table 4-2: Possible Human Exposure Pathways at Rodney Street

Media	Exposure Route	Pathway	Retained	Rationale
Air	Inhalation	Inhalation of metals on re-entrained soil and dust in indoor air and outdoor air	Yes	The current assessment assumes that a person will be exposed to elevated levels of nickel and other metals in indoor and outdoor air and that the annual average from air monitoring data will be representative.
Soil	Ingestion	Ingestion of soil	Yes	The ingestion of metals in soil/dust represents a potential exposure pathway for residents in the Rodney Street community.
		Uptake into plants and consumption of plants	Yes	Fruits and vegetables grown in backyard gardens in the Rodney Street community properties may contain metals taken up from the soil. The consumption of this produce represents a potential exposure pathway for residents in the Rodney Street community.
		Uptake into animals through plants and consumption of animal products	No	The homes in the Rodney Street community are not used for the production of livestock. Therefore exposure to metals through the consumption of livestock raised on the Rodney Street properties will not occur.
	Dermal Uptake	Dermal contact with soil	Yes	Exposure to metals through skin contact with metal bearing soil/dust on the Rodney Street properties is a potential exposure pathway for residents in the Rodney Street community.
Groundwater	Ingestion	Ingestion of metals in water derived from groundwater	No	Groundwater is not used as a source of domestic supply.
	Dermal Uptake	Dermal contact with metals in the groundwater	No	Groundwater is not used as a source of domestic supply.

4.1.3 Identifying Receptor Parameters

In addition to knowing who will be exposed to the metals in the soil and what routes contribute to the total exposure, it is necessary to have an understanding of the amount of exposure that could be expected for people in each of the age groups identified in Table 4-1. In order to accomplish this, it is necessary to characterize the physiological and behaviourial characteristics of each receptor group. For example, the amount of soil ingested will determine the level of direct exposure to metals in the soil, and the amount of air inhaled will govern the inhalation exposures experienced. These factors, and others, known as receptor parameters will govern the exposures experienced by the residents of the Rodney Street community. Several sources were considered in the selection of these parameters. The Compendium of Canadian Human Exposure Factors for Risk Assessment (O'Connor, 1997) was used as a primary source of receptor data. This source was selected since it characterizes Canadian populations; it relies on published and reliable reference sources such as Health Canada, Statistics Canada and the Canadian Fitness and Lifestyles Research Institute; and, has been used in the past on several assessments conducted by the ministry and the CCME (Canadian Council of Ministers of the Environment). In cases where this data set was unable to adequately describe certain time activity patterns and/or behaviourial/physiological characteristics, other data sources, such as the US EPA Exposure Factors Handbook (US EPA, 1997) were used. A summary of receptor parameters including: body weight, inhalation rate, drinking water intake, soil ingestion, soil adhesion to skin, consumption rates for backyard garden produce, and activity patterns used to assess exposures for the residents of the Rodney Street community are presented in Table 4-3. A detailed discussion of the selection and derivation of the values listed in Table 4-3 is provided in Appendix 6 of the report.

Table 4-3: Receptor Parameters Used to Estimate Daily Exposures

Parameter	Units	Infant 0 - 0.5 yrs	Toddler 0.5 - <5 yrs	Child 5 - <12 yrs	Teen 12 - <20 yrs	Adult 20+	Source
Number of Years	years	0.5	4.5	7	8	50	
Body Weight	kg	8.2	16.5	32.9	59.7	70.7	O'Connor, 1997; CCME, 2000
Inhalation Rate	m^3/day	2.1	9.3	14.5	15.8	15.8	O'Connor, 1997
Drinking Water Intake	L/day	0.3	0.6	0.8	1	1.5	O'Connor, 1997; CCME, 2000
Soil Ingestion	g/day	0.035	0.1	0.1	0.02	0.02	US EPA, 1997; 2000; Health Canada, 1995; CEPA, 1994a
Soil Adhesion to Skin	mg/cm^2	0.2	0.2	0.2	0.07	0.07	US EPA, 2000
Surface Area of Hands	m^2	0.032	0.043	0.059	0.08	0.089	O'Connor, 1997
Surface Area of Arms	m^2	0.055	0.089	0.148	0.223	0.25	O'Connor, 1997
Surface Area of Legs	m^2	0.091	0.169	0.307	0.497	0.572	O'Connor, 1997
Surface Area of Feet	m^2	0.025	0.043	0.072	0.108	0.119	O'Connor, 1997
Backyard Root Vegetables	g/day	6.05	7.7	11.7	16.5	13.7	MOEE, 1995
Backyard Other Vegetables	g/day	5.25	4.88	7.14	8.7	10	MOEE, 1995
Backyard Fruit	g/day	3.96	6.81	7.8	7.5	14.1	MOEE, 1995
Supermarket Food	g/day	822	1478	1798	1945	1598	Health Canada, 1995
Amount of time spent outdoors-summer	hrs/day	3	4.3	4.3	4.3	3	Derived from US EPA[1]
Amount of time spent outdoors-winter	hrs/day	2	2	2	2	2	Derived from US EPA[1]
Amount of time spent indoors-summer	hrs/day	21	19.7	19.7	19.7	21	Derived from US EPA[1]
Amount of time spent indoors-winter	hrs/day	22	22	22	22	22	Derived from US EPA[1]
Amount of time spent in Rodney Street Area (July and August)	hrs/day	23	23	23	23	23	Derived from US EPA[1]
Amount of time spent in Rodney Street Area (remainder of year)	hrs/day	23	23	16	14	23	Derived from US EPA[1]

[1]based on time activity pattern taken from the US EPA Exposure Factors Handbook (US EPA, 1997).

4.1.4 Exposure Assessment Assumptions

The objective of the assessment is to provide exposure estimates that are representative of reasonable upper bound exposures that could be experienced by Rodney Street residents. When selecting these parameters and assumptions for use in the current assessment, MOE considered guidance provided in documents by RAGS (Risk Assessment Guidance for Superfund), CCME, and MOE (e.g., Site Specific Risk Assessment guidelines) to estimate potential exposures that are conservative in nature, and would not be expected to underestimate potential exposures or risks to sensitive members of the Rodney Street community. A list of the assumptions used in this assessment and the effect that each will have on exposure estimates is provided in Table 4-4.

Default assumptions for human receptor characteristics are conservatively derived from extensive surveys of these characteristics in human populations and are protective of most segments of society. These human receptor characteristics are used to develop health based exposure limits (standards) and to assess the risks associated with various exposures to contaminants in air, water, soil, food and consumer products. When setting standards to protect human health and the environment under conditions of relatively low pollution (rural or urban background), assumptions about exposure to specific chemicals in air, water, soil and food assume that most of the chemical being inhaled, ingested or contacted by the skin is completely available for absorption into the body. This assumption is highly conservative and generally overestimates actual conditions and provides an additional margin of safety to the standard or risk being assessed. In reality, the amount of chemical available for absorption into the human body from any environmental medium is dependent on a complex interaction between the form and properties of the chemical and how that chemical is presented to the body. Consequently, when assessing exposures and risks where contamination of air, water or soil is elevated above background, greater attention must be paid to making exposure estimates more realistic and reassessing the validity of conservative default assumptions.

Default assumptions for human receptor characteristics can vary depending on how they are being used and the amount of information available to assess the extent of the exposure. In a situation where the assessment is preliminary and the information on exposure levels is limited, the assessment will be at the screening level and most exposure assumptions will be conservative, i.e., the maximum levels in environmental media are used, all the chemical in the medium is assumed to be available for uptake and upper range receptor intake factors are applied. Use of a worst case exposure estimate approach like this is sometimes used to screen and prioritize chemicals in environmental media. The underlying assumption being that if the concentration of a chemical assessed under these conditions is less than the relevant environmental criteria or exposure limits, there should be no further concern about that chemical.

In situations where knowledge of levels of chemicals in environmental media is extensive, the exposure assessment scenario can be tailored more closely to the information and a more realistic exposure estimate derived. In this situation, receptor intake factors can be more reflective of the

general population and use more central estimates of the range of the receptor characteristics in the population. For example, under RAGS (Risk Assessment Guidance for Superfund) (US EPA, 1989), the US EPA recommends use of 200 mg/day for soil ingestion by children even though this is considered a screening or upper range level of intake. Use of 80 mg/day or 100 mg/day reflects a mid range level of intake and may be more appropriate and sufficiently protective. Generally, when the assessor has a better understanding of the range of exposures, a combination of mid range receptor characteristics and upper (but not maximum) measurements of environmental chemical concentrations results in exposure estimates that are realistic and protective of the population. This latter approach is what is meant by the reasonable upper bound exposure. In addition, as more information is available concerning the form (speciation) of the chemical and the environmental matrix it is found in, further adjustments can be made to take into account how much of the chemical is biologically available.

Default assumptions for human receptor characteristics and bioavailability from various environmental media have been established by major jurisdictions over the past two to three decades. When characterizing exposure, it is important that the exposure parameters are the same as the exposure limit. That is, if the exposure limit is based on doses administered to test animals (intakes) or concentrations in media that humans were exposed to, the exposure estimate should be on the same basis. If the exposure limit is based on internal or absorbed doses (uptakes), the exposure estimate should be an absorbed dose. Since the majority of exposure limits are based on administered doses or other intake estimates, exposure estimates are generally concerned with estimating how much chemical is presented to the body in air inhaled, water, food or soil ingested or skin exposure to air, water and soil. These intakes take the chemical to the place where it may be absorbed, such as the upper and lower parts of the respiratory tract, the digestive tract or the skin. The portion of soil bound metal available *in vitro* for uptake is termed "bioaccessible" (see Appendix 5 for further discussion). Depending on the form and chemical properties of the chemical, some proportion of the bioaccessible chemical can be absorbed into the bloodstream (uptake or internal dose) and become bioavailable. As discussed in Appendix 5, the focus of the exposure assessment part of the risk assessment is on developing intake estimates, not bioavailability or uptake estimates. The use of *in vitro* bioaccessiblity factors based on soil sieved to 250 μm (which is more likely to adhere to the skin and be transferred to the mouth by children) to adjust soil ingestion intakes will tend to overestimate relative bioavailability factors, and is a conservative and realistic method of estimating metal intakes from soil ingestion.

Table 4-4: Summary of Exposure Assessment Assumptions

Parameter	Assumption	Effect on Assessment
Residency Time	A person has been assumed to spend up to 23 hours per day at their residence every day for a full 70 year life-time (a total of 25,550 days).	This approach over estimates all potential exposures because it likely underestimates the amount of time that people would be away from the home.
Soil/dust Ingestion	The rate of soil ingestion has been assumed to remain constant throughout the year. For each metal it was further assumed that all of the soil ingested in a day comes from the area of where the highest level of each individual metal was found. Adjustments were made to compensate for summer/winter and indoor/outdoor differences in soil concentrations and exposure patterns.	This approach will over estimate soil/dust ingestion exposures to metals because it assumes that people will have access to the soil 365 days per year. It allows for periods when access to the soil will be limited due to snow cover and ground freezing, as well as adjusting for exposure differences indoors and outdoors. It does not account for people moving about between areas of varying metal concentrations in the soil.
	This assessment also assumed that surface soils (0 - 20 cm) are the only contributors to daily soil ingestion.	Contact with soils deeper than a few cm will be limited.
Backyard Produce Consumption	Backyard produce consumption has been assumed to occur every day throughout the year. The amount of produce grown and consumed has been estimated based on previous studies conducted by the MOE in other communities in Ontario.	This assumption may over estimate potential exposures for people in the area who do not grow or consume home produce, or produce less than has been assumed in this assessment.
	Metal concentrations used in the current assessment were based upon the 95th percentile concentrations detected in recent sampling conducted in both the Rodney Street and general Port Colborne areas.	This assumption will likely overestimate potential exposures as most produce grown and consumed will have lower metal concentrations than those corresponding with the 95th percentile value.
Skin Contact with Soil	The total amount of skin contact with soil was assumed to take place in the area of highest reported concentration for each metal. It was also assumed that dermal contact with both outdoor soil and indoor dust would occur every day, 365 days per year, every year over a 70 year life-time. It was also assumed that once on the skin, soil would remain in place for a full 24 hours before it was removed by washing.	This approach will over estimate exposures that occur through skin contact. It does account for periods during the year when access to the soil would be limited either through snow cover and/or ground freezing, however, it does not account for soil being washed off the skin before the end of a 24 hour period.
Drinking Water Intakes	People living in the Rodney Street community were assumed to get all domestic water from the municipal supply. Exposures were also assumed to occur every day over a 70 year life-time. The highest level of each metal reported in the drinking water was assumed to be present in the drinking water over the entire 70 year exposure period.	Using upper bound values will over estimate the drinking water exposures to metals for residents of the Rodney Street community. The maximum reported values for the 1996 to 1999 monitoring period range between 1.5 and 2 fold greater than the average levels reported over the same period (see Appendix 1).

Table 4-4: Summary of Exposure Assessment Assumptions (continued)

Parameter	Assumption	Effect on Assessment
Supermarket Food	Daily food consumption estimates developed by Health Canada for the general population were assumed to be representative of the food consumption rates and patterns for residents of the Rodney Street community. For Rodney Street community residents, it was further assumed that any backyard garden produce consumed would be in addition to the daily intakes of supermarket food. That is; the Health Canada daily consumption estimates for root and other vegetables were not lowered to account for decreases in the intakes of supermarket produce when home produce was being used.	Daily food consumption from supermarket sources was also added to the amount of food consumption arising from home garden produce. The estimated daily dietary intakes of metals from supermarket foods will be marginally over estimated using this approach.
Inhalation of Airborne Metals	Inhalation exposures to metals in the air of Port Colborne have been assumed to occur over a 24 hour period. It has been assumed that the levels of metals in indoor air are related to those found in ambient outdoor air. It was further assumed that a person would be exposed to either the highest or the average of the reported annual average levels for each metal every day over a 70 year life-time (based on the1992 through 1995 local air monitoring data). This approach also assumes that the levels of metals reported in the air are present as freely available metal and are not bound to particulate matter. However, in ambient air, metals are generally bound to particulate matter. Therefore ambient air monitoring results reflect the levels of metals bound to particulates in the air and provide a truer estimate of the levels of metals present in the air than measurement of particulate matter would.	This assumption will over estimate inhalation exposures to metals. Assuming a 70 year life-time residency does not allow for decreases in exposures that may occur when a person would be away from the Rodney Street community. This assumption also assumes 100% bioavailability of the metal on the particulate. By assuming that the monitored levels represent metal that is available for absorption in the lung, the assessment over estimates actual doses. Potential re-entrainment of soil to air issues are discussed in section 7.1.

4.2 Metal Concentrations in Environmental Media

As noted above, the risk assessment is intended to provide reasonable upper bound estimates of exposure for residents in the Rodney Street community. Therefore upper bound levels of each metal reported in drinking water and soil, and an upper bound level (95th percentile) for backyard garden vegetables have been used to assess potential exposures from these sources. Metal levels in ambient air are not based on the maximum reported levels, but rather on the average of reported annual average values. The rationale for these assumptions is provided in Appendix 3. The concentrations of metals in the various media are summarized in Table 4-5.

Metal levels in individual supermarket food items are not considered directly in the current assessment. Rather, the exposure assessment has relied on estimates of the total daily dietary intake of each metal provided by regulatory agencies such as Health Canada and the US EPA or detailed intake surveys published in the scientific literature. A detailed discussion of the derivation of the daily dietary intakes of metals for all age groups is provided in Appendix 4. These values have been used directly in the estimation of total daily intake for receptors in each age group.

The values listed in Table 4-5 have been used in conjunction with the daily dietary intake estimates for each metal to develop estimates of total daily exposure for all residents in the Rodney Street community.

Table 4-5: Metal Concentrations Used to Assess Residential Exposures*

Medium	Units	Metal Concentrations					
		Antimony	Beryllium	Cadmium	Cobalt	Copper	Nickel
Drinking Water	µg/L	0.97	0.2	0.083	0.04	44	1.3
Ambient Air	µg/m^3	0.0011	0.00012	0.0007	0.002	0.112	0.033
Soil	µg/g	91.1	4.56	35.3	262	2720	17000
Backyard Root Vegetables	µg/g	0.021	0.007	0.063	0.048	1.92	2.44
Backyard Other Vegetables	µg/g	0.021	0.007	0.063	0.048	1.92	2.44
Backyard Fruits	µg/g	0.021	0.007	0.063	0.048	1.92	2.44

* See also Appendix 4 for further details.

4.3 Metal Exposures in Individual Media

In assessing the total daily intakes of each metal of concern in the Rodney Street community, it is necessary to determine the contribution that each individual exposure pathway makes to the daily total. Each of the potential exposure pathways has been assessed individually for the residents of the Rodney Street community. These individual contributions of each pathway are then combined to provide estimates of the total daily intake of each metal from all sources for each receptor age group (Section 4.4). Exposures from the individual pathways identified in Section 4.2 are summarized in the following sections. Detailed discussions of all pathways are provided in Appendices 3, 4 and 6 of the report.

4.3.1 Intake of Metals from Supermarket Foods

Estimates of the daily dietary intakes of metals from supermarket foods are generally limited and the amount of information available varies widely between metals. The metals of concern in Port Colborne include, antimony, beryllium, cadmium, cobalt, copper, and nickel. Information regarding daily dietary intakes of these metals has been taken from regulatory agencies in Canada and internationally. Additional information has been taken from the available literature. For the purposes of assessing likely daily dietary metal intakes for the residents of the Rodney Street community, preference has been given to data generated from the Canadian population. It was felt that information from Canadian sources would provide the best reflection of likely dietary habits and metal intakes for Rodney Street community residents. The daily dietary intake of metals is discussed in detail in Appendix 4. A summary of the daily dietary intake of metals for all age groups is presented in Table 4-6. These estimates have been used in conjunction with those from the other media to develop total daily intake values for each metal (Section 4.4).

Table 4-6: Estimated Daily Intakes of Metals from Supermarket Food

Receptor	Daily Intakes of Metals from Supermarket Food (µg/day)					
	Antimony	Beryllium	Cadmium	Cobalt	Copper	Nickel
Infant	1.3	4.8	5.08	4.18	518	109.2 (72.2-146.2)*
Toddler	2.3	8.6	10.6	7	822	190
Child	3.5	13.2	16.8	10	1230	251
Teen	4	15	17.3	12	1520	313
Adult	3.4	12.7	14.8	11	1430	307

* This range represents three separate estimates of infant dietary intakes. Refer to Table A4-4 for further details.

4.3.2 Intake of Metals from Drinking Water

The intake of metals from drinking water depends upon the level of metal in the water and the amount of water consumed by the average person in a day. Residents in the Rodney Street community are supplied with municipal water that is not derived from groundwater, but rather, from Lake Erie. Therefore, drinking water monitoring data for the town of Port Colborne was used to estimate the exposures to metals in drinking water for residents of the Rodney Street community. The concentration of each metal in the municipal supply is listed in Table 4-5. These values have been used to estimate the daily intake of antimony, beryllium, cadmium, cobalt, copper and nickel, for each receptor age group considered. The daily intake estimates for each metal for each age group are summarized in Table 4-7. A detailed discussion of the calculations used to estimate the daily intakes is provided in Appendix 3 of the report. The data in Table 4-7 shows that the daily intakes of most metals are generally below 2 µg/day. The notable exception is copper, where daily intakes for all age groups range between 13 and 66 µg/day. The contribution that drinking water makes to total daily exposure is discussed in Section 4.4.

Table 4-7: Estimated Daily Intakes of Metals from Drinking Water

Receptor	Daily Intakes of Metals from Drinking Water (µg/day)					
	Antimony	Beryllium	Cadmium	Cobalt	Copper	Nickel
Infant	0.29	0.06	0.025	0.012	13	0.39
Toddler	0.58	0.12	0.050	0.024	26	0.78
Child	0.78	0.16	0.066	0.032	35	1.0
Teen	0.97	0.2	0.083	0.040	44	1.3
Adult	1.5	0.3	0.12	0.060	66	2.0

4.3.3 Intake of Metals from Ambient Air

The risks associated with inhalation exposures to metals in the Rodney Street community have been assessed in two ways in this report;

First, the potential ingestion exposures associated with inhalation exposures to metal bearing particles is considered. This type of exposure is considered in this section of the report.

Second, the potential human health risks directly associated with inhaled metals were assessed by comparing the average of annual average air concentrations (1992 to 1995) in the MOE Port Colborne or Environment Canada air monitoring data for Ontario (Table A3-3, Appendix 3) with the appropriate inhalation exposure limit. This latter exposure has been directly assessed in the Risk Characterization section of the report (Section 5.0).

Air monitoring in the Rodney Street area of Port Colborne began in the summer of 2001. While annualized data is not available, the levels of the metals of concern found during the recent monitoring program by the MOE are comparable to those in other parts of the province. The comparison of TSP, PM_{10}, and $PM_{2.5}$ levels in the Rodney Street area with Federal data obtained between 1986 and 1994 show that the TSP levels are comparable to national averages (Table A1-2b, Appendix 1) while the PM_{10} and $PM_{2.5}$ levels in the area are lower. The MOE continues to monitor the metal-in-air levels in the Rodney Street area to obtain annualized data.

In Port Colborne, inhaled metals will be associated with particulate matter and will not be present as freely available metal or metal compounds. Therefore, there is a potential for the inhaled particulate matter to be cleared from the lungs, through mucocilliary transport, and swallowed. Material cleared from the lungs in this fashion will add to the total daily ingestion of metal. The amount of particulate delivered to the stomach by this process is difficult to predict with any accuracy. Therefore, to provide conservative estimates of the amount of metal ingested as a result of the clearance of inhaled particles, it has been assumed that all inhaled metal is cleared from the lung and passed to the stomach. This approach will over estimate the contribution that inhalation exposures make to the total daily intakes of metals. The reported annual average level of each metal (Table 4-5) has been used to estimate the daily ingestion intake of metals following inhalation for people living in the Rodney Street community. The rationale for using annual average ambient air quality monitoring information, and the calculations used to estimate daily intakes of each metal for each age group are discussed in detail in Appendix 3. The data in Table 4-8 shows that inhalation exposures make a very small contribution to ingestion exposures for the metals in soil in the Rodney Street community, even when it has been assumed that all inhaled material is passed to the stomach. Thus, it can be concluded that inhalation exposure to metals does not make a significant contribution to ingestion exposures to metals for the residents of the Rodney Street community. However, these intake estimates have been used in conjunction with the other values to develop total ingestion intake estimates for each metal (Section 4.4).

Table 4-8: Estimated Daily Intakes of Metals from Ambient Air

Receptor	Daily Intakes of Metals from Ambient Air (µg/day)					
	Antimony	Beryllium	Cadmium	Cobalt	Copper	Nickel
Infant	0.0018	0.0002	0.0011	0.0033	0.18	0.053
Toddler	0.0081	0.0009	0.0051	0.015	0.82	0.24
Child	0.013	0.0014	0.0080	0.023	1.28	0.37
Teen	0.014	0.0015	0.0087	0.025	1.39	0.41
Adult	0.014	0.0015	0.0086	0.025	1.38	0.40

4.3.4 Intake of Metals from Backyard Produce

Eating produce grown in backyards where metal levels are above typical levels, represents a potential exposure pathway if the metals present in the soil are taken up into the produce. The exposures received by people eating such produce depends upon the concentration of the metals in the produce and the amount of produce consumed from backyard gardens on an annual basis. The current assessment has assumed that backyard garden produce is consumed on a daily basis throughout the year. The amount of backyard garden produce consumed on an annual averaged daily basis is discussed in detail in Appendix 6.

As part of the ongoing work in Port Colborne, samples of backyard produce have been collected by the MOE and Jacques Whitford Environmental Limited (JWEL) from the Rodney and Mitchell Streets in the Rodney Street community and from the general Port Colborne area. The levels of individual metals in the various types of produce tested are provided in Appendix 1 of this report. For the purpose of this assessment, backyard garden produce has been divided into three general categories;

root vegetables	includes: beet root, carrot, onion and radish
other vegetables	includes: beet tops, celery, lettuce, peppers, rhubarb, squash, leeks and tomatoes
fruits	includes: pear, apple, cantaloupe, peach, plum, watermelon, and grapes

A review of the available produce data indicated that the concentrations of metals in produce is not strongly affected by the levels of metals present in the soil and as such it was not possible to derive appropriate uptake factors for the Rodney Street area. As a result, upper bound produce concentrations measured in the area were assumed for all gardens in the assessment. With the exception of nickel, maximum values were selected due to limited data sets (see Appendix 1). For nickel, over 200 relevant plant samples were available and as a result it was considered more appropriate to select an upper bound plant concentration to represent all gardens in the

assessment (Appendix 1, Table A1-12b presents the data available at the time of this assessment). The 95th percentile concentration was selected based on the following rationale: (i) others have considered the 95th percentile of a non-normal distribution to be representative of an upper bound value (the data set appears to be log-normally distributed); (ii) the data set is highly skewed and maximum values would not be reflective of reasonable upper bound exposures; (iii) a typical diet would consist of a composite of the available produce types, while the maximum level is only reflective of a single plant type and garden location. No distinction was made for different produce types, rather the selected upper bound concentration was assumed to represent all plant types. Summaries of the daily intake estimates for each metal from root vegetables, other vegetables and fruits, for each age group are shown in Table 4-9, Table 4-10 and Table 4-11 respectively.

The intake estimates for root and other vegetables for each metal and each receptor age group, were used in conjunction with intake estimates from the other sources to develop total daily intake estimates for each metal and age group (Section 4.4).

Table 4-9: Estimated Daily Intakes of Metals from Backyard Root Vegetables

Receptor	Daily Intakes of Metals from Backyard Root Vegetables (µg/day)					
	Antimony	Beryllium	Cadmium	Cobalt	Copper	Nickel
Infant	0.127	0.04	0.38	0.29	12	-*
Toddler	0.161	0.05	0.48	0.37	15	19
Child	0.25	0.08	0.74	0.56	22	29
Teen	0.35	0.12	1.0	0.8	32	40
Adult	0.29	0.10	0.86	0.66	26	33

* see section 5.6.1, Tables A3-8 and A4-4 for discussion of infant dietary nickel intakes.

Table 4-10: Estimated Daily Intakes of Metals from Other Backyard Vegetables

Receptor	Daily Intakes of Metals from Other Backyard Vegetables (µg/day)					
	Antimony	Beryllium	Cadmium	Cobalt	Copper	Nickel
Infant	0.11	0.037	0.33	0.25	10.1	-*
Toddler	0.10	0.034	0.31	0.23	9.4	12
Child	0.15	0.050	0.45	0.34	14	17
Teen	0.18	0.061	0.55	0.42	17	21
Adult	0.21	0.070	0.63	0.50	19	24

* see section 5.6.1, Tables A3-8 and A4-4 for discussion of infant dietary nickel intakes.

Table 4-11: Estimated Daily Intakes of Metals from Backyard Fruits

Receptor	Daily Intakes of Metals from Backyard Fruits (μg/day)					
	Antimony	Beryllium	Cadmium	Cobalt	Copper	Nickel
Infant	0.08	0.028	0.25	0.19	7.6	-*
Toddler	0.14	0.048	0.43	0.33	13.1	17
Child	0.16	0.055	0.49	0.37	15	19
Teen	0.16	0.053	0.47	0.36	14	18
Adult	0.15	0.050	0.45	0.30	14	17

* see section 5.6.1, Tables A3-8 and A4-4 for discussion of infant dietary nickel intakes.

4.3.5 Intake of Metals from Soil

The ingestion of soil that contains metal represents a potential exposure pathway for people who live in the homes in the Rodney Street community. The daily intake of metal from soil depends upon the amount of soil ingested and the level of metal bound to soil particles. The soil monitoring program conducted by the MOE in the Rodney Street community showed that metal levels in the soil varied across the community. It also showed that metal levels in soil varied across the sampling horizon of 20 cm. The results of the sampling program are discussed in detail in Part A, Section 6.0. A summary of the soil monitoring results is presented in Appendix 1 of this report. Because elevated levels of metals appear to be mostly confined to the top 20 cm of soil, it is possible that typical gardening activities could bring materials to the surface and thereby be available for exposure. Therefore, the highest level of each metal reported in the top 20 cm of soil was used to assess exposure for residents of the Rodney Street community. This approach will provide conservative estimates of reasonable upper bound exposures for all receptor age groups.

In addition to the amount of metal ingested with soil, the effective intake of metal is also dependent upon the amount of metal released from the soil during digestion. Only metal that is released from soil into the stomach or intestines during digestion can be considered to be bioaccessible to the body and available for uptake. Any metal not released from soil is excreted in the feces and does not have the opportunity to cause adverse health effects. Therefore, in assessing exposure and potential human health risks, it is necessary to consider the amount of metal actually released from the soil into the gut and not just the total amount of metal ingested with the soil, when assessing exposures and the potential for human health effects to occur.

The metals in the soil in the Rodney Street community have varying solubility under neutral pH conditions (pH 6 - pH 8). However, the solubility of the metals increases under acidic conditions. Therefore, under the acidic conditions of the stomach, it is reasonable to expect that some metal will be released and be accessible to the body and available for uptake. The amount of metal released from soil from the Rodney Street community has been examined by subjecting the soil to both simulated stomach acid digestion and bioaccessibility testing measuring the amount of

each metal released from the soil into acid and neutral pH extraction fluids (Appendix 5). These results, expressed as a percentage of the total metal level in the original soil sample have been used to correct the estimates of metal intake from soil ingestion. The stomach acid leach and bioaccessibility testing used to determine the adjustment factors for each metal is discussed in detail in Appendix 5. The equations used to estimate the adjusted metal intake from ingested soil are provided in Appendix 3. The results of this assessment are summarized in Table 4-12. The estimates of daily metal intakes from ingested soil for each receptor age group have been used in conjunction with the intake estimates from other sources to provide total daily intake estimates for each metal (Section 4.4).

Table 4-12: Estimated Daily Intakes of Metals from Soil Ingestion

Receptor	Daily Intakes of Metals from Soil Ingestion (µg/day)					
	Antimony	Beryllium	Cadmium	Cobalt	Copper	Nickel
Infant	0.54	0.05	0.5	1.41	21.7	59.9
Toddler	1.54	0.14	1.42	4.02	61.9	171
Child	1.54	0.14	1.42	4.02	61.9	171
Teen	0.31	0.029	0.28	0.81	12.4	34.2
Adult	0.31	0.029	0.28	0.81	12.4	34.2

4.3.6 Dermal Contact with Metals in Soil

Daily contact with metals through soil present on the skin represent a potential route of exposure. However, the insoluble nature of most metals in soil limits their bioaccessibility for uptake into and through the skin. Where data is available, it shows that dermal uptake of metals is low (Paustenbach, 2000). In determining the amount of metal that could be delivered to the skin from soil, a number of conservative assumptions have been used to provide upper bound estimates of potential exposure. It was assumed that soil on the skin would remain in place for a full 24 hour period and that bathing would only remove soil from the skin once every 24 hours. In addition, conservative or default assumptions were made regarding the amount of metal that would be released from the soil to the skin. Detailed discussions of the derivation of the dermal uptake coefficient for each metal and the calculation of the dermal contact exposures are presented in Appendix 7. The dermal contact/uptake values were assumed to represent intake values for each metal in order to facilitate their comparison with intakes from the other exposure routes. Estimates of dermal contact/intake are summarized in Table 4-13.

Table 4-13: Estimated Daily Intakes of Metals from Dermal Contact

Receptor	Daily Intakes of Metals from Dermal Contact (µg/day)					
	Antimony	Beryllium	Cadmium	Cobalt	Copper	Nickel
Infant	0.098	0.005	0.038	0.011	2.93	0.70
Toddler	0.17	0.0085	0.065	0.019	4.99	1.19
Child	0.28	0.014	0.11	0.032	8.23	1.96
Teen	0.15	0.0074	0.057	0.017	4.37	1.04
Adult	0.16	0.0079	0.061	0.018	4.66	1.11

4.4 Estimating Total Daily Intakes Of Metals

In order to estimate the potential health effects associated with exposure to metals for the residents of the Rodney Street community, it is necessary to know the total daily intakes of metals from all sources. In the Rodney Street community, two types of exposures can be considered to occur;

Non Soil Related (General) Exposures: these can be defined as; *non soil related exposures that are common across the Rodney Street community, Port Colborne and the Ontario population.* These include metal intakes from supermarket food, drinking water and ambient air.

Rodney Street Soil Related Exposures: these can be defined as; *exposures that are directly affected by the metals present in the soil on the properties in the Rodney Street community.* These include metal intakes from backyard garden produce, ingestion of soil and dermal contact with soil.

Total metal intakes from general and Rodney Street community specific exposures have been assessed separately to provide an indication of any additional exposure burdens that may be experienced by the residents of the Rodney Street community as a result of elevated levels of metals in the community. General and Rodney Street community specific exposures for each metal for all receptor age groups are provided in the following sections. In addition to providing estimates of total daily intakes on a µg/day basis, each of the following sections provide estimated daily intake (EDI) values for each receptor group on a per body weight basis, expressed as µg/kg-day. These can be considered as dose estimates and are necessary in the estimation of chronic daily intakes (CDI), which are used to estimate lifetime CDI values. Further details on the calculation of the CDI values are provided in Section 5 (Risk Characterization).

4.4.1 Total Daily Intakes of Antimony

The contributions that general and Rodney Street community specific exposures make to the total

daily intake of antimony are summarized in Table 4-14 and 4-15 respectively. The data shows that supermarket food makes the largest contribution to the total daily intake of antimony for all age groups, contributing 46% of the total exposure (general plus Rodney Street specific exposure) for the toddler receptor. It also suggests that for the toddler receptor, general exposures (air, water and market food) contribute 58% of the total exposure, with Rodney Street specific soil related pathways (soil ingestion, dermal contact and backyard produce) contributing the remaining 42%. Similar trends are observed for the other receptor groups. The implications of these exposures and the potential for health effects to develop as a result of these exposures is discussed in Section 5.0.

Table 4-14: Total Daily Intakes of Antimony: General Exposures

| Receptor | Intake for Individual Media (μg/day) | | | Total (μg/day) | Body Weight (kg) | EDI[1] (μg/kg-day) |
	Supermarket Food	Drinking Water	Ambient Air			
Infant	1.3	0.29	0.0018	1.59	8.2	0.19
Toddler	2.3	0.58	0.0081	2.89	16.5	0.18
Child	3.5	0.78	0.013	4.29	32.9	0.13
Teen	4	0.97	0.014	4.98	59.7	0.083
Adult	3.4	1.5	0.014	4.87	70.7	0.069

1: EDI = Estimated Daily Intake expressed in μg/kg-day.

Table 4-15: Total Daily Intakes of Antimony: Rodney Street Community Specific Exposures

| Receptor | Intake for Individual Media (μg/day) | | | | | Total (μg/day) | Body Weight (kg) | EDI[1] (μg/kg-day) |
	Root Vegetables	Other Vegetables	Fruits	Soil Ingestion	Dermal			
Infant	0.13	0.11	0.08	0.54	0.098	0.96	8.2	0.12
Toddler	0.16	0.10	0.14	1.54	0.17	2.11	16.5	0.13
Child	0.25	0.15	0.16	1.54	0.28	2.38	32.9	0.07
Teen	0.35	0.18	0.16	0.31	0.15	1.15	59.7	0.019
Adult	0.29	0.21	0.15	0.31	0.16	1.12	70.7	0.016

1: EDI = Estimated Daily Intake expressed in μg/kg-day.

4.4.2 Total Daily Intakes of Beryllium

The contributions that general and Rodney Street community specific exposures make to the total daily intake of beryllium are summarized in Table 4-16 and 4-17 respectively. The data shows that supermarket food and drinking water make the largest contributions to the total daily intakes of beryllium for all receptor groups, contributing more than 96% of the total daily intake (general plus Rodney Street specific exposure) for all receptor groups. This suggests that the daily exposures to beryllium experienced by residents in the Rodney Street community of Port Colborne do not differ from those experienced by the general Ontario population. The implications of these exposures and the potential for health effects to develop as a result of theses exposures is discussed in Section 5.0.

Table 4-16: Total Daily Intakes of Beryllium: General Exposures

| Receptor | Intake for Individual Media (µg/day) | | | Total (µg/day) | Body Weight (kg) | EDI[1] (µg/kg-day) |
	Supermarket Food	Drinking Water	Ambient Air			
Infant	4.8	0.06	0.0002	4.86	8.2	0.59
Toddler	8.6	0.12	0.00088	8.72	16.5	0.53
Child	13.2	0.16	0.0014	13.4	32.9	0.41
Teen	15.0	0.2	0.0015	15.2	59.7	0.25
Adult	12.7	0.3	0.0015	13.0	70.7	0.18

1: EDI = Estimated Daily Intake expressed in µg/kg-day.

Table 4-17: Total Daily Intakes of Beryllium: Rodney Street Community Specific Exposures

| Receptor | Intake for Individual Media (µg/day) | | | | | Total (µg/day) | Body Weight (kg) | EDI[1] (µg/kg-day) |
	Root Vegetables	Other Vegetables	Fruit	Soil Ingestion	Dermal			
Infant	0.04	0.037	0.028	0.050	0.005	0.16	8.2	0.0195
Toddler	0.05	0.034	0.048	0.14	0.0085	0.28	16.5	0.0170
Child	0.08	0.050	0.055	0.14	0.014	0.34	32.9	0.0103
Teen	0.12	0.061	0.053	0.029	0.0074	0.27	59.7	0.0045
Adult	0.10	0.070	0.050	0.029	0.0079	0.26	70.7	0.0036

1: EDI = Estimated Daily Intake expressed in µg/kg-day.

4.4.3 Total Daily Intakes of Cadmium

The contributions that general and Rodney Street community specific exposures make to the total daily intake of cadmium are summarized in Table 4-18 and 4-19 respectively. The data shows that supermarket food makes the largest contributions to total daily intakes for all age groups. For the toddler receptor, general exposures (air, water and market food) contribute 80% of the total exposure (general plus Rodney Street specific exposure), with Rodney Street specific soil related pathways (soil ingestion, dermal contact and backyard produce) contributing the remaining 20%. Similar trends are observed for the other receptor groups. The implications of these exposures and the potential for health effects to develop as a result of theses exposures is discussed in Section 5.0.

Table 4-18: Total Daily Intakes of Cadmium: General Exposures

| Receptor | Intake for Individual Media (µg/day) | | | Total (µg/day) | Body Weight (kg) | EDI[1] (µg/kg-day) |
	Supermarket Food	Drinking Water	Ambient Air			
Infant	5.08	0.025	0.0011	5.11	8.2	0.62
Toddler	10.6	0.05	0.0051	10.7	16.5	0.65
Child	16.8	0.066	0.008	16.9	32.9	0.51
Teen	17.3	0.083	0.0087	17.4	59.7	0.29
Adult	14.8	0.13	0.0086	14.9	70.7	0.21

1: EDI = Estimated Daily Intake expressed in µg/kg-day.

Table 4-19: Total Daily Intakes of Cadmium: Rodney Street Community Specific Exposures

| Receptor | Intake for Individual Media (µg/day) | | | | | Total (µg/day) | Body Weight (kg) | EDI[1] (µg/kg-day) |
	Root Vegetables	Other Vegetables	Fruits	Soil Ingestion	Dermal			
Infant	0.38	0.33	0.25	0.50	0.038	1.50	8.2	0.18
Toddler	0.48	0.31	0.43	1.42	0.065	2.71	16.5	0.164
Child	0.74	0.45	0.49	1.42	0.11	3.21	32.9	0.098
Teen	1.0	0.55	0.47	0.28	0.057	2.36	59.7	0.039
Adult	0.86	0.63	0.45	0.28	0.061	2.28	70.7	0.032

1: EDI = Estimated Daily Intake expressed in µg/kg-day.

4.4.4 Total Daily Intakes of Cobalt

The contributions that general and Rodney Street community specific exposures make to the total daily intake of cobalt are summarized in Table 4-20 and 4-21 respectively. The data shows that supermarket food makes the largest contributions to total daily intakes for all age groups. For the toddler receptor, general exposures (air, water and market food) contribute 59% of the total exposure (general plus Rodney Street specific exposure), with Rodney Street specific soil related pathways (soil ingestion, dermal contact and backyard produce) contributing the remaining 41%. Similar trends are observed for the other receptor groups. The implications of these exposures and the potential for health effects to develop as a result of theses exposures is discussed in Section 5.0.

Table 4-20: Total Daily Intakes of Cobalt: General Exposures

| Receptor | Intake for Individual Media (µg/day) | | | Total (µg/day) | Body Weight (kg) | EDI[1] (µg/kg-day) |
	Supermarket Food	Drinking Water	Ambient Air			
Infant	4.18	0.012	0.0033	4.20	8.2	0.51
Toddler	7.00	0.024	0.015	7.04	16.5	0.43
Child	10.0	0.032	0.023	10.1	32.9	0.31
Teen	12.0	0.040	0.025	12.1	59.7	0.20
Adult	11	0.060	0.025	11.1	70.7	0.16

1: EDI = Estimated Daily Intake expressed in µg/kg-day.

Table 4-21: Total Daily Intakes of Cobalt: Rodney Street Community Specific Exposures

| Receptor | Intake for Individual Media (µg/day) | | | | | Total (µg/day) | Body Weight (kg) | EDI[1] (µg/kg-day) |
	Root Vegetables	Other Vegetables	Fruits	Soil Ingestion	Dermal			
Infant	0.29	0.25	0.19	1.41	0.011	2.15	8.2	0.26
Toddler	0.37	0.23	0.33	4.02	0.019	4.97	16.5	0.30
Child	0.56	0.34	0.37	4.02	0.032	5.32	32.9	0.16
Teen	0.80	0.42	0.36	0.81	0.017	2.41	59.7	0.040
Adult	0.66	0.50	0.30	0.81	0.018	2.29	70.7	0.032

1: EDI = Estimated Daily Intake expressed in µg/kg-day.

4.4.5 Total Daily Intakes of Copper

The contributions that general and Rodney Street community specific exposures make to the total daily intake of copper are summarized in Table 4-22 and 4-23 respectively. The data shows that supermarket food makes the largest contributions to total daily intakes for all age groups. For the toddler receptor, general exposures (air, water and market food) contribute 89% of the total exposure (general plus Rodney Street specific exposure), with Rodney Street specific soil related pathways (soil ingestion, dermal contact and backyard produce) contributing the remaining 11%. Similar trends are observed for the other receptor groups. The implications of these exposures and the potential for health effects to develop as a result of theses exposures is discussed in Section 5.0.

Table 4-22: Total Daily Intakes of Copper: General Exposures

| Receptor | Intake for Individual Media (µg/day) | | | Total (µg/day) | Body Weight (kg) | EDI[1] (µg/kg-day) |
	Supermarket Food	Drinking Water	Ambient Air			
Infant	518	13	0.18	531	8.2	65
Toddler	822	26	0.82	849	16.5	51
Child	1230	35	1.28	1267	32.9	38
Teen	1520	44	1.39	1565	59.7	26
Adult	1430	66	1.38	1498	70.7	21

1: EDI = Estimated Daily Intake expressed in µg/kg-day.

Table 4-23: Total Daily Intakes of Copper: Rodney Street Community Specific Exposures

| Receptor | Intake for Individual Media (µg/day) | | | | | Total (µg/day) | Body Weight (kg) | EDI[1] (µg/kg-day) |
	Root Vegetables	Other Vegetables	Fruits	Soil Ingestion	Dermal			
Infant	12	10.1	7.6	21.7	2.93	54.3	8.2	7
Toddler	15	9.4	13.1	61.9	4.99	104.4	16.5	6
Child	22	14	15	61.9	8.23	121.1	32.9	4
Teen	32	17	14	12.4	4.37	79.8	59.7	1
Adult	26	19	14	12.4	4.66	76.1	70.7	1.1

1: EDI = Estimated Daily Intake expressed in µg/kg-day.

4.4.6 Total Daily Intakes of Nickel

The contributions that general and Rodney Street community specific exposures make to the total daily intake of nickel are summarized in Table 4-24 and 4-25 respectively. The data shows that while supermarket food makes the largest single contribution to total daily nickel intakes for all receptor age groups, Rodney Street community specific exposures also make a significant contribution. For the toddler receptor, general exposures (air, water and market food) contribute 47% of the total exposure (general plus Rodney Street specific exposure), with Rodney Street specific soil related pathways (soil ingestion, dermal contact and backyard produce) contributing the remaining 53%. Similar trends are observed for the other receptor groups. For the assessment of exposures in the Rodney Street community, gastro-intestinal absorption of nickel was assumed to be 100% for both food and drinking water sources.

The implications of these exposures and the potential for health effects to develop as a result of these exposures is discussed in Section 5.0.

Table 4-24: Total Daily Intakes of Nickel: General Exposures

Receptor	Intake for Individual Media (µg/day)			Total (µg/day)	Body Weight (kg)	EDI[1] (µg/kg-day)
	Supermarket Food	Drinking Water	Ambient Air			
Infant	109	0.39	0.053	109	8.2	13
Toddler	190	0.78	0.24	191	16.5	12
Child	251	1.04	0.37	252	32.9	8
Teen	313	1.30	0.41	315	59.7	5.3
Adult	307	1.95	0.41	309	70.7	4.4

1: EDI = Estimated Daily Intake expressed in µg/kg-day.

Table 4-25: Total Daily Intakes of Nickel: Rodney Street Community Specific Exposures

Receptor	Intake for Individual Media (µg/day)					Total (µg/day)	Body Weight (kg)	EDI[1] (µg/kg-day)
	Root Vegetables	Other Vegetables	Fruits	Soil Ingestion	Dermal			
Infant	nc	nc	nc	59.9	0.70	61	8.2	7.4
Toddler	19	12	17	171	1.19	220	16.5	13.3
Child	29	17	19	171	1.96	238	32.9	7.2
Teen	40	21	18	34.2	1.04	114	59.7	1.9
Adult	33	24	17	34.2	1.11	109	70.7	1.5

1: EDI = Estimated Daily Intake expressed in µg/kg-day.

nc = home garden was not considered for the infant (see Appendix 3).

5.0 Risk Characterization

The potential health risks for residents of the Rodney Street community were characterized using two procedures:

1. The general and Rodney Street community specific exposures were combined into the total metal intake from all exposure pathways and were compared with the oral exposure limit (RfD, etc.) (Table 3-1) selected for that metal;

2. Potential health risks from inhaling airborne metals were assessed by comparing the annual average air concentration in the MOE air monitoring data for Port Colborne or Environment Canada air monitoring data for Ontario (Table A3-3) with the selected inhalation exposure limit (RfC, unit cancer risk, etc.) (Table 3-1 and Table 3-2).

In order to compare the estimated daily exposures to each metal calculated for each of the receptor age groups, it is necessary to convert the individual exposures into a life-time averaged chronic daily intake (Σ CDI). The rationale for using life-time averaged daily doses for estimating the risks associated with life-time exposures is provided in Appendix 2. The total life-time CDI is calculated as shown in equation 5-1.

Eq 5-1:
$$Total\,CDI = \sum_{1}^{n} \left(\frac{EDI_{1..n} * Time_{1..n}}{70\,years} \right)$$

Where:

Total CDI	=	Total Chronic Daily Intake	μg/kg-day
$EDI_{1..n}$	=	Estimated Daily Intake of age group n	μg/kg-day
$Time_{1..n}$	=	Time spent in each age group	years
n	=	Age group	

From equation 5-1 it can be seen that the total CDI is a sum of the fractional CDI contributions made by exposures that occur during each life stage (receptor age groups). For the purpose of this assessment, the fractional CDIs for general and Rodney Street community specific exposures have been calculated for each receptor age group (identified as CDI in the tables). The total CDIs for the general and Rodney Street community specific exposures are also listed to provide an indication of what factors make the greatest contributions to the final CDI. The final CDIs are compared to their respective oral RfD values in making the final estimates of potential risk. CDIs that are lower than their respective RfD values indicate that exposures are below the identified exposure limit and that human health effects would not be expected to occur.

Graphical representations of the EDIs for the individual receptor age groups as well as the CDI compared to the RfD are also provided for the six metals carried through the detailed risk assessment. In comparing the individual EDI values to the RfD values, it should be remembered that the US EPA recommends that this serve only as a screening tool to indicate potential risk but

that they are not to be considered as predictive of potential human health effects. Predictive estimates of potential risk should only be based on a comparison between the CDI and the R*f*D (US EPA, 1989). For non-carcinogenic end points, the conservative approach of assessing the situation on the basis of the most sensitive receptor age group (from an outdoor soil exposure perspective) has been undertaken for this assessment. In cases where a particular age group exceeds the established health benchmark from general exposures alone, chemical and receptor specific factors are considered to determine the relevance and health significance of this exceedance. In general, this situation will only arise for the infant receptor group, which is a very short duration age group (<6 months) and the relevance of a life-time R*f*D to an exposure of this duration is questionable.

In the Rodney Street community HHRA, life-time cancer risks based on inhaling ambient air levels of several metals that are potentially carcinogenic (Be, Cd and Ni) were estimated by comparing inhalation intakes with cancer slope factors (risk per (μg/kg/day)) or unit risk (risk per μg/m^3). This is in accordance with risk assessment procedures used by US EPA and WHO (World Health Organization).

5.0.1 Development of Health-Based Site Specific Intervention Levels

An important outcome of characterizing the risk is determining the need for developing an intervention level which would be used as a tool in evaluating and cleaning up contaminated soils. The need for developing such a level is based on a number of key considerations including:

1. the nature, extent and duration of the risk and the uncertainties in how these are estimated;

2. evidence or lack of evidence of actual harm to health in the community; and

3. outcomes of assessments in other communities with similar or higher levels of exposure.

These considerations are not exclusive, and other legal, financial and community concern based issues, may also have an important role in developing intervention levels.

In cases where exposures to a particular metal exceed the established health benchmark, a site specific soil intervention level may be set at the soil concentration at which the combined site specific exposure to the metal from all exposure pathways, experienced by the most sensitive receptor age group, either approaches or exceeds the established health benchmark.

5.1 Antimony
5.1.1 Ingestion Exposure to Antimony

To characterize the potential health risks for residents of the Rodney Street community, the total antimony intake from all exposure pathways (Table 5-1) was compared with the 0.4 μg/kg-day oral exposure limit (US EPA IRIS, 1998a - oral R/D assessment last revised 1991; WHO, 1996) (Table 3-1).

Inspection of Table 5-1 and Figure 5-1 show that the presence of up to 91.1 μg/g (Table A1-4) antimony in soil in the Rodney Street community is unlikely to be associated with any adverse health effects. For the toddler, dietary intake is the predominant contributor to the total daily intake of antimony accounting for 46% of the total daily intake for the Rodney Street community, air and water pathways contribute 0.2% and 12%, respectively, and surface soil, through ingestion, dermal absorption and backyard produce, accounts for approximately 42% of the total daily intake.

Similar trends are observed for the other receptor groups. The total chronic daily intake is approximately 0.1 μg/kg-day, which lies well below the R/D of 0.4 μg/kg-day. For the infant, the receptor with the estimated highest exposure, the total daily intake, under even these extreme conditions, is below 0.4 μg/kg-day.

5.1.2 Inhalation Exposure for Antimony

Potential health risks from inhaling airborne antimony were assessed by comparing the highest maximum and annual average air concentrations in the Environment Canada air monitoring data for Ontario (Table A3-3, Appendix 3) with the US EPA R/C of 0.2 μg/m³ (US EPA IRIS, 1998a -inhalation R/C assessment last revised 1995) (Table 3-1). In this case, both the maximum antimony concentration (0.012 μg/m³) and the highest annual average concentration (0.0011 μg/m³) were well below the R/C. Consequently, there appears to be no potential for health related effects from inhalation of antimony.

Table 5-1: Life-time Averaged Daily Antimony Intakes for the Rodney Street Community

Metal	Receptor	Years	General Exposures			Rodney St Specific Exposures			Total CDI[4]
			EDI[1]	CDIf[2]	Σ CDI[3]	EDI[1]	CDIf[2]	Σ CDI[3]	
Antimony	0 - 6 months	0.5	0.19	0.001	0.08	0.12	0.0009	0.030	0.11
	7 mo - <5 yrs	4.5	0.18	0.011		0.13	0.0084		
	5 - <12 years	7	0.13	0.013		0.07	0.0070		
	12 - <20 years	8	0.083	0.009		0.019	0.0022		
	20 + years	50	0.07	0.049		0.016	0.0114		

1. Estimated Daily Intake (µg/kg-day).
2. Chronic Daily Intake Fraction (µg/kg-day).
3. ΣCDI = sum of CDIf for General or Rodney Street community specific exposures (µg/kg-day).
4. Total CDI = Chronic Daily Intake for a life-time exposure to metal in the Rodney Street community (µg/kg-day).

Figure 5-1: Summary of Total Exposures to Antimony
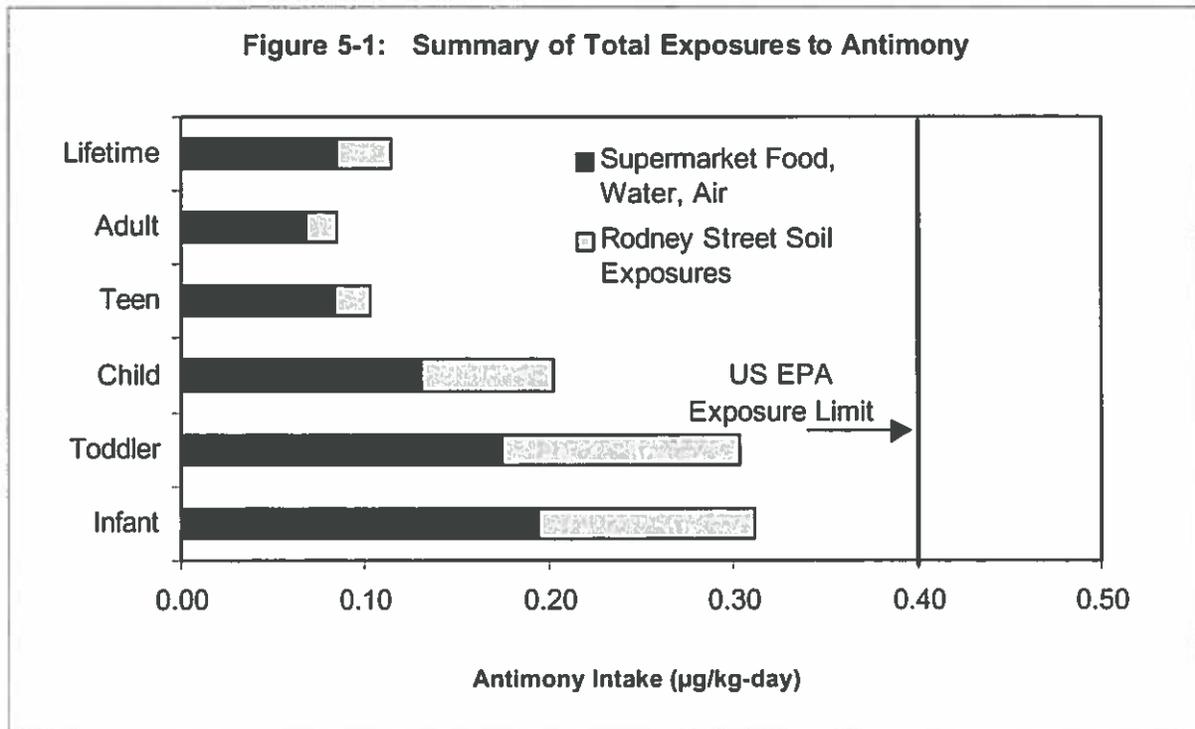
5.2 Beryllium
5.2.1 Ingestion Exposure to Beryllium

To characterize the potential health risks for residents of the Rodney Street community, the total beryllium intake from all exposure pathways (Table 5-2) was compared with the 2 µg/kg-day oral exposure limit (US EPA IRIS, 1998b - oral R/D assessment last revised 1998) (Table 3-1).

Inspection of Table 5-2 and Figure 5-2 show that the presence of up to 4.56 µg/g (Table A1-4) beryllium in soil in the Rodney Street community is unlikely to be associated with any adverse health effects since these conservatively estimated total intakes did not exceed the US EPA R/D (2 µg/kg-day) for any age class. Figure 5-3 shows the estimated daily intakes of beryllium on an expanded scale, to make it easier to see the relative contributions made by general exposures and those received from Rodney Street soil. From Figure 5-3 it can be seen that Rodney Street community specific exposures to beryllium in soil do not make an appreciable contribution to the total daily intakes of beryllium. For the toddler, dietary intake is the predominant contributor to the total daily intake of beryllium accounting for 97% of the total daily intake for the Rodney Street community, air and water pathways contribute 0.01% and 1.3%, respectively, and surface soil, through ingestion, dermal absorption and backyard produce, accounts for approximately 3% of the total daily intake. Similar trends are observed for the other receptor groups.

5.2.2 Inhalation Exposure to Beryllium

Potential health risks from inhaling airborne beryllium were assessed by comparing the estimated airborne concentration of beryllium in TSP (Total Suspended Particulate) with both the R/C of 0.02 µg/m^3 for non-cancer effects (US EPA IRIS, 1998b -inhalation R/C assessment last revised 1998) (Table 3-1) and with the US EPA inhalation unit risk of 0.0024 (µg/m^3)$^{-1}$ (US EPA IRIS, 1998b-carcinogenicity assessment last revised 1998) (Table 3-2). In both cases, the estimated airborne beryllium concentration (0.00012 µg/m^3) in the Rodney Street community (see Appendix 3) is less than the R/C and the air concentration at the 10^{-6} life-time cancer risk level. Consequently, there appears to be no potential for health related effects from inhalation of beryllium.

Table 5-2: Life-time Averaged Daily Beryllium Intakes for the Rodney Street Community

Metal	Receptor	Years	General Exposures			Rodney St Specific Exposures			Total CDI[4]
			EDI[1]	CDIf[2]	Σ CDI[3]	EDI[1]	CDIf[2]	Σ CDI[3]	
Beryllium	0 - 6 months	0.5	0.59	0.004	0.24	0.0198	0.0001	0.0054	0.24
	7 mo - <5 yrs	4.5	0.53	0.034		0.0174	0.0011		
	5 - <12 years	7	0.41	0.041		0.0105	0.0011		
	12 - <20 years	8	0.25	0.028		0.0045	0.0005		
	20 + years	50	0.18	0.128		0.0036	0.0026		

1. Estimated Daily Intake (µg/kg-day).
2. Chronic Daily Intake Fraction (µg/kg-day).
3. ΣCDI = sum of CDIf for General or Rodney Street community specific exposures (µg/kg-day).
4. Total CDI = Chronic Daily Intake for a life-time exposure to metal in the Rodney Street community (µg/kg-day).

Figure 5-2:   Summary of Total Exposures to Beryllium

Figure 5-3:   Exposures to Beryllium (expanded scale)
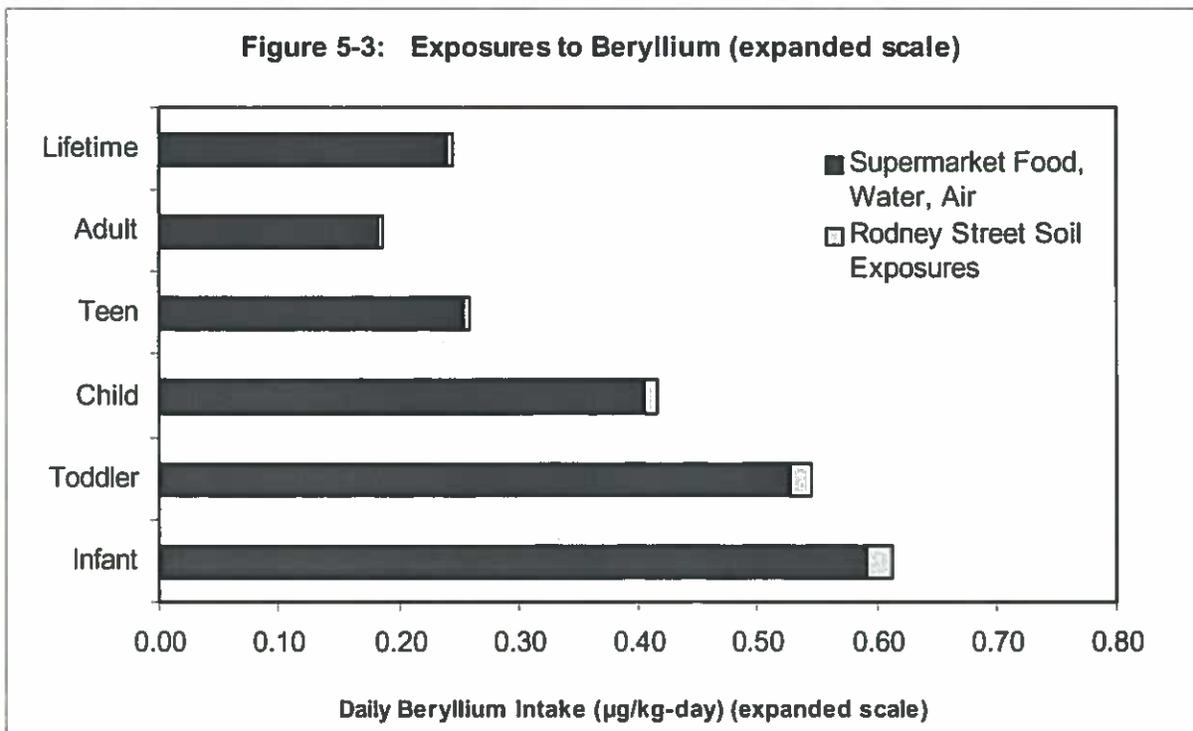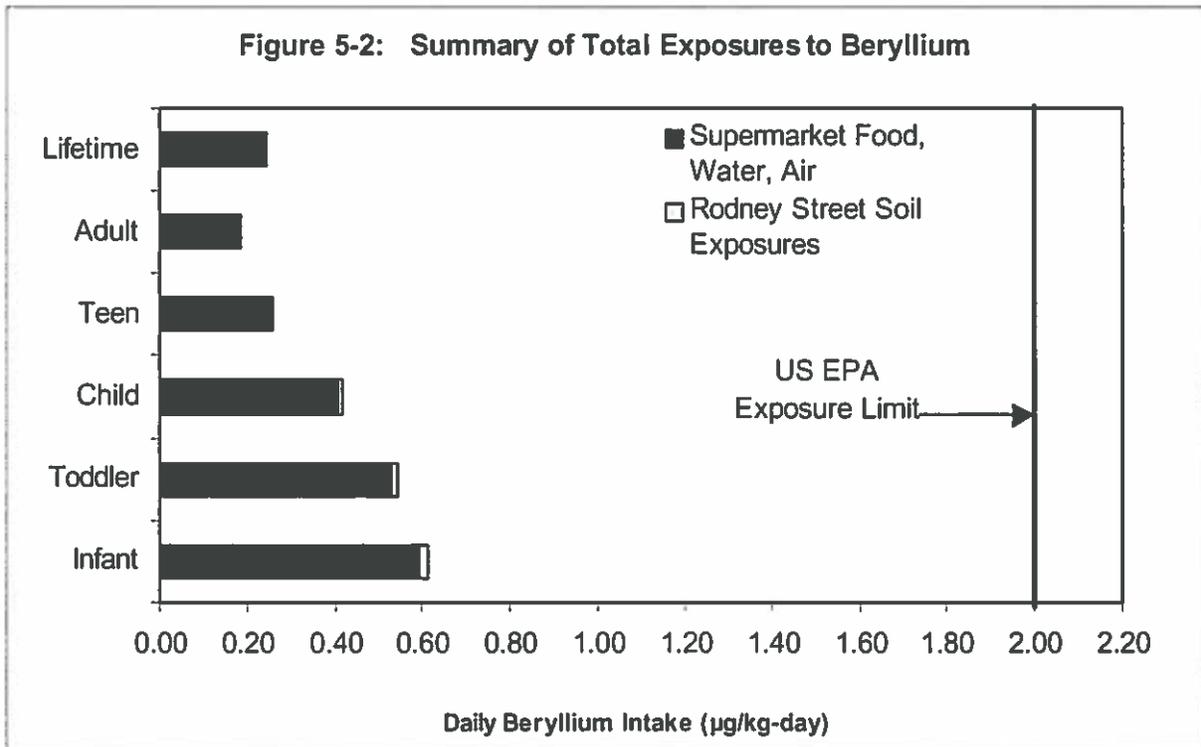
5.3 Cadmium
5.3.1 Ingestion Exposure to Cadmium

To characterize the potential health risks for residents of the Rodney Street community, the total cadmium intake from all exposure pathways (Table 5-3) was compared with the 1 μg/kg-day oral exposure limit proposed by the US EPA IRIS (1998c - oral R/D assessment last revised 1994) and WHO (1998a) (Table 3-1). This higher intake limit was used because intakes were estimated for all exposures not just drinking water or diet.

Inspection of Table 5-3 and Figure 5-4 show that the presence of up to 35.3 μg/g (Table A1-4) cadmium in soil in the Rodney Street community is unlikely to be associated with any adverse health effects since these conservatively estimated total intakes did not exceed the US EPA R/D (1 μg/kg-day) for any age class. For the toddler, dietary intake is the predominant contributor to the total daily intake of cadmium accounting for 80% of the total daily intake for the Rodney Street community, air and water pathways contribute 0.04% and 0.4%, respectively, and surface soil, through ingestion, dermal absorption and backyard produce, accounts for approximately 20% of the total daily intake. Similar trends are observed for the other receptor groups.

5.3.2 Inhalation Exposure to Cadmium

There are no appropriate air monitoring data for estimating life-time inhalation cancer risks for cadmium in Port Colborne. Recent air monitoring at the Rodney Street site for cadmium based on 26 sample dates over a five month period, yield the following interim results: average Cd concentration from TSP sampling = 0.042 μg/m^3 (max. = 0.3 μg/m^3); average Cd concentration from PM$_{10}$ sampling = 0.0002 μg/m^3 (max. = 0.0005 μg/m^3). Over this period, the airborne particulate readings were: average TSP concentration = 46.1 μg/m^3 (max. = 100 μg/m^3); average PM$_{10}$ concentration = 18.3 μg/m^3 (max. = 66 μg/m^3). The particulate data show a typical mass concentration relationship, i.e., TSP mass about twice the PM$_{10}$ mass (see section Appendix 2-9.2.1.1). However, the cadmium concentrations for the PM$_{10}$ monitoring are less than 1% of the cadmium concentrations found in the TSP monitoring data. This PM$_{10}$ data is consistent with the range of federal 1995 - 1999 PM$_{10}$ cadmium air monitoring data for Ontario (average = 0.0007 μg/m^3 (range 0.0001 μg/m^3 to 0.0067 μg/m^3) (Table A1-3). Potential cancer risks from inhaling airborne cadmium were assessed by comparing the highest average annual average air concentration in the Environment Canada air monitoring data for Ontario (Table A1-3) with the US EPA inhalation unit risk (0.0018 (μg/m^3)$^{-1}$ (US EPA IRIS, 1998c - carcinogenicity assessment last revised 1992) (Table 3-2). In this case, the maximum average cadmium concentration found in the 1995-1999 Environment Canada monitoring of nine sites spread across Ontario was 0.0007 μg/m^3, or about 1.2 x 10^{-6} life-time risk.

The WHO (2000) considers the US EPA unit risk to be a serious overestimate due to concomitant exposure to arsenic in the populations used to derive this unit risk. WHO (2000) estimates that the lowest estimate of cumulative exposure to airborne cadmium associated with

lung cancer extrapolated for continuous life-time exposure is around 0.3 $\mu g/m^3$. Converting the Health Canada TC_{05} for cadmium of 5.1 $\mu g/m^3$ to a unit risk factor (0.05 / 5.1 $\mu g/m^3$ = 9.8 X 10^{-3} $(\mu g/m^3)^{-1}$), and applying this to the mean of the annual average air cadmium concentration of the Environment Canada data (0.0007 $\mu g/m^3$), the life-time inhalation cancer risk estimate is 6.9 X 10^{-6}. This life-time inhalation risk estimate is six times greater than the US EPA risk estimate.

Two factors should be noted:

1. The Health Canada TC_{05} is based on animal studies and the US EPA unit risk is based on cadmium smelter workers. Calculation of human lifetime cancer risks using extrapolations from human data is preferable; and

2. The use of PM_{10} monitoring data to estimate lung cancer risks using unit risks based on workplace air particulate concentrations of different particle size distributions may require further adjustment (see section A2-9.2.1.1). PM_{10} monitoring samplers collect a lower particle mass and a smaller particle size range than the workplace air monitors.

Until additional air quality data for cadmium in Rodney Street Community air is available to determine an annual air concentration, the potential for lung cancer risk from this exposure cannot be reliably estimated. If cadmium air concentrations in the Rodney Street community remain at their 2001 interim results, the estimated life-time cancer risks from inhaling cadmium are in the 10^{-6} risk range or below.

Table 5-3: Life-time Averaged Daily Cadmium Intakes for the Rodney Street Community

Metal	Receptor	Years	General Exposures			Rodney St Specific Exposures			Total CDI[4]
			EDI[1]	CDIf[2]	Σ CDI[3]	EDI[1]	CDIf[2]	Σ CDI[3]	
Cadmium	0 - 6 months	0.5	0.62	0.004	0.28	0.18	0.0013	0.049	0.33
	7 mo - <5 yrs	4.5	0.65	0.041		0.164	0.0105		
	5 - <12 years	7	0.51	0.051		0.098	0.0098		
	12 - <20 years	8	0.29	0.033		0.040	0.0046		
	20 + years	50	0.21	0.15		0.032	0.0229		

1. Estimated Daily intake (μg/kg-day).
2. Chronic Daily Intake Fraction (μg/kg-day).
3. ΣCDI = sum of CDIf for General or Rodney Street community specific exposures (μg/kg-day).
4. Total CDI = Chronic Daily Intake for a life-time exposure to metal in the Rodney Street community (μg/kg-day).

Figure 5-4: Summary of Total Exposures to Cadmium
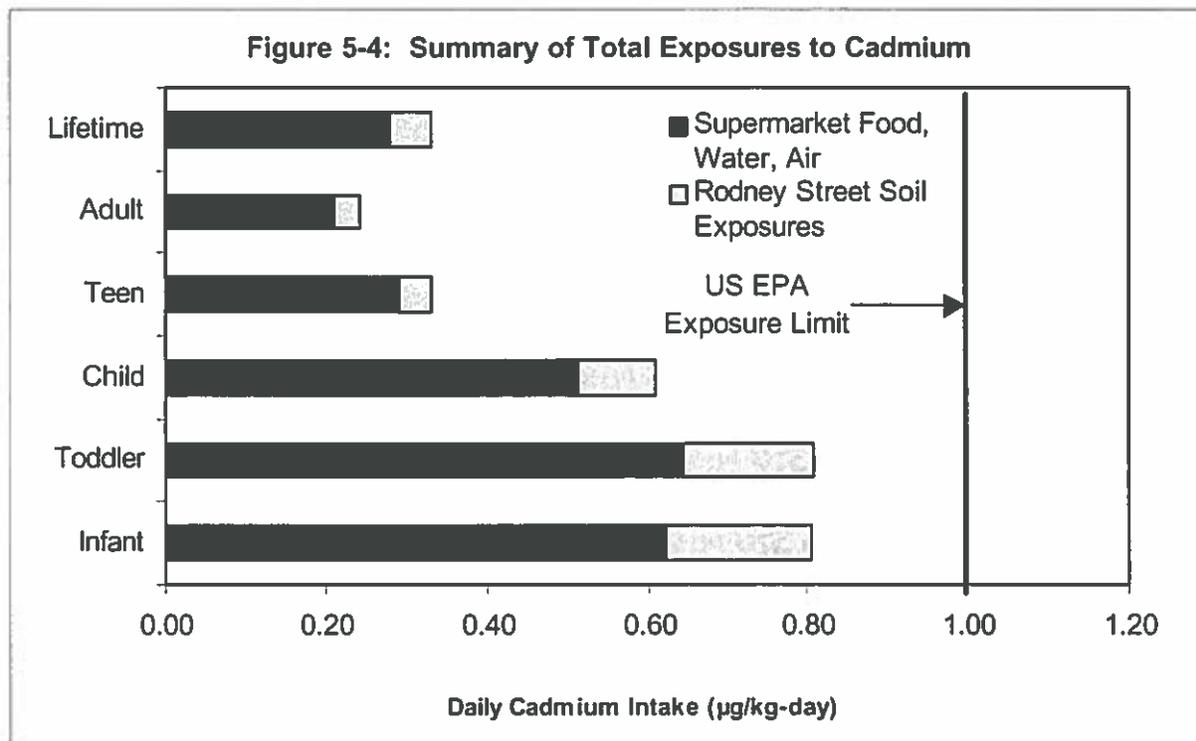
5.4 Cobalt
5.4.1 Ingestion Exposure to Cobalt

To characterize the potential health risks for residents of the Rodney Street community, the total cobalt intake from all exposure pathways (Table 5-4) was compared with the 20 µg/kg-day oral exposure limit proposed by the US EPA Region III (Table 3-1).

Inspection of Table 5-4 and Figure 5-5 show that the presence of up to 262 µg/g (Table A1-4) cobalt in soil in the Rodney Street community is unlikely to be associated with any adverse health effects since these conservatively estimated total intakes did not exceed the US EPA R/D (20 µg/kg-day) for any age class. Figure 5-6 shows the contributions to total daily intakes of cadmium made by general and Rodney Street community specific exposures. For the toddler, dietary intake is the predominant contributor to the total daily intake of cobalt accounting for 59% of the total daily intake for the Rodney Street community, air and water pathways contribute 0.12% and 0.2%, respectively, and surface soil, through ingestion, dermal absorption and backyard produce, accounts for approximately 41% of the total daily intake. Similar trends are observed for the other receptor groups.

5.4.2 Inhalation Exposure to Cobalt

Potential health risks from inhaling airborne cobalt were assessed by comparing the highest annual average air concentration in the Environment Canada air monitoring data for Ontario (Table A3-3) with the ATSDR (Agency for Toxic Substances & Disease Registry) (1992) inhalation minimal risk level (MRL) (0.03 µg/m³) (Table 3-1). In this case, the maximum cobalt concentration found in the 1995-1999 Environment Canada monitoring of nine sites spread across Ontario (0.017 µg/m³ (Table A3-3)) and the highest annual average concentration (0.002 µg/m³), are well below this inhalation MRL. Consequently, there appears to be no potential for health related effects from inhalation of cobalt.

Table 5-4: Life-time Averaged Daily Cobalt Intakes for the Rodney Street Community

Metal	Receptor	Years	General Exposures			Rodney St Specific Exposures			Total CDI[4]
			EDI[1]	CDI*f*	Σ CDI[3]	EDI[1]	CDI*f*	Σ CDI[3]	
Cobalt	0 - 6 months	0.5	0.51	0.003		0.26	0.0019		
	7 mo - <5 yrs	4.5	0.43	0.027		0.30	0.0193		
	5 - <12 years	7	0.31	0.031	0.19	0.16	0.0160	0.065	0.26
	12 - <20 years	8	0.20	0.022		0.040	0.0046		
	20 + years	50	0.15	0.11		0.033	0.024		

1. Estimated Daily intake (µg/kg-day).
2. Chronic Daily Intake Fraction (µg/kg-day).
3. ΣCDI = sum of CDI*f* for General or Rodney Street community specific exposures (µg/kg-day).
4. Total CDI = Chronic Daily Intake for a life-time exposure to metal in the Rodney Street community (µg/kg-day).
Note: Due to rounding of values, total intakes may not add up to exactly the sum of the intakes for each row, in particular the final decimal place.

Figure 5-5: Summary of Total Exposures to Cobalt

Legend:
- ■ Supermarket Food, Water, Air
- □ Rodney Street Soil Exposures

← US EPA Exposure Limit

Categories (top to bottom): Lifetime, Adult, Teen, Child, Toddler, Infant

X-axis: Daily Cobalt Intake (μg/kg-day) — 0.00, 10.00, 20.00, 30.00, 40.00, 50.00

Figure 5-6: Exposures to Cobalt (expanded scale)

Legend:
- ■ Supermarket Food, Water, Air
- □ Rodney Street Soil Exposures

Categories (top to bottom): Lifetime, Adult, Teen, Child, Toddler, Infant

X-axis: Daily Cobalt Intake (μg/kg-day) (expanded scale) — 0.00, 0.10, 0.20, 0.30, 0.40, 0.50, 0.60, 0.70, 0.80, 0.90
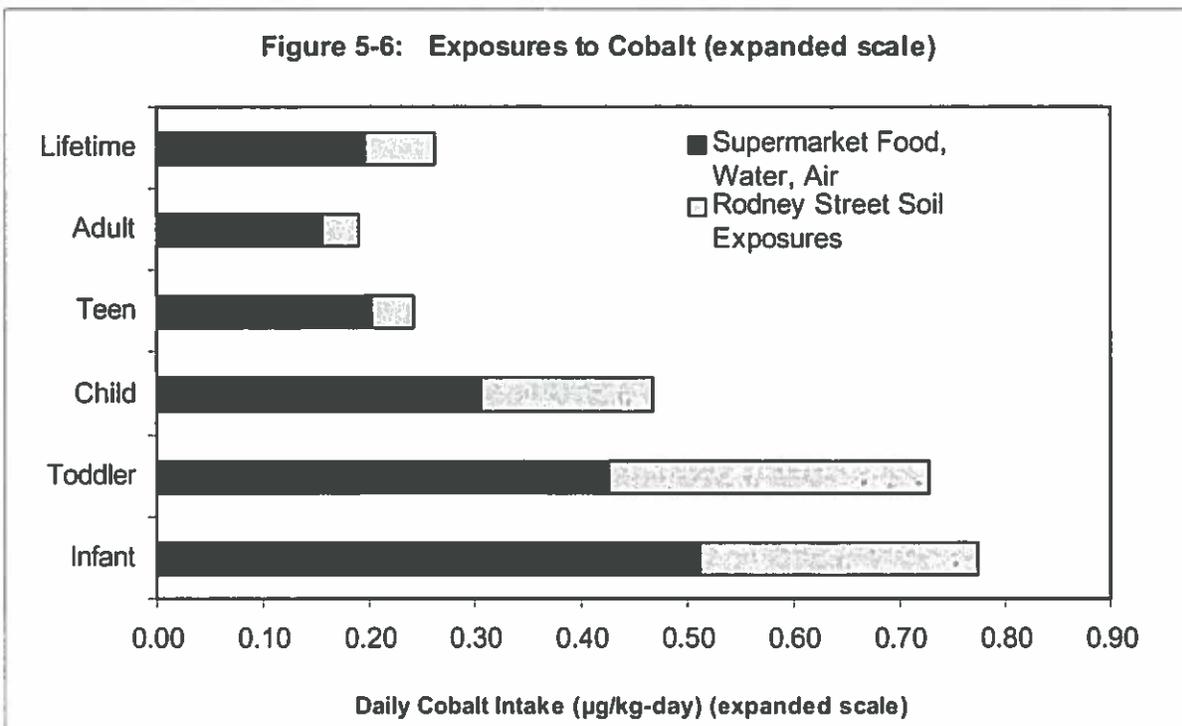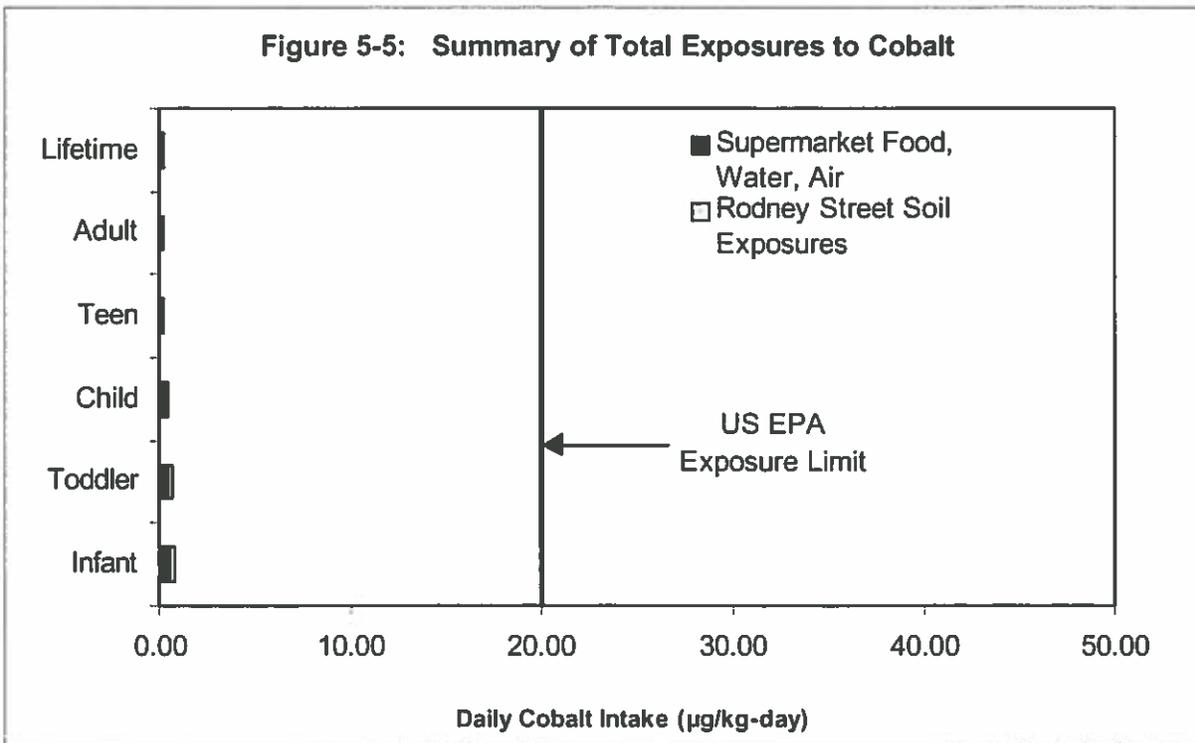
5.5 Copper
5.5.1 Ingestion Exposure to Copper

To characterize the potential health risks for residents of the Rodney Street community, the total copper intake from all exposure pathways (Table 5-5) was compared with the Recommended Daily Allowances (RDA) for adults (30 µg/kg-day) or children (50 µg/kg-day) and the tolerable upper intake limit of 140 µg/kg-day proposed by the WHO (1998b) and IOM (Institute of Medicine), (2001) (Table 3-1).

Inspection of Table 5-5 and Figure 5-7 show that the presence of up to 2,720 µg/g (Table A1-4) copper in soil in the Rodney Street community is unlikely to be associated with any adverse health effects since these conservatively estimated total intakes did not exceed the tolerable upper intake limit (140 µg/kg-day) for any age class. For the toddler, dietary intake is the predominant contributor to the total daily intake of copper accounting for 89% of the total daily intake for the Rodney Street community, air and water pathways contribute 0.09% and 2.8%, respectively, and surface soil, through ingestion, dermal absorption and backyard produce, accounts for approximately 11% of the total daily intake. Similar trends are observed for the other receptor groups.

5.5.2 Inhalation Exposure to Copper

Potential health risks from inhaling airborne copper were assessed by comparing the highest annual average air concentration in the MOE air monitoring data for Port Colborne (Table A3-3) with the chronic air quality criteria for copper used by California (Table 3-1). In this case, the maximum copper concentration found (0.56 µg/m³) (Table A3-3) and the highest annual average concentration (0.112 µg/m³), are well below California's chronic inhalation reference exposure limit (REL) (2.4 µg/m³) (Table 3-1). The maximum copper concentration in the 2001 air monitoring data for the Rodney Street location was 0.08 µg/m³. Consequently, there appears to be no potential for health related effects from inhalation of copper.

Table 5-5: Life-time Averaged Daily Copper Intakes for the Rodney Street Community

Metal	Receptor	Years	General Exposures			Rodney St Specific Exposures			Total CDI[4]
			EDI[1]	CDI[2]	Σ CDI[3]	EDI[1]	CDI[2]	Σ CDI[3]	
Copper	0 - 6 months	0.5	65	0.46	26	6.6	0.05	1.8	27
	7 mo - <5 yrs	4.5	51	3.3		6.3	0.41		
	5 - <12 years	7	38	3.8		3.7	0.4		
	12 - <20 years	8	26	3.0		1.3	0.1		
	20 + years	50	21	15		1.1	0.8		

1. Estimated Daily Intake (µg/kg-day).
2. Chronic Daily Intake Fraction (µg/kg-day).
3. ΣCDI = sum of CDIf for General or Rodney Street community specific exposures (µg/kg-day).
4. Total CDI = Chronic Daily Intake for a life-time exposure to metal in the Rodney Street community (µg/kg-day).

Figure 5-7: Summary of Total Exposures to Copper
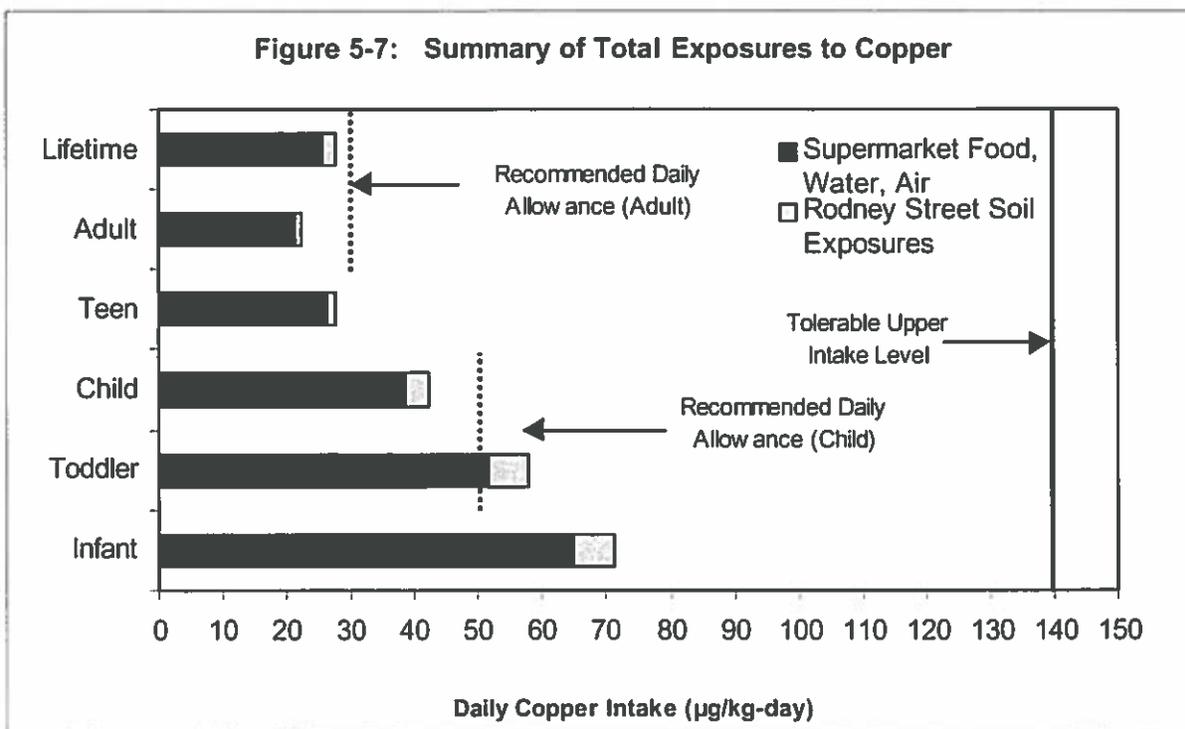
5.6 Nickel
5.6.1 Ingestion Exposure to Nickel

To characterize the potential health risks for residents of the Rodney Street community, the total nickel intake from all exposure pathways (Table 5-6), was compared with the US EPA RfD of 20 µg/kg-day (US EPA IRIS, 1998e - oral RfD assessment last revised 1996) (Table 3-1). Table 5-6 and Figure 5-8 shows that total nickel intakes are below the RfD for age groups over the age of five years. This situation is also true for people who live in the Rodney Street community throughout their entire life-time even when soil nickel levels are as high as 17,000 µg/g. The total chronic daily intake for people exposed to soil containing 17,000 µg/g nickel for their entire life-time averages 8.4 µg/kg-day (about 50% of the US EPA RfD).

Table 5-6: Life-time Averaged Daily Nickel Intakes for the Rodney Street Community

Metal	Receptor	Years	General Exposures			Rodney St Specific Exposures			Total CDI[4]
			EDI[1]	CDIf[2]	Σ CDI[3]	EDI[1]	CDIf[2]	Σ CDI[3]	
Nickel	0 - 6 months	0.5	13	0.09		7.4	0.053		
	7 mo - <5 yrs	4.5	12	0.8		13.0	0.84		
	5 - <12 years	7	8	0.8	5.4	7.2	0.72	3.0	8.4
	12 - <20 years	8	5.3	0.61		1.9	0.22		
	20 + years	50	4.4	3.1		1.6	1.1		

1. Estimated Daily Intake (µg/kg-day).
2. Chronic Daily Intake Fraction (µg/kg-day).
3. ΣCDI = sum of CDIf for General or Rodney Street community specific exposures (µg/kg-day).
4. Total CDI = Chronic Daily Intake for a life-time exposure to metal in the Rodney Street community (µg/kg-day).
Note: Due to rounding of values, total intakes may not add up to exactly the sum of the intakes for each row, in particular the final decimal place.

There is a great deal of uncertainty related to the assessment of infant exposures, most notably those related to soil and dietary exposures. The current assessment has defined infants as ranging from zero to six months of age. Most infants do not start moving around on their own until seven to nine months of age and as a result it is highly unlikely that infants would have much opportunity for direct soil contact. As a result, the amount of soil intake experienced by infants via the oral and dermal routes is likely minimal. To ensure conservatism these pathways were included in the assessment. With respect to food intakes, CEPA (1994b) and Dabeka (1989) have based infant diets on dietary surveys taken in the 1970s. A comparison of the infants exposures under the following scenarios is provided below and is also discussed in Appendix 4:

- Diet A - assumes that the infant only consumes breast milk for six months;
- Diet B - assumes that the infant only consumes formula for six months; and
- Diet C - assumes that the infant consumes breast milk or formula only for the first three months, and this diet is then supplemented by vegetables (backyard garden not considered), cereal and bread, and, fruit and fruit juices.

Diet A - Recent studies of nickel in human milk using the ICP-MS analytical method indicate low levels in European mothers. Biego et al., (1998) did not detect nickel in 17 breast milk samples from French mothers at a reported detection limit of 2.9 µg/L. Krachler et al., (2000) analyzed milk samples from 27 Austrian mothers and reported a median value of 0.79 µg/L (range - detection limit 0.13 µg/L to 6.35 µg/L). In a recent Canadian study, milk from 43 mothers living in Newfoundland was analyzed during the first 12 weeks of breast feeding (Friel et al., 1999). Median values for nickel ranged from 0 to 28 µg/L. If an infant consumes 850 mL breast milk per day (Emmett et al., 2000), nickel intakes resulting from Diet A could range from 0.67 µg/day to 23.8 µg/day.

Diet B - Dabeka (1989) reported milk based formulas as having half the nickel content of soy based formulas. For the 0 - 6 month age group, reported nickel intakes from formula range from 35.7 µg/day (evaporated milk) to 74.7 µg/day (soy based formula).

Diet C - The combination diet (Diet C) suggested above uses Nutrition Canada's values for vegetable, cereal and fruit intakes for this age group (33g, 34.4g and 75.7g/day, respectively). Using the overall nickel intake for each of these food groups based on Dabeka and McKenzie (1995) and the Nutrition Canada food intake factors, this results in a nickel intake estimate of 143 µg/day. Diet A and three months of vegetable, cereal and fruit intakes suggests overall intakes of 72.2 µg/day to 95.3 µg/day over the first six months. Similarly, Diet B and three months of vegetable, cereal and fruit intakes suggests overall intakes of 107.2 µg/day to 146.2 µg/day over this period. The mid point of this range (72.2 to 146.2 µg/day) is 109.2 µg/day.

Consideration of these reasonable infant diets indicate that at 17,000 µg/g (Table A1-4) it is unlikely that the infant's exposure exceeds the established exposure limit. For the toddler exposure scenario, the estimated total daily intake is 25 µg/kg-day at the 17,000 µg/g level. A large and fixed percentage of these intake estimates is due to exposures not influenced by soil nickel concentrations (supermarket food, drinking water and ambient air; see Figure 5-9) which account for 46.3% of the total intake. Other age groups do not exceed the US EPA RfD at the 17,000 µg/g level. The relationships of total daily intake, age class and the US EPA RfD at 17,000 µg/g nickel in the soil are shown in Figure 5-8.

Figure 5-8: Summary of Total Exposures to Nickel (17,000 µg/g)

- ■ Supermarket Food, Water, Air
- □ Rodney Street Soil Exposures

US EPA Exposure Limit (Lifetime Protection)

Daily Nickel Intake @ 17,000 µg/g in soil (µg/kg-day)

Figure 5-9 Pathway Breakdown for the Toddler (@ 17,000 µg/g Nickel in the Soil)

air 0.1%

water 0.2%

market basket 46.3%

soil/dust 42.0%

home garden 11.5%
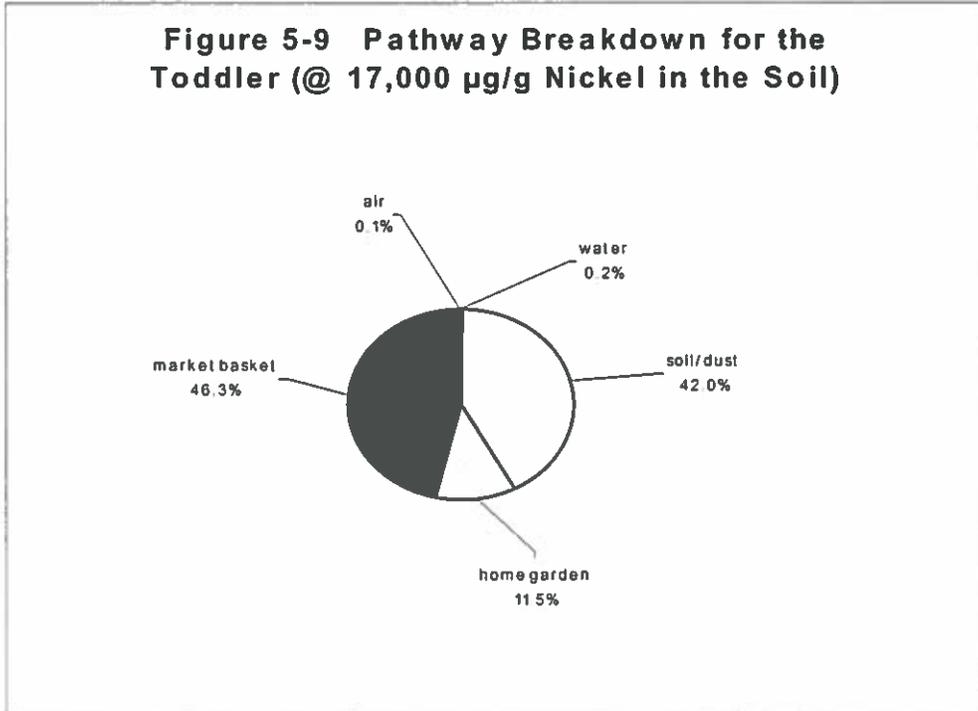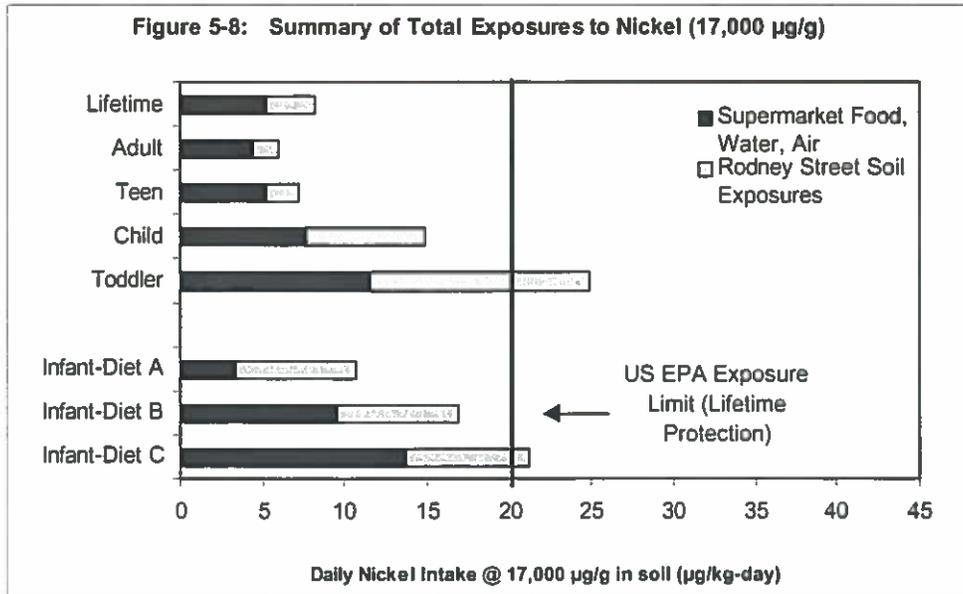
Since the toddler receptor group exceeds the US EPA RfD the need for an intervention level was evaluated. In the case of nickel:

1. The health benchmark used is the US EPA (IRIS) (1998e - oral RfD assessment last revised 1996) and Institute of Medicine (2001) RfD for soluble nickel salts. This RfD is for life-time protection and the endpoints of concern are reduced organ weight and reproductive endpoints found in animal studies.

2. It is generally assumed by most authorities that the RfD given in terms of μg/kg body weight /day is valid for children as well as for adults, while recommending that the exposure of infants and children to contaminants be kept as low as practicable. Although it is generally recognized that children may be more susceptible to the effects of contaminants than adults, the evidence often comes from differences in exposure rather than from animal studies. All major regulatory agencies (Health Canada, US EPA, OECD/WHO) have placed a high priority on protecting children's environmental health. Currently, there are no standard national or international regulatory approaches for developing criteria to protect sensitive groups, such as children, although research is now underway in this area.

For nickel, the most exposed life-stage is the toddler age group and as such it is considered prudent, in this case, to establish a soil intervention level that does not exceed the RfD for this group. Consideration of the lifetime CDI for total nickel exposures, including Rodney Street community specific exposures, indicates that by setting a soil intervention level to protect the toddler aged group, that the RfD would not be exceeded by older age groups.

3. At the present time, evidence of actual harm from exposure to soils containing elevated levels of nickel in Port Colborne is being assessed in a separate ongoing health study administered through the Community Based Risk Assessment (Ventana CHAP).

4. There are no similar assessments of other communities with similar or higher levels of nickel exposure.

At a concentration of 9,061 μg/g of nickel in the soil, the calculated total nickel exposure (general plus Rodney Street specific) which is 20 μg/kg-day, is equivalent to the RfD (20 μg/kg-day) for the toddler. Under the 'reasonable' diet scenarios, the infant's exposure is less than the RfD as well. All the other age groups do not exceed the US EPA RfD at this level. The relationships of total daily intake, age class and the US EPA RfD, a concentration of 9,061 μg/g nickel in the soil are shown in Figure 5-10. Figure 5-11 shows that market food is still the predominate exposure pathway.

Figure 5-10: Summary of Total Exposures to Nickel (@ 9,061 µg/g Nickel in the Soil)

Legend:
- ■ Supermarket Food, Water, Air
- □ Rodney Street Soil Exposures

US EPA Exposure Limit (Lifetime Protection)

Categories (y-axis): Lifetime, Adult, Teen, Child, Toddler, Infant-Diet A, Infant-Diet B, Infant-Diet C

x-axis: Daily Nickel Intake @ 9,061 µg/g in soil (µg/kg-day) — 0.00, 5.00, 10.00, 15.00, 20.00, 25.00, 30.00, 35.00, 40.00

Figure 5-11 Pathway Breakdown for the Toddler (@ 9,061 µg/g Nickel in the Soil)

- air 0.1%
- water 0.2%
- soil/dust 27.8%
- home garden 14.3%
- market basket 57.6%

5.6.2 Inhalation Exposure to Nickel

Levels of nickel in ambient air in Port Colborne are well below MOE's 24 hour air standard for nickel (see Table A1-2a and Table A1-3). In assessing potential cancer risks it was assumed that all of the nickel in air is in the form of nickel oxide. This likely is a conservative assumption, given no information on the form of nickel in the TSP is available.[1] Potential cancer risks from inhaling airborne nickel were assessed by comparing the annual average air concentrations for 1992 to 1995 in the MOE air monitoring data for Port Colborne (Table A1-2a) with inhalation unit risk factors from various agencies (Table 3.2). In this case, the mean annual average nickel concentration for 1992 to 1995 was 0.033 $\mu g/m^3$. Recent air concentration data for the Rodney Street community collected in 2001 is also used (see data in Appendix A1, Section A1-1.2).

Using the US EPA inhalation unit risk of 2.4×10^{-4} $(\mu g/m^3)^{-1}$ (Table 3.2) for nickel refinery dusts (US EPA IRIS, 1998d - carcinogenicity assessment last revised 1991), the air concentration of nickel at the 10^{-6} life-time cancer risk (one-in-1,000,000) level is 0.004 $\mu g/m^3$.

Using the WHO air quality guideline for nickel (WHO, 2000), the air concentration at the 10^{-6} life-time cancer risk level is 0.0025 $\mu g/m^3$.

Using the Health Canada TC_{05} of 0.04 mg/m^3 for combined oxidic, sulphidic and soluble nickel, division by 50,000 provides a 10^{-6} life-time cancer risk benchmark to judge the adequacy of indoor and ambient air (Health Canada, 1996). This 10^{-6} life-time cancer risk air concentration is 0.0008 $\mu g/m^3$.

In each case, the predicted 10^{-6} life-time cancer risk air concentration ranges from 1/2,500,000th to 1/12,500,000th of the air nickel concentrations known to cause lung cancer in nickel refinery workers (10 mg/m^3) (Doll et al., 1990).

As discussed in section A2-9.2.3, all three agencies considered lung cancer mortality data from overlapping sets of occupational studies (e.g., all three agencies used the Norwegian data, and both the US EPA and Health Canada also used the Ontario data). As noted in section A2-9.2.3, all the underlying epidemiology studies used by these three agencies have been updated. The fact that the predicted air concentration of nickel associated with a 10^{-6} life-time cancer risk ranges from 0.0008 to 0.004 $\mu g/m^3$ (a five fold range) is a consequence of how each agency selected lung cancer mortality data and mathematically extrapolated the dose-response information.

The US EPA IRIS (1998d - carcinogenicity assessment last revised 1991), WHO (2000) and Health Canada (1996) inhalation unit risk factors were developed for nickel refinery dust which

---

[1] The ministry has submitted air samples from the MOE ambient air monitor in the Rodney Street Community to the Stanford Synchrotron Radiation Laboratory (the same lab that conducted nickel speciation analysis on the Rodney Street soil samples - see Part A Main Document Section 6.2.2). This is research level work that the ministry has recently learned is possible for particulate in air samples.

contains different percentages of soluble and insoluble nickel than ambient air. In order to see how representative nickel refinery dust is of Port Colborne air, a comparison of the nickel refinery dusts inhaled by workers and associated with lung and nasal cancer in these workers was examined with airborne nickel in Port Colborne (Section A2-9.3.1).

It would appear that apart from the electrowinning process and nickel plating where the dust is mainly soluble nickel salts, the proportions of soluble and insoluble nickel species (i.e., a large insoluble percentage) in nickel refinery dust and ambient air are comparable. Similarly, the old "total" sampler and TSP sampler data are comparable in the particle size range collected. Consequently, inhalation cancer risks estimated on this basis, are not unreasonable. However, estimates based on using unit risks with air sampler data with other particle size cut points may over or underestimate lung cancer risk potential.

To provide further context to the estimates of potential inhalation cancer risks for the 1992 to 1995 air monitoring data, it should be noted that the 1995 annual average air concentration of nickel was 0.023 $\mu g/m^3$. In addition, recent air monitoring at the Rodney Street site for nickel over a five month period in 2001 (Tables A1-2a, A1-2b and A1-3) yield the following interim results:

> average Ni concentration from TSP sampling = 0.013 $\mu g/m^3$ (max. = 0.12 $\mu g/m^3$);

> average Ni concentration from PM_{10} sampling = 0.006 $\mu g/m^3$ (max. = 0.026 $\mu g/m^3$).

Over this period, the average airborne particulate readings were: TSP concentration = 46.1 $\mu g/m^3$ (max. = 100 $\mu g/m^3$); PM_{10} concentration = 21.9 $\mu g/m^3$ (max. = 66 $\mu g/m^3$). The particulate data show a typical mass concentration relationship, i.e., TSP mass about twice the PM_{10} mass (see section A2-9.2.1.1). The nickel concentrations for the PM_{10} and TSP monitoring follow a similar relationship. This recent PM_{10} nickel air monitoring data fits in with the range of federal 1995-1999 PM_{10} nickel air monitoring data for Ontario (average = 0.003 $\mu g/m^3$ (range 0.0007 $\mu g/m^3$ to 0.035 $\mu g/m^3$) (Table A1-3).

Based on the above and Table A1-2a, the air concentrations of nickel in Port Colborne indicate a continuing downward trend since 1992 with a corresponding lowered inhalation cancer risk. If the current annualized average air concentration of nickel is 0.013 $\mu g/m^3$, the corresponding life-time inhalation cancer risk range, using the US EPA (US EPA IRIS, 1998d), WHO (2000) and Health Canada (1996) inhalation risk factors, is 3.1 X 10^{-6} to 1.6 X 10^{-5}. This risk estimate is interim until additional air sampling data is available for 2002 and the ministry receives results from the speciation analysis.

5.7 Arsenic
5.7.1 Human Health Significance of Measured Soil Arsenic Levels

Arsenic is a known human carcinogen. Long term chronic ingestion of arsenic has been associated with skin changes including skin cancer and is reported to increase the risk of cancer of the liver, bladder, kidney and lung (ATSDR, 1993). Major public health agencies base their quantitative assessment on skin cancer as the most critical effect. Unlike lead, exposures over the entire life-time are more important than exposures during childhood only. Because of the extensive experience with risk evaluation of arsenic in soil in other Ontario communities (Port Hope, (MOE, 1991); Deloro (MOE, 1999); and Wawa (MOE, 2001a), it is considered that replication of similar calculations would not shed any light of additional value on arsenic risks in this situation. Rather, Port Colborne is compared to these other Ontario communities to determine whether the levels here are out of the ordinary and whether it is plausible that increased health risk could occur.

An important consideration regarding potential exposure to arsenic in these soils is that arsenic ions form insoluble salts with a number of cations in soils and are adsorbed by soil constituents, such as organic colloids and iron and aluminum oxides. Arsenic is held quite strongly by soils, especially fine-textured ones, and is leached very slowly. As such, relatively high levels of arsenic in soil may pose little risk if they are indeed highly insoluble and therefore not available for absorption if swallowed. In fact, the measured average solubility of arsenic in these soil samples using bioaccessibility testing is 35% (Appendix 5). However, it should be noted that very low and non-detectable arsenic levels were measured in backyard vegetables.

People everywhere, including Ontario, are chronically exposed to low levels of arsenic in the environment and as such everyone has a certain amount of risk. These exposures can occur by a number of different pathways including normal diet and drinking water. To understand the potential relevance of the measured soil levels in Port Colborne it is useful to compare the levels found with levels elsewhere in the province and in particular with the findings of health studies around arsenic conducted in the province (MOE; 1991, 1999, 2001).

The average level of arsenic in the Rodney Street community is approximately 16 µg/g. This is just below the Ontario background value of 17 µg/g, which is the 98th percentile of typical urban parklands in Ontario. Out of all properties sampled there were two which had arsenic levels greater than 100 µg/g in the soil. Background soil arsenic levels in North America range from 1 to 40 µg/g. Measured arsenic levels in Port Hope, Ontario average 20 µg/g, with a maximum of around 250 µg/g (MOE, 1991). Most typical urban communities show this type of distribution with a few elevated properties within a given area (see Maps 4, 5 and 6, Part A). A comparison of soil arsenic levels in the Rodney Street community with other communities where arsenic-related health studies have been conducted is shown in Table 22 (Part A).

In the Port Hope risk assessment study (MOE, 1991) average soil arsenic levels were roughly double what they are in the Rodney Street community, with several properties in excess of 100

µg/g. Incremental cancer risk levels for soil arsenic exposure were calculated to be less than one-tenth of the calculated cancer risk from arsenic in the normal diet and determined to be non-significant. The assumed bioavailability of arsenic in soil for Port Hope has a similar range to the arsenic bioaccessibility in the Rodney Street community soils. It can be concluded that exposures to the arsenic in soil in Port Colborne would not produce a significantly different cancer risk. Also, it is not anticipated that these levels would lead to increased arsenic levels in people as measured in urine.

Similarly, a more recent health study (the most comprehensive of its kind undertaken in Ontario) considered arsenic soil contamination in the village of Deloro, Ontario (MOE 1999) where levels averaged 111 µg/g (seven-fold greater than the average in the Rodney Street community) and ranged up to 605 µg/g. In this study no adverse health effects were identified or predicted related to the arsenic in the village. Measured urinary arsenic levels were almost identical to a control population from a nearby community not exposed to elevated concentrations of arsenic in soil. Most significantly, there was no association between soil arsenic levels and urinary arsenic levels in the residents. A detailed health survey revealed no arsenic associated health effects in the community. Further, it was estimated that removal of all arsenic in soil would reduce the overall calculated risk to the residents by no more than 4%.

After the remediation of nickel contaminated properties the maximum soil arsenic levels in the Rodney Street community is about 1/4 the maximum level in Port Hope, about 1/10th the maximum level in Deloro, and about 1/17th the maximum concentration of arsenic in soil in Wawa. Wawa and Deloro have undertaken health studies and a health risk assessment was conducted in Port Hope (see Part A Main Document Section 9.5 and Table 22). These studies concluded:

Wawa

This study did not produce evidence that Wawa residents are at an increased risk of cancer due to exposure to arsenic (Goss Gilroy 2000).

Deloro

Estimated arsenic exposures are not measurably higher than those of typical Ontario residents (Cantox 1999).

Port Hope

Estimated intakes from contact with these soils yield risk estimates in the range generally considered negligible and below the WHO permissible intake (MOE 1991).

Based on these recent studies conducted in these three Ontario communities with higher soil arsenic levels, it is unlikely the soil arsenic concentrations in Port Colborne will result in increased exposures to Rodney Street community residents.

5.8 Lead
5.8.1 Human Health Significance of Measured Soil Lead Levels

Lead in soil has long been recognized as posing potential risk, particularly to younger children ages one to four years, who may play in lead contaminated areas (MOEE, 1994). Because of their higher contact rates with soil and higher rates of intestinal absorption for lead as compared with adults, young children will generally have greater exposures by this pathway. Although exposures of women of child bearing age due to fetal exposure issues merit consideration, such exposures will generally be much smaller and result in smaller absorbed intakes than for children. Therefore, young children may be considered the most susceptible receptor for exposures for direct soil/dust ingestion, and therefore characterization of risk should focus on this subgroup. Exposure to lead in soil occurs predominantly through the eating of soil or dust. Breathing of dust and skin absorption are considered minimal.

Lead may be taken up into edible plants from the soil; therefore home gardening may also contribute to exposure if the produce is grown in soil containing high lead concentrations. Simple measures such as thorough washing of vegetables prior to preparation and consumption can minimize exposure to lead on the surface of home produce. Other measures to reduce personal lead exposure are contained in the MOE's Fact Sheet entitled "Frequently asked questions about lead contamination" (MOE, 2001c).

5.8.2 Typical Urban Soil Lead Concentrations

It is useful to compare the reported levels of lead in soil in this neighborhood with those in other Ontario urban areas in order to postulate whether exposures to lead here could be greater. Bearing in mind that there is no "typical" urban residential site, one may examine other Ontario residential sites in built-up areas that are not obviously associated with any lead-related industry (although the areas may have been influenced to some degree by other industry, vehicle exhaust deposition, etc.).

The mean lead content of Windsor soils was 45 µg/g with some samples approaching 300 µg/g (Weis and Barclay, 1985). In 1990, MOE (1994) found that lead concentrations in Windsor surface soils ranged from 23 to 128 µg/g with arithmetic and geometric means of 59 and 40 µg/g and a median concentration of 36 µg/g. Linzon et al., (1976) report in a survey of an Ontario downtown area, serving as control for samples collected near a lead industry, lead levels in surface soils (0 - 5 cm) averaging 482 µg/g, with a range of 18 to 1,450 µg/g. Also, lead levels near roadways and major intersections in urban areas can exceed 500 µg/g (MOE, 1986). O'Heany et al., (1988) reported levels of lead in surface soil from school play areas (excluding sandy and baseball diamond soils of unknown origin) in urban (Windsor and Toronto) and suburban (Scarborough and Etobicoke) Ontario. The levels ranged from 11 to 180 µg/g (average of the means 63 µg/g) in urban soils and from 6 to 81 µg/g (average of the means 21 µg/g) in suburban soils. Samples of house dust, street dust and yard soil were analyzed for lead and other elements in a survey of surficial yard soil from fifty one homes in the Ottawa area. Lead

concentrations in surficial garden soils ranged from 16 to 547 µg/g and the arithmetic and geometric means, and median concentrations were reported to be 65 µg/g, 42 µg/g and 34 µg/g, respectively (Rasmussen et al., 2001). MOE sampling data from 1999 demonstrate mean soil lead concentrations from properties in Welland and Sarnia of 329 and 629 µg/g, respectively (see Table 18 in Part A). It can thus be suggested that average soil lead levels in the Rodney Street community area (mean, median, and 90th percentile of 217, 173, and 406 µg/g respectively) are in a similar range to other urban residential sites in Ontario.

As well, the pattern of lead levels on these residences is consistent with very localized spots of higher (> 1,000 µg/g) contamination related to leaded paint, automotive batteries or fuel use. For example, mean soil lead concentrations of samples the MOE collected from less than one metre from structures on properties in Welland and Sarnia were 475 and 1,150 µg/g, respectively (see Table 18 in Part A). By corollary, estimated exposures in Port Colborne (and hence blood lead levels) would be predicted to be on average similar to those for other urban Ontario populations.

Urban areas outside of Ontario also have similar reported soil lead concentrations. Sutton et al., (1995) reported residential soil lead concentration geometric means (and ranges) of 188 (30 - 1,973) µg/g, 234 (26 - 2,664) µg/g, and 897 (56 - 88,176) µg/g in L.A., Sacramento, and Oakland California, respectively. There have also been two recent US wide studies of lead in soil. The US EPA performed a study (US EPA, 1996a) where 381 housing units constructed before 1980 across the US were inspected for lead in paint, dust, and exterior soil. Of the privately-owned homes, 23% exceeded 400 µg/g soil lead concentrations and 11% exceeded 1,000 µg/g. The mean soil lead concentration (for soils collected away from buildings but still on the properties) was 183 µg/g for homes built pre 1940. A similar study by the U.S. Department of Housing and Urban Development (US HUD, 2001) reported that 21% of homes had maximum soil lead concentrations ≥ 400 µg/g and 12% ≥ 1,200 µg/g.

Generally, older residential areas typically have soils with higher lead concentrations. Soil lead concentrations in the three Californian communities discussed above (Sutton et al., 1995) were highly and significantly correlated with age of housing (construction year). This study determined that 86% of pre 1920 homes, 67% of 1950-1970 homes, and 11% of homes built after 1970 had maximum soil lead concentrations ≥ 500 µg/g. In a US wide study by the US HUD (2001) the results showed that 67% of pre 1940 homes, 31% of 1940-1959 homes, 9% of 1960-1977 homes, and virtually none of 1978-1998 homes had maximum soil concentrations over 400 µg/g. It was determined that the homes with earlier construction dates were more likely to have maximum soil concentrations over 400 µg/g. Similarly, the US EPA US wide study (US EPA, 1996a) reported geometric means for soil lead concentrations (collected away from structures on the properties) of 183 µg/g for homes built pre 1940, 67 µg/g for 1940-1949 homes, 44 µg/g for 1950-1959 homes, and 23 µg/g for 1960-1979 homes. It was concluded that the strongest predictor of soil lead was the housing unit's date of construction. The Rodney Street community is one of the oldest urban residential areas in Port Colborne. About 95% of the homes were built before 1949, and about 40% were built before 1920. The average house age is 78.

Construction year is probably a measure of historical paint lead concentrations, the deterioration of paint, and the deposition of atmospheric lead in soil over time (Sutton et al., 1995). Lead-based paint and lead additives to gasoline have added massive quantities of lead to the urban environment in modern times (Mielke and Reagan, 1998). Indeed the US wide studies by the US EPA (1996a) and the US HUD (2001) indicate that soil lead levels are consistently higher closer to structures. The US EPA (1996a) study reported geometric means, for soil lead concentrations collected close to structures, of 480 µg/g for pre 1940 homes, 151 µg/g for 1940-1949 homes, 70 µg/g for 1950-1959 homes, and 27 µg/g for 1960-1979 homes. Approximately 35% of discharged lead from leaded gasoline was in the form of tiny particles able to disperse over large areas from the roadway, and in a typical urban environment may have spread over the majority of the city (US EPA, 1998a). Regarding lead from paint, although the highest amount of leaded paint falls to the dripline area (along the walls of structures), it can travel quite widely within a neighbourhood (Gulson et al., 1995). Since lead does not biodegrade, decay or wash away, the lead deposited in dust and soil becomes a long-term source of lead exposure (ATSDR, 1988; Shinn et al., 2000). An estimated 4 to 5 million tons of lead previously used in gasoline remain in dust and soil to which people continue to be exposed (ATSDR, 1988). In the Rodney Street community, 40% of the houses were built before 1920, and 54% were constructed between 1920 and 1949, and 6% were built after 1950 (see Part A, Section 8.7.1).

5.8.3 Relationship Between Soil Lead and Blood Lead

It is also relevant to discuss briefly the current scientific information relating to lead in soil and blood lead levels in young children (for review see MOEE, 1994; Davies, 1988). This question has been examined to some extent in a number of epidemiological investigations. Some studies have found positive correlations between soil lead and blood lead levels in children, particularly where soil lead levels exceed 1,000 µg/g. Blood lead appears to vary directly with soil lead concentrations in some cases. The range of reported average slope factors (which attempt to describe this relationship numerically) is 0.6-8.0 µg/dL per 1,000 µg/g soil lead (MOEE, 1994; Davies, 1988; Steele et al., 1990), based on roughly 20 studies using a range of data analysis methods. For example, the study of Baltrop et al., (1975) in Derbyshire, England, concluded that soil lead contributed 0.6 µg/dL per 1,000 µg/g soil in a rural area where industrial point sources of lead no longer operate. Another study has demonstrated no apparent elevation in mean blood lead concentrations (compared to low exposure groups) for children in two English villages with mean soil lead levels of greater than 1,000 µg/g (Baltrop et al., 1975). In a review of blood lead studies by Steele et al., (1990), it was concluded that mine waste-derived lead in soil has a lesser impact on blood lead (slope factors ranging between 0 and 4 µg/dL per 1,000 µg/g soil lead) than lead in soil derived from operational smelters or urban settings (vehicle or paint sources) (slope factors ranging between 1 and 8).

A recent analysis (Lanphear et al., 1998) pooling 12 epidemiologic lead studies estimated the contributions of lead-contaminated dust and soil to children's blood lead concentrations. Results demonstrated that soil lead concentrations of 100, 500, 1000, and 1500 µg/g predicted blood lead concentration ranges of 2.9-7.6, 3.5-9.0, 3.8-9.7, and 4.0-10.2 µg/dL, respectively, depending on

the house dust lead loading. In another analysis (Jin et al., 1997), several studies were reviewed where soil exposure could be distinguished from other sources of lead. It was reported that large changes in ambient soil lead are required to achieve relatively modest changes in blood lead. Compared to children exposed to soil lead levels of 100 µg/g, those exposed to levels of 1000 µg/g had mean blood lead concentrations that were 1.10 to 1.86 times higher.

In a recent study in the Township of West Carleton, Ontario, reported mean soil lead levels exceeding background concentrations were associated with low blood lead concentrations (ROCHD, 2000; MOE, 2001b). Soil lead concentrations on residential properties averaged 306 µg/g and the highest concentration found was 3,900 µg/g. The average blood lead concentration from a blood lead screening in the target area was 0.092 µmol/L (1.9 µg/dL) for children under the age of seven and 0.113 µmol/L (2.34 µg/dL) for non-pregnant persons over six years old.

It is important to realize that environmental conditions greatly influence this relationship. Generally those that exhibit slope factors at the upper end of the range typically involve settings which are arid and lacking grass cover where the soil lead will be present as lead dust. These sites generally involve operating lead-based industry emissions (lead smelters, mining and battery plants). In contrast, those with the lowest slopes tend to not involve lead dusts or arid conditions. For example, in the study of Baltrop et al., (1975) in Derbyshire, almost all soil was grass covered and there appeared to be little influence of the soil lead upon children's blood lead levels. Although one can not rely on this pattern entirely, it would suggest that in the Rodney Street community where there is not a great deal of bare soil in sampled areas nor a lead-based industry, that a large influence on blood lead by soil lead would not be expected. For illustrative purposes only, conservatively assuming that a very high slope factor operated in this situation, say a 7 - 8 µg/dL increase per 1,000 µg/g lead in soil, and knowing that background blood lead levels in Ontario children are on average 3.1 - 3.5 µg/dL (Wang et al., 1997; Langlois et al., 1996), it would require soil lead levels of approximately 1,000 µg/g to cause blood lead levels to increase above the level of concern of 10 µg/dL recommended by the US Center for Disease Control (CDC, 1991), Health Canada (1996), the National Research Council (NRC, 1993b), and the MOE (MOEE, 1994). From this perspective it can be concluded that based on a highly conservative assumption, soil levels below 1,000 µg/g should not pose an appreciable risk, whereas those at 1,000 µg/g and greater, allowing for some individual variability between different children, should be considered for risk-reduction measures. Recent blood lead data collected from children under the age of seven in the Rodney Street area support this conclusion.

5.8.4 Blood Lead Concentrations in Port Colborne

The Regional Niagara Public Health Department (RNPHD, 2001) undertook ten blood lead screening test clinics between April and June 2001, for residents living or frequently spending time in the Rodney Street community area. Pregnant women, women of reproductive age and children under seven, were strongly encouraged to participate in the blood lead level screening tests. Over 1,000 people participated in the blood lead level screening tests. The results indicated there is no immediate health concern for residents, and that the average blood lead concentrations in the Eastside community of Port Colborne (the residential community adjacent to the Inco refinery that includes the Rodney Street community) are low and are similar to those for the rest of Port Colborne and elsewhere in the province. Further, there was no correlation demonstrated between properties with elevated soil lead levels and elevated blood lead levels in residents. In particular, the study notes that all children who lived on properties with surface soil lead concentrations greater than 400 µg/g tested below the level for medical review (10 µg/dL) and that the five adults with elevated blood lead concentrations (mean of 13.1 µg/dL) did not live on properties with elevated soil lead concentrations.

The blood lead screening report concluded that children under seven years and pregnant women in the Rodney Street community are not at increased risk of lead exposure as compared to other communities in Ontario, even considering the localized elevated soil lead levels. The mean blood lead concentration of 2.3 µg/dL for Eastside community children of Port Colborne was less than the mean of 3.5 µg/dL found in the Ontario Blood Lead Control community (Langlois et al., 1996).

In general, based upon consideration of; a) typical urban lead levels in Ontario and in the US, b) consideration of findings regarding the observed relationship between soil lead and blood lead in other communities, and c) the recently measured blood lead levels in children under the age of seven from the Rodney Street area, it can be concluded that exposure to lead in these soils should not result in undue health effects in this community. It cannot be concluded that the reported values on average would lead to undue elevation of blood lead levels overall in this community. At the same time, based upon findings in the literature it is prudent to conclude that in the few residences with reported levels above 1,000 µg/g in soil, there may be some possibility for exposures that result in some elevation in blood lead levels in children who routinely play in these areas.

5.8.5 Consideration of Lead Intervention Levels and Exposure Reduction of Lead in Soil

Individuals can substantially reduce their exposure to lead in soil in many ways. Regular hand and face washing to remove lead dust from young children, especially before meals, can lower the possibility of accidentally swallowing lead in dust while eating. Regularly cleaning the home of tracked in soil and removal of shoes after having been in soil areas will also reduce exposure. Planting of grass, or other coverings over bare areas of a yard can lower contact that children and pets may have with soil and the tracking of soil into homes.

With respect to identifying a specific soil lead level which requires intervention through soil removal or other form of remediation, it must be remembered that a large variety of risk factors influence lead exposure in any given situation and as such there is not one universal lead in soil standard that can be applied to all cases. Determining the specific contribution of any particular environmental variable like soil/dust to blood lead level is extremely difficult. This difficulty is also confounded by significant other factors such as socioeconomic status and dietary exposure. For instance, the numerous variables studied in Ontario blood lead studies (MOH, 1984; MOH/MOE, 1990) were unable to account for more than 30% of the variations seen in blood levels in children. The range of observations on the relationship between soil lead and blood lead seen in various studies is a further reflection of the difficulties of determining such associations. As such selection of a single value for this situation involves considerable judgement.

An intervention level or other exposure reduction controls should have some reasonably clear potential for not elevating blood lead levels in children to medical levels of concern (10 µg/dL blood lead). The range of slope factors relating soil lead to blood lead is quite wide but consideration of the upper end of the range suggests that levels above 1,000 µg/g could result in elevation of blood lead, possibly to levels of concern. As a result, it would seem prudent to err on the side of caution and select 1,000 µg/g as an intervention level for remediation/control at a residence in the absence of individual blood lead testing data for the nine residences which fall into this category. This would be applied to both bare and grass covered soils.

Another approach to development of a soil lead level of concern is to utilize multi-pathway exposure modeling. One such tool is the US EPA Integrated Exposure Uptake Biokinetic (IEUBK) Model for Lead in Children (US EPA, 2001a). In simple terms, this model converts estimates of lead exposure from different routes and predicts a blood lead level in children. Utilizing dietary (including homegrown produce), air, and drinking water intakes and exposure factors for a Rodney Street community child (2 - 3 years) and assuming conservative soil lead bioavailability of 30%, the model predicted blood lead levels ranging from 3.7 to 6.4 µg/dL over the soil lead range of 200 to 1,000 µg/g. This is consistent with the analysis above that suggests an intervention level of 1,000 µg/g as sufficiently protective under typical exposure conditions. It should be noted that a lead bioavailability of 30%, is conservative and is the default used by US EPA in the IEUBK model (US EPA, 2001a).

As noted in the preceding section, the mean blood lead concentration for Eastside community children of Port Colborne under seven years was 2.3 µg/dL indicating that the IEUBK model

may over predict the soil lead - blood lead relationship for this community.

Also relevant to the choice of an appropriate intervention level are existing regulatory standards or guidelines from other jurisdictions. Most recently US EPA (2001) has developed a new lead in soil hazard standard under section 403 of the Toxic Substances Control Act. After initial consideration of a 2,000 µg/g standard and extensive public comment, the following standards were established: a soil lead hazard standard of 400 µg/g for bare soil in play areas and an average of 1,200 µg/g for bare soil in non-play areas of the yard. The EPA view is that this is a pragmatic approach which focuses exposure reduction actions on those areas where exposures may be highest for children. This approach would appear reasonable and adoption of a similar stratified approach for this situation seems sensible. Use of a 400 µg/g soil lead level in bare soil of children's play area is prudent given the possibility of higher exposures in these areas for some children.

One other important consideration is the role of soil removal and other lead abatement activities in reducing actual exposure. Some studies have examined the effectiveness of soil lead abatement on blood lead concentrations. Soil remediation alone has been shown to result in a reduction of children's mean blood lead concentrations, but remediation is usually more effective on reducing children's blood lead levels when their blood levels are initially quite high. Soil abatement accompanied by house dust abatement and education of the residents may be more successful. When considering the effectiveness of soil abatement, it is important to recognize that childhood lead exposure comes from a number of media (e.g., interior paint, exterior paint, soil, house dust) across a range of environments (e.g., child's residence, school, playground, friend's residence) (US EPA, 1998b).

A number of large studies regarding followup to soil lead abatement in inner city environments (Aschengrau et al., 1997; Burgoon et al., 1994; Farrell et al., 1998; US EPA, 1996b; 1998b; 2000b) and communities near smelters (Gagne, 1994; Langlois et al., 1996; Hilts et al., 1998) where high lead levels were prevalent throughout the community have been reported. The focus of these studies has been whether re-contamination has occurred following remediation, and, how effective the lead abatement program has been in reducing children's blood lead levels. By contrast, the Rodney Street community properties exceeding the 400 ppm and 1000 ppm soil lead intervention levels appear to have higher soil lead levels than the surrounding properties and re-contamination with lead is not likely (see Maps A19, A20 and A21 in Part A). As already noted the mean blood lead concentration in the Eastside community children tested in 2001 (RNPHD, 2001) is well below 10 ug/dL, the medical level of concern.

5.9 Combined Risks of Multiple Contaminants in the Rodney Street Community

A number of successful risk assessment models that combine the effects of multiple contaminants with similar modes of action and disease endpoints exist (US EPA, 1996c, 2000c, 2000d, 2001c). However, there are no biologically convincing models that combine the risks of multiple contaminants with dissimilar modes of action and disease endpoints. The current report has considered the combined exposure of all environmental exposure pathways, including

background exposures, such as, supermarket food, and normal Ontario-wide background levels of chemicals in air, drinking water and soil for each individual chemical of concern. This combined exposure was compared to the exposure limit for the individual chemical. As explained below a number of conditions must be met before meaningful addition of different chemicals can be done. Currently, for most regulatory bodies, the accepted *de minimis* risk level for a single chemical in a single environmental pathway is 1-in-one-million. So, just by having several pathways for the same chemical, the cumulative risk could rise above 1-in-one-million. Adding multiple pathway risks for multiple chemicals also distorts the application of the *de minimis* risk concept.

Some risk assessments do include estimates of the overall cancer risk of contaminant mixtures by adding cancer risks for contaminants with similar modes of action and endpoints. These overall risk estimates can be compared with annual incidences for specific types of cancer for the local population, this may not be meaningful for the Rodney Street community because relatively large populations are needed.

For non-cancer endpoints, the ratio of the estimated exposure to the selected exposure limit (called an exposure ratio (ER) or hazard quotient (HQ)) can be calculated. These ER or HQs can be summed to estimate overall non-cancer risks for specific endpoints (sometimes called a hazard index (HI)). As indicated above, it is not clear how meaningful these estimates of overall risk are since mixtures of contaminants may have antagonistic effects as well as additive effects. Current risk characterization methodologies consider that the HQ for a single environmental pathway for an individual chemical should be less than some fraction of the HQ, e.g., 0.1 or 0.2. If multiple exposure pathways are combined, then the overall HQ for an individual chemical should be less than or equal to one. In a multiple chemical situation, with multiple environmental pathways, the interpretation of HI greater than one is problematical. The addition of multiple risks or HQ is a mathematical construct which can only be related to health effects under highly restricted conditions.

The US EPA has a number of initiatives that are being developed to address what is termed "cumulative risk assessment". The US EPA released a preliminary draft of its first cumulative risk assessment in early December 2001 (US EPA, 2001c). This is the first time the US EPA has produced a major analysis of the combined risks of organophosphate pesticides, rather than each substance in isolation. This cumulative risk assessment considers pesticides that have a common mechanism of toxicity, meaning that all the pesticides have the same effect on a given organ or tissue in the human body. In addition, the US EPA Risk Assessment Forum has provided technical guidance for conducting health risk assessment of chemical mixtures (US EPA, 2000). It should be noted that the emphasis of these US EPA initiatives has been on mixtures of chemically similar or related mixtures, e.g., organophosphate pesticides, PCBs, dioxins and furans, PAHs, and not on chemically unrelated mixtures with different mechanisms of toxicity, such as metals and metalloids.

Combined risk assessments of multiple chemicals are commonly used when environmental reviews of the emissions from large facilities such as incinerators, landfills and other industrial

processes are conducted. These combined risk assessments attempt to sum the modeled exposures and health effects of proposed facilities as a screening exercise when evaluating the terms of approval or rejection of proposals for these facilities. These risk assessments are used to set operating parameters and specify pollution control devices for the proposed facility. In most cases, the modeled population exposures are theoretical and bear no relationship to actual populations. Consequently, the application of this type of combined risk assessment to the Rodney Street community has limitations. More research is required before the combined risk of multiple contaminants that have the potential to affect different health endpoints can be confidently assessed in health risk assessments.

6.0 Discussion of Uncertainties
6.1 General Discussion of Uncertainty

The risk assessment process requires that many assumptions be made, either because of gaps in available monitoring data, or because of an improper or incomplete understanding of how people are likely to be exposed to the contaminants of concern. For example, when estimating daily exposures to a chemical, it is necessary to assume specific body weights in order to determine daily doses on a per body weight basis, which is necessary in order to make predictive estimates of potential health effects. However, large variations in body weights are normal between people in any of the age groups considered. The use of such assumptions results in a degree of uncertainty in the overall estimates of exposure and risk and in the final conclusions of the risk assessment. As regulators, conservative or precautionary assumptions are made to err on the side of caution and to ensure that the risk assessment does not under estimate the potential for adverse effects.

Another way of approaching uncertainty is to say "how reliable" are the conclusions of the risk assessment, or, what is the "confidence" in the process? When discussing the degree of confidence one holds in the results of the risk assessment, it is useful to illustrate the distinction between variability and uncertainty.

6.1.1 Variability In the Data

Variability is the most common type of uncertainty found within an assessment, and is based upon the differences or diversity inherent within populations or samples of exposure parameters or individuals. Sources of variability are generally viewed as the result of natural random processes, as well as environmental or genetic differences among the species or elements being evaluated. For example, the analytical results for the testing of air, water, soil and backyard produce from Port Colborne and the Rodney Street community have some degree of sampling and analytical error. Unlike uncertainty, variability in the data can be quantified statistically, and our understanding of the overall variability can be improved through further sampling exercises. To indicate the variability in the key receptor and intake parameters used in the HHRA, and, to indicate whether the value used was a central tendency, upper confidence level or maximum value, Tables 6-1 to 6-3 were prepared.

Table 6-1: Critical Receptor Parameters Used to Estimate Daily Exposures

Parameter	Units	Toddler 0.5 - <5 yrs	Metric	Source
Body Weight	kg	16.5 ± 4.5	Mean values from empirical Canadian data. These data were derived from three Canadian surveys conducted in 1970-72, 1981 and 1988 (Demirjian 1980, CFLRI 1981, CFLRI 1988 - described in O'Connor et al., 1997). Toddler body weight was based on data from Demirjian (1980), but adjusted for evident weight increases in the Canadian population observed between 1970 and 1988.	O'Connor, 1997; CCME, 2000
Inhalation Rate	m³/day	9.3 ± 2.6	Mean inhalation rates were based on a Monte Carlo simulation incorporating quantitative time-activity data with minute volume data for various levels of physical activity for each age group considered (O'Connor, 1997).	O'Connor, 1997,
Drinking Water Intake	L/day	0.6 ± 0.4	For toddlers, Canadian data do not exist. Therefore, a mean rate was derived by calculating a weighted mean for sub-groups reported by Ershow & Cantor (1989 - cited in O'Connor, 1997) within the desired age range.	O'Connor, 1997; CCME, 2000
Soil Ingestion	g/day	0.1	90th percentile	US EPA, 1997; 2000; Stanek et al., 2001
Soil Adhesion to Skin	mg/cm²	0.2	95th confidence interval	US EPA, 2000, Holmes et al., 1999
Surface Area of Hands	m²	0.043 ± 0.005	Mean values are based on equations developed by US EPA for estimating skin surface area from measurements of weight and height; Canadian weight and height data were then employed for calculations of skin surface areas of various body parts.	O'Connor, 1997
Surface Area of Arms	m²	0.089 ± 0.024	"	O'Connor, 1997
Surface Area of Legs	m²	0.169 ± 0.034	"	O'Connor, 1997
Surface Area of Feet	m²	0.043 ± 0.008	"	O'Connor, 1997
Backyard Root Vegetables	g/day	7.7	Fixed percentage of Canadian per capita food consumption rates based on assumed crop yield from a 30 m² vegetable garden (Southern Ontario).	MOEE 1995, Section A6-7
Backyard Other Vegetables	g/day	4.9	"	MOEE 1995, Section A6-7

Table 6-1: Critical Receptor Parameters Used to Estimate Daily Exposures

Parameter	Units	Toddler 0.5 - <5 yrs	Metric	Source
Backyard Fruit	g/day	6.8	"	MOEE 1995, Section A6-7
Supermarket Food	g/day	1478	Mean intakes estimated from Canadian food consumption data obtained from the 1973 Nutrition Canada National Survey.	Health Canada, 1995
Amount of time spent outdoors-summer	hrs/day	4.3	Based on assumed time activity patterns and time spent outdoors taken from the US EPA Exposure Factors Handbook.	Section A6-8, US EPA, 1997
Amount of time spent outdoors-winter	hrs/day	2	"	Section A6-8, US EPA, 1997
Amount of time spent indoors-summer	hrs/day	19.7	"	Section A6-8, US EPA, 1997
Amount of time spent indoors-winter	hrs/day	22	"	Section A6-8, US EPA, 1997
Amount of time spent in Rodney Street Area (July and August)	hrs/day	23	"	Section A6-8, US EPA, 1997
Amount of time spent in Rodney Street Area (remainder of year)	hrs/day	23	"	Section A6-8, US EPA, 1997

Table 6-2: Critical Environmental Nickel Concentration Parameters Used to Assess Residential Exposures

Medium	Units	Nickel Concentrations	Metric	Source
Drinking Water	μg/L	1.3	Maximum concentration found in Port Colborne municipal drinking water distribution system (mean concentration = 0.94; range = 0.6 to 1.3).	Table A1-1
Ambient Air	μg/m³	0.033	Mean of annual average air concentrations measured on hi-vol TSP air samplers at Davis & Fraser for 1992-1995 (range = 0.023 to 0.053).	Table A1-2
Soil	μg/g	17000	Maximum soil concentration at 0-20 cm depth (mean = 2544; range = 35 to 17,000).	Table A1-4
Backyard Root Vegetables	μg/g	2.44	95th percentile of over 180 relevant plant samples (Mean = 0.63; range = 0.03 to 5.17).	Table A1-12a and Table A1-12b
Backyard Other Vegetables	μg/g	2.44	"	Table A1-12a and Table A1-12b
Backyard Fruits	μg/g	2.44	"	Table A1-12a and Table A1-12b
Supermarket Food	μg/day	190	Based on mean nickel concentration per food item and Canadian food consumption factors.	Dabeka & McKenzie, 1995

Table 6-3: Critical Receptor Intake Parameters Used to Assess Residential Exposures

Medium	Units	Metric	Source
Soil Bioaccessibility	% soil ingestion	The maximum mean measured soil bioaccessibility (19 for nickel) under both acidic and neutral extraction conditions and both ground and unground soil samples (range = 12 to 23).	Appendix 5 and Table A5-17
Indoor air to Outdoor air ratio	% outdoor air concentration	Midrange value (0.75) of several reported ratios from studies of the contribution of outdoor sources to indoor concentrations.	Section A3-1.3
Indoor dust to Outdoor soil ratio	% outdoor soil concentration	Midrange value (0.39) of several reported ratios from contaminated sites and the US EPA default value for Pb in soil (0.70) (range = 0.20 to 0.70).	section A6-5.2
Dermal uptake coefficient	unitless	Based on the details of 3 studies of nickel penetration of excised human skin. US EPA (1992) default factors for other metals.	Appendix 7

6.1.2 Scientific Judgement

This type of uncertainty is introduced into the process when scientific judgement must be used to bridge gaps in analytical, toxicological or receptor characteristic data. For example, in estimating dermal exposure to metals it is necessary to use scientific judgement in selecting reasonable dermal uptake factors for metals where direct information is not available. In this type of situation, uncertainty in the parameter may be mitigated either by obtaining more data or by the use of conservative estimates that are applied in a consistent manner throughout the risk assessment.

Several areas of uncertainty exist within the current risk assessment. These are discussed in the following sections. In addition, a discussion of the implications that each has on the overall conclusions of the risk assessment is provided as a summary.

6.2 Uncertainties in Environmental Media Concentrations

There are several areas in the estimates of metal concentrations in environmental media (air, water, soil, backyard vegetables, diet) where uncertainties could have been introduced into the risk assessment including:

6.2.1 Estimates of Dietary Intakes of Metals

Information on the level of metals in typical foods and the daily intake of metals from the diet is limited. Reasonable data is available for nickel and several of the other metals considered in this report. However, even within these data sets there are discrepancies in the estimates of daily dietary intakes between the populations examined. For instance, estimates of daily dietary intakes of nickel by the Canadian population are two to three times the levels estimated for the US and UK populations (Appendix 4).

The Canadian data (as reported by Dabeka and McKenzie, 1995) was used in this assessment because it was felt that this provided the best reflection of likely dietary intakes for the residents of the Rodney Street community. This approach avoids under estimates of the likely dietary intake of nickel for the residents of the Rodney Street community, which in turn, ensures that upper bound estimates of total daily intakes are calculated. Similar approaches are used for the other metals considered in the detailed assessment of risk. It should be noted that this Canadian data was based upon one study conducted in the city of Montreal, and as such, may not be fully representative of the dietary habits of the Rodney Street community, or the Port Colborne population as a whole. Studies from other jurisdictions (e.g., U.S. and U.K. market surveys) have shown significantly lower level of nickel intake from foods than estimated based upon the Canadian data (see Table A4-4 in Appendix 4). While some uncertainty does exist in the Canadian data, these other studies do provide confidence that the nickel intake from diet would not be underestimated in this assessment. Additionally, Dabeka and McKenzie (1995) indicated that stainless steel cooking utensils (e.g., oven pan and roasting pan) appear to contribute to the higher levels of nickel in cooked steak, ground beef, port, lamb, and poultry. This may provide some rationale as to why the intake concentrations reported by Dabeka and his colleagues were higher than those presented in other jurisdictions.

6.2.2 Metal Levels in Backyard Garden Produce

The current assessment has had the benefit of more metal in backyard garden produce information than the previous assessment carried out for Port Colborne (MOE, 1998). However, even the current data only provides limited information on metal levels in a limited selection of crops. To address any uncertainties that may be introduced due to the limited nature of the present data, all available literature was considered. For the current assessment, metal levels in the examined crops were assumed to be representative of the levels found in all crops of an equivalent type. In addition, the 95th percentile of the reported levels found in the root and other vegetable categories were used to estimate exposures. This approach will over estimate likely intakes of metals. While it may be possible that future site-specific sampling may locate produce with marginally higher metal concentrations, there is sufficient conservatism built into the calculation of the soil intervention level to accommodate variability in produce metal concentrations within the Rodney Street community.

6.2.3 Metal Levels in Ambient Air

Long term ambient air monitoring specific to Port Colborne was available for copper, lead and nickel. The remainder of the metal levels relied on Environment Canada data for southern Ontario. While there is no reason to believe that southern Ontario data for these other metals is not representative of ambient air levels in Port Colborne, the lack of direct data introduces a level of uncertainty into the assessment. However, the detailed exposure assessment clearly showed that inhalation exposures to metals in ambient air make a very minor contribution to the total daily exposure and do not, in themselves, represent a risk. For the metals that are potential inhalation carcinogens, this uncertainty has a large impact on inhalation risk estimates.

6.2.4 Metal Levels in Soil

An extensive sampling program was undertaken in the Rodney Street community. Metal levels were assessed in almost 2,000 samples. However, even with a large data set there is a potential for an error of up to 20% in the reported metal level for any one sample. To address this, the highest level of each metal was used to assess exposures for all residents of the Rodney Street community regardless of where they resided. The soil data from the properties were statistically evaluated to determine the confidence interval around the maximum value of all the samples collected from any single property. Given the large data base and the log-normal distribution of the soil nickel levels across the Rodney Street community, the asymptotic distribution (also known as the Gumbel distribution - see Johnson and Kotz, 1995) of the maximum likelihood estimates (normal) was used to construct confidence intervals around the maximum concentration for any property. From the human health risk assessment, it was determined that at a soil nickel concentration of 9,061 µg/g, the nickel exposures from all sources including the Rodney Street community soil-specific exposure is less than the R$_f$D for the toddler and all other age groups. Using the confidence intervals from the Gumbel distribution of the combined 2000 and 2001 Rodney Street residential soil data, it can be shown that, if the maximum soil nickel level of any single sample at any depth from a property is 8,139 µg/g or less, it is 99% certain that the total nickel exposure including the Rodney Street community soil-specific exposure is less than the R$_f$D for the toddler and all other age groups. Therefore, to ensure that no property is missed from remediation due to sample variability, the soil intervention level was further lowered to 8,000 µg/g (See Part A, section 9.3). Soil sampling and analytical variability and data confidence is discussed in Part A (sections 5.1, 5.4, and 5.5).

6.2.5 Metal Levels in Indoor Dust

Metal levels were predicted in indoor dust based on literature information comparing indoor dust levels to outdoor soil levels (section A6-6.2). It was assumed that the daily intake of soil and dust would occur from the soil and dust associated with the highest reported level of each metal. It was further assumed that this would occur throughout the year including in winter conditions when direct exposure to residential outdoor soils is limited.

It is also important to note that some data (Shorten and Hooven, 2000) has demonstrated the

existence of indoor dust "reservoirs" on inaccessible surfaces within schools which have slowly accumulated based upon historical dust levels within the buildings (potentially arising from historical air emissions). Lead dust concentrations were significantly elevated on the inaccessible surfaces (tops of filing cabinets and light fixtures) yet they were uniformly low on accessible surfaces (desktops and windowsills) and the children's palms. Lead in the inaccessible surfaces had no effect on blood lead levels in the children over a six month period indicating that the mere presence of lead in inaccessible dust does not automatically constitute a health hazard. If indoor dust reservoirs are not disturbed (i.e., extensive renovations), one would not expect indoor dust concentrations to be significantly elevated. Although events such as renovations may result in an elevation of ambient metal concentrations in indoor air and dust, these exposures would be of a very short term nature and expected to occur infrequently. Further investigations are currently underway in the Port Colborne region which should provide a more detailed data set to evaluate the relative contribution of indoor dust "reservoirs" to metal exposures. Indoor dust in Port Colborne homes will be further explored as part of the indoor environment monitoring program planned in the CBRA.

6.2.6 Estimates of Metal Bioaccessibility from Soil

The metals in the soil, particularly nickel, are largely present in insoluble forms that are tightly bound to the soil matrix. Therefore their accessibility to the body, either in the gut, or through the skin, will depend upon their release from the soil. While the metals may be less soluble in water at neutral pH (6 - 8), their solubility increases under acidic conditions. To address the potential for metal release from the soil during digestion, soils were subjected to both a simulated stomach acid leach test and bioaccessibility testing (Appendix 5).

There is also some uncertainty with respect to the relative bioavailabilities of metals in outdoor soils *versus* those present with indoor dust. Indoor dust is typically composed of much finer-sized particulate than that found in the outdoor ambient environment. While very little data is available to determine differences in relative bioavailabilities, one study on arsenic concentrations administered to Cynomolgus monkeys indicated that the oral bioavailability of metals in indoor dust was slightly higher than that present in outdoor soils (Freeman et al., 1995). However, as noted elsewhere, *in vitro* bioaccessibility values were used to estimate intakes.

6.3 Uncertainties in Receptor Characteristics

There are several areas related to the characteristics and activity patterns of the residents where uncertainties can be introduced into the assessment of exposure and risk.

6.3.1 Receptor Characteristics

As noted above, the use of single point values to characterize the population does not account for the wide variation that exists within any community. The receptor parameters used in this assessment have been taken from Canadian sources and are based on statistical surveys of the Canadian population. As such, they can be considered to be reasonably representative of the residents of the Rodney Street community.

Statistical methods exist to address these variations and provide ranges of exposures and risk for a community. However, these techniques are really only beneficial when an initial worst case assessment demonstrates that potential risks exist. The current assessment made use of conservative assumptions to provide reasonable upper bound estimates of exposure and risk. These showed that even at the highest predicted exposures for antimony, beryllium, cadmium, cobalt and copper, risks do not exist within the community. Therefore a refinement of exposure estimates to account for variation within the population was not deemed necessary.

6.3.2 Activity Patterns

The current assessment assumed that a person would be living in the Rodney Street community for a full 70 year life-time. All exposures were assumed to occur within this community. Correction was made for time spent away for this area. This approach will provide an upper-bound estimate of potential exposures from the site.

6.3.3 Dermal Contact with Soil

Dermal contact for metals is generally considered to be a very minimal pathway of exposure. However, in the current assessment, dermal exposure was estimated to ensure that this contribution to the total daily exposure was examined.

6.3.4 Consumption of Home Grown Produce

There is some uncertainty associated with the actual amount of home grown produce a family could consume. For the current assessment, it was conservatively assumed that a family of four would consume 100% of the total garden yield. In addition to this assumption, annual backyard garden yields were based on an assumed garden size and an estimated average crop yield. Depending on the actual family size, garden size, and crop yield, this may be an over or under-estimate of individual exposure. However, given that there was no reduction of exposures to home garden produce due to crop loss (e.g., browsing by wildlife and birds or spoilage), it is concluded that the estimates employed in this assessment would be conservative.

This assumption results in a calculated home grown produce consumption rate which represent approximately 9.9% of the total produce (i.e., both home grown and store bought produce) consumed by the Rodney Street residents, in addition to the 100% food consumption assumed from market basket produce. It should also be noted that very few of the residences in the

Rodney Street area have home gardens. However, this pathway has been considered in all cases to ensure that the assessment is sufficiently conservative so as to enable any resident to grow and consume home garden produce without any health risk based upon soil metal concentrations.

6.4 Uncertainties in Toxicity Assessment
6.4.1 Non-Cancer Endpoints

Each of the toxicologically based exposure limits used to estimate potential health risks related to non-cancer endpoints have uncertainty factors associated with them. These factors account for the strength of the toxicological data and incorporate uncertainty factors to account for intraspecies and interspecies extrapolations of toxicological data. These uncertainty factors reflect the adequacy of the toxicological data available for each compound. Where toxicological data is poor or limited to one or two studies, large uncertainty factors are applied to ensure adequate protection of sensitive members of the population. The result is a general over-estimation of potential risks from exposure. Thus, exposures which exceed the exposure limits may not always result in adverse health effects. The uncertainty factor attached to each exposure estimate gives a measure of this potential. The lower the uncertainty factor, the more certain the data and the more predictive of adverse health effects an exposure limit is. In these cases, the probability that exceedances of exposure limits will result in adverse health effects is higher. For exposure limits with higher uncertainty factors, the probability of adverse effects occurring as a result of limited exceedance of exposure limits is thought to decline. A more detailed discussion of uncertainty factors and their derivation is provided in Section 1 of Appendix 2.

The toxicity and exposure limits selected in this study also have conservative assumptions incorporated in them. Since these parts of the risk assessment were taken from the reviewed literature and from recognized regulatory agencies, discussion of their uncertainty is beyond the scope of this report. However, it should be noted that the toxicological basis of exposure limits is updated as new information becomes available so every effort was made to ensure that recent information was used.

The US EPA is a reliable source of exposure limits or reference doses (R$_f$D) for ingestion exposures and reference concentration (R$_f$C) for inhalation exposures, that are developed from toxicological studies of human or animal populations. These are set to ensure that chronic exposures to a chemical at concentrations that are at or below the exposure limit will not result in adverse effects. The US EPA (US EPA IRIS, 1998a-e)defines the R$_f$D/R$_f$C as;

> *A quantitative estimate (with uncertainty spanning perhaps an order of magnitude) of a daily exposure to the human population (including sensitive subgroups) that is likely to be without an appreciable risk of non-carcinogenic, deleterious effects during a life-time.*

There may be uncertainty as to whether the current US EPA R$_f$D of 20 µg nickel/kg-day (US EPA IRIS, 1998e - oral R$_f$D assessment last revised 1996) completely protects against the contact dermatitis experienced by a percentage of the population, mainly female, who are already sensitized to nickel through wearing jewelry, dental or surgical prostheses or other contact with

metallic nickel and stainless steel.

6.4.2 Cancer Endpoints

Derivation of exposure limits for cancer endpoints usually involves extrapolating from animal data to humans and from high doses (the doses which cause measurable effects in animals) to low doses (where there will be no effect in humans). More recently, where there is better information on human exposure, epidemiological information can be used to extrapolate to human risks removing the animal to man extrapolation uncertainty.

Carcinogens are assumed to have no threshold for their effects. Mathematical models have been used to extrapolate from animal cancer bioassay studies to levels where the risk of exposure is negligible in a large population (10^{-5} to 10^{-6} risk level) (Health Canada, 1996). These mathematical models are extremely conservative and make several assumptions which may not hold true for the behavior of chemicals in the body. Some of these assumptions introduce uncertainty into the mathematical extrapolation:

- the no-threshold assumption, i.e., that a single molecule can cause an event leading to cancer development. Carcinogenesis is a multi-step process with three generally recognized processes, namely, initiation, promotion and progression. It is also known that non-genetic processes can trigger or control gene expression and that specific genes are involved in various cancers. Chemical theory and behavior of biological systems suggests that a sufficient population of molecules must be present to trigger an event such as initiation and also to promote the subsequent stages of carcinogenesis. Consequently, in some cases, a "practical" or operational threshold concentration may be involved;

- the genotoxicity assumption - mathematical models assume that direct action on the genetic material is involved. Several carcinogens are indirect in that they cause cancer without causing direct damage to genetic material. The "standard" mathematical models do not account for this;

- the mathematical models extrapolate several orders of magnitude from the realm of measured effects (high dose) in biological systems to a range where nothing can be verified biologically.

For these reasons, likelihood estimates are used for carcinogens with a range of risks over which some description of whether the impact is negligible or not can be assigned. Most major jurisdictions consider the level of life-time cancer risk of one incidence per million (10^{-6}) population to be a "de minimis" risk. Ontario's drinking water, and effects-based air standards are both based on a cancer risk level of 10^{-6} where attainable. With respect to the ministry's guidance on the development of site specific soil criteria (Level 1 risk management), the ministry specifies the "use of an excess life-time cancer risk of one in a million (10^{-6}) for each exposure pathway for non-threshold parameters (carcinogens)." However, a distinction must be made between calculating a cancer risk estimate based on environmental sampling data (a normal

output from a HHRA), and setting a standard based on a cancer risk level of 10^{-6}. In one case, a risk estimate is calculated, and in the second case, an environmental exposure limit or concentration is set to prevent exposure risks exceeding a lifetime cancer risk level of 10^{-6}.

Human epidemiological studies have many limitations related to proving that there is a causal association between chemical exposure and effects in the population, i.e., the healthy worker effect, mis-classification of workers, confounding variables, such as smoking, latency period, the fact that associations can only be used for populations not individuals, and other difficulties in showing dose-response relationships.

6.4.3 Extrapolation from Occupational Air Data

The uncertainties associated with extrapolating from the occupational air sampling data used to estimate lung cancer risk in nickel refinery workers (section A2-9.3.1), to potential lung cancer risks due to current levels of airborne nickel in Port Colborne are described in section A2-9.2.1.1. A comparison of the particle size distributions, masses and speciation of nickel compounds in occupational settings and ambient air and the sampling methods available to sample airborne nickel indicates that TSP-based measurements can be compared with the older "total" personal air sampling data from the nickel refinery workers for estimating potential lung cancer risk associated with breathing Port Colborne air. The use of PM_{10} and $PM_{2.5}$ data introduces more uncertainty and a requirement to prorate older workplace exposure data so that "respirable" fractions are comparable.

6.5 Uncertainties in the Risk Characterization
6.5.1 Using an Exposure Limit for Nickel Soluble Salts

In estimating potential risks associated with ingestion and dermal exposures to nickel, the report made use of the non-cancer oral exposure limit for soluble nickel salts put forward by the US EPA (US EPA IRIS, 1998e - oral RfD assessment last revised 1996) and the Institute of Medicine (IOM, 2001). The nickel in soil in the Rodney Street community has been identified predominantly as nickel oxide which is insoluble in water. Therefore there are some potential uncertainties associated with using an oral exposure limit set for a soluble form of the metal. However, the risk assessment made use of both a stomach acid leach test and bioaccessibility testing to determine the amount of nickel that could be released from the soil matrix during digestion. This digestion would, in fact convert nickel oxide to soluble forms. Once released from the soil matrix, the nickel would be in the form of a soluble salt. Thus, the comparison of this acid soluble fraction of soil nickel to an exposure limit set for a soluble form of nickel is appropriate.

6.5.2 Use of Life-time Chronic Daily Intakes (CDI)

The current assessment developed life-time chronic daily intakes (CDI) estimates from all age groups (life stages) to estimate the life-time exposure. These values were compared to the reference dose limits set by the US EPA. This is the approach recommended by the US EPA. The

use of a life-time CDI is appropriate for the current assessment because exposures have been considered to occur nearly every day over a 70 year life-time, a truly life-time exposure.

Estimates of exposure for individual age groups have also been compared to the reference dose. If exposures for individual age groups exceed the reference dose as in the case of nickel exposure to the preschool toddler, then a soil intervention level was established. However, the likelihood that life-time exposures will result in adverse effects is limited. This approach is conservative.

6.6 Implications of Uncertainties
6.6.1 Systemic Health Effects

There are a number of areas where uncertainties may have been introduced into the current assessment of exposure and risk. Throughout, conservative assumptions have been used in an effort to provide estimates of the reasonable upper bound exposures. The objective was to determine if these exposures had the potential to cause adverse health effects in the residents of the Rodney Street community. The risk characterization has shown that even under the conservative conditions that have been assumed to exist in the Rodney Street community, exposures to metals in the soil in the community would not be expected to result in adverse health effects. In most cases, the estimated exposures were significantly lower than the exposure limits identified for each metal.

6.6.2 Contact Dermatitis

The current assessment for nickel shows that the risks of systemic effects occurring as a result of exposure to nickel in the soil are limited. However, the potential for contact dermatitis to occur in response to skin contact with soil, or through the ingestion of nickel bearing soil in individuals who have already been sensitized to nickel cannot been addressed at the present time. In the absence of exposure limits set to protect against this effect, the risk assessment process employed here cannot effectively address this issue. A more complete discussion of contact dermatitis can be found in Appendix 2 (section A2-9.2.4). The ongoing Ventana CHAPs health study initiated as part of the Port Colborne CBRA will investigate allergic responses to metals.

7.0 Recommendations and Conclusions

The intent of the Rodney Street community risk assessment was two-fold. One objective was to assess the potential for health risks from current exposure pathways (soil, air, drinking water and food). The second objective was to use pathway-specific information to develop soil intervention levels to protect against any immediate or future health risk for this community.

As part of the first objective, a reasonable upper bound exposure estimate was modeled using the maximum reported metal levels or other upper-bound estimates in surface soil within the Rodney Street community, in municipal drinking water, in backyard produce from the Rodney Street community, and average concentrations in supermarket food and in air monitoring data for Port Colborne or for nine other sites in Ontario. The exposure assessment looked at receptors for each age class (infant, toddler, child, teen and adult) and modeled exposures for inhalation, ingestion and dermal contact using standard exposure assessment methods. Adjustments were made for the fact that the predominant form of nickel in the Rodney Street community is insoluble nickel oxide. Acid leachate tests and bioaccessibility studies were used to adjust for the amount of each metal that would be bioaccessible in the digestive tract. A dermal uptake factor for nickel was developed based on several published studies of nickel permeation of human skin. In other cases, accepted dermal exposure factors were taken from the scientific literature.

Metal exposures for residents of the Rodney Street community were divided into two main components, those related either to dermal contact with metals in the soil or the ingestion of soil and/or backyard garden produce from the Rodney Street community, and general exposures to metals such as those experienced by people elsewhere in Ontario. These general exposures include supermarket food, municipal drinking water and ambient air. The major contributor, in all cases, to total daily intakes of metals is supermarket food which is independent of any local soil metal exposures experienced in the Rodney Street community.

When total metal exposures are broken down by age group, the highest exposures are for the infant and toddler age classes (up to five years old). Reasonable upper bound exposures for these age classes only exceeded the US EPA RfD for nickel.

Potential health risks from inhaling airborne metals were assessed by comparing either the highest or mean annual average air concentration in the MOE air monitoring data for Port Colborne or Environment Canada air monitoring data for Ontario (Table A3-3) with the selected inhalation exposure limit (RfC, unit cancer risk, etc.). In the case of antimony, beryllium, cadmium, cobalt and copper, there appears to be little or no potential for health related effects from inhalation of these metals in ambient air in the Rodney Street community.

The potential health risks associated with exposures to lead and arsenic in all environmental media in the Rodney Street community were assessed by comparison with human health risk assessments and health studies of other Ontario communities with elevated soil concentrations of these metals that were generally higher than those in the Rodney Street community. Conclusions and recommendations for arsenic and lead are described in separate sections below.

The second objective of this study was to develop intervention levels as needed to prevent any immediate or future health risk for this community.

7.1 Nickel

For nickel, the major soil exposure pathways (soil ingestion, dermal contact with soil and backyard produce consumption) were used to develop the nickel soil intervention level. Only the soluble portion of the insoluble nickel compounds (mainly nickel oxide) found in Rodney Street community soils, and the soluble nickel component of the other soil exposure pathways was considered. Soluble nickel has not been proven to be carcinogenic via ingestion. The inhalation pathway was only used to consider the non-cancer aspects of ingesting inhaled air particles. As illustrated in Section 4.4.6, the ingestion of inhaled particles accounts for less than 0.1% of the toddler's total nickel exposure and is a very minor soil exposure pathway.

The total chronic daily intake for people exposed to soil containing 17,000 µg/g nickel for their entire life-time is about 50% of the US EPA R/D. For the infant age class, total nickel intakes from the assumed diets may exceed the US EPA R/D; however, consideration of more reasonable infant diets indicate that even at 17,000 µg/g it is unlikely that the infant's exposure exceeds the established exposure limit (Table 5-6). At the maximum soil nickel concentration in the Rodney Street community (17,000 µg/g), the toddler receptor exceeds the R/D level of exposure. Additionally, the following should be noted:

1. The health benchmark used is the US EPA (IRIS) (1998e - oral R/D assessment last revised 1996) and is supported by the Institute of Medicine (2001) R/D for soluble nickel salts. This R/D is for life-time protection and the endpoints of concern are reduced organ weight and reproductive endpoints found in animal studies.

2. It is generally assumed by most authorities that the R/D given in terms of µg/kg body weight/day is valid for children as well as for adults, while recommending that the exposure of infants and children to contaminants be kept as low as practicable. Although it is generally recognized that children may be more susceptible to the effects of contaminants than adults, the evidence often comes from differences in exposure rather than from animal studies. All major regulatory agencies (Health Canada, US EPA, OECD/WHO) have placed a high priority on protecting children's environmental health. Currently, there are no standard national or international regulatory approaches for developing criteria to protect sensitive groups, such as children, although research is now underway in this area.

    For nickel, the most exposed life-stage is the toddler age group and as such it is considered prudent to establish a soil intervention level that does not exceed the R*f*D for this group. Consideration of the life-time CDI for total nickel exposures, including Rodney Street community specific exposures, indicates that by setting a soil intervention level to protect the toddler aged group, that the R/D would not be exceeded by older age groups.

3.   At the present time, evidence of actual harm from exposure to soils containing elevated levels of nickel in Port Colborne is being assessed by a separate ongoing health study (Ventana CHAP) as part of the Community Based Risk Assessment.

4.   There are no similar assessments of other communities with similar or higher levels of nickel exposure.

At a concentration of 9,061 µg/g nickel in the soil, the total exposure to nickel from general and Rodney Street soil specific exposures is equivalent to the R/D for the toddler. Under the 'reasonable' diet scenarios, the infants exposure is less that the R/D as well. All the age groups do not exceed the US EPA R/D at this level.

Part A, Section 7.1 identified that the soil samples collected in the Rodney Street community had variability. To ensure there is a high level of confidence that no property is missed from remediation due to sample variability the soil intervention level was lowered to 8,000 µg nickel/g soil.

This site specific soil intervention level of 8,000 µg nickel/g soil was developed specifically for the Rodney Street community. The available scientific data on nickel dermal sensitization are inadequate to determine a soil intervention level that would be protective to nickel dermatitis in sensitized individuals, or that would protect people from being sensitized to nickel. However, proposed health research studies in the Port Colborne community as part of the CBRA (Ventana CHAP) will be testing people for nickel contact dermatitis over the next year, which should provide additional data to allow for better characterization of this information gap.

The inhalation risk of airborne nickel was estimated using lung cancer potency factors from three agencies (Table 3-2). Taking a cautious approach, it was assumed all of the nickel in air is nickel oxide (the predominant form of nickel in soil in the Rodney Street community). In the case of inhaled nickel, the ministry calculated that the current life-time cancer risk from inhaling airborne nickel based on the 1992 to 1995 air monitoring data was mainly in the 1:100,000 risk range. This estimate of cancer risk from air in Port Colborne is likely overestimated because the mean of Port Colborne air data from 1992 to 1995 was used and the level of nickel in air was decreasing in each of those four years. The current level of nickel in air from the MOE sampling conducted in 2001 in Rodney Street is about 1/3 of the level used in the risk assessment. If the life-time cancer risk is calculated using the 2001 Rodney Street air data the calculated risk ranges from 3.1×10^{-6} to 1.6×10^{-5}, a range which is considered to be very low. This risk estimate is interim until additional air sampling data is available for 2002 and the ministry receives results from the speciation analysis.

With respect to the ministry's guidance on the development of site specific soil criteria (Level 1 risk management), the ministry specifies the "use of an excess life-time cancer risk of one in a million (10^{-6}) for each exposure pathway for non-threshold parameters (carcinogens)." The 10^{-6} guideline is applied when developing a site specific soil criterion and in this case, the air pathway was not used to develop the soil intervention level. The reasons for not applying the air pathway

include the facts that airborne nickel in the Rodney Street community is not exclusively derived from surface soils and Inco continues to emit nickel to Rodney Street community air from its ongoing cobalt refining operation. In urban areas, industrial emissions, traffic and residential heating emissions provide local contributions to the chemical composition of air particulate. Crustal emissions from local and larger areas of surface soils also contribute to particulate in air. Since the link between soil levels and ambient air concentrations cannot be reliably estimated the cancer risk from inhalation exposure was appropriately not used to derive the soil intervention level. MOE will continue to monitor ambient air in 2002 to confirm the declining trends in air nickel levels. In addition, the ministry will determine the form of nickel present in air to better estimate the risk. Inco, at the ministry's request, is also assessing air emissions from their Port Colborne refinery. The issue of soil re-entrainment as it relates to particulate and contaminants in air is being investigated by the air dispersion modeling being conducted for the greater Port Colborne area as part of the CBRA.

7.1.1 Recommendations for Nickel

1.   The site specific soil intervention level of 8,000 μg nickel/g for non-cancer effects developed specifically for the Rodney Street community, should be used to facilitate remediation of affected properties in the Rodney Street community.

7.2 Arsenic

It is concluded that the measured levels of arsenic in these soils are unlikely to pose an undue health risk to residents of this community based upon consideration of:

1.   Comparison to typical levels found elsewhere in Ontario.

2.   Knowledge of outcomes of health studies involving arsenic in soil exposure in other Ontario communities where the average and maximum soil arsenic levels were higher than those found in Port Colborne.

7.2.1 Recommendations for Arsenic

1.   Residents living on properties with arsenic levels above the MOE guideline should be provided with the MOE Greenfact Sheet entitled, "Arsenic in the Environment" (MOE, 2001d) which outlines simple measures related to reducing exposure.

2.   The Inco-sponsored CBRA Ventana CHAPs study will be examining levels of urinary arsenic in Port Colborne. If the results indicate exceedences of medical levels of review, a management strategy will be undertaken.

7.3 Lead

The weight of evidence supports an intervention level of 400 μg/g for residential or play areas of

bare soil in which children might possibly come into repeated contact. The weight of evidence supports an intervention level of 1000 µg/g for residential soils that are covered by grass or other barriers to exposure such as asphalt or concrete. These conclusions are based on:

1. The value of 10 micrograms of lead per decilitre of blood (10 µg/dL) in children has been broadly recognized as an accepted benchmark trigger for concern since 1991 (USCDCP, 1991).

2. Conservatively assuming a very high slope factor for soil lead contribution to blood lead, such as a 7 - 8 µg/dL increase per 1,000 µg/g lead in soil, and knowing that average blood lead levels in Port Colborne children are 2.3 µg/dL. It would require soil lead levels of approximately 1,000 µg/g or greater to cause blood lead levels to increase to the medical level of review of 10 µg/dL (MOEE, 1994; Davies, 1988; Steele et al., 1990).

3. Results from a recent blood lead screening study in the Township of West Carleton, Ontario indicate that children under the age of seven, from an area where residential properties had a mean soil lead concentration of 306 µg/g (MOE, 2001b) (approximately 100 µg/g greater than mean soil lead concentrations found in Port Colborne), had a mean blood lead concentration of 1.9 µg/dL (ROCHD, 2000), which is slightly less than the mean blood lead concentration found in Port Colborne children (2.3 µg/dL) (RNPHD, 2001).

4. A recent analysis (Lanphear et al., 1998) pooled 12 epidemiologic lead studies to estimate the contributions of lead-contaminated soil and dust to children's blood lead. Results demonstrated that a soil lead concentration of 1,000 µg/g predicted mean blood lead concentrations of 3.8 to 9.7 µg/dL, the range depending on the house dust lead loading.

5. Exhaustive evaluation of the relationship between soil lead and children's blood lead using the Integrated Exposure Uptake and Biokinetic (IEUBK) Model for Lead in Children (US EPA, 1994) links the 10 µg/dL value to a soil lead concentration of 400 µg/g (bare soil to which children have regular access).

6. The 400 µg/g intervention level is based on observations that there is an important relationship between direct contact with bare soil or dust and children's blood lead (Lumens et al., 1991; Freeman et al., 1997).

7. The ministry recognizes that the 400 µg/g intervention level for bare soil in play areas is conservative and that related work by other offices of the US EPA has indicated higher levels of lead in soil as appropriate intervention levels (US EPA, 1998c; 2001) for cases with lesser degrees of exposure. The ministry considers the 400 µg/g intervention level appropriate for bare soil to ensure that the lead burden of young children in urban areas in Canada is not increased by controllable environmental factors such as direct contact with lead in soil.

8.   A soil lead intervention level of 1000 µg/g is established for soils covered with grass based on related work by other offices of the US EPA that has indicated higher levels of lead in soil as appropriate intervention levels (US EPA, 1998c; 2001) for cases with lesser degrees of exposure. Offices in the US EPA have indicated that an average lead concentration of 1,200 µg/g in bare soil as appropriate in the non-play areas of the yard (US EPA, 2001b).

In addition, adoption of the US EPA stratified approach to a soil standard seems desirable to focus resources and efforts on those areas which have the highest exposure potential for children. Therefore, an intervention level of 400 µg/g for lead in bare soil play areas is reasonable to apply. Bare soil allows for greater contact of soil particles with children and therefore a more stringent value than 1,000 µg/g is warranted for these specific areas on a property.

7.3.1 Recommendations for Lead

1.   An intervention level be established for this community at a soil lead level of 400 µg/g for children's play areas with bare soil on residential properties or in public areas, and at a level of 1,000 µg/g for all other areas of these properties to which children have access.

2.   Soil removal, where conducted, should take into account the depth of the contamination. Wet methods of dust control should be employed.

3.   Residents living at properties exceeding these intervention levels for lead in soil should minimize/avoid contact with these soils and not consume vegetables from backyard gardens.

4.   All households with measured lead levels above the Ministry screening guideline (200 µg/g) receive the MOE fact sheet on "Frequently Asked Questions about Lead Contamination" (MOE, 2001c) to provide a better understanding of lead exposure and simple measures that can reduce potential exposure.

7.4 Antimony, Beryllium, Cadmium, Cobalt and Copper

For the metals, antimony, beryllium, cadmium, cobalt and copper, estimated total daily intakes for all age classes were well below stringent oral or inhalation exposure limits from major recognized jurisdictions, such as the US EPA, WHO and Health Canada.

No soil intervention levels for the metals, antimony, beryllium, cadmium, cobalt and copper, in soil in the Rodney Street community are recommended.

8.0 References

The references listed here pertain to the citations contained in this, the main document. Lists of the references used in each of the appendices are provided at the end of each appendix.

Aschengrau, A., A. Beiser, D. Bellinger, D. Copenhafer, M. Weitzman, 1997. *Residential Lead-Based-Paint Hazard Remediation and Soil Lead Abatement: Their Impact among Children with Mildly Elevated Blood Lead Levels*. Am. J. Public Health. 87:1698-1702.

ATSDR (Agency for Toxic Substances and Disease Registry). 1988. *The Nature and Extent of Lead Poisoning in Children in the United States: A Report to Congress*, July 1988.

ATSDR (Agency for Toxic Substances and Disease Registry). 1992. *Toxicological Profile for Cobalt*. US Department of Health and Human Services - Public Health Service (CDROM version, 2000).

ATSDR (Agency for Toxic Substances and Disease Registry). 1993. *Toxicological Profile for Arsenic*. US Department of Health and Human Services, Atlanta, Georgia, USA.

Baltrop, D., I.. Thornton, C.D. Strehlow, and J.S. Webb. 1975. *Absorption of lead from dust and soils*. Graduate Medical Journal. 51:801-804.

Biego, G.H., M. Joyeux, P. Hartemann and G. Debry. 1998. *Determination of mineral contents in different kinds of milk and estimation of dietary intake in infants*. Food Additives Contam. 15(7):775-781.

Burgoon, D.A., S.W. Rust, and B.D. Schultz, 1994. *A Summary of Studies Addressing the Efficacy of Lead Abatement*. In: Beard, M.E. and S.D. Allen Iske (eds.). *Lead in Paint, Soil, and Dust: Health Risks, Exposure Studies, Control Measures, Measurement Methods, and Quality Assurance*, ASTM STM 1226. American Society for Testing and Materials, Philadelphia, Pennsylvania, USA.

Cantox Environmental Inc. 1999. *Deloro Village Exposure Assessment and Health Risk Characterization for Arsenic and Other Metals*. Final Report Prepared for the Ontario Ministry of the Environment, and CH2H Gore & Storie Ltd., December 1999

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

CDC. 1991. *Preventing Lead Poisoning In Young Children: A Statement by the Centers for Disease Control.*

CEPA (Canadian Environmental Protection Act). 1994a. *Human Health Risk Assessment for Priority Substances.* Health Canada. ISBN 0-662-22165-5.

CEPA (Canadian Environmental Protection Act). 1994b. *Nickel and its compounds. Priority substances list assessment report.* Government of Canada: Environment Canada, Health Canada. ISBN 0-662-22340-3.

CFLRI. 1981. Unpublished data. Canadian Fitness and Lifestyle Research Institute. Ottawa. Cited in: O'Connor, 1997.

CFLRI. 1988. Unpublished data. Canadian Fitness and Lifestyle Research Institute. Ottawa. Cited in: O'Connor, 1997.

Dabeka, R.W. 1989. *Survey of lead, cadmium, cobalt and nickel in infant formulas and evaporated milks and estimation of dietary intakes of the element by infants 0-12 months old.* The Science of the Total Environment. 89:279-289.

Dabeka, R.W. and A.D. McKenzie. 1995. *Survey of lead, cadmium, fluoride, nickel and cobalt in food composites and estimation of dietary intakes of these elements by Canadians in 1986-1988.* J.A.O.A.C. 78:897-909.

Davies. 1988. *Lead in Soil: Issues and Guidelines.* Environmental Geochemistry and Health Monograph Series 4.

Demirjian, A. 1980. *Anthropometry Report: Height, Weight and Body Dimensions. A report from Nutrition Canada.* Health Promotion Directorate, Bureau of Nutritional Sciences, Health and Welfare Canada, Ottawa. 133 pages + unpublished data of the Nutrition Canada Survey. Cited in: O'Connor, 1997.

Doll, R., A. Anderson, W.C. Copper, I. Cosmatos, D.L. Cragle, D. Easton, P. Enterline, M. Goldberg, L. Metcalfe, T. Norseth, J. Peto, J.P. Rigaut, R. Roberts, S.K. Seilkop, H. Shannon, F. Speizer, F.W. Sunderman Jr., P. Thornhill, J.S. Warner, J. Weglo, and M. Wright, 1990. *Report of the international committee on nickel carcinogenesis in man.* Scand J Work Environ. Health. 16:1-82.

Emmett, P., K. North, S. Noble, and the ALSPAC Study Team. 2000. *Types of drinks consumed by infants 4 and 8 months of age: A descriptive study.* Public Hlth. Nutr. 3:211-217.

Ershow, A.G. and K.P. Cantor. 1989. *Total water and tap water intake in the United States: population-based estimates of quantiles and sources*. Life Sciences Research Office, Federation of American Societies for Experimental Biology. Cited in: O'Connor, 1997.

Farrell, K.P., M.C. Brophy, J.J. Chisolm Jr., C.A. Rohde, and W.J. Strauss, 1998. *Soil Lead Abatement and Children's Blood Lead Levels in an Urban Setting*. Am. J. Public Health. 88:1837-1839.

Freeman, N., A. Ettinger, M. Berry, and G. Rhoads, 1997. *Hygiene and Food Related Behaviors Associated with Blood Lead Levels of Young Children from Lead-Contaminated Homes*. Jour. Exp. Anal. Environ. Pathol. 7:103-118.

Freeman, G.B., R.A. Schoof, M.V. Ruby, A.O. Davis, J.A. Dill, S.C. Liao, C. A. Lapin, and P.D. Bergstrom. 1995. *Bioavailability of Arsenic in Soil and House Dust Impacted by Smelter Activities Following Oral Administration in Cynomolgus Monkeys*. Fund Appl. Toxicol. 28:215-222.

Friel, J.K., W.L. Andrew, S.E. Jackson, H.P. Longerich, C. Mercer, A. McDonald, B. Dawson and B. Sutradhar. 1999. *Elemental composition of human milk from mothers of premature and full-term infants during the first 3 months of lactation*. Biol. Trace Element Res. 67: 225-247.

Gagne, D. 1994. *Blood Lead Levels in Noranda Children Following Removal of Smelter-Contaminated Yard Soil*. Canadian J. Public Health. 85:163-166.

Gizyn, W.I. 1994. *Windsor Air Quality Study: Soil and Garden Produce Survey Results*. Phytotoxicology Section, Standards Development Branch, Ontario Ministry of Environment and Energy. ISBN 0-7778-03018-3. Fall 1994. pp. 15.

Goss Gilroy Inc. 2001. *Survey of Arsenic Exposure for Residents of Wawa*. Submitted to the Wawa Environmental Steering Committee, January 2001.

Gulson, B.L., J.J. Davis, and J. Bawden-Smith, J. 1995. *Paint as a source of recontamination of houses in urban environments and its role in maintaining elevated blood lead in children*. Sci. Total Environ. 164:221-235.

Health Canada. 1995. *Investigating Human Exposure to Contaminants in the Environment: A Handbook for Exposure Calculations*. Ottawa, Ontario, Canada. ISBN-0-662-23543-6. pp. 66.

Health Canada. 1996. *Health-based tolerable daily intakes/concentrations and tumorigenic doses/ concentrations for priority substances*. ISBN 0-662-24858-9.

Hilts, S.R., S.E. Bock, T.L. Oke, C.L. Yates, and R.A. Copes, R.A. 1998. *Effect of Interventions on Children's Blood Lead Levels*. Environ. Health Perspect. 106:79-83.

Holmes, Jr., K.K., J.H. Shirai, Y. Richter, and J.C. Kissel. 1999. *Field measurement of dermal soil loadings in occupational and recreational activities*. Environ. Res. Section A. 80: 148-157.

IOM (Institute of Medicine - Food and Nutrition Board). 2001. *Dietary Reference Intakes for Vitamin A, Vitamin K, Arsenic, Boron, Chromium, Copper, Iodine, Iron, Manganese, Molybdenum, Nickel, Silicon, Vanadium, and Zinc*. National Academy Press, Washington, D.C.

Jin, A., K. Teschke, and R. Copes, 1997. *The Relationship of Lead in Soil to Lead in Blood and Implications for Standard Setting*. Sci. Total Environ. 208:23-40.

Johnson, N. and S. Kotz. 1995. *Extreme Value Distributions*. In: *Continuous Univariate Distributions*. John Wiley & Sons. Toronto.

Krachler, M., T. Prohaska, G. Koellensperger, E. Rossipal and G. Stingeder. 2000. *Concentrations of selected trace elements in human milk and infant formulas determined by magnetic sector field inductively coupled plasma-mass spectrometry*. Biol. Trace Element Res. 76: 97-111.

Lanphear, B.P., T.D. Matte, J. Rogers, R.P. Clickner, B. Dietz, R.L. Bornschein, P. Succop, K.R. Mahaffey, S. Dixon, W. Galke, M. Rabinowitz, M. Farfel, C. Rohde, J. Schwartz, P. Ashley, and D.E. Jacobs, 1998. *The Contribution of Lead-Contaminated House Dust and Residential Soil to Children's Blood Lead Levels*. A Pooled Analysis of 12 Epidemiologic Studies. Environ. Res. A79:51-68.

Langlois, P., L. Smith, S. Fleming, R. Gould, V. Goel, and B. Gibson, 1996. *Blood lead levels in Toronto Children and Abatement of Lead-contaminated Soil and House Dust*. Archives of Environmental Health. 51:59-67.

Linzon, S.N., B.L. Chai, P.J. Temple, R.G. Pearson, and M.L. Smith, 1976. *Lead contamination of urban soils and vegetation by emissions from secondary lead industries*. J. Air Pollut. Contr. Assoc. 26:650-654.

Lumens, M.E., P. Uhlenbelt, H. Geron, and R. Herber, 1991. *The Impact of Hygienic Behavior on the Relation Between Environmental and Biological Monitoring*. Proceedings of the International Symposium, Luxembourg. eds. Brown, R., Curtis, M., Saunders, K., and Vandendriessche, S. pp. 83-85.

Mielke, H.W. and P.L. Reagan, 1998. *Soil is an Important Pathway of Human Lead Exposure.* Environ. Health Perspect. 106 (Suppl. 1). 217-229.

MOE. 1991. *Assessment of Human Health Risk of Reported Soil Levels of Metals and Radionuclides in Port Hope*, S. Fleming et al., pp. 117.

MOEE. 1994. *Scientific Criteria Document for the Development of Multimedia Environmental Standards: Lead.* Ontario Ministry of Environment and Energy. ISBN 0-7778-2529-5. pp. 332.

MOEE. 1995. *Health Risk Assessment of Mercury Contamination in the Vicinity of ICI Forest Products Cornwall, Ontario.* Ontario Ministry of Environment and Energy. May 1995. PIBS 3352.

MOEE. 1996. *Rationale for the Development and Application of Generic Soil, Groundwater and Sediment Criteria for Use at Contaminated Sites in Ontario.* Revised, December 1996, ISBN 0-7778-5906-8.

MOEE. 1997. *Guideline for Use at Contaminated Sites in Ontario.* Ontario Ministry of the Environment. Revised February 1997. ISBN 0-7778-6114-3.

MOE. 1998. *Assessment of Potential Health Risk of Reported Soil Levels of Nickel, Copper and Cobalt in Port Colborne and Vicinity, May 1997.* Ontario Ministry of the Environment. ISBN 0-7778-7884-4.

MOE. 1999. *Deloro Environmental Health Risk Study: Overall Technical Summary.* Ontario Ministry of the Environment.

MOE. 2001a. *Survey of Arsenic Exposure for Residents of Wawa.* Goss Gilroy Inc., Ottawa, Canada. Ontario Ministry of the Environment.

MOE. 2001b. *Phytotoxicology Survey Report: Lead in Soil on Residential Properties, Morris Island - West Carleton (2000).* Phytotoxicology Technical Memorandum. Report No. SDB-060-3511-2001. Ontario Ministry of the Environment. June, 2001.

MOE. 2001c. Fact Sheet. *Frequently Asked Questions about Lead Contamination.* Ontario Ministry of the Environment. March, 2001. [www.ene.gov.on.ca/cons/index.htm] Accessed Mar. 6, 2002.

MOE. 2001d. Fact Sheet. *Arsenic in the Environment.* Ontario Ministry of the Environment. March, 2001. [www.ene.gov.on.ca/cons/index.htm] Accessed March 6, 2002.

MOH (Ontario Ministry of Health). 1984. *Blood Lead Concentrations and Associated Risk Factors in Ontario Children.*

MOH/MOE. 1990. Ontario Ministry of Health (MOH) and Ontario Ministry of the Environment (MOE). *The Northern Ontario Blood Lead Study 1987-88.*

NRC (National Research Council, US). 1983a. *Risk Assessment in the Federal Government: Managing the Process.* National Academy Press, Washington, D.C.

NRC (National Research Council, US). 1993b. *Measuring lead exposure in infants, children, and other sensitive populations.* 1st ed. v. 1. National Academy Press, Washington, D.C.

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

O'Heany, J., R. Kusiak, C.E. Duncan, J.F. Smith, L.F. Smith, and L. Spielberg, 1988. *Blood lead levels and associated risk factors in Ontario children.* Sci. Total Environ. 71: 477-483.

Paustenbach, D.J. 2000. *The Practice of Exposure Assessment: A State-of-the-Art Review.* J. Toxicol. Environ. Health, Part B. 3:179-291.

Rasmussen, P.E., K.S. Subramanian and B.J. Jessiman. 2001. *A multi-element profile of housedust in relation to exterior dust and soils in the city of Ottawa, Canada.* Sci. Total Environ. 267: 125-140.

Rinne, R. 1986. *Soil lead levels in urban areas of Ontario.* Ministry of the Environment, Air Resources Branch.

RNPHD. 2001. Regional Niagara Public Health Department. *Lead Screening Report, Eastside Community, Port Colborne, April - June, 2001.* M.L. Decou, R. Williams and E. Ellis. August 2001.

ROCHD. 2000. Region of Ottawa-Carleton Health Department. *Results of West Carleton Blood Lead Screening.* May 2000.

Shinn, N.J., J. Bing-Canar, M. Cailas, N. Peneff, and H.J. Binns, 2000. *Determination of Spatial Continuity of Soil Lead Levels in an Urban Residential Neighbourhood.* Environ. Res. 82:46-52.

Shorten, C.V. and M.K. Hooven. 2000. *Methods of exposure assessment: Lead-contaminated dust in Philadelphia schools.* Environ. Health Perspect. 108(7): 663-666.

Stanek, E.J., E.J. Calabrese and M. Zorn. 2001. *Soil ingestion distributions for Monte Carlo risk assessment in children.* Human Ecol. Risk Assess. 7(2): 357-368.

Steele, M.J., B.D. Beck, B.L. Murphy, H.S. Strauss, 1990. *Assessing the contribution from lead in mining wastes to blood lead.* Regul. Toxicol. Pharmacol. 11:158-190.

Sutton, P.M., M. Athanasoulis, P. Flessel, G. Guirguis, M. Haan, R. Schlag, and L.R. Goldman, 1995. *Lead levels in the Household Environment of Children in Three High-Risk Communities in California.* Environ. Res. 68:45-57.

Stern, A. 1994. *Derivation of a Target Level of Lead in Soil at Residential Sites Corresponding to a De Minimis Contribution to Blood Lead Concentration.*

USCDCP. 1991. United States Centers for Disease Control and Prevention. *Preventing Lead Poisoning in Young Children.* A Statement by the Centers for Disease Control.

US EPA. 1989. *Risk Assessment Guidance for Superfund, Volume I, Human Health Evaluation Manual (Part A), Interim Final.* Office of Emergency and Remedial Response, US Environmental Protection Agency. EPA/540/1-89/002.

US EPA. 1992. U.S. Environmental Protection Agency. *Dermal Exposure Assessment: Principles and Applications.* EPA/600/8-91/001B.

US EPA. 1994. *Technical Support Document: Parameters and Equations used in Integrated Uptake Biokinetic Model for Lead in Children (v.099d).* United States Environmental Protection Agency. EPA/540/R-94/040.

US EPA. 1996a. *Distributions of Soil Lead in the Nation's Housing Stock.* Office of Pollution Prevention and Toxics. May, 1996.

US EPA. 1996b. *Urban Soil Lead Abatement Demonstration Project (USLADP): Volume I, EPA Integrated Report.* EPA/600/P-93/001aF. April, 1996.

US EPA. (1996c) *PCBs: cancer dose-response assessment and application to environmental mixtures.* National Center for Environmental Assessment, Washington, DC. EPA/600/P-96/001F.

US EPA. 1997. *Exposure Factors Handbook.* Office of Research and Development, National Center for Environmental Assessment, US Environmental Protection Agency. EPA/600/P-95/002Fa.

US EPA. 1998a. *Sources of Lead in Soil: A Literature Review.* Final Report. U.S. Environmental Protection Agency, Office of Pollution Prevention and Toxics. February, 1998. EPA 747-R-98-001a.

US EPA. 1998b. *Review of Studies Addressing Lead Abatement Effectiveness: Updated Edition.* US Environmental Protection Agency. EPA 747-B-98-001. December, 1998.

US EPA. 1998c. *Risk Analysis to Support Standards for Lead in Paint, Dust and Soil. Volume I.* Office of Pollution Prevention and Toxics. United States Environmental Protection Agency. 747-R-97-006.

US EPA. 2000. *Risk Assessment Guidance for Superfund Volume I: Human Health Evaluation Manual (Part E, Supplemental Guidance for Dermal Risk Assessment), Interim Guidance.* EPA/540/R-99/005. Office of Solid Waste and Emergency Response, Washington, DC. PB99-963312. As cited by US EPA Region IX.

US EPA. 2000b. *Bunker Hill Populated Areas Operable Unit. First Five Year Review Report.* U.S. Environmental Protection Agency. September, 2000.

US EPA. 2000c. *Supplementary Guidance for Conducting Health Risk Assessment of Chemical Mixtures.* Risk Assessment Forum, U.S. Environmental Protection Agency, Washington, DC 20460. EPA/630/R-00/002 August 2000.

US EPA. 2000d. *Chapter 9. Toxicity equivalence factors for dioxin and related compounds.* In: *Exposure and human health reassessment of 2,3,7,8-tetrachlorodibenzo-p-dioxin and related compounds.* External review draft. http://www.epa.gov/ncea/pdfs/dioxin/part2/drich9.pdf.

US EPA. 2001a. *Integrated Exposure Uptake Biokinetic Model for Lead in Children Windows version* (IEUBKwin v1.0 build 244). [www.epa.gov/oerrpage/superfund/programs/lead/ieubk.htm] (Accessed March 7, 2002).

US EPA. 2001b. *Lead; Identification of Dangerous Levels of Lead: Final Rule.* Federal Register. 66:1205-1240. 40 CFR Part 745.

US EPA. 2001c. *Technical Briefing on the Preliminary Organophosphate Pesticide.* Cumulative Risk Assessment Environmental Protection Agency (EPA). Notice. Federal Register: December 28, 2001. 66 (249):67246-67247.

US EPA IRIS. 1998a. *Antimony. Integrated Risk Information System (IRIS).* Environmental Criteria and Assessment Office, Office of Health and Environmental Assessment, Cincinnati, OH. Online at http://www.epa.gov/iris/index.html. (Accessed October 17, 2001).

US EPA IRIS. 1998b. *Beryllium and compounds. Integrated Risk Information System (IRIS).* Environmental Criteria and Assessment Office, Office of Health and Environmental Assessment, Cincinnati, OH. Online at http://www.epa.gov/iris/index.html. (Accessed October 17, 2001).

US EPA IRIS. 1998c. *Cadmium. Integrated Risk Information System (IRIS)*. Environmental Criteria and Assessment Office, Office of Health and Environmental Assessment, Cincinnati, OH. Online at http://www.epa.gov/iris/index.html. (Accessed October 17, 2001).

US EPA IRIS. 1998d. *Nickel refinery dust. Integrated Risk Information System (IRIS)*. Environmental Criteria and Assessment Office, Office of Health and Environmental Assessment, Cincinnati, OH. Online at http://www.epa.gov/iris/index.html. (Accessed Oct. 17, 2001).

US EPA IRIS. 1998e. *Nickel, soluble salts. Integrated Risk Information System (IRIS)*. Environmental Criteria and Assessment Office, Office of Health and Environmental Assessment, Cincinnati, OH. Online at http://www.epa.gov/iris/index.html. (Accessed October 17, 2001).

US HUD. 2001. *National Survey of Lead and Allergens in Housing; Final Report; Volume I: Analysis of Lead Hazards*, Revision 6.0. Office of Lead Hazard Control, U.S. Department of Housing and Urban Development. April, 2001.

Wang, S.T., S. Pizzolato, H.P. Demshar, and L.F. Smith, 1997. *Decline in Blood Lead in Ontario Children Correlated to Decreasing Consumption of Leaded Gasoline, 1983-1992*. Letter to the Editor. Clinical Chemistry 43:1251-1252.

Weis, M. and G.F. Barclay, 1985. *Distribution of heavy metals and organic contaminants in plants and soils of Windsor and Essex County, Ontario*. J. Great Lakes Res. 11(3):339-346.

World Health Organization (WHO). 1996. *Antimony. Guidelines for drinking-water quality, 2nd ed. Vol. 2 Health criteria and other supporting information*. WHO, Geneva. pp. 147-156.

World Health Organization (WHO). 1998a. *Cadmium. Guidelines for drinking-water quality, 2nd ed. Addendum to Vol. 2 Health criteria and other supporting information*. WHO, Geneva. pp. 195-201.

World Health Organization (WHO). 1998b. *Copper. Guidelines for drinking-water quality, 2nd ed. Addendum to Vol. 2 Health criteria and other supporting information*. WHO, Geneva. pp. 31-46.

World Health Organization (WHO) Regional Office for Europe, Copenhagen. 2000. *Air Quality Guidelines for Europe*, Second Edition. ISBN 92 890 1358 3.

Human Health Risk Assessment

Appendix 1

Environmental Monitoring of Metals
in the Rodney Street Community and Port Colborne

List of Tables
Human Health Risk Assessment - Appendix 1
Environmental Monitoring of Metals in the Rodney Street Community and Port Colborne

Drinking Water Monitoring Data

Table A1-1: Summary of Port Colborne Municipal Drinking Water Data, 1996 -1999
(Number of Samples = 8)

Element	Range of Drinking Water Concentrations (µg/L)	Average Drinking Water Concentrations (µg/L)	MOE Drinking Water Standard (µg/L)	World Health Organization Drinking Water Guidelines (µg/L)	EPA Maximum Contaminant Levels (µg/L)
Treated Drinking Water					
Antimony	0.31 - 0.96	0.60	none	5 (provisional)	6
Arsenic	0.2 - 0.6	0.34	25	10 (provisional)	5
Beryllium	0.11 - 0.2	0.16	none	none	4
Cadmium	0.0041 - 0.051	0.02	5	3	5
Cobalt	0.025 - 0.054	0.03	none	none	none
Copper	0.33 - 1	0.65	none	2,000 (provisional)	1,300 (aesthetics)
Lead	0.05 - 1.9	0.37	10	10	15
Nickel	0.4 - 1.1	0.77	none	20 (provisional)	none
Distributed Water at Charlotte Street					
Antimony	0.45 - 0.97	0.64	none	5 (provisional)	6
Arsenic	0.14 - 0.40	0.28	25	10 (provisional)	5
Beryllium	0.03 - 0.2	0.11	none	none	4
Cadmium	0.022 - 0.083	0.06	5	3	5
Cobalt	0.026 - 0.04	0.03	none	none	none
Copper	5 - 44.1	14.50	none	2,000 (provisional)	1,300 (aesthetics)
Lead	0.067 - 0.71	0.38	10	10	15
Nickel	0.6 - 1.3	0.94	none	20 (provisional)	none

* EPA Region III Risk Based Concentrations are not standards or guidelines and were used for comparison purpose only when the MOE or EPA did not have a standard/guideline for a particular substance.

Air Monitoring Data

Table A1-2a: MOE TSP (Total Suspended Particulate) Nickel Annual Air Monitoring Data (1992-1995) from Monitoring Station 27047 at Davis and Fraser and Summer 2001 Monitoring at Rodney Street

Year (# of Samples)	Percentiles						Maximum $\mu g/m^3$	Mean $\mu g/m^3$	Geom. Mean $\mu g/m^3$
	10%	30%	50%	70%	90%	99%			
1992 (54)	0.005	0.005	0.020	0.038	0.130	0.496	0.690	0.053	0.020
1993 (49)	0.002	0.006	0.010	0.020	0.090	0.302	0.390	0.034	0.013
1994 (48)	0.004	0.007	0.011	0.021	0.067	0.141	0.160	0.025	0.014
1995 (55)	0.003	0.008	0.011	0.019	0.049	0.135	0.140	0.023	0.011
2001 (22)	INS	INS	INS	INS	INS	INS	0.12	0.013	INS

INS = insufficient data

Table A1-2b: Comparison of PM_{10} (Particulate Matter $\leq 10 \ \mu m$) Nickel-In-Air Concentrations in Port Colborne With Other Areas in Southern Ontario Based on the Most Recently Available Results

Location	Start	End	Number of Samples	Number Below Detection Limit	Mean $\mu g/m^3$	Maximum $\mu g/m^3$	Minimum $\mu g/m^3$
Ottawa	03-Jan-95	25-Dec-98	217	104	0.0030	0.0351	0.0008
Point Petre	25-Aug-96	31-Dec-98	117	64	0.0018	0.0068	0.0009
Windsor	03-Jan-95	31-Dec-98	181	38	0.0043	0.0226	0.0010
Toronto	03-Jan-95	06-Sep-95	37	10	0.0036	0.0088	0.0010
Toronto	21-Feb-97	31-Dec-98	93	25	0.0030	0.0084	0.0009
Toronto	02-Jun-95	31-Dec-98	179	27	0.0041	0.0109	0.0010
Hamilton	03-Jan-95	25-Dec-98	193	45	0.0034	0.0133	0.0007
Walpole Island	03-Jan-95	04-Mar-95	8	4	0.0018	0.0036	0.0010
Egbert	10-Mar-95	31-Dec-98	179	93	0.0023	0.0109	0.0009
Port Colborne	Summer 2001		21	9	0.0059	0.0260	0.0010*

*The minimum value for nickel-in-air represents ½ detection limit and was used in the calculation of the average value.

Table A1-3: Summary of Annual Air Monitoring Data for Port Colborne

Metal	Minimum Air Concentration ($\mu g/m^3$)	Maximum Air Concentration ($\mu g/m^3$)	Average Air Concentration ($\mu g/m^3$)	MOE Air Standard (24 hour) ($\mu g/m^3$)
Ministry of the Environment - Davis and Fraser, Port Colborne Air Monitoring Data - 1992 - 1995				
Copper (TSP)	0.058	0.56	0.112	50
Lead (TSP)	0.01	0.06	0.02	2
Total Suspended Particulate	9	222	51.7	120
Ministry of the Environment - Rodney Street, Port Colborne Air Monitoring Data - Summer 2001				
Antimony (TSP)	0.0004	0.0026	0.0011	25
Antimony (PM_{10})	0.0001	0.0025	0.0007	25
Arsenic (TSP)	0.0006	0.0116	0.0043	0.30
Arsenic (P Beryllium (TSP) M_{10})	0.0006	0.0120	0.0040	0.30
	0.00001	0.0004	0.0002	0.01
Beryllium (PM_{10})	0.00001	0.0006	0.0001	0.01
Cadmium (TSP)	0.05	0.3	0.086	2
Cadmium (PM_{10})	0.00003	0.0005	0.0002	2
Copper (TSP)	.0005	0.0800	0.0193	50
Copper (PM_{10})	.0003	0.4700	0.1124	50
Lead (TSP)	.005	0.0500	0.014	2
Lead (PM_{10})	.005	0.0700	0.014	2
Total Suspended Particulate	20	100	46.1	100
PM_{10}	8	66	21.9	50
Jacques Whitford Environmental - Air Monitoring Data for Port Colborne Schools - Summer 2000				
Arsenic (TSP)	0.001	0.005	0.002	0.3
Cobalt (TSP)	0.004	0.01	0.0075	0.1
Copper (TSP)	0.01	0.08	0.035	50
Nickel (TSP)	0.01	0.11	0.05	2
Total Suspended Particulate	24	63	48.7	120
PM_{10}	21	44	34	50
Environment Canada Air Monitoring Program - Typical Ontario Air Concentrations - 1995 - 1999				
Antimony (PM_{10})	0.0001	0.0115	0.0011	25
Arsenic (PM_{10})	0.0003	0.016	0.0016	0.3
Beryllium (PM_{10})	No data available			0.01
Cadmium (PM_{10})	0.0001	0.0067	0.0007	2
Cobalt (PM_{10})	0.001	0.017	0.002	0.1
Copper (PM_{10})	0.001	0.101	0.018	50
Lead (PM_{10})	0.0005	0.13	0.0077	2
Nickel (PM_{10})	0.0007	0.035	0.003	2

Soil Monitoring Data

Table A1-4: Summary of Soil Data (All Depths Combined)

Element	Concentration (µg/g)											
	Minimum	Maximum	Mean	10th Percentile	20th Percentile	30th Percentile	40th Percentile	50th Percentile (Median)	60th Percentile	70th Percentile	80th Percentile	90th Percentile
Aluminum	2200	47300	17346	8700	11300	13500	15300	17300	19030	20600	22520	26300
Antimony	0.1	91.1	2.2	0.4	0.5	0.7	0.9	1.2	1.5	1.9	2.9	5.5
Arsenic	0	350	16	6	8	9	11	13	15	18	21	29
Barium	19	956	169	88	101	122	139	154	169	188	211	266
Beryllium	0.2	4.6	1.0	0.6	0.7	0.8	0.9	1.0	1.1	1.2	1.3	1.4
Cadmium	0.0	35.3	1.2	0.4	0.6	0.8	0.9	1.1	1.2	1.5	1.7	2.2
Calcium	1920	93400	20543	10620	13300	15000	17000	19000	21200	23900	26300	32000
Chromium	7	245	30	19	22	25	27	29	31	33	36	40
Cobalt	5	262	51	20	24	30	34	39	47	55	70	95
Copper	4	2720	246	70	106	134	161	196	230	284	360	471
Iron	8820	140000	29706	18030	20900	23020	25200	27000	29200	31900	35500	42600
Lead	6	1800	217	74	96	120	148	173	202	244	299	406
Magnesium	990	31900	7903	4500	5600	6220	6726	7320	8000	8800	9950	11900
Manganese	131	5620	506	340	389	425	453	478	500	540	588	671
Molybdenum	0.2	12.1	3.7	0.9	1.7	2.8	3.6	3.9	4.2	4.4	4.8	5.8
Nickel	19	17000	2508	518	800	1070	1400	1730	2200	2810	3784	5592
Selenium	0.1	19.4	2.4	0.4	0.7	1.0	1.3	1.8	2.3	2.9	3.9	5.2
Strontium	14	690	78	40	49	56	62	68	76	87	99	121
Vanadium	10	75	37	25	28	31	34	37	39	42	45	50
Zinc	23	1750	365	140	187	223	260	305	356	418	520	665

Data are µg/g dry weight.

N.B. Does not include the 2000 trench samples.

Backyard Garden Produce Monitoring Data

Table A1-5: Levels of Antimony in Produce from Rodney Street Residences

Location	Antimony Soil Concentration (µg/g)	Produce Type	Dry Weight Antimony Concentration in Produce (µg/g)	Conversion Factor* (Dry to Fresh Weight)	Fresh Weight Antimony Concentration in Produce[1] (µg/g)
JWEL 2000 Samples					
3	<0.5	Beet Root	0.06	0.13	0.0078
		Celery	<0.05	0.059	-
		Tomato	<0.1	0.065	-
9	Not analyzed	Tomato	<0.1	0.065	-
25	1.1	Pepper	<0.1	0.066	-
		Lettuce	0.46	0.045	0.021
		Beet Root	<0.1	0.13	-
33	Not analyzed	Lettuce	<0.1	0.045	-
		Pepper	<0.05	0.066	-
34	Not analyzed	Radish	<0.05	0.0516	-
		Pepper	0.27	0.066	0.018
41	Not analyzed	Tomato	<0.1	0.065	-
Control Samples					
Food Store Control	n/a	Beet	<0.05	0.13	-
		Pepper	<0.1	0.066	-
		Lettuce	<0.15	0.045	-
Wainfleet Bog (Background Control)	n/a	Beet Root	<0.05	0.13	-
		Pepper	<0.1	0.066	0.0066
		Beet Top	<0.1	0.091	-

[1] Fresh weight concentrations were not calculated when dry weight values were below the detection limit.

[2] n/a - not available.

* refer to Table A3-6 for dry weight to wet weight conversion factors.

Table A1-6: Levels of Beryllium in Produce from Rodney Street Residences

Location	Beryllium Soil Concentration (µg/g)	Produce Type	Dry Weight Beryllium Concentration in Produce (µg/g)	Conversion Factor* (Dry Weight to Fresh Weight)	Fresh Weight Beryllium Concentration in Produce (µg/g)
JWEL 2000 Samples					
3	0.7	Beet Root	<0.1	0.13	0.0065
		Celery	<0.1	0.059	0.00295
		Tomato	<0.1	0.065	0.00325
9	Not Analyzed	Tomato	<0.2	0.065	0.0065
25	0.5	Pepper	<0.2	0.066	0.0066
		Lettuce	<0.2	0.045	0.0045
		Beet Root	<0.1	0.13	0.0065
33	Not Analyzed	Lettuce	<0.2	0.045	0.0045
		Pepper	<0.1	0.066	0.0033
34	Not Analyzed	Radish	<0.1	0.0516	0.00258
		Pepper	<0.3	0.066	0.0099
41	Not Analyzed	Tomato	<0.2	0.065	0.0065
MOE Samples					
Sample #1	0.75	Tomato	0.1	0.065	0.0065
		Green Pepper	0.1	0.066	0.0066
Sample #2	0.425	Pepper	0.1	0.066	0.0066
		Tomato	0.1	0.065	0.0065
Control Samples					
Food Store Control	n/a	Beet	<0.1	0.13	0.0065
		Pepper	<0.2	0.066	0.0066
		Lettuce	<0.3	0.045	0.00675
Wainfleet Bog (Background Control)	0.5	Beet Root	<0.1	0.13	0.0065
		Pepper	<0.2	0.066	0.0066
		Beet Top	<0.2	0.091	0.0091

One half the detection limit used for values reported as <detection limit.
n/a - not available.

* refer to Table A3-6 for dry weight to wet weight conversion factors.

Table A1-7: Levels of Cadmium in Produce from Rodney Street Residences

Location	Cadmium Soil Concentration (µg/g)	Produce Type	Dry Weight Cadmium Concentration in Produce (µg/g)	Conversion Factor* (Dry Weight to Fresh Weight)	Fresh Weight Cadmium Concentration in Produce (µg/g)
JWEL 2000 Samples					
3	<0.5	Beet Root	0.38	0.13	0.049
		Celery	0.85	0.059	0.05
		Tomato	0.23	0.065	0.015
9	Not Analyzed	Tomato	0.13	0.065	0.0085
25	1.1	Pepper	0.15	0.066	0.0099
		Lettuce	0.34	0.045	0.015
		Beet Root	0.24	0.13	0.031
33	Not Analyzed	Lettuce	0.45	0.045	0.02
		Pepper	0.22	0.066	0.015
34	Not Analyzed	Radish	0.16	0.0516	0.0083
		Pepper	0.28	0.066	0.018
41	Not Analyzed	Tomato	0.13	0.065	0.0085
MOE Samples					
Sample #1	0.8	Tomato	0.2	0.065	0.013
		Green Pepper	0.05	0.066	0.0033
Sample #2	0.1	Pepper	0.2	0.066	0.013
		Tomato	0.2	0.065	0.013
Control Samples					
Food Store Control	n/a	Beet	0.21	0.13	0.027
		Pepper	0.17	0.066	0.011
		Lettuce	1.06	0.045	0.048
Wainfleet Bog (Background Control)	0.3 - 0.4	Beet Root	0.31	0.13	0.04
		Pepper	0.89	0.066	0.059
		Beet Top	0.69	0.091	0.063

n/a - not available.

* refer to Table A3-6 for dry weight to wet weight conversion factors.

Table A1-8: Levels of Cobalt in Produce from Rodney Street Residences

Location	Cobalt Soil Concentration ($\mu g/g$)	Produce Type	Dry Weight Cobalt Concentration in Produce ($\mu g/g$)	Conversion Factor* (Dry Weight to Fresh Weight)	Fresh Weight Cobalt Concentration in Produce[1] ($\mu g/g$)
JWEL 2000 Samples					
3	20.1	Beet Root	0.37	0.13	0.048
		Celery	0.14	0.059	0.0083
		Tomato	0.09	0.065	0.0059
9	Not Analyzed	Tomato	0.06	0.065	0.0039
25	28.6	Pepper	0.05	0.066	0.0033
		Lettuce	0.1	0.045	0.0045
		Beet Root	0.11	0.13	0.014
33	Not Analyzed	Lettuce	0.33	0.045	0.015
		Pepper	0.04	0.066	0.0026
34	Not Analyzed	Radish	0.13	0.0516	0.0067
		Pepper	<0.03	0.066	-
41	Not Analyzed	Tomato	0.04	0.065	0.0026
MOE Samples					
Sample #1	44.5	Tomato	0.1	0.065	0.0065
		Green Pepper	0.1	0.066	0.0066
Sample #2	58	Pepper	0.1	0.066	0.0066
		Tomato	0.1	0.065	0.0065
Control Samples					
Food Store	n/a[2]	Beet	0.02	0.13	0.0026
		Pepper	0.05	0.066	0.0033
		Lettuce	0.28	0.045	0.013
Wainfleet Bog (Background Control)	4.5	Beet Root	0.11	0.13	0.014
		Pepper	0.56	0.066	0.037
		Beet Top	0.22	0.091	0.02

[1] Fresh weight concentrations were not calculated when dry weight values were below the detection limit.

[2] n/a - not available.

* refer to Table A3-6 for dry weight to wet weight conversion factors.

Table A1-9: Levels of Arsenic in Produce from Rodney Street Residences

Location	Arsenic Soil Concentration (μg/g)	Produce Type	Dry Weight Arsenic Concentration in Produce (μg/g)	Conversion Factor* (Dry Weight to Fresh Weight)	Fresh Weight Arsenic Concentration in Produce[1] (μg/g)
JWEL 2000 Samples					
3	<0.2	Beet Root	<0.2	0.13	-
		Celery	<0.2	0.059	-
		Tomato	<0.4	0.065	-
9	Not Analyzed	Tomato	<0.4	0.065	-
25	18.6	Pepper	<0.4	0.066	-
		Lettuce	<0.4	0.045	-
		Beet Root	<0.4	0.13	-
33	Not Analyzed	Lettuce	<0.4	0.045	-
		Pepper	<0.2	0.066	-
34	Not Analyzed	Radish	0.2	0.0516	0.01
		Pepper	<0.6	0.066	-
41	Not Analyzed	Tomato	<0.4	0.065	-
MOE Samples					
Sample #1	13	Tomato	0.1	0.065	0.0065
		Green Pepper	0.1	0.066	0.0066
Sample #2	17.5	Pepper	0.1	0.066	0.0066
		Tomato	0.1	0.065	0.0065
Control Samples					
Food Store (Control)	n/a[2]	Beet	<0.2	0.13	-
		Pepper	<0.4	0.066	-
		Lettuce	<0.6	0.045	-
Wainfleet Bog (Background Control)	1.3 - 1.4	Beet Root	<0.2	0.13	-
		Pepper	<0.4	0.066	-
		Beet Top	<0.4	0.091	-

[1] Fresh weight concentrations were not calculated when dry weight values were below the detection limit.

[2] n/a - not available.

* refer to Table A3-6 for dry weight to wet weight conversion factors.

Table A1-10: Levels of Copper in Produce from Rodney Street Residences

Location	Copper Soil Concentration (µg/g)	Produce Type	Dry Weight Copper Concentration in Produce (µg/g)	Conversion Factor* (Dry Weight to Fresh Weight)	Fresh Weight Copper Concentration in Produce (µg/g)
JWEL 2000 Samples					
3	134	Beet Root	14.8	0.13	1.92
		Celery	5.14	0.059	0.3
		Tomato	10.1	0.065	0.66
9	Not Analyzed	Tomato	11.3	0.065	0.73
25	194	Pepper	10.4	0.066	0.69
		Lettuce	11.8	0.045	0.53
		Beet Root	9.71	0.13	1.26
33	Not Analyzed	Lettuce	8.81	0.045	0.4
		Pepper	7.09	0.066	0.47
34	Not Analyzed	Radish	5.2	0.0516	0.27
		Pepper	1.6	0.066	0.106
41	Not Analyzed	Tomato	7.93	0.065	0.52
MOE Samples					
Sample #1	220	Tomato	4.9	0.065	0.32
		Green Pepper	5.9	0.066	0.39
Sample #2	325	Pepper	9.4	0.066	0.62
		Tomato	4.6	0.065	0.3
Control Samples					
Food Store (Control)	n/a	Beet	7.78	0.13	1.01
		Pepper	18.7	0.066	1.23
		Lettuce	6.54	0.045	0.29
Wainfleet Bog (Background Control)	14.9 - 22.2	Beet Root	7.93	0.13	1.03
		Pepper	14.9	0.066	0.98
		Beet Top	7.21	0.091	0.66

n/a - not available.

* refer to Table A3-6 for dry weight to wet weight conversion factors.

Table A1-11: Levels of Lead in Produce from Rodney Street Residences

Location	Lead Soil Concentration (μg/g)	Produce Type	Dry Weight Lead Concentration in Produce (μg/g)	Conversion Factor* (Dry Weight to Fresh Weight)	Fresh Weight Lead Concentration in Produce (μg/g)
JWEL 2000 Samples					
3	379	Beet Root	6.26	0.13	0.81
		Celery	4.16	0.059	0.25
		Tomato	0.1	0.065	0.0065
9	Not Analyzed	Tomato	0.12	0.065	0.0078
25	371	Pepper	0.15	0.066	0.0099
		Lettuce	0.93	0.045	0.042
		Beet Root	8.11	0.13	1.05
33	Not Analyzed	Lettuce	2.43	0.045	0.11
		Pepper	2.55	0.066	0.17
34	Not Analyzed	Radish	2.55	0.0516	0.13
		Pepper	0.58	0.066	0.038
41	Not Analyzed	Tomato	<0.1	0.065	-
MOE Samples					
Sample #1	91.5	Tomato	0.25	0.065	0.016
		Green Pepper	0.25	0.066	0.017
Sample #2	88	Pepper	0.6	0.066	0.04
		Tomato	1.9	0.065	0.12
Control Samples					
Food Store Control	n/a[2]	Beet	0.17	0.13	0.022
		Pepper	0.13	0.066	0.0086
		Lettuce	0.23	0.045	0.01
Wainfleet Bog (Background Control)	10.9 - 11	Beet Root	0.1	0.13	0.013
		Pepper	0.15	0.066	0.0099
		Beet Top	0.35	0.091	0.032

[1] Fresh weight concentrations were not calculated when dry weight values were below the detection limit.

[2] n/a - not available.

* refer to Table A3-6 for dry weight to wet weight conversion factors.

Table A1-12a: Levels of Nickel in Produce from Rodney Street Residences
(JWEL & MOE 2000 Samples)

Location	Nickel Soil Concentration (µg/g)	Produce Type	Dry Weight Nickel Concentration in Produce (µg/g)	Conversion Factor* (Dry Weight to Fresh Weight)	Fresh Weight Nickel Concentration in Produce (µg/g)
JWEL 2000 Samples					
3	764	Beet	14	0.13	1.82
		Celery	4.2	0.059	0.248
		Tomato	3.2	0.065	0.208
6	99.5	Onion	0.3	0.11	0.033
		Pear	0.6	0.17	0.102
		Pepper	2.7	0.066	0.178
8	188	Apple	0.05**	0.15	0.0075
		Lettuce	5.7	0.045	0.257
		Pepper	6	0.066	0.396
17	157	Beet	1	0.13	0.13
		Pepper	1.3	0.066	0.0858
19	380	Apple	0.2	0.15	0.03
		Beet	1.1	0.13	0.143
		Cantaloupe	3.3	0.1	0.33
		Peach	2.8	0.11	0.308
		Pear	3.2	0.17	0.544
		Pepper	4.1	0.066	0.271
		Plum	1.6	0.21	0.336
		Tomato	2.5	0.065	0.163
		Watermelon	7.1	0.1	0.71
20	333	Beet	1.9	0.13	0.247
		Pepper	6.1	0.066	0.403
23	472	Grapes	0.2	0.19	0.038
		Pepper	8.3	0.066	0.548
24	296	Apple	0.2	0.15	0.03
		Lettuce	4.2	0.045	0.189
		Pepper	4.9	0.066	0.323
25	1570	Pepper	7.9	0.066	0.521
		Lettuce	7	0.045	0.315
		Beet	10.5	0.13	1.365
26	516	Rhubarb	1.2	0.052	0.0624
		Squash	1.5	0.057	0.0855

Location	Nickel Soil Concentration (µg/g)	Produce Type	Dry Weight Nickel Concentration in Produce (µg/g)	Conversion Factor* (Dry Weight to Fresh Weight)	Fresh Weight Nickel Concentration in Produce (µg/g)
		Tomato	1.2	0.065	0.078
37	293	Celery	1.5	0.059	0.0885
		Pepper	10.4	0.066	0.686
42	302	Leeks	1.9	0.124	0.236
		Pepper	12.2	0.066	0.805
43	135	Carrot	0.7	0.12	0.084
		Pepper	2.9	0.066	0.191
MOE Samples					
MOE Sample	2750	Pepper	14	0.066	0.924
		Tomato	5.3	0.065	0.345
Control Samples					
Food Store Control	n/a	Beet	0.1	0.13	0.013
		Pepper	0.2	0.066	0.013
		Lettuce	0.6	0.045	0.027
Wainfleet Bog (Background Control)	15.4 - 15.8	Beet Root	0.3	0.13	0.039
		Pepper	4.8	0.066	0.317
		Beet Top	0.6	0.091	0.055

* refer to Table A3-6 for dry weight to wet weight conversion factors
** This value is one half the detection limit.

Table A1-12b: Levels of Nickel in Produce from Port Colborne Residences
(Data Provided by JWEL at Time of Report Finalization)

Date Collected	JWEL Sample #	Produce Sampled	Dry Weight Nickel Concentration (µg/g)	Dry to Wet Conversion Factor*	Wet Weight Concentration (µg/g)
Sept. 5	FBS5S32B	apples	0.2	0.15	0.03
Sept. 7	FBS7S62B	apples	0.4	0.15	0.06
Sept. 5	FBS5S25B	apples	0.5	0.15	0.075
Sept. 6	FBS6S48A	apples	6.7	0.15	1.005
Sept. 7	FBS7S63A	apples	0.3	0.15	0.045
Sept. 7	FBS7S64E	apples	0.5	0.15	0.075
Sept. 7	FBS7S62G	basil	32.7	0.045	1.472
Sept. 5	FBS5S15A	beans	11.3	0.099	1.119
Sept. 7	FBS7S53D	beans	9	0.099	0.513
Sept. 7	FBS7S61B	beans	5.8	0.099	0.891
Sept. 5	FBS5S16A	beets	4.2	0.13	0.546
Sept. 5	FBS5S26A	beets	2.1	0.13	0.273
Sept. 5	FBS5S28B	beets	0.7	0.13	0.091
Sept. 6	FBS6S42B	beets	14.6	0.13	1.898
Sept. 7	FBS6S51A	beets	2.1	0.13	2.73
Sept. 7	FBS7S61C	beets	38.4	0.13	4.992
Sept. 7	FBS7S64D	beets	9.4	0.13	1.222
Sept. 11	FBS11S68A	beets	5.9	0.13	0.767
Sept. 11	FBS11S70A	beets	1.8	0.13	0.234
Sept. 11	FBS12S72A	beets	3.1	0.13	0.403
Sept. 7	FBS7S62I	broccoli	30.9	0.093	2.874
Sept. 6	FBS6S36B	cabbage	9.6	0.0468	0.449
Sept. 5	FBS5S22C	carrots	3.2	0.12	0.384
Sept. 5	FBS5S28A	carrots	0.6	0.12	0.072
Sept. 5	FBS5S30C	carrots	0.7	0.12	0.084
Sept. 6	FBS6S34A	carrots	1.2	0.12	0.144
Sept. 6	FBS6S40A	carrots	0.4	0.12	0.048
Sept. 6	FBS6S42A	carrots	9.2	0.12	1.104
Sept. 6	FBS6S49D	carrots	2.6	0.12	0.312
Sept. 7	FBS6S51B	carrots	3.4	0.12	0.408
Sept. 7	FBS7S59B	carrots	19.6	0.12	2.352
Sept. 7	FBS7S63C	carrots	3.6	0.12	0.432
Sept. 7	FBS7S64B	carrots	9.4	0.12	1.128
Sept. 11	FBS11S69A	carrots	6.6	0.12	0.792
Sept. 11	FBS11S70B	carrots	2.9	0.12	0.348
Sept. 11	FBS11S71A	carrots	3.3	0.12	0.396
Sept. 12	FBS12S74A	carrots	22.2	0.12	2.664
Sept. 5	FBS5S21B	celery	0.8	0.059	0.0472
Sept. 5	FBS5S33A	celery	19.4	0.059	1.145
Sept. 6	FBS6S36C	celery	0.5	0.059	0.0295
Sept. 6	FBS6S49B	celery	30.9	0.059	1.823
Sept. 5	FBS5S17B	cucumber	4.3	0.05	0.215
Sept. 5	FBS5S26C	cucumber	2.7	0.05	0.135

Date Collected	JWEL Sample #	Produce Sampled	Dry Weight Nickel Concentration (µg/g)	Dry to Wet Conversion Factor*	Wet Weight Concentration (µg/g)
Sept. 7	FBS7S52C	cucumber	2.7	0.05	0.135
Sept. 7	FBS7S53B	cucumber	7.6	0.05	0.38
Sept. 7	FBS7S55C	cucumber	14.7	0.05	0.735
Sept. 7	FBS7SS60B	cucumber	4	0.05	0.2
Sept. 5	FBS5S19B	eggplant	3.1	0.093	0.288
Sept. 6	FBS6S49C	eggplant	1.3	0.093	0.121
Sept. 7	FBS7S62F	fennel	7.8	0.0121	0.0944
Sept. 7	FBS7S62A	grapes	0.8	0.19	0.152
Sept. 12	FBS12S73A	horseradish	9.2	0.0516	0.475
Sept. 11	FBS11S66C	Jerusalem potato	13.5	0.133	1.796
Sept. 5	FBS5S22A	leeks	3.5	0.124	0.434
Sept. 5	FBS5S29B	leeks	2.4	0.124	0.298
Sept. 7	FBS7S62H	leeks	6.4	0.124	0.794
Sept. 7	FBS7S64C	leeks	11.6	0.124	1.438
Sept. 5	FBS5S23B	leeks	1.9	0.124	0.236
Sept. 5	FBS5S19A	lettuce	8.1	0.045	0.365
Sept. 5	FBS5S26B	lettuce	2.8	0.045	0.126
Sept. 5	FBS5S33C	lettuce	15.9	0.045	0.716
Sept. 6	FBS6S36A	lettuce	8	0.045	0.36
Sept. 6	FBS6S49A	lettuce	1.9	0.045	0.0855
Sept. 11	FBS11S65B	muskmelon	2	0.1	0.2
Sept. 5	FBS5S20C	onion	0.6	0.11	0.066
Sept. 5	FBS5S21A	onion	4.5	0.11	0.495
Sept. 5	FBS5S30B	onion	4.8	0.11	0.528
Sept. 6	FBS6S47A	onion	1.6	0.11	0.176
Sept. 7	FBS7S55D	onion	2.6	0.11	0.286
Sept. 7	FBS7SS60C	onion	4.6	0.11	0.506
Sept. 7	FBS7S61D	onion	15.6	0.11	1.716
Sept. 7	FBS7S62E	onion	9.5	0.11	1.045
Sept. 11	FBS11S67A	onion	1.4	0.11	0.154
Sept. 6	JFBS6S43B	parsnip	7.2	0.21	1.512
Sept. 7	FBS7S64A	parsnip	14.1	0.21	2.961
Sept. 5	FBS5S19C	peach	4.8	0.11	0.528
Sept. 5	FBS5S32A	peach	1	0.11	0.11
Sept. 5	FBS5S33D	peach	2.7	0.11	0.297
Sept. 7	FBS7S62C	peach	9.7	0.11	1.067
Sept. 5	FBS5S19D	pears	2.6	0.17	0.442
Sept. 5	FBS5S31A	pears	0.7	0.17	0.119
Sept. 6	FBS5S31B	pears	1	0.17	0.17
Sept. 5	FBS5S25A	pears	1.7	0.17	0.289
Sept. 5	FBS5S23C	pears	1.1	0.17	0.187
Sept. 6	FBS6S35A	pears	0.8	0.17	0.136
Sept. 6	FBS6S36E	pears	5.4	0.17	0.918
Sept. 6	FBS6S45B	pears	0.3	0.17	0.051
Sept. 7	FBS7S62D	pears	2.1	0.17	0.357

Date Collected	JWEL Sample #	Produce Sampled	Dry Weight Nickel Concentration (µg/g)	Dry to Wet Conversion Factor*	Wet Weight Concentration (µg/g)
Sept. 7	FBS7S64F	pears	1.6	0.17	0.272
Sept. 6	FBS6S48B	pears	0.3	0.17	0.051
Sept. 6	FBS6S49E	pears	1.9	0.17	0.323
Sept. 6	FBS7S63B	pears	4.7	0.17	0.799
Sept. 5	FBS5S17C	peppers	11.1	0.066	0.733
Sept. 5	FBS5S22D	peppers	15.3	0.066	1.01
Sept. 5	FBS5S33B	peppers	8.6	0.066	0.568
Sept. 6	FBS6S34B	peppers	3.5	0.066	0.231
Sept. 6	FBS6S42D	peppers	16.5	0.066	1.089
Sept. 6	FBS6S44B	peppers	5.5	0.066	0.363
Sept. 6	FBS6S50A	peppers	41.5	0.066	0.957
Sept. 7	FBS7S52A	peppers	19.9	0.066	1.313
Sept. 7	FBS7S55B	peppers	26.9	0.066	1.775
Sept. 7	FBS7S59C	peppers	40.2	0.066	2.653
Sept. 11	FBS11S67B	peppers	11.9	0.066	0.785
Sept. 5	FBS5S28E	plum	0.3	0.21	0.063
Sept. 7	FBS6S40B	plum	0.3	0.21	0.063
Sept. 6	FBS6S43C	plum	0.4	0.21	0.084
Sept. 5	FBS5S21C	potato	2.9	0.133	0.386
Sept. 5	FBS5S28D	potato	0.5	0.133	0.0665
Sept. 5	FBS5S29A	potato	1	0.133	0.133
Sept. 5	FBS5S23A	potato	0.5	0.133	0.067
Sept. 6	FBS6S43A	potato	0.7	0.133	0.093
Sept. 11	FBS11S66B	potato	1.8	0.133	0.239
Sept. 7	FBS7S62J	radicchio	14.1	0.045	0.635
Sept. 7	FBS7S59A	radish	10.8	0.0516	0.557
Sept. 7	FBS7S51C	rhubarb	2.4	0.052	0.125
Sept. 7	FBS7S53C	rhubarb	2.1	0.052	0.109
Sept. 5	FBS5S17A	squash	13.6	0.057	0.775
Sept. 5	FBS5S30A	squash	2.3	0.057	0.131
Sept. 11	FBS11S65A	squash	1.4	0.057	0.0798
Sept. 11	FBS11S66A	squash	5.2	0.057	0.296
Sept. 5	FBS5S15C	Swiss chard	35.9	0.089	3.195
Sept. 5	FBS5S28C	swiss chard	0.8	0.089	0.0712
Sept. 5	FBS5S15B	tomato	6.1	0.065	0.397
Sept. 5	FBS5S16B	tomato	0.9	0.065	0.0585
Sept. 5	FBS5S18A	tomato	6	0.065	0.39
Sept. 5	FBS5S20B	tomato	0.5	0.065	0.0325
Sept. 5	FBS5S22B	tomato	2.6	0.065	0.169
Sept. 5	FBS5S24A	tomato	0.8	0.065	0.052
Sept. 5	FBS5S27A	tomato	0.5	0.065	0.0325
Sept. 6	FBS6S38A	tomato	0.6	0.065	0.039
Sept. 6	FBS6S39A	tomato	1.3	0.065	0.0845
Sept. 6	FBS6S42C	tomato	5.1	0.065	0.332
Sept. 6	FBS6S44A	tomato	6.9	0.065	0.449
Sept. 6	FBS6S45A	tomato	1	0.065	0.065

Date Collected	JWEL Sample #	Produce Sampled	Dry Weight Nickel Concentration (µg/g)	Dry to Wet Conversion Factor*	Wet Weight Concentration (µg/g)
Sept. 6	FBS6S46A	tomato	23.1	0.065	1.502
Sept. 7	FBS7S52B	tomato	0.9	0.065	0.0585
Sept. 7	FBS7S53A	tomato	1.4	0.065	0.091
Sept. 7	FBS7S55A	tomato	2.9	0.065	0.189
Sept. 7	FBS7S59D	tomato	28	0.065	1.82
Sept. 7	FBS7SS60A	tomato	31.1	0.052	2.022
Sept. 7	FBS7S61S	tomato	8.3	0.065	0.54
Sept. 5	FBS5S20A	watermelon	1.1	0.1	0.11
Sept. 5	FBS5S24B	zucchini	1.6	0.054	0.0864

* refer to Table A3-6 for dry weight to wet weight conversion factors.

Table A1-12c: Summary of Nickel in Produce from Rodney Street Residences (2000 and 2001 Samples Combined)

Summary Statistic	Wet Weight Nickel Concentration ($\mu g/g$)
average	0.62
95th% percentile	2.32
maximum	4.99
median	0.33

Human Health Risk Assessment

Appendix 2

Toxicity Assessment

Table of Contents
Human Health Risk Assessment – Appendix 2
Toxicity Assessment

Toxicity Assessment

A2-1 Introduction

The chemical screening section of the main report identified eight metals as being potential human health concerns in the Rodney Street area of Port Colborne. A toxicity assessment was compiled for these metals. The objectives of the toxicity assessment were:

- **S** to provide the reader with a brief understanding of the toxicological effects that have been reported to be associated with exposure to the chemicals of concern;
- **S** to identify whether each metal of concern is considered to have carcinogenic or non-carcinogenic effects; and
- **S** to identify suitable exposure limits against which exposures can be compared to provide estimates of potential health risks.

The toxicological profiles are **not** intended to:
- **S** be exhaustive examinations of all the toxicological information available for each metal;
- **S** be used to develop exposure limits for exposure routes where no exposure limits are available; or
- **S** critically review and/or modify currently existing exposure limits set by Agencies such as the US EPA, WHO, Health Canada, etc.

This toxicity assessment outlines the toxicological effects that have been reported to be associated with inhalation, ingestion and dermal contact exposures to antimony, arsenic, beryllium, cadmium, cobalt, copper, lead and nickel, and identifies whether each metal should be considered as a carcinogen or a non-carcinogen based on the exposure pathway. The type of exposure limit selected is dependent upon whether a compound is considered to have non-carcinogenic or carcinogenic effects, however, in some cases, e.g., lead and cobalt, cancer potency factors are not available. The types of exposure limits associated with both types of compounds are discussed below.

The relevance of toxicological endpoints derived from animal studies to humans is a challenging task from a risk assessment perspective. The primary difficulty is that the contaminant levels the study animals are typically exposed to are much higher than exposures faced by human populations. The effects of these high exposures are assessed by a variety of histopathological methods.

In most cases, the molecular mechanisms causing the metal related adverse effects are not well understood, and in many cases, there is the uncertainty that the mechanisms leading to an adverse effect may differ between species as well as between the route of exposure. In addition, many animal studies are based on a metal species that is convenient to administer but may not be the

one to which human populations are exposed.

The finding of adverse non-cancer effects in animals is then related to human populations by the use of uncertainty factors. Uncertainty factors provide a safety margin in the extrapolation of the estimates of adverse effects when the mechanisms leading to adverse effects are not well understood (discussed later in this section).

Carcinogenic compounds are generally considered to work through a non-threshold mechanism. This means that an adverse effect is assumed to have the potential to occur at any level of exposure, i.e., any exposure to a carcinogen is associated with some level of risk. At very low doses, the probability that an adverse effect (cancer) will occur is very small. The probability of developing cancer increases as the dose increases. Cancer potency factors are developed by extrapolating from animal test data or cancer incidences in exposed human populations.

There are cases where animal studies and occupational health studies of the adverse effects of nickel have shown certain similarities. For example, as discussed in section A2-9.2.1, an occupational exposure study carried out by Chashschin et al., 1994 clearly indicated various health impairments such as respiratory, skin, cardiovascular diseases and adverse reproductive outcomes in Russian nickel refinery workers directly exposed to at least 130 ug/m^3, and as high as 200 ug/m^3 nickel.

These findings parallel respiratory and reproductive effects shown in animal studies (see sections A2-9.2.1, A2-9.2.3). While the adverse effects of nickel are apparent in controlled animal studies, it is difficult to draw conclusive parallels with the occupational health study as the routes of exposure differ.

Similarly, nickel refinery workers exposed to elevated levels of nickel refinery dust exhibited changes in lung opacity (non-cancer endpoint) as well as nasal and lung cancer (see sections A2-9.2.1). Test animals exposed to nickel refinery dust aerosols and aerosols of individual insoluble nickel species (nickel oxide, nickel sulphide) developed increased incidences of lung tumors in some cases (see section A2-9.2.3.2).

However, in many cases, regulators are faced with evaluation of studies that are not exactly applicable but are the best available. This makes it a difficult task to correlate the findings from animal studies to humans. This is a point of uncertainty in relating the findings of a controlled animal study to an uncontrolled human health survey.

The toxicological profiles also examine the effect that the route of exposure has on the toxicological activity of each compound. For some compounds, the route by which the compound enters the body can have a marked effect on the toxicological effects that occur. In cases where the toxicological effects of a chemical differ between the routes of exposure, it is necessary to assess inhalation and ingestion exposures independently. For example, exposure to beryllium,

cadmium and nickel by inhalation may be carcinogenic, but exposure via ingestion, has not been shown to be carcinogenic. However, arsenic may be carcinogenic following either inhalation or ingestion exposure. Therefore, where route-specific exposure limits are available, the toxicological profiles will provide both. In cases where exposure limits are available for a single route of exposure, the toxicological profiles will not develop exposure limits by route-to-route extrapolation. Although complex route-to-route extrapolation is undertaken in some situations, it is typically discouraged by the US EPA and similar regulatory agencies because it requires detailed knowledge of pharmacokinetic and pharmacodynamic factors and extensive modeling. All of which are beyond the scope of the current assessment.

The health risk of antimony (Sb)(MOE, 1991), arsenic (As)(MOE, 1991; 1999; 2001), cadmium (Cd)(MOE, 1991; 1999), cobalt (Co)(MOE, 1991; 1998; 1999), copper (Cu)(MOE, 1991; 1998; 1999), lead (Pb)(MOE, 1991; MOEE, 1994; MOE, 1999) and nickel (Ni)(MOE, 1991; 1998; 1999) exposure in Ontario soils has been assessed in detail for several Ontario communities. These appendices offer supplementary information to chapter 3 of the main report (Part B). The references used in the development of each toxicological profile are provided at the end of each profile.

The selection of exposure limits (cancer and non-cancer) requires consideration of a number of important attributes of the available values. The framework and methods for developing exposure limits are well described by all major agencies.

Evaluation of any study used to estimate human health risk based on either toxicity testing in laboratory animals or an epidemiological study of exposed humans, utilizes extensive scientific criteria to determine the reliability of the testing protocols used. Other factors, such as the health of the animals during the test, the appropriate determination of toxicity endpoints, the statistical significance of any dose-response relationships found, and the occurrence of any confounding factors that affect the results are also considered. Epidemiological studies have a different set of rules to determine causal association between disease endpoints and human exposure patterns.

In terms of animal testing studies, the age of the study is not necessarily as important as the following of recognized and approved protocols for treating and exposing the animals to the substance being tested and identifying the resulting adverse effects. The statistical reliability of the data demonstrating the presence or absence of adverse health endpoints or any apparent dose-response relationship arising from the study are an important factor. More recent testing information generally benefits from more recent developments in toxicology and adds to the weight of evidence used to develop exposure limits.

Adoption of a particular animal testing (or epidemiology) study by regulatory agencies as supporting documentation for their exposure limits, is an important criterion for evaluation and selection of relevant toxicological information. Regulatory agencies then use such criteria to select studies to support the development of exposure limits, e.g., reference dose (R/D), tolerable daily

intake (TDI), etc. In addition, using the dose-response (or comparable extrapolation techniques from epidemiology studies), agencies apply uncertainty factors (UFs) for threshold, non-cancer endpoints, or mathematical low dose extrapolation methods for non-threshold, cancer endpoints.

For chemicals that exhibit threshold-type dose-response relationships, it is assumed that there exists an exposure dose below which adverse effects are very unlikely, i.e., where no measurable impacts would occur. This threshold or limit is commonly referred to as a reference dose (RfD), or allowable daily intake (ADI). Conservative estimates of this threshold are based on the application of "safety factors" or "uncertainty factors" (US FDA, 1982; US EPA, 1989; Health Canada, 1993) to a NOAEL (no observed adverse effect level) that has been determined experimentally, usually in animals and occasionally through controlled studies in people. The magnitude of these "safety factors" or "uncertainty factors", depends on the level of confidence in the use of available data as a basis for extrapolation to the exposure scenario of the risk assessment. This confidence is dependent on differences in species and duration of exposure, information on the variability in sensitivity among individuals, and the quality of available data (i.e., the weight of evidence of the supporting data).

Uncertainty factors are derived for a specific chemical on a case-by-case basis, depending principally on the quality of the available toxicological database. Generally, a factor of one to ten is used to account for intraspecies variation and interspecies variation. An additional factor of 1 to 100 is used to account for inadequacies of the database which include but are not necessarily limited to, lack of adequate data on developmental, chronic or reproductive toxicity, use of a LO(A)EL (lowest observed adverse effect level) versus a NO(A)EL and inadequacies of the critical study. An additional uncertainty factor ranging between one and five may be incorporated where there is sufficient information to indicate a potential for interaction with other chemical substances commonly present in the general environment. If the chemical substance is essential or beneficial for human health, the dietary requirement is also taken into consideration in derivation of the Tolerable Daily Intake or Concentration. Exceptionally, in deriving an exposure limit for severe effects (e.g., teratogenicity), an additional uncertainty factor of one to ten may be incorporated. Numerical values of the uncertainty factor normally range from 1 to 10,000. Uncertainty factors greater than 10,000 are not applied since the limitations of such a database are sufficient to preclude development of a reliable exposure limit.

Expert judgement is required in both the application of uncertainty factors to the identified threshold value and the extrapolation to low dose estimates. While RfD or risk-based concentrations can be generated using all the appropriate risk assessment protocols and principles, the resulting exposure limits may not be immediately achievable. This may be due to background concentrations, intakes from specific pathways, technical feasibility or other reasons, though none of these reasons should necessarily preclude the use of well documented and reviewed exposure limits. It should be noted that just because one exposure limit is lower than another, it does not necessarily imply that the limit provides additional safety, since most exposure limits incorporate adequate margins of safety. How an exposure limit is applied to reduce risk can have implications

for the ultimate "safety" of the value. Consequently, while exposure limits from different agencies using current, reliable toxicological information may vary, any claims as to their relative safety vis-a-vis each other requires an assessment of how the exposure limits are applied.

In this document, the term *µg/kg body weight-day* will be abbreviated as µg/kg-day.

A2-1.1 Exposure Limits for Non-Carcinogenic Compounds

Non-carcinogenic compounds are generally considered to act on the body through threshold mechanisms. This means that at low doses the body is able to detoxify and remove the compounds from the body without the compounds causing adverse or toxic effects. As the dose or exposure to a compound increases, the body's ability to detoxify and clear the compound is reduced. When exposure exceeds the body's ability to detoxify and excrete the compound, it can cause adverse or toxic effects. The point at which this occurs is called the threshold. The threshold is different for every compound. The exposure limits developed for each compound reflect the threshold for each chemical.

Contaminants of concern were assessed for both cancer potential and adverse health effects other than cancer. Toxic effects other than cancer may play an important role in the overall toxicity of the metals. Table A2-1 provides a brief summary of the major toxic effects, other than cancer, of the selected metals.

Non-cancer exposure limits by inhalation and oral routes are summarized in tables A2-2 and A2-3. The specific exposure limits selected for use in the human health risk assessment are shown in Table 3-1.

Table A2-1: Major Toxic Effects (Other Than Cancer) in Humans Following Elevated Exposures to Metals of Concern

Substance	Health Effects	Reference
Antimony	Nausea, vomiting, diarrhea, cardiovascular effects and GI disorders	ATSDR, 1990a; US EPA IRIS, 1998a; WHO,1996a
Arsenic	Abdominal pain, vomiting, diarrhea, muscular pain, flushing of skin, dermal lesions, neuropathy, and peripheral vascular disease	US EPA IRIS, 1998b; WHO,1998a; ATSDR, 1993a
Beryllium	Lung disease, no reported oral toxicity	US EPA IRIS, 1998c; ATSDR, 1993b
Cadmium	Kidney damage, also respiratory effects that tend to be reversible with discontinuation of exposure	US EPA IRIS, 1998d; WHO,1996b; ATSDR, 1999
Cobalt	Essential element (part of vitamin B_{12}), cardiovascular effects, lung disease, allergic contact dermatitis, rhinitis and asthma	ATSDR, 1992
Copper	Essential nutrient, lung disease, kidney disease, skin irritation and eczema, abdominal pain, nausea, vomiting, diarrhea, liver damage	US EPA IRIS 1998e; WHO, 1998b; ATSDR, 1990b
Lead	Tiredness, sleeplessness, irritability, headaches, joint pain, GI symptoms, peripheral neuropathy, kidney disease, hypertension, anemia, damage to central and peripheral nervous system, effects in behavior and intellectual development	US EPA IRIS 1998f; WHO,1996c; ATSDR, 1993c; MOEE, 1993
Nickel	Allergic lung disease, reproductive effects, kidney disease and allergic contact dermatitis	US EPA IRIS 1998g,h,i; WHO,1993, 1996d, 1998c; ATSDR, 1997

Table A2-2: Non-Cancer Inhalation Exposure Limits of Metals of Concern, Expressed as Air Concentrations (μg/m³)

Substance	Sources		
	US EPA[1]	WHO[2]	CEPA/CCME[3]
Antimony	0.2	NA	NA
Arsenic	NA	NA	NA
Beryllium	0.02	NA	NA
Cadmium	NA	0.005	NA
Cobalt	NA (0.03-ATSDR)	NA	NA
Copper	NA (2.4 California)	NA	NA
Lead	NA	0.5	NA
Nickel			
Metallic	NA	NA	0.018
Soluble Salts	NA (0.2 ATSDR[4])	NA	0.0035
Nickel Refinery Dust	NA	NA	0.02 (nickel oxide)
1. US EPA IRIS, 1998a-i; 2. WHO, Air Quality Guidelines for Europe, 2000; 3. Health Canada, 1996a; 4. ATSDR, 1997			

Table A2-3: Non-Cancer Oral Exposure Limits of Metals of Concern,
Expressed as Intakes (µg/kg/day)

Substance	Sources		
	US EPA[1]	WHO[2]	CEPA/CCME[3]
Antimony	0.4	0.86	NA
Arsenic	0.3	2	NA
Beryllium	2	NA	NA
Cadmium	1	1	NA
Cobalt	20 [4]	NA	NA
Copper	NA (140 [5])	2000	(100)
Lead	NA	3.5	3.57
Nickel			
Soluble Salts	20	(5)[6]	1.3 (nickel chloride) 50 (nickel sulphate)
Nickel Refinery Dust	NA	NA	NA
1. US EPA IRIS, 1998a-i, 2. WHO Drinking Water Guideline documentation, 1996-1998, 3. Health Canada, 1996a,b and CCME, 1997; 4. US EPA Region III, 2001; 5. IOM, 2001; 6. see section A2-9.3.2			

The US EPA is a reliable source of exposure limits or reference doses (RfD) for ingestion exposures and reference concentration (RfC) for inhalation exposures, that are developed from toxicological studies of human or animal populations. These are set to ensure that chronic exposures to a chemical at concentrations that are at or below the exposure limit will not result in adverse effects. The US EPA defines the RfD/RfC as;

A quantitative estimate (with uncertainty spanning perhaps an order of magnitude) of a daily exposure to the human population (including sensitive subgroups) that is likely to be without an appreciable risk of non-carcinogenic, deleterious effects during a life-time.

The US EPA RfD/RfC values are based on life-time averaged exposures. This means that limited exposures to a compound that exceed an exposure limit will not result in adverse effects, provided that over a life-time, the averaged daily dose does not exceed the exposure limit. The exposure limit is set to prevent the accumulation of the compound in the body at levels that exceed the threshold and therefore limit the possibility of adverse health effects occurring.

The RfD/RfC values are intended to be used as life-time average daily exposures and therefore, in assessing potential risks for an exposed individual or population, life-time averaged daily doses should be used if the exposures are expected to occur over a life-time. The US EPA Risk Assessment Guidance for Superfund (RAGS) recommends that in the assessment of risks associated with exposures to non-carcinogenic compounds, that life-time averaged exposures be used to assess risks when exposures occur throughout a life-time (US EPA, 1989). The RAGS further notes that the comparison of short-term or non-life-time exposures to RfD/RfC values should only be used as a screening exercise to determine if a potential human health risk would be predicted based on estimated exposures. Short-term exposures that are below the chronic exposure limit are not a concern for adverse human health effects (US EPA, 1989). Life-time chronic daily intakes (CDI) are calculated as shown in equation A2-1.

$$\textbf{Eq A2-1} \qquad \textbf{\textit{Total CDI}} = \sum_{1}^{n} \frac{(\textit{Intake}_{(1..n)} \times \textit{Time}_{(1..n)})}{(\textit{B.W.}_{(1..n)} \times 70 \textit{years})}$$

Where:
Total CDI	=	Total Chronic Daily Dose		μg/kg-day
Intake$_{l..n}$	=	Estimated Daily Intake (EDI) of age group n		μg/kg-day
Time$_{l..n}$	=	Time spent in each age group		years
BW$_{l..n}$	=	Body weight of receptor in each age group		kg

It is important to note that the total CDI value can also be referred to as a life-time averaged daily dose (LADD).

A2-1.2 Exposure Limits for Carcinogenic Compounds

The assessment of the carcinogenic potential of chemicals is undertaken by several major agencies and MOE relied on their classifications to characterize the potential for health effects and to select appropriate exposure limits.

A2-1.2.1 Cancer Classification

The International Agency for Research in Cancer (IARC), was the first organization to develop a weight of evidence scheme whereby a panel of international experts systematically evaluates the evidence for and against carcinogenicity of a given agent. The IARC panel then publishes the carcinogenic ranking for a given agent, which summarizes the weight of evidence for its potential to induce cancer in humans. IARC is a part of the World Health Organization (WHO).

Although the IARC ranking continues to be highly respected, other agencies have developed similar ranking schemes. Of these, the one published by the US EPA (1986) is probably the most influential. In 1996, US EPA replaced its ranking scheme based on letter ranks with a new descriptive scheme, which takes into account a wider range of data. The US EPA's 1986 scheme is still widely used, in part because the evaluations based on this earlier ranking scheme continues to be reported in the Integrated Risk Information System (IRIS) database. The ranking schemes by IARC and US EPA (1986) are quite similar. Although both organizations place a greater emphasis on reliable human epidemiological studies than on animal data, US EPA has traditionally placed heavier emphasis on animal data than IARC. The new US EPA (1996) ranking scheme has only been in use for a few years, so the number of agents ranked by this scheme is still relatively small.

In Canada, Health Canada has developed a carcinogen-ranking scheme under the Canadian Environmental Protection Act (CEPA) (Meek et al., 1994) based on the IARC ranking scheme. CEPA's scheme consists of more categories and subcategories and is not very compatible with those of IARC and US EPA. CEPA distinguishes between genotoxic and non-genotoxic carcinogens, and gives the latter group a lower ranking.

Some US states, including California, have their own rankings. A comparison of the key ranking schemes is summarized in table A2-4 below.

Table A2-4: Comparison of the Three Major Cancer Classification Schemes

Strength/Type of Evidence	Weight of Evidence Classification		
	US EPA[1]	IARC[2]	CEPA[3]
Sufficient/Strong Human Evidence	A	1	I
Limited Human + Sufficient Animal Evidence	B1	2A	III
Some Human, Limited Animal Evidence	B2	2B	II
Weak Evidence from Human and Animal Data	C	(2B)	III
Little Evidence for or Against Carcinogenicity	D	3	VI
Good Evidence for Absence of Carcinogenicity	E	4	V

1. US EPA, 1986, 2. IARC Monographs website, 2001, 3. Meek et al., 1994.

The route of exposure is an important issue that has to be considered in determining the basis of the cancer classification. Some metals appear to be carcinogenic when inhaled. Although they may have other toxic effects when they are taken up by another route, the evidence for carcinogenicity via these other routes is either absent or not very strong. Most cancer ranking schemes do not provide separate ranking for each route of exposure, although some information may be provided in the text accompanying the ranking. Table A2-5 presents the original ranking by the three agencies for the selected contaminants. For these substances, the carcinogenicity ranking describes the health impact resulting from inhalation exposure, but except for arsenic, does not describe the impact resulting from oral exposure.

Table A2-5: Cancer Classification by Inhalation and/or Oral Route

Substance	General Ranking		
	US EPA[1]	IARC[2]	CEPA[3]
Arsenic	A (oral & inhalation)	1 (oral & inhalation)	1 (oral & inhalation)
Beryllium	B1 (inhalation)	2A (inhalation)	NA
Cadmium	B1 (inhalation)	1 (inhalation)	II (inhalation)
Nickel			
Metallic	NA	2B (inhalation)	VI
Soluble Salts	NA	1 (inhalation)	1 (inhalation)
Insoluble Dusts	A (inhalation)	1 (inhalation)	I (inhalation)
1. US EPA IRIS, 1998b; 1998c; 1998d; 1998g; 1998h; 1998i, 2. IARC Monograph website, 2001, 3. Health Canada, 1996a.			

A2-1.2.2 Types and Sites of Cancer

Different carcinogens have a tendency to induce tumours at different sites and different cancer types. Table A2-6 below summarizes the site and the type of tumour induced by the selected contaminants. The list is not necessarily complete and only the best documented tumours are included in the table. Note that for some contaminants, the site and the type of tumour depend on the route of exposure.

Table A2-6: Sites and Types of Tumours

Substance	Cancer Types	Reference
Arsenic	skin, lung and other internal cancers	US EPA IRIS, 1998b; IARC, 1998; CEPA, 1996
Beryllium	lung cancer	US EPA IRIS, 1998c; IARC, 1997a; CEPA, 1996
Cadmium	lung cancer	US EPA IRIS, 1998d; IARC, 1997b; CEPA, 1996
Nickel	nasal and lung cancer	US EPA IRIS, 1998g; h; i, IARC, 1990; CEPA, 1996

Carcinogenic compounds, that act by damaging DNA, are generally considered to work through a non-threshold mechanism which means that an adverse effect is assumed to occur at any level. Any exposure to a carcinogen is considered to be associated with some level of risk. At very low doses, the probability that an adverse effect (cancer) will occur is extremely small. The probability of developing cancer increases as the dose increases. Incremental increases in life time cancer risk are estimated by comparing the established potency for each compound with the calculated CDI for that compound. In addition, potential cancer risks from inhaling airborne metals can be assessed by comparing the annual average concentrations with inhalation unit risk factors from various agencies (Table 3.2).

A2-1.2.3 Carcinogenic Potency

Carcinogenic potency by inhalation and oral routes is summarized in tables A2-7 and A2-8.

Table A2-7: Inhalation Potency of Selected Metals of Concern,
Expressed as Unit Risks $(\mu g/m^3)^{-1}$ (The recommended values are underlined)

Substance	Sources		
	US EPA[1]	WHO[2]	CEPA[3]
Arsenic	4.3×10^{-3}	1.5×10^{-3}	$6.4 \times 10^{-3,\,(4)}$ $(TC_{05} = 7.8)$
Beryllium	$\underline{2.4 \times 10^{-3}}$	NA	NA
Cadmium	$\underline{1.8 \times 10^{-3}}$	NA	$9.8 \times 10^{-3,\,(4)}$ $(TC_{05} = 5.1)$
Nickel			
Soluble Salts	-	-	$7.1 \times 10^{-4\,(4)}$ $(TC_{05} = 70)$
Nickel Refinery Dust	$\underline{2.4 \times 10^{-4}}$	3.8×10^{-4}	$1.25 \times 10^{-3\,(4)}$ $(TC_{05} = 40)$
1. US EPA IRIS, 1998b; c; d; g; h; i, 2. WHO, 2000, 3. Health Canada, 1996a, 4. Calculated from TC_{05} value reported by Health Canada (1996a) (unit risk = $0.05/TC_{05}$).			

Table A2-8: Oral Potencies of Selected Metals of Concern,
Expressed as Slope Factors $(\mu g/kg/day)^{-1}$

Substance	Sources		
	US EPA[1]	WHO[2]	CEPA[3]
Arsenic	1.5×10^{-3}	1.5×10^{-3}	2.8×10^{-3}
Beryllium	NA	NA	NA
Cadmium	NA	NA	NA
Nickel			
Soluble salts	NA	NA	NA
Nickel refinery dust	NA	NA	NA
1. US EPA IRIS, 1998, 2. WHO, 1996b; 1998d; 1998a; 1998c, 3. Health Canada, 1996a, 4. NA = Not Applicable.			

The US EPA is a reliable source of estimates of carcinogenic potency for numerous chemicals. The US EPA expresses carcinogenic potencies as cancer slope factors (*Risk per ($\mu g/kg$ body weight-day)*) or as a *Unit Risk (UR $(\mu g/m^3)^{-1}$)* for inhalation exposures or *Unit Risk (UR $(\mu g/L)^{-1}$)* for exposures to chemicals in drinking water. The slope factor is defined by the US EPA as;

> *An upper-bound on a maximum likelihood estimate developed from dose-response data using one of several models incorporating low-dose linearity.*

The Unit Risk is defined as;

> *The upper-level increased likelihood that an individual will develop cancer when exposed to a chemical over a life-time at a concentration of 1 μg/L in drinking water or 1 μg/m³ in air for a continuous inhalation exposure.*

WHO Guidelines for drinking water quality (1998c) state:

> *For substances that are considered to be carcinogenic, the guideline value is the concentration in drinking-water associated with an excess lifetime cancer risk of 10^{-5} (one additional cancer per 100 000 of the population ingesting drinking-water containing the substance at the guideline value for 70 years). Concentrations associated with estimated excess lifetime cancer risks of 10^{-4} and 10^{-6} can be calculated by multiplying and dividing, respectively, the guideline value by 10.*

> *In cases in which the concentration associated with an excess lifetime cancer risk of 10^{-5} is not feasible as a result of inadequate analytical or treatment technology, a provisional guideline value is recommended at a practicable level and the estimated associated excess lifetime cancer risk presented.*

> *It should be emphasized that the guideline values for carcinogenic substances have been computed from hypothetical mathematical models that cannot be verified experimentally and that the values should be interpreted differently from TDI-based values because of the lack of precision of the models. At best, these values must be regarded as rough estimates of cancer risk. However, the models used are conservative and probably err on the side of caution. Moderate short-term exposure to levels exceeding the guideline value for carcinogens does not significantly affect the risk.*

WHO Air quality guidelines for Europe (2000) describe incremental unit risks which are cancer risk estimates for lifetime exposure to a concentration of 1 μg/m³.

Health Canada provides cancer potency estimates as Tumorigenic Doses (TD_{05}) and Tumorigenic Concentrations (TC_{05}). These values represent life-time exposure levels that would result in cancers in 5% of the population.

A2-1.3 Dermal Exposure Limits

There are no dermal exposure limits for the metals of concern in Rodney Street community soils. US EPA (1989, 1992, 2001) provides limited guidance on a) determining the relative contribution of the dermal route of exposure and its importance to total exposure, and b) route-to-route extrapolation of exposure limits from another route of exposure (usually oral) to a dermal exposure limit. The approach described involves calculating dermal exposures as absorbed doses, which are compared to an oral exposure limit also expressed as an absorbed dose. Information on

the uptake and absorption of the chemical following oral and dermal exposure is required. In addition, the toxicological endpoint should be similar for both routes of exposure. Situations where the dermal route causes an endpoint through direct action on the skin would make route-to-route extrapolation from the oral exposure limit inappropriate. For the metals of concern:

a) estimated dermal intakes from exposure to metals in Rodney Street community soils were less than 1% of the total estimated metal intake from all pathways indicating that this exposure route was not significant. An exception to this was the dermal exposure estimate for antimony which was about 2% to 4% of the total exposure;

b) lead, cadmium, nickel and cobalt are known skin sensitizers and can cause irritation under some conditions. In terms of adverse effect endpoints following systemic absorption, there is no evidence suggesting significantly different toxicological sequelae following absorption from either dermal or oral routes.

Based on a) and b), no adjustments were made to the dermal uptake estimates and these were combined with the oral intake estimates and evaluated using oral exposure limits.

A2-1.4 References

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

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

ATSDR (Agency for Toxic Substances and Disease Registry). 1992. Toxicological Profile for Cobalt. U.S. Department of Health and Human Services, Public Health Service, Atlanta, GA.

ATSDR (Agency for Toxic Substances and Disease Registry). 1993a. Toxicological Profile for Arsenic. U.S. Department of Health and Human Services, Public Health Service, Atlanta, GA.

ATSDR (Agency for Toxic Substances and Disease Registry). 1993b. Toxicological Profile for Beryllium. U.S. Department of Health and Human Services, Public Health Service, Atlanta, GA.

ATSDR (Agency for Toxic Substances and Disease Registry).1993c. Toxicological Profile for Lead. U.S. Department of Health and Human Services, Public Health Service, Atlanta, GA.

ATSDR (Agency for Toxic Substances and Disease Registry). 1997. Toxicological Profile for Nickel. U.S. Department of Health and Human Services, Public Health Service, Atlanta, GA.

ATSDR (Agency for Toxic Substances and Disease Registry). 1999. Toxicological Profile for Cadmium. U.S. Department of Health and Human Services, Public Health Service, Atlanta, GA.

CCME (Canadian Council of Ministers of the Environment). 1997. Canadian Soil Quality Guidelines for Copper. CCME Subcommittee on Environmental Quality Criteria for Contaminated Sites. ISBN 0-662-25520-8.

CEPA. 1996. Canadian Environmental Protection Act. Priority Substances List. Supporting Documentation: Health-Based Tolerable Daily Intakes/Concentrations and Tumourigenic Doses/Concentrations for Priority Substances (Unedited Version). Health Canada, August, 1996.

Chashschin, V.P., G.P. Artunina, T. Norseth, 1994. Congenital defects, abortion and other health effects in nickel refinery workers. Sci. Total Environ. 148:287-291.

Health Canada. 1993. Health Risk Determination. The Challenge of Health Protection. ISBN 0-662-20842-0.

Health Canada. 1996a. Health-based tolerable daily intakes/concentrations and tumorigenic doses/concentrations for priority substances. ISBN 0-662-24858-9.

Health Canada. 1996b. Canadian Soil Quality Guidelines for Contaminated Sites. Human Health Effects: Inorganic Lead. Final Report. The National Contaminated Sites Remediation Program.

IARC (International Agency for Research on Cancer). 1998. Arsenic and arsenic compounds. Monographs on the evaluation of the carcinogenic risk of chemicals to humans. http://193.51.164.11/htdocs/Monographs/Suppl7/Arsenic.html. (Accessed October 17, 2001).

IARC (International Agency for Research on Cancer). 1997a. International Programme on Chemical Safety (IPCS), INCHEM. Beryllium and beryllium compounds. http://www.inchem.org/documents/iarc/iarc/iarc740.htm. (Accessed October 17, 2001).

IARC (International Agency for Research on Cancer). 1997b. International Programme on Chemical Safety (IPCS), INCHEM. Cadmium and cadmium compounds. http://www.inchem.org/documents/iarc/iarc/iarc741.htm. (Accessed October 17, 2001).

IARC (International Agency for Research on Cancer). 1990. Nickel and nickel compounds. Monographs on the evaluation of the carcinogenic risk of chemicals to humans. 49: 257-445.

IARC (International Agency for Research on Cancer) Monograph website. 2001. Lists of IARC Evaluations. http://193.51.164.11/monoeval/grlist.html. (Accessed October 17, 2001).

IOM. 2001. Institute of Medicine (Food and Nutrition Board). Dietary Reference Intakes for Vitamin A, Vitamin K, Arsenic, Boron, Chromium, Copper, Iodine, Iron, Manganese, Molybdenum, Nickel, Silicon, Vanadium, and Zinc. National Academy Press, Washington, D.C.

Meek, M.E., R. Newhook, R.G. Liteplo, and V.C. Armstrong, 1994. Approach to Assessment of Risk to Human Health for Priority Substances under the Canadian Environmental Protection Act. Environ. Carcino. Ecotox. Revs. C12:105-134.

MOE. 1991. Assessment of Human Health Risk of Reported Soil Levels of Metals and Radionuclides in Port Hope. pp.117. ISBN 0-7729-9065-4.

MOEE.1993. Rationale Document for the Development of Soil, Water and Air Quality Criteria for Lead. Ontario Ministry of Environment and Energy.

MOEE. 1994. Scientific Criteria Document for the Development of Multimedia Environmental Standards: Lead. Ontario Ministry of Environment and Energy. ISBN 0-7778-2529-5. pp. 332.

MOE. 1998. Assessment of Potential Health Risk of Reported Soil Levels of Nickel, Copper and Cobalt in Port Colborne and Vicinity, May 1997. Ontario Ministry of the Environment. ISBN 0-7778-7884-4.

MOE. 1999. Deloro Village Environmental Risk Study: Overall Technical Summary - Final Report. Ontario Ministry of the Environment, December, 1999.

MOE. 2001. Survey of Arsenic Exposure for Residents of Wawa. Goss Gilroy Inc., Ottawa, Canada.

US EPA.1986. U.S. Environmental Protection Agency. Guidelines for Carcinogen Risk Assessment. Federal Register 51(185):33992-34003. EPA/630/R-00/004.

US EPA. 1989. Risk Assessment Guidance for Superfund. U.S. Environmental Protection Agency, Washington, DC. EPA/540/01.

US EPA. 1992. Dermal Exposure Assessment: Principles and Applications. Washington, D.C. U.S. Environmental Protection Agency. EPA/600/8-91/011B.

US EPA. 1996. Proposed Guidelines for Carcinogen Risk Assessment. U.S. Environmental Protection Agency. Office of Research and Development. EPA/600/P-92/003C.

US EPA IRIS. 1998a. Antimony. Integrated Risk Information System (IRIS). Environmental Criteria and Assessment Office, Office of Health and Environmental Assessment, Cincinnati, OH. Online at http://www.epa.gov/iris/index.html. (Accessed Oct. 17, 2001).

US EPA IRIS. 1998b. Arsenic, inorganic. Integrated Risk Information System (IRIS). Environmental Criteria and Assessment Office, Office of Health and Environmental Assessment, Cincinnati, OH. Online at http://www.epa.gov/iris/index.html. (Accessed Oct. 17, 2001).

US EPA IRIS. 1998c. Beryllium and compounds. Integrated Risk Information System (IRIS). Environmental Criteria and Assessment Office, Office of Health and Environmental Assessment, Cincinnati, OH. Online at http://www.epa.gov/iris/index.html. (Accessed Oct. 17, 2001).

US EPA IRIS. 1998d. Cadmium. Integrated Risk Information System (IRIS). Environmental Criteria and Assessment Office, Office of Health and Environmental Assessment, Cincinnati, OH. Online at http://www.epa.gov/iris/index.html. (Accessed Oct. 17, 2001).

US EPA IRIS. 1998e. Copper. Integrated Risk Information System (IRIS). Environmental Criteria and Assessment Office, Office of Health and Environmental Assessment, Cincinnati, OH. Online at http://www.epa.gov/iris/index.html. (Accessed Oct. 17, 2001).

US EPA IRIS. 1998f. Lead and compounds (inorganic). Integrated Risk Information System (IRIS). Environmental Criteria and Assessment Office, Office of Health and Environmental Assessment, Cincinnati, OH. Online at http://www.epa.gov/iris/index.html. (Accessed Oct. 17, 2001).

US EPA IRIS. 1998g. Nickel refinery dust. Integrated Risk Information System (IRIS). Environmental Criteria and Assessment Office, Office of Health and Environmental Assessment, Cincinnati, OH. Online at http://www.epa.gov/iris/index.html. (Accessed Oct. 17, 2001).

US EPA IRIS. 1998h. Nickel subsulfide. Integrated Risk Information System (IRIS). Environmental Criteria and Assessment Office, Office of Health and Environmental Assessment, Cincinnati, OH. Online at http://www.epa.gov/iris/index.html. (Accessed Oct. 17, 2001).

US EPA IRIS. 1998i. Nickel, soluble salts. Integrated Risk Information System (IRIS). Environmental Criteria and Assessment Office, Office of Health and Environmental Assessment, Cincinnati, OH. Online at http://www.epa.gov/iris/index.html. (Accessed Oct. 17, 2001).

US EPA Region III. 2001. Risk-Based Concentration Table. US Environmental Protection Agency, Region III, Philadelphia, Penn. http://www.epa.gov/reg3hwmd/risk/riskmenu.htm. (Accessed Oct. 18, 2001).

U.S. Food and Drug Administration (FDA). 1982. Toxicological Principles for the Safety Assessment of Direct Food Additives and Color Additives Used in Food. (U.S.) Food and Drug Administration, Bureau of Foods, Washington, DC.

WHO (World Health Organization). 1993. Guidelines for drinking-water quality, 2nd ed. Vol. 1. Recommendations. Geneva, World Health Organization.

WHO (World Health Organization). 1996a. Antimony. Guidelines for drinking-water quality, 2nd ed. Vol. 2 Health criteria and other supporting information. WHO, Geneva. pp. 147-156.

WHO (World Health Organization). 1996b. Cadmium. Guidelines for drinking-water quality, 2nd ed. Addendum to Vol. 2 Health criteria and other supporting information. WHO, Geneva. pp. 195-201.

WHO (World Health Organization). 1996c. Lead. Guidelines for drinking-water quality, 2nd ed. Vol. 2 Health criteria and other supporting information. WHO, Geneva.

WHO (World Health Organization). 1996d. Guidelines for drinking-water quality, 2nd ed. Addendum to Vol. 2. Health criteria and other supporting information. Geneva.

WHO (World Health Organization). 1998a. Arsenic. Addendum to Guidelines for drinking-water quality, 2nd ed. Vol. 2 Health criteria and other supporting information. WHO, Geneva.

WHO (World Health Organization). 1998b. Copper. Guidelines for drinking-water quality, 2nd ed. Addendum to Vol. 2 Health criteria and other supporting information. WHO, Geneva. pp. 31-46.

WHO (World Health Organization). 1998c. Guidelines for drinking-water quality, 2nd ed. Addendum to Vol. 2 Health criteria and other supporting information. WHO, Geneva.

WHO. 2000. World Health Organization, Regional Office for Europe, Copenhagen. 2000. Air Quality Guidelines for Europe, Second Edition. ISBN 92-890-1358-3.

A2-2 Toxicological Profile for Antimony

Antimony is a naturally occurring metal that is used in various manufacturing processes. It is generally found as a sulphide or oxides. The natural sulphide of antimony was known and used in Biblical times as medicine and as a cosmetic. The most important use of antimony metal is as a hardener in lead for storage batteries. The metal also finds applications in solders and other alloys. Antimony trioxide is the most important of the antimony compounds and is primarily used in flame-retardant formulations. These flame retardant applications include such markets as children's clothing, toys, aircraft and automobile seat covers.

A2-2.1 Pharmacokinetics

Exposure to antimony may be via inhalation, oral and dermal routes (ATSDR, 1990). Antimony is sparingly absorbed following ingestion or inhalation (Felicetti et al., 1974; Gerber et al., 1982; US EPA IRIS, 1998a,b). Both gastrointestinal and pulmonary absorption are a function of compound solubility. Trivalent antimony is more readily absorbed than pentavalent forms. Antimony is transported in the blood. Antimony is not metabolized but may bind to macromolecules and react covalently with sulfhydryl and phosphate groups (ATSDR, 1990). Excretion of antimony is primarily via the urine and feces (Cooper et al., 1968; Ludersdorf et al., 1987; ATSDR, 1990).

A2-2.2 Toxicology
A2-2.2.1 Non-Cancer Effects

Acute oral exposure of humans and animals to high doses of antimony or antimony-containing compounds (antimonials) may cause gastrointestinal disorders (vomiting, diarrhea), respiratory difficulties, and death at extremely high doses (Bradley and Frederick, 1941; Beliles, 1979; ATSDR, 1990). Subchronic and chronic oral exposure may affect hematologic parameters (ATSDR, 1990). Long term exposure to high doses of antimony or antimonials has been shown to adversely affect longevity in animals (Schroeder et al., 1970). Limited data suggest that prenatal and postnatal exposure of rats to antimony interferes with vasomotor responses (Marmo et al., 1987; Rossi et al., 1987).

Acute occupational exposure may cause gastrointestinal disorders (probably due to ingestion of airborne antimony) (ATSDR, 1990). Exposure of animals to high concentrations of antimony and antimonials (especially stibine gas) may result in pulmonary edema and death (Price et al., 1979). Long term occupational exposure of humans has resulted in electrocardiac disorders, respiratory disorders, and possibly increased mortality (Renes, 1953; Breiger et al., 1954). Antimony levels for these occupational exposure evaluations ranged from 2,200 to 11,980 µg Sb/m^3. Based on limited data, occupational exposure of women to metallic antimony and several antimonials has reportedly caused alterations in the menstrual cycle and an increased incidence of spontaneous abortions (Belyaeva, 1967). Reproductive dysfunction has been demonstrated in rats exposed to antimony trioxide (Belyaeva, 1967).

No data were available indicating that dermal exposure of humans to antimony or its compounds results in adverse effects. Eye irritation due to exposure to stibine gas and several antimony oxides has been reported for humans (Stevenson, 1965; Potkonjak and Pavlovich, 1983).

A2-2.2.2 Cancer Effects

The US EPA has not evaluated antimony or antimonials for carcinogenicity. IARC (1989) has classified antimony trioxide as possibly carcinogenic to humans (Group 2B) by inhalation. Antimony trisulphide was not classifiable as to its carcinogenicity to humans (Group 3) (IARC, 1989).

A2-2.2.3 Susceptible Populations

No studies were located regarding unusual susceptibility of any human sub-population to antimony. A susceptible population will exhibit a different or enhanced response to antimony than will most persons exposed to the same level of antimony in the environment. Reasons include genetic make-up, developmental stage, health and nutritional status, and chemical exposure history. These parameters result in decreased function of the detoxification and excretory processes (mainly hepatic and renal) or the pre-existing compromised function of target organs. For these reasons the elderly with declining organ function and the youngest of the population with immature and developing organs are expected to be generally more vulnerable to toxic substances than healthy adults (ATSDR, 1992).

A2-2.3 Current Exposure Limits
A2-2.3.1 Oral Exposure Limits

The US EPA (US EPA IRIS, 1998a - oral R$_f$D assessment last revised 1991) has calculated subchronic and chronic oral reference doses (R$_f$Ds) of 0.4 µg/kg-day based on decreased longevity and alteration of blood chemistry in rats chronically exposed to potassium antimony tartrate in the drinking water (5 ppm equivalent to 350 µg Sb/kg-day) (Schroeder et al., 1970). More recently, the NRC (2000) has proposed an oral R$_f$D for antimony trioxide of 200 µg/kg-day based on increases in serum enzymes and liver weight in female rats from a study conducted by Hext et al., (1999). Because there is an inadequate toxicity database, the NRC's confidence in this derived R$_f$D is low to medium. Use of the US EPA's R$_f$D is a more conservative approach.

A2-2.3.2 Inhalation Exposure Limits

The US EPA (US EPA IRIS, 1998b - inhalation R$_f$C assessment last revised 1995) has calculated a reference concentration for chronic inhalation exposure (R$_f$C) of 0.2 µg/m^3 based on pulmonary toxicity and chronic interstitial inflammation in a one year inhalation toxicity study in rats exposed to antimony trioxide (Newton et al., 1994). This R$_f$C was also proposed by the NRC (NRC, 2000).

A2-2.3.3 Selection of Exposure Limits

The exposure limits used to assess the potential risks associated with ingestion and inhalation exposures to antimony are summarized in Table A2-9.

Table A2-9: Selected Exposure Limits for Antimony

Route of Exposure	Exposure Limit	Toxicological Basis	Source Agency
Non-Cancer Effects			
Ingestion	0.4 µg/kg-day	decreased longevity and altered blood chemistry in rats	US EPA IRIS, 1998a
Inhalation	0.2 µg/m³	pulmonary toxicity in rats	NRC, 2000; US EPA IRIS, 1998b
Dermal Contact	-	-	-
Cancer Effects			
Ingestion	N.A.[1]	-	-
Inhalation	N.A.	-	-
Dermal Contact	N.A.	-	-

1. Not Applicable

A2-2.4 Antimony References

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

ATSDR (Agency for Toxic Substances and Disease Registry). 1992. Toxicological profile for antimony. Atlanta, GA: U.S. Department of Health and Human Services, Public Health Service.

Beliles, R.P. 1979. The lesser metals. In Oehme, F. W., Ed., Toxicity of heavy metals in the environment, Marcel Dekker, New York. pp. 547-615.

Belyaeva, A.P. 1967. The effect of antimony on reproduction. Gig. Truda. Prof. Zabol. 11:32. (Cited in ATSDR, 1990).

Bradley, W.R. and W.G. Frederick. 1941. The toxicity of antimony - animal studies. Ind. Med. 10:15-22. (Cited in ATSDR, 1990).

Breiger, H., C.W. Semisch, J. Stasney and D.A. Piatnek. 1954. Industrial antimony poisoning. Indust. Med. Health. 23:521-523. (Cited in ATSDR, 1990).

Cooper, D.A., E.P. Pendergrass, A.J. Vorwald, R.L. Mayock, H. Brieger 1968. Pneumoconiosis among workers in an antimony industry. Am. J. Roentgenol. Rad. Ther. Nuclear Med. 103:495-508. (Cited in ATSDR).

Felicetti, S.W., R.G. Thomas and R.O. McClellan. 1974. Retention of inhaled antimony-124 in the beagle dog as a function of temperature of aerosol formation. Health Phys. 26(6): 525-531.

Gerber, G.B., J. Maes, and B. Eykens, 1982. Transfer of Antimony and Arsenic to the Developing Organism. Arch. Toxicol. 49:159-168.

Hext, P.M., P.J. Pinto and B.A. Rimmel. 1999. Subchronic feeding study of antimony trioxide in rats. J. Appl. Toxicol. 19(3): 205-209.

International Agency for Research on Cancer (IARC). 1989. Antimony trioxide and antimony trisulfide. In some organic solvents, resin monomers and related compounds, pigments and occupational exposures in paint manufacture and painting. IARC Monographs on the evaluation of carcinogenic risks to humans. Volume 47: 291-442.

Ludersdorf, R., A. Fuchs, P. Mayer, G. Skulsuksai, G. Schacke 1987. Biological assessment of exposure to antimony and lead in the glass-producing industry. Int. Arch. Occup. Environ. Health 59:469-474. (Cited in ATSDR, 1990).

Marmo, E., M.G. Matera, R. Acampora, C. Vacca, D. De Santis, S. Maione, V. Susanna, S. Chieppa, V. Guarino, et al., 1987. Prenatal and postnatal metal exposure: Effect on vasomotor reactivity development of pups. Cur. Ther, Res. 42:823-838.

MOE & MOL. 1991. Assessment of Human Health Risk of Reported Soil Levels of Metals and Radionuclides in Port Hope. Ministry of the Environment and Ministry of Labour. PIBS 1727.

Newton, P.E., H.F. Bolte, I.W. Daly, B.D. Pillsbury, J.B. Terrill, R.T. Drew, R. Ben-Dyke, A.W. Sheldon, L.F. Rubin 1994. Subchronic and chronic inhalation toxicity of antimony trioxide in the rat. Fund. and Appl. Tox. 22:561-576.

NRC (National Research Council). 2000. Toxicological risks of selected flame-retardant chemicals. National Academy Press, Washington, D.C.

Potkonjak, V. and M. Pavlovich. 1983. Antimoniosis: A particular form of pneumoconiosis. I. Etiology, clinical and x-ray findings. Int. Arch. Occup. Environ. Health. 51:199-207. (Cited in ATSDR, 1990).

Price, N. H., W.G. Yates and S.D. Allen. 1979. Toxicity evaluation for establishing IDLH values. National Institute for Occupational Safety and Health, Cincinnati, OH. PB87-229498. (Cited in ATSDR, 1990).

Renes, L.E. 1953. Antimony poisoning in industry. Arch. Ind. Hyg. 7:99-108.

Rossi, F., R. Acampora, C. Vacca, et al., 1987. Prenatal and postnatal antimony exposure in rats: Effect on vasomotor reactivity development of pups. Teratogen. Carcinogen. Mutagen. 7:491-496.

Schroeder, H.A., M. Mitchener and A.P. Nason. 1970. Zirconium, niobium, antimony, vanadium and lead in rats: Life-time studies. J. Nutr. 100:59-68.

Stevenson, C. J. 1965. Antimony spots. Trans. St. John's Hospital Dermat. Soc. 51:40-42. (Cited in ATSDR, 1990).

US EPA IRIS. 1998a. Antimony. Integrated Risk Information System (IRIS). Environmental Criteria and Assessment Office, Office of Health and Environmental Assessment, Cincinnati, OH.

US EPA IRIS. 1998b. Antimony Trioxide. Integrated Risk Information System (IRIS). Environmental Criteria and Assessment Office, Office of Health and Environmental Assessment, Cincinnati, OH.

A2-3 Toxicological Profile for Arsenic

Arsenic (As) is a brittle, gray metal that tarnishes in air. It is a natural component of the earth's crust and occurs in small amounts in rock, soil, water and underwater sediments. It is commonly found in combination with sulphur and iron in minerals such as arseno-pyrite. Arsenic is used mainly to preserve wood and to control insects and weeds.

Elemental arsenic is not soluble in water; calcium arsenate, and calcium arsenites are sparingly soluble in water; the remaining arsenicals are soluble in water. Arsenic, arsenic pentoxide, arsenic trioxide, the calcium arsenites, lead arsenate, and potassium arsenate are soluble in various acids (ATSDR, 1993).

A2-3.1 Pharmacokinetics

The oral bioavailability of arsenic compounds is dependent on the chemical species and on the matrix (e.g., soil or dust) in which it is administered. Based on published literature, the absorption of water-soluble inorganic arsenic compounds in an aqueous solution is about 95 percent (ATSDR, 1993). For soil and house dust impacted by operating smelters containing arsenic, the oral bioavailability in Cynomolgus monkeys is about 14 and 19 percent, respectively (Freeman et al., 1995). The bioavailability of inorganic arsenic for exposure via inhalation would be in the range of 30 – 34 percent (ATSDR, 1993). Dermal absorption through human skin *in vitro* ranged from 0.8 to 1.9 percent (Wester et al., 1993).

Distribution of arsenic within the body is affected by the route through which exposure occurs. Given sufficient time for equilibration, arsenic generally tends to be evenly distributed amongst tissues within the body. The interaction of arsenic with various tissues is dependent on the chemical form of the arsenic. The primary pathway of elimination of inorganic arsenic is excretion via the urine. Because of the importance of urinary excretion as the primary route of elimination of arsenic, concentrations of arsenic compounds in the urine is considered to be a reliable index of recent exposure to arsenic (ATSDR, 1993).

A2-3.2 Toxicology
A2-3.2.1 Non-Cancer Effects

There are numerous studies that have looked at human exposures to inorganic arsenic in the air, but there are no reports of fatalities associated with short term occupational exposures to arsenic levels as high as 100 mg As/m^3 (ATSDR, 1993).

Inhalation exposures to inorganic arsenic dusts in the workplace have been reported to cause irritation of the nose and throat, laryngitis, bronchitis and cases of very high exposures have been reported to result in perforation of the nasal septum (ATSDR, 1993). However, respiratory effects have not been noted at exposure levels that range between 0.1 and 1.0 mg/m^3 (ATSDR, 1993). There is some limited evidence of respiratory tract effects following oral exposure to inorganic

arsenic, but this is thought to be a secondary effect that is due to the vascular damage which results from the ingestion of arsenic (ATSDR, 1993).

There is limited and equivocal epidemiological evidence that suggests that inhalation exposures to arsenic trioxide dust may result in cardiovascular effects. However, there are a number of studies that indicate that oral exposures to inorganic arsenic can lead to serious damage of the cardiovascular system (ATSDR, 1993). Both acute and long-term exposures can result in myocardial depolarization and cardiac arrhythmias. Long term exposures to low levels of arsenic can also result in damage to the vascular system, characterized by a progressive loss of circulation in the hands and feet (ATSDR, 1993). In areas of Taiwan, with elevated levels of arsenic in the drinking water, evidence of circulatory effects related to arsenic exposures were seen at a dose of approximately 0.014 mg As/kg-day (ATSDR, 1993).

There are several studies that have indicated that inhalation exposures to inorganic arsenic can lead to a number of neurological effects in humans, including peripheral neuropathy of sensory and motor neurons that are manifested as numbness, loss of reflexes and muscle weakness. In extreme cases, frank encephalopathy including hallucinations and memory loss have been reported (ATSDR, 1993). These effects generally cease once exposures have ended (ATSDR, 1993).

Acute effects of oral arsenic exposure include vomiting, nausea, diarrhea, gastrointestinal haemorrhage and death. There are a large number of cases of human fatalities following the ingestion of inorganic arsenicals. In most cases, the doses resulting in death have been difficult to quantify. However, two reports, indicate that doses ranging between 1 and 22 mg As/kg body weight per day (mg/kg-day) have resulted in death. Although similar effects are often seen with long-term exposures to lower doses of arsenic, effects are not generally reported at doses lower than 0.01 mg As/kg-day (ATSDR, 1993).

There are a large number of studies that indicate that the acute ingestion of large amounts of inorganic arsenic can cause a number of injuries to the nervous system including; headache, lethargy, mental confusion, hallucination, seizures and in extreme cases, coma (ATSDR, 1993). Chronic exposures to lower levels of arsenic, ranging between 0.019 and 0.5 mg/kg-day, are typically characterized by a peripheral neuropathy similar to that seen with inhalation exposures. Neurological effects have not been detected in populations chronically exposed to arsenic levels of less than 0.01 mg/kg-day (ATSDR, 1993).

A number of hematological effects including anemia and leukopenia have been reported in humans as a result of acute, intermediate and chronic oral exposures to arsenic (ATSDR, 1993). These effects are usually not seen in persons exposed to levels of arsenic lower than 0.07 mg/kg-day (ATSDR, 1993).

Oral exposures to inorganic arsenic have been reported to cause several toxic effects in the liver including, elevated levels of hepatic enzymes in the blood, portal tract fibrosis and swelling of the

liver (ATSDR, 1993). These effects are generally seen in cases where chronic exposures range between 0.019 to 0.1 mg/kg-day (ATSDR, 1993). It has been suggested by several researchers that these effects are secondary to the damage of hepatic blood vessels resulting from the damaging effects that inorganic arsenic has on the circulatory system. However, there is insufficient clinical information available to confirm this (ATSDR, 1993).

There is little clinical evidence of renal damage following oral exposures to inorganic arsenic compounds (ATSDR, 1993). A few cases of renal failure have been reported in cases of arsenic poisoning, but this is felt to be due to fluid imbalances of vascular damage caused by arsenic, and not directly attributable to arsenic (ATSDR, 1993).

The most common dermal effect associated with the ingestion of inorganic arsenic is the development of a pattern of skin changes which include hyperkeratosis, the development of hyperkeratotic warts, areas of hyperpigmentation and hypopigmentation (ATSDR, 1993). Numerous studies have shown that dermal effects are common in humans exposed to inorganic arsenic levels that range between 0.01 and 0.1 mg As/kg-day. These studies have also demonstrated that, below a dose level of 0.01 mg As/kg-day, dermal effects are not reported (ATSDR, 1993).

A2-3.2.2 Cancer Effects

There is sufficient convincing epidemiological evidence to show that inhalation exposure to inorganic arsenic can increase the risk of developing lung cancer. Many studies provide only qualitative evidence of an association between the duration of and/or level of exposure to arsenic and the increase in the rate of lung cancer. There is sufficient epidemiological information available from occupational studies for the US EPA to develop cancer potency estimates for inhalation exposures to inorganic arsenic (US EPA IRIS, 1998 - carcinogenicity assessment last revised 1998). Health Canada (1996) classified inorganic arsenic compounds as Group I (carcinogenic to humans).

There are a large number of epidemiological studies that provide convincing evidence that the ingestion of inorganic arsenic increases the risk of developing skin cancer. The most common effect is the development of squamous cell carcinomas. Basal cell carcinomas also occur. In the majority of cases, skin cancer only develops after prolonged exposure (ATSDR, 1993). There is sufficient human epidemiological data available for the US EPA to develop estimates of cancer risk associated with oral exposure to inorganic arsenic (US EPA IRIS, 1998 - carcinogenicity assessment last revised 1998).

A2-3.2.3 Susceptible Populations

No studies were located regarding unusual susceptibility of any human sub-population to arsenic. A susceptible population will exhibit a different or enhanced response to arsenic than will most persons exposed to the same level of arsenic in the environment. Reasons include genetic make-

up, developmental stage, health and nutritional status, and chemical exposure history. These parameters result in decreased function of the detoxification and excretory processes (mainly hepatic and renal) or the pre-existing compromised function of target organs. For these reasons the elderly with declining organ function and the youngest of the population with immature and developing organs are expected to be generally more vulnerable to toxic substances than healthy adults (ATSDR, 1993).

A2-3.3 Current Exposure Limits
A2-3.3.1 Oral Exposure Limits

The US EPA IRIS (1998 - oral R/D assessment last revised 1993) calculated an oral R/D of 0.3 µg As/kg-day based on epidemiological studies of chronic exposure to arsenic through drinking water. This limit was selected for non-carcinogenic effects.

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. The cancer slope factor of 0.0015 (µg As/kg-day)$^{-1}$ and corresponding risk specific dose (RSD) of 0.00067 µg As/kg-day are based on an acceptable risk level of one-in-one million.

On January 22, 2001, the US EPA established an enforceable Maximum Contaminant Level (MCL) of 10 µg/L for arsenic (Fed. Reg. 66: 6976-7059). On March 23, 2001, US EPA issued a delay of the effective date for this Final rule (Fed. Reg. 66: 16134-16135). On May 22, 2001 EPA announced that it would delay the effective date for the rule until February 22, 2002 allowing time to complete the reassessment process and to afford the public a full opportunity to provide further input. Following a request from US EPA to the National Academy of Sciences' (NAS) National Research Council's subcommittee on arsenic to review the science and update its 1999 *Arsenic in Drinking Water* report, a revised *Arsenic in Drinking Water, 2001 Update* report was prepared. This update report assessed new information on arsenic exposure in Taiwan and northern Chile. At this time the US EPA IRIS file for inorganic arsenic has not been updated, so the current IRIS file oral cancer slope factor is as described above.

WHO (1996) calculated an arsenic in drinking water risk of 10^{-5} (µg/ L)$^{-1}$ based on prevalence of skin cancer in Taiwanese studies and the US multistage model.

Health Canada (Health Canada, 1996; CEPA, 1996) developed a TC_{05} of 840 µg/L for drinking water based on the Taiwanese studies and the US EPA's 1988 model (US EPA, 1988, as cited in CEPA, 1996) adjusted for Canadian ingestion volumes.

A2-3.3.2 Inhalation Exposure Limits

The US EPA (US EPA IRIS, 1998 - carcinogenicity assessment last revised 1998) calculated an inhalation unit risk for arsenic of 0.0043 $(\mu g/m^3)^{-1}$ based on epidemiological studies of lung cancer in workers at arsenic smelters.

WHO (2000) calculated an inhalation unit risk for arsenic of 0.0015 $(\mu g/m^3)^{-1}$ based on epidemiological studies of lung cancer in workers at arsenic smelters in Sweden and the USA.

Health Canada (1996) developed a TC_{05} of 7.8 $\mu g/m^3$ based on occupational exposure data at the Anaconda copper smelter in Montana and a relative risk model.

A2-3.3.3 Selection of Exposure Limits

See discussion in Section 5.7 (Main document - Part B).

A2-3.4 Arsenic References

ATSDR. 1993. Agency for Toxic Substances and Disease Registry, Toxicological Profile for Arsenic. U.S. Department of Health and Human Services, Atlanta, Georgia, USA.

CEPA. 1996. Canadian Environmental Protection Act. Priority Substances List. Supporting Documentation: Health-Based Tolerable Daily Intakes/Concentrations and Tumourigenic Doses/Concentrations for Priority Substances (Unedited Version). Health Canada, August, 1996.

Freeman, G.B., R.A. Schoof, M.V. Ruby, A.O. Davis, J.A. Dill, S.C. Liao, C.A. Lapin and P.D. Bergstrom. 1995. Bioavailability of arsenic in soil and house dust impacted by smelter activities following oral administration Cynomolgus monkeys. Fundam. Appl. Toxicol. 28: 215-222.

Health Canada. 1996. Health-based tolerable daily intakes/concentrations and tumorigenic doses/ concentrations for priority substances. ISBN 0-662-24858-9.

NRC (National Research Council). 2001. Arsenic in Drinking Water, 2001 Update. Washington, D.C: National Academy Press.

US EPA. 1988. Special report on ingested inorganic arsenic: Skin cancer; nutritional essentiality. Washington, D.C.: U.S. Environmental Protection Agency, Risk Assessment Forum. EPA/625/3-87/013.

US EPA IRIS. 1998. Arsenic, inorganic. Integrated Risk Information System (IRIS). Environmental Criteria and Assessment Office, Office of Health and Environmental Assessment, Cincinnati, OH. Online at http://www.epa.gov/iris/index.html. (Accessed Oct. 17, 2001).

US EPA. 2001. Environmental Protection Agency. 40 CFR Parts 9, 141 and 142. Final Rule. Federal Register 66:16134-16135.

Wester, R.C., H.I. Maibach, L. Sedik, J. Melendres and M. Wade. 1993. *In vivo* and *in vitro* percutaneous absorption and skin decontamination of arsenic from soil and water. Fundam. Appl. Toxicol. 20: 336-340.

WHO (World Health Organization). 1996. Arsenic. Guidelines for drinking-water quality, 2nd ed. Vol. 2 Health criteria and other supporting information. WHO, Geneva.

WHO (World Health Organization). 2000. WHO Regional Office for Europe, Copenhagen. Air Quality Guidelines for Europe, Second Edition. ISBN 92-890-1358-3.

A2-4 Toxicological Profile for Beryllium

Beryllium (Be) is a hard, grayish, odorless metal. The element occurs naturally as a chemical component of certain rocks, coal and oil, volcanic dust, and soil. Two kinds of mineral rocks, bertrandite and beryl, are mined commercially for the recovery of beryllium (ATSDR, 1993). Beryllium is used in beryllium-copper alloys, in microelectronics, in aerospace technology, as a solid-propellant in rocket fuels (Lewis, 1993), in aircraft brakes, in X-ray windows, and in neutron reflectors (Ashford, 1994).

A2-4.1 Pharmacokinetics

Inhaled beryllium is absorbed through the lungs, however insufficient data are available to determine the rate and extent of absorption. The biological half-life of beryllium in serum is estimated to be between two to eight weeks.

Toxicity through oral exposure is not very likely since animal studies show that beryllium is not efficiently absorbed through the gastrointestinal tract. Soluble salts are precipitated by reaction with proteins in the alimentary tract (Browning, 1969). No human data are available regarding the absorption of beryllium after oral exposure.

Beryllium does not appear to be absorbed through intact skin as exposed workers demonstrated skin rashes and ulcerations only when the skin was cut accidently.

A2-4.2 Toxicology
A2-4.2.1 Non-Cancer Effects

Inhalation of high concentrations of soluble beryllium compounds has caused pneumonia in occupationally exposed workers. Chronic inhalation exposure to somewhat lower concentrations can lead to an obstructive lung disease known as chronic beryllium disease (CBD). Chronic beryllium disease is caused by genetically regulated cell-mediated immune responses (US EPA IRIS, 1998; Chang, 1996).

Swallowing beryllium has not been reported to cause effects in humans because very little beryllium can move from the stomach and intestines into the bloodstream. No human data are available regarding ingestion of beryllium. However, animal studies show lesions on the stomach as well as the small and large intestines as the result of ingestion of beryllium sulphate in the diet.

Skin lesions have been reported in a few individuals occupationally exposed to beryllium. Skin ulceration occurred only if the skin had been accidently cut.

A2-4.2.2 Cancer Effects

Several epidemiological studies show an increase incidence of lung cancer deaths amongst workers employed at beryllium factories (ATSDR, 1993). However, historical exposure levels were not reported so no correlation could be drawn between the incidence of lung cancer deaths and beryllium exposure.

The United States Environmental Protection Agency classifies inhaled beryllium and beryllium compounds as a probable human carcinogen (Group B1) based on limited evidence for humans and sufficient data for animals (US EPA IRIS,1998 - carcinogenic assessment last revised 1998). The United States Environmental Protection Agency also indicates that there are no studies on the potential carcinogenicity of ingested beryllium for humans and that the available animal studies do not indicate any adverse effects (US EPA IRIS, 1998 - carcinogenic assessment last revised 1998). The United States National Toxicology Program classifies beryllium and compounds as reasonably anticipated carcinogens (NTP, 2001).

The International Agency for Research on Cancer (IARC, 1997) has classified beryllium and beryllium compounds as carcinogenic to humans (Group 1) based on sufficient animal and human data.

A2-4.2.3 Susceptible Populations

A genetic predisposition for a human leukocyte antigen (HLA) class II may make some individuals more susceptible to chronic beryllium disease (ATSDR, 1993). Other factors which may increase susceptibility to beryllium are lowered adrenal gland or liver function (ATSDR, 1993).

A2-4.3 Current Exposure Limits
A2-4.3.1 Oral Exposure Limits

The United States Environmental Protection Agency has developed an oral reference dose of 2.0 µg/kg-day for beryllium (US EPA IRIS, 1998 - oral R$_f$D assessment last revised 1998). The reference dose is based on a benchmark dose derived from dose response modeling of data for a study of lesions on the small intestines of dogs (Morgareidge et al., 1976). A benchmark dose is the dose at the 95% confidence interval of a dose-response model and corresponds to a 10% increase in effects (stomach lesions) in comparison to the control population. An uncertainty factor of 300 (ten for interspecies differences, ten for differences in human populations and three for database deficiencies) was applied to the benchmark dose to determine the reference dose.

ATSDR has developed a minimal risk level (MRL) for beryllium based on the same dog study (Morgareidge et al., 1976) of 1.0 µg beryllium/kg-day. The MRL was determined by applying an uncertainty factor of 100 to the no-observed-adverse-effect level (NOAEL) at 100 µg beryllium/kg-day.

A2-4.3.2 Inhalation Exposure Limits

The US EPA (US EPA IRIS, 1998 - inhalation R*f*C assessment last revised 1998) has developed an inhalation R*f*C of 0.02 $\mu g/m^3$ based on beryllium sensitization and progression to chronic beryllium disease in beryllium workers (Kreiss et al., 1996) and the Eisenbud et al., (1949) study of community residents living near a beryllium plant.

The US EPA (US EPA IRIS, 1998 - carcinogenic assessment last revised 1998) has developed an inhalation unit risk of 0.0024 $(\mu g/m^3)^{-1}$ based on lung cancer mortality in male beryllium manufacturing workers (Wagoner et al., 1980). The air concentration at the 10^{-6} life-time cancer risk levels is 0.0004 $\mu g/m^3$, respectively.

A2-4.3.3 Selection of Exposure Limits

The estimates of the carcinogenic potencies of inhaled beryllium and non-cancer effects of inhaled and ingested beryllium, developed by the US EPA will be used to assess potential human health risks associated with exposure to beryllium at this site. The selected exposure limits established by the US EPA and the health effects upon which they are based are summarized below (Table A2-10).

Table A2-10: Selected Exposure Limits for Beryllium

Route of Exposure	Exposure Limit	Toxicological Basis	Source Agency
Non-Cancer Effects			
Ingestion	2 $\mu g/kg$-day	intestinal lesions in dogs	US EPA IRIS, 1998
Inhalation	0.02 $\mu g/m^3$	beryllium sensitization in human populations	US EPA IRIS, 1998
Cancer Effects			
Ingestion	N/A[1]	-	-
Inhalation	0.0024 $(\mu g/m^3)^{-1}$	lung cancer in humans	US EPA IRIS, 1998

1. Not Applicable.

A2-4.4 Beryllium References

Ashford, R.D. 1994. Ashford's Dictionary of Industrial Chemicals: Properties, Production, Uses. London, England: Wavelength Publ, Ltd. pp.12. (as cited in HSDB, 2001).

ATSDR. 1993. Agency for Toxic Substances and Disease Registry. Toxicological Profile for Beryllium. U.S. Department of Health and Human Services - Public Health Service (CDROM version, 2000).

Browning, E. 1969. Toxicity of Industrial Metals. 2nd ed. New York: Appleton-Century-Crofts. (as cited in HSDB, 2001).

Chang, L.W. (ed.). 1996. Toxicology of Metals. Boca Raton, FL: Lewis Publishers, pp.929-30. (as cited in HSDB, 2001).

Eisenbud, M., R.C. Wanta, C. Dustan, L.T. Steadman, W.B. Harris, B.S. Wolf, 1949. Non-Occupational Berylliosis. J. Indust. Hyg. Toxicol. 31:282-294.

HSDB. 2001. Hazardous Substance Data Bank. National Library of Medicine, National Toxicology Information Program, Bethesda, MD. [http://toxnet.nlm.nih.gov/cgi-bin/sis/htmlgen/HSDB]. (Accessed October 17, 2001).

IARC (International Agency for Research on Cancer). 1997. International Programme on Chemical Safety (IPCS), INCHEM. Beryllium and beryllium compounds. http://www.inchem.org/documents/iarc/iarc/iarc740.htm. (Accessed October 17, 2001).

Kreiss, K., M.M. Mroz, L.S. Newman, J. Martyny, B. Zhen, 1996. Machining risk of beryllium disease and sensitization with median exposures below 2 μg /m^3. Am J Ind Med. 30(1): 16-25.

Lewis, R.J., Jr. 1993. Hawley's Condensed Chemical Dictionary 12th ed NY, NY: Van Nostrand Reinhold Co. pp.139. (as cited in HSDB, 2001).

Morgareidge, K., G.E. Cox, and M.A. Gallo, 1976. Chronic feeding studies with beryllium in dogs. Food and Drug Research Laboratories, Inc. Submitted to the Aluminum Company of America, Alcan Research & Development, Ltd., Kawecki-Berylco Industries, Inc., and Brush-Wellman, Inc.

National Toxicology Program (NTP). 2001. 9th Report on Carcinogens. Revised January 2001. U.S. Department of Health and Human Services, Public Health Service, National Toxicology Program.

US EPA IRIS. 1998. Beryllium and Compounds. Integrated Risk Information System (IRIS). Environmental Criteria and Assessment Office, Office of Health and Environmental Assessment, Cincinnati, OH. Online at http://www.epa.gov/iris/index.html. (Accessed October 17, 2001).

Wagoner, J.K., P.F. Infante and D.L. Bayliss. 1980. Beryllium: an etiologic agent in the induction of lung cancer, nonneoplastic respiratory disease, and heart disease among industrially exposed workers. Environ. Res. 21:15-34.

A2-5 Toxicological Profile for Cadmium

Cadmium is a natural element that is usually found as a mineral combined with other elements such as oxygen, chloride and sulphur. Cadmium forms both organic and inorganic compounds. It is extracted mostly during the production of other metals, and is used in batteries, pigments, metal coatings and plastics.

A2-5.1 Pharmacokinetics

Following inhalation, the major site of cadmium absorption in humans is the alveoli of the lung. Human data are not available for absorption in the lung. A kinetic respiratory tree model has been developed to predict cadmium particle deposition in the lung (Nordberg et al., 1985). This model suggests that only 5% of the particles that are greater than 10 µm in diameter will be deposited and that about 50% of the particles less than 0.1 µm will be deposited. The respiratory tree model also predicts that 50 to 100% of the cadmium deposited in the alveoli will be absorbed.

The majority of ingested cadmium tends to pass through the gastrointestinal tract without being absorbed and is excreted in the feces. Absorption of ingested cadmium is influenced by nutritional status, with absorption increased by low intake of calcium, iron, zinc and copper (Nordberg et al., 1985). Absorbed (from the lungs and gastrointestinal tract) cadmium tends to be excreted very slowly and is found in equal proportions in the urine and feces. The main target organ for cadmium following ingestion is the kidney. The half life of cadmium in the human body is very long. An estimated half life for cadmium in the kidney ranges from 6 to 38 years and the liver from 4 to 19 years (ATSDR, 1998). The placenta may act as a partial barrier to fetal cadmium exposure.

Cadmium is not metabolized, rather it binds to proteins and other molecules. In particular it binds to the protein, albumin in the bloodstream which transports cadmium to the liver. Once cadmium enters the liver it becomes bound to another protein called metallothionein and is released to the bloodstream. The metallothionein bound cadmium is then filtered by the kidney glomerulus and is then reabsorbed by the proximal tubule cells. Lysozymes degrade the cadmium-metallothionein complex and cause free cadmium to be released in the kidney. The free cadmium initiates the synthesis of metallothionein in the proximal tubule cells and can also cause damage to the kidneys in excessive amounts.

Currently, information to determine the potential absorption of cadmium via the dermal route of exposure is limited (ATSDR, 1998). Dermal absorption factors have been recommended (US EPA, 1992, 2001) based on a human skin *in vitro* study of the dermal absorption of cadmium chloride from soil and water (Wester et al., 1992).

A2-5.2 Toxicology

Cadmium and cadmium compounds possess moderately acute toxicity via both ingestion and inhalation. Cadmium is slowly excreted by the body, and therefore bioaccumulates in humans. Chronic cadmium poisoning can be associated with both inhalation and ingestion.

A2-5.2.1 Non-Cancer Effects

Based on studies of cadmium production workers, the route of entry for cadmium with the most immediate health effects is inhalation of fumes or dust. Localized health effects caused by cadmium exposure include irritation to the respiratory tract and to the mucous membrane lining of the inner surface of the eyelid. This is often accompanied by dyspnea (severe difficulty in breathing) and general weakness. Troubled breathing may become more pronounced as pulmonary edema and tracheobronchitis develop. The most common result of acute systemic cadmium exposure is emphysema, but in some instances, mortality may occur. Prolonged exposure may also result in anosmia (loss of sense of smell) and discoloration of the teeth.

Ingestion of high acute doses of cadmium may cause gastrointestinal effects such as nausea, vomiting, and abdominal pain (Nordberg et al., 1973). Cadmium causes kidney damage, particularly to the proximal renal tubules in the early stages and as the disease progresses, or the dose increases, glomerular damage is also observed. Renal dysfunction has been demonstrated to be a consequence of chronic low level exposure to both inhaled and ingested cadmium (Bernard et al., 1994).

Chronic cadmium exposure coupled with poor nutrition can lead to changes in the way which the kidney metabolizes vitamin D. This can result in painful bone diseases such as osteomalacia and osteoporosis, mainly in women. There is limited data to suggest that cadmium exposures in pregnant women may result in decreased birth weight in their babies.

Epidemiologic studies have found an association between cadmium exposure and osteoporosis. Findings in a study on workers exposed to cadmium for up to five years suggest dose-response relationships between cadmium dose and bone mineral density and between cadmium dose and osteoporosis (Järup et al., 1998). In a study on residents living within known proximities to zinc smelters, low to moderate environmental exposure to cadmium was associated with skeletal demineralization and incidence of fractures and height loss (Staessen et al., 1999).

Cadmium appears to have a relatively low dermal toxicity based on studies that showed that workers who were occupationally exposed to high levels of cadmium dust, did not report any dermal effects. Cadmium does not appear to cause sensitization by repeated dermal contact.

A2-5.2.2 Cancer Effects

Epidemiological studies demonstrate increased incidence of lung cancer in workers exposed to

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

The United States Environmental Protection Agency has classified cadmium as a probable human carcinogen (Group B2) when inhaled, based on limited human and sufficient animal data (US EPA IRIS, 1998 - carcinogenicity assessment last revised 1992). Health Canada (Newhook et al., 1994) has classified cadmium as a Group II carcinogen.

A2-5.2.3 Susceptible Populations

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

A2-5.3 Current Exposure Limits
A2-5.3.1 Oral Exposure Limits

ATSDR (1998) has developed a chronic oral minimum risk level (MRL) of 0.2 µg/kg-day for cadmium. The chronic MRL is derived from a NOAEL (no observed adverse effect level) of 2.1 µg/kg-day from a study of cadmium accumulation in the kidneys of Japanese farmers living in an area of Japan with highly elevated cadmium levels (ATSDR, 1998). An uncertainty factor of ten was used to account for variability in the human population.

The United States Environmental Protection Agency has developed oral reference doses for cadmium for food and water. The oral reference dose for food is 1.0 µg/kg-day and for water is 0.5 µg/kg-day (US EPA IRIS, 1998 - oral R$_f$D assessment last revised 1994). The highest cadmium level in the human kidney which does not produce proteinuria (excretion of low weight molecular proteins into the urine) has been determined to be 200 µg cadmium/g of wet kidney cortex. A toxicokinetic model was used to determine the level of chronic oral exposure that would result in a cadmium kidney concentration of 200 µg cadmium/g of wet kidney cortex. The toxicokinetic model assumes that 0.01% of the body cadmium kidney burden is eliminated daily and that absorption of cadmium from food and water are 2.5% and 5% respectively. A No-Observed-Adverse-Effect Level (NOAEL) for chronic cadmium exposure was determined to be 5.0 and 10 µg/kg-day. An uncertainty factor of ten to account for human variability was applied to the NOAELs to develop the reference doses for food and water.

Joint FAO/WHO Expert Committee on Food Additives (WHO, 1993) proposed that the total daily intake of cadmium should not exceed 1 µg/kg body weight/day. This intake was designed to keep the cadmium levels in the renal cortex below 50 µg/g, and assumed an absorption rate for dietary

cadmium of 5% and a daily excretion rate of 0.005% of body burden (WHO, 1996).

Health Canada has not determined a tolerable daily intake for cadmium (Health Canada, 1996).

A2-5.3.2 Inhalation Exposure Limits

The US EPA (US EPA IRIS, 1998 - carcinogenicity assessment last revised 1992) has developed an inhalation unit risk of 1.8×10^{-3} $(\mu g/m^3)^{-1}$. This unit risk is based on lung and upper respiratory tract cancers in cadmium production workers (Thun et al., 1985). The air concentration at the 10^{-6} life-time cancer risk level (1-in-1,000,000) is 0.0006 $\mu g/m^3$.

The WHO has an annual guideline value (non-cancer) of 0.005 $\mu g/m^3$ (WHO, 2000).

Health Canada has calculated a TC_{05} of 5.1 $\mu g/m^3$. This TC_{05} is based on lung tumor incidence in an inhalation exposure bioassay with rats exposed to cadmium chloride for 18 months (Takenaka et al., 1983, Oldiges et al., 1984). This TC_{05} was amortized over the standard life-time of a rat, and converted to an equivalent concentration in humans using standard values for breathing volumes and body weights of rats and humans. It can be divided by 50,000 to obtain a 10^{-6} life-time cancer risk air concentration of 0.0001 $\mu g/m^3$.

A2-5.3.3 Selection of Exposure Limits

The estimates of the carcinogenic potencies of inhaled cadmium and non-cancer effects of ingested cadmium, developed by the US EPA will be used to assess potential human health risks associated with exposure to cadmium at this site. The selected exposure limits established by the US EPA and the health effects upon which they are based are summarized below (Table A2-11).

Table A2-11: Selected Exposure Limits for Cadmium

Route of Exposure	Exposure Limit	Toxicological Basis	Source Agency
Non-Cancer Effects			
Ingestion	1 µg/kg-day	kidney damage in humans	US EPA IRIS, 1998; WHO (JECFA), 1993
Inhalation	N/A[1]	-	-
Cancer Effects			
Ingestion	N/A[1]	-	-
Inhalation	1.8×10^{-3} $(\mu g/m^3)^{-1}$	lung cancer in cadmium workers	US EPA IRIS, 1998

1. Not Applicable.

A2-5.4 Cadmium References

ATSDR (Agency for Toxic Substances and Disease Registry). 1998. Toxicological Profile for Cadmium. U.S. Department of Health and Human Services - Public Health Service (CDROM version, 2000).

Bernard, A.M., H. Roels, A. Cardenas and R. Lauwerys. 1994. Assessment of urinary protein 1 and transferrin as early markers of cadmium nephrotoxicity. Brit. J. Ind. Med. 47:559-565.

Health Canada. 1996. Health-Based Tolerable Daily Intakes/Concentrations and Tumorigenic Doses/Concentrations for Priority Substances. ISBN 0-662-24858-9.

Järup, L., T. Alfvén, B. Persson, G. Toss, and C.G. Elinder, 1998. Cadmium may be a risk factor for osteoporosis. Occup. Environ. Med. 55:435-439.

Newhook, R., G. Long, M.E. Meek, R.G. Liteplo, P. Chan, J. Argo and W. Dormer. 1994. Cadmium and its compounds: Evaluation of the risks to health from environmental exposure in Canada. Environ. Carcino. & Ecotox. Revs., C12(2): 195-217.

Nordberg, G.F., S. Slorach, and T. Stenstrom, 1973. Cadmium poisoning caused by a cooled soft drink machine. Lakartidn. 70:601-604 (in Swedish with English summary).

Norberg, G.F., T. Kjellstrom and M.. Nordberg. 1985. Kinetics and Metabolism. In: Friberg, L., C.G. Elinder, T. Kjellstrom and G. Nordberg. Cadmium and Health: A toxicological and epidemiological appraisal. Vol. 1, Exposure, dose and metabolism. CRC Press, Inc., Boca Raton, Florida. pp. 103-178.

Oldiges, H., D. Hochrainer, S. Takenaka, G. Oberdorster and H. Konig.1984. Lung carcinimas in rats after low level cadmium inhalation. Toxicol. Environ. Chem. 9:41-51.

Staessen, J.A., H.A. Roels, D. Emelianov, T. Kuznetsova, L. Thijs, and J. Vangronsveld, 1999. Environmental exposure to cadmium, forearm bone density, and risk of fractures: Prospective population study. Lancet. 353:1140-1144.

Takenaka, S., H. Oldiges, H. Konig, D. Hochrainer and G. Oberdorster. 1983. Carcinogenicity of cadmium chloride aerosols in W rats. J. Natl. Cancer Inst. 70:367-373.

Thun, M.J., T.M. Schnorr, A.B. Smith and W.E. Halperin. 1985. Mortality among a cohort of U.S. cadmium production workers: An update. J. Natl. Cancer Inst. 74(2): 325-333.

US EPA IRIS. 1998. Cadmium. Integrated Risk Information System (IRIS). Environmental Criteria and Assessment Office, Office of Health and Environmental Assessment, Cincinnati, OH. Online at http://www.epa.gov/iris/index.html. (Accessed October 17, 2001).

US EPA. 1992. Dermal Exposure Assessment: Principles and Applications. Washington, D.C. U.S. Environmental Protection Agency. EPA/600/8-91/011B.

US EPA. 2001. Risk assessment guidance for Superfund. Volume 1: Human health evaluation manual (Part E, Supplemental guidance for dermal risk assessment). Interim. Review Draft for Public comment. EPA.

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

WHO (World Health Organization - JECFA). 1993. Evaluation of certain food additives and contaminants: Forty-first report of the Joint FAO/WHO Expert Committee on Food Additives (JECFA). WHO, Geneva (WHO Technical Report Series, No. 837).

WHO (World Health Organization). 1996. Cadmium. Guidelines for drinking-water quality, 2nd ed. Addendum to Vol. 2 Health criteria and other supporting information. WHO, Geneva. pp. 195-201.

WHO. 2000. World Health Organization, Regional Office for Europe, Copenhagen. 2000. Air Quality Guidelines for Europe, Second Edition. ISBN 92-890-1358-3.

A2-6 Toxicological Profile for Cobalt

Cobalt exists in nature as a brittle hard metal, closely resembling iron and nickel in appearance. It has two valence states (Co(II) and Co(III)), which form numerous organic and inorganic salts. It is alloyed with iron and nickel to make Alnico. Cobalt is used in Stellite alloys, and stainless steel alloys used in jet and gas turbines. Cobalt salts have been used for centuries for the production of brilliant and permanent blue colours in porcelain, glass, pottery and enamel.

Cobalt is an essential nutrient for humans as it is needed to make vitamin B_{12}. Vitamin B_{12} is a coenzyme in many biological reactions including the production of red blood cells. Cobalt has, therefore, also been used to treat anemia. As cobalt is an essential element, it is found in most body tissues with the highest concentrations occurring in the liver, kidney and bones.

A2-6.1 Pharmacokinetics

Inhaled cobalt particles accumulate in the respiratory tract depending on particle size (see section A2-9.2.1.1). From the lungs, cobalt particles either dissolve into the bloodstream or are transferred to the gastrointestinal tract by mucocilliary action and swallowing. Approximately 50% of the cobalt transferred to the gastrointestinal tract is actually absorbed and the rest is eliminated in the feces. About 50% of the portion of the initial lung burden can remain up to six months after exposure (Foster et al., 1989 as cited in ATSDR, 1992).

Cobalt consumed by the oral route of exposure is absorbed by the gastrointestinal tract. The amount of cobalt absorbed ranges from18 to 97% in humans and is dependent upon the dose and type (form) of cobalt as well as the nutritional status of the individuals involved. Cobalt absorption tends to increase in subjects which have iron deficiencies in their diet. Elimination in the feces is the primary excretion method for oral cobalt exposures.

Absorption of cobalt through intact, or unbroken skin does not generally occur (ATSDR, 1992). However, cobalt may be absorbed through broken or injured skin.

A2-6.2 Toxicology
A2-6.2.1 Non-Cancer Effects

Acute effects of exposure to cobalt-containing dust occupationally are typically inflammation of the nasopharynx. Inhalation of cobalt can affect the respiratory system and if sufficient quantities are inhaled ($3\mu g$ cobalt/m^3), irritation, wheezing, asthma and pneumonia can result. The occupational exposure levels noted here are approximately 10,000 times the typical outdoor air concentration. Individuals can also develop a sensitivity to cobalt if exposed continually in an occupational setting to concentrations of about 7 μg cobalt/m^3 and subsequent exposures can result in skin rashes or asthma attacks (ATSDR, 1992).

Oral exposure to cobalt has occurred in humans who consumed beer containing cobalt salts. In the

1960s, cobalt salts were added to beer to improve its foaming qualities. This practice has been discontinued as it led to several deaths amongst heavy beer drinkers (8 to 30 pints per day) who consumed doses ranging from 3 to 10 mg cobalt per day ("beer drinkers cardiomyopathy"). Less serious effects associated with the consumption of beer containing cobalt compounds included nausea, vomiting and diarrhea. Increased production of red blood cells also occurs in humans after oral exposure to cobalt. Decreased uptake of iodine by the thyroid gland has been observed in humans exposed to short term doses of 1000 μg cobalt/kg-day or longer term doses of 540 μg cobalt/kg-day (ATSDR, 1992).

Developmental effects were not observed in babies born to mothers who were taking medication containing cobalt to regulate anemia while pregnant (Holly, 1955 as cited in ATSDR, 1992). Reproductive effects were not observed in the people who died after exposure to high cobalt levels in beer. Some effects have been observed in animals (adverse effects on the testes and increased length of the estrous cycle). However, the significance of these effects for humans is not clear as the cobalt doses used in these studies were much higher than those to which humans are usually exposed.

Contact dermatitis has also been consistently reported as an outcome of acute dermal exposure to cobalt compounds in occupational settings.

A2-6.2.2 Cancer Effects

Hamsters exposed to cobalt oxide dust did not develop an increased incidence of lung tumours in comparison to the control population. Intramuscular injection of cobalt oxide resulted in the production of tumours in rats but not in mice (Gilman 1962 as cited in ATSDR, 1992). Based on animal data, the International Agency for Research on Cancer (1991) has classified cobalt as 2B; possibly carcinogenic for humans.

There is insufficient evidence to implicate cobalt or cobalt compounds as human carcinogens. Cobalt has not been shown to cause cancer in humans.

A2-6.2.3 Susceptible Populations

People who are already sensitized to cobalt may be unusually susceptible because subsequent cobalt exposure may trigger an asthma attack. Cobalt sensitization can be determined by cobalt-specific changes to serum antibodies (IgE and IgA).

A2-6.3 Current Exposure Limits
A2-6.3.1 Oral Exposure Limits

The recommended daily intake of cobalt as vitamin B_{12} is 2 μg/day for adults and 0.3 μg/day for children less than two years old (Health Canada, 2000).

The US EPA Region III derived an oral R/D of 20 µg/kg-day for cobalt based on cobalt intake levels in food (US EPA, 2001). This R/D was based on the upper range of average intake for children, that is below the levels of cobalt needed to induce polycythemia in both renally compromised patients. However, the current US EPA IRIS list of chemicals does not include cobalt.

A2-6.3.2 Inhalation Exposure Limits

An intermediate minimal risk level (MRL) of 0.03 µg cobalt/m^3 is proposed by the Agency for Toxic Substances and Disease Registry (ATSDR, 1992). This inhalation R/C is based on a Lowest-Observed-Adverse Effect Level (LOAEL) of 110 µg/m^3 (as cobalt sulphate) for squamous metaplasia of the larynx in rats and mice exposed for 13 weeks in the NTP (1991) and Bucher et al., (1990) studies. This dose was adjusted for intermittent exposure, converted to an equivalent concentration in humans, and a safety factor of 1000 was applied.

No regulatory dermal exposure limits for cobalt were identified in the literature reviewed for the current assessment.

A2-6.3.3 Selection of Exposure Limits

The estimates of the carcinogenic potencies of inhaled cobalt and non-cancer effects of ingested cobalt, developed by the US EPA will be used to assess potential human health risks associated with exposure to cobalt at this site. The selected exposure limits established by the US EPA and the health effects upon which they are based are summarized below (Table A2-12).

Table A2-12: Selected Exposure Limits for Cobalt

Route of Exposure	Exposure Limit	Toxicological Basis	Source Agency
Non-Cancer Effects			
Ingestion	20 µg/kg-day	effects in renally compromised patients	US EPA, 2001
Inhalation	0.03 µg/m^3	squamous metaplasia in rodent larynx	ATSDR, 1992
Cancer Effects			
Ingestion	N/A[1]	-	-
Inhalation	N/A	-	-

1. Not Applicable.

A2-6.4 Cobalt References

ATSDR (Agency for Toxic Substances and Disease Registry). 1992. Toxicological Profile for Cobalt. U.S. Department of Health and Human Services - Public Health Service (CDROM version, 2000).

Bucher, J.R., M.R. Elwell, M.B. Thompson, B.J. Chou, R. Renne, and H.A. Ragan, 1990. Inhalation toxicity studies of cobalt sulfate in F344 rats and B6C3F1 mice. Fundam. Appl. Toxicol. 15:357-372.

Foster P.P., I. Pearman, and D. Ramsden 1989. An Interspecies Comparison of the Lung Clearance of Inhaled Monodisperse Cobalt Oxide Particles Part II: Lung Clearance of Inhaled Cobalt Oxide in Man. Journal of Aerosol Science. 20:189-204.

Gilman, J.P.W. 1962. Metal carcinogenesis II. A study on the carcinogenic activity of cobalt, copper, iron and nickel compounds. Cancer Res. 22:158-162.

Health Canada. 2000. Health Canada Food Program. Departmental Consolidation of the Food and Drugs Act and of the Food and Drug Regulations with Amendments to December 20, 2000. Issued by Department of Health. Part D: Vitamins, Minerals and Amino Acids. [http://www.hc-sc.gc.ca/food-aliment/english/publications/acts_and_regulations/food_and_drugs_ acts/] (Accessed October 17, 2001).

Holly, R.G. 1955. Studies on iron and cobalt metabolism. J. Am. Med. Assoc. 158:1349-1352.

IARC (International Agency for Research on Cancer). 1991. Chlorinated drinking water; Chlorination by-products; Some other halogenated compounds; Cobalt and cobalt compounds. Monographs on the evaluation of the carcinogenic risk of chemicals to humans. 52:363-472.

NTP. 1991. National Toxicology Program - technical report series no.5. Cobalt sulfate heptahydrate (CAS No. 10026-24-1) in F344/N rats and B6C3F$_1$ mice (inhalation studies). Research Triangle Park, NC: U.S. Department of Health and Human Services, Public Health Service, National Institutes of Health. NIH publication no. 91-3124.

US EPA Region III. 2001. Risk-Based Concentration Table. US Environmental Protection Agency, Region III, Philadelphia, Penn. http://www.epa.gov/reg3hwmd/risk/riskmenu.htm. (Accessed October 18, 2001).

A2-7 Toxicological Profile for Copper

Copper is a natural element that is also an essential nutrient for the human body. It is used as a conductive agent in electrical equipment, reducing agent, catalyst, as wire material, and can be found in some pesticides.

Copper can be ingested from drinking water or eating certain foods. Another possible route of exposure includes the inhalation of roadway dust containing copper from the use of car brakes. It could also be ingested from foods that have absorbed it from copper cookware. Copper sulphate is used as a pesticide, fungicide and nutritional supplement in animal feed and fertilizer.

Copper is an essential element for humans and is found widely throughout the body. Adverse health effects can be linked to both copper deficiency as well as excessive copper levels. Copper deficiency is demonstrated by anemia, neutropenia and bone abnormalities, but is rarely observed in clinical situations. Copper is considered essential for the development of structural and enzymatic proteins. Enzymes regulating cellular respiration, free radical detoxification, iron metabolism, neurotransmitter function and synthesis of connective tissue contain copper. Regulation (activation and repression) of gene transcription also requires copper. Copper concentrations are regulated in the body by a process called homeostasis (ATSDR, 1990).

Copper regulates the mechanism which controls its intracellular homeostasis. Copper enters the liver where it is reduced and then complexes with glutathione. Metallothionein is the primary protein to which copper binds and these proteins are involved in the detoxification and binding of excess copper. Copper binds to the transcription factor which causes the production of metallothionein. When cellular copper levels are high then copper will bind to the metallothionein transcription factor causing metallothionein production, thereby detoxifying excess copper concentrations. If cellular copper levels are low, it is unlikely that there will be enough copper to bind to the metallothionein transcription factor, thereby limiting the production of metallothionein so that the copper can be used for metabolism (Gollan et al., 1973).

A2-7.1 Pharmacokinetics

No studies were found which document absorption, distribution or elimination of copper following inhalation exposure.

Absorption of copper occurs primarily through the gastrointestinal tract. Copper absorption is related to the amount of copper in the diet. For example, when adults were administered a low copper diet (780 µg copper per day), 55.6% of the administered copper was absorbed by the gastrointestinal tract as determined by the use of isotopes. For adults who were administered an adequate dose of copper in their diet (1,680 µg copper per day), 36.3% absorption was observed and for adults with a high daily copper intake (7,530 µg copper per day), only 12.4 % absorption was found. Copper absorption in adults is saturable and the percentage of copper absorbed decreases as the daily intake of copper increases. Total retention of copper increased with dietary

intake and appropriate balance was maintained even at the lowest concentration studied (780 μg copper per day). Copper absorption and metabolism decreases as a result of competition with high levels of other metals such as iron and zinc for binding sites on metallothionein. Molybdenum inhibits copper retention.

Recent studies with an isotopic tracer indicate that infants absorb sufficient copper to meet their growth needs (Ehrenkranz, 1989). Infants appear to reduce copper intake at high dietary concentrations by increasing fecal elimination and decreasing absorption.

The liver is the major organ involved in the distribution of copper throughout the body. Distribution of copper to other tissues throughout the body occurs through the blood stream. The highest concentrations of copper are found in the brain, kidney, heart, liver and pancreas. Ceruloplasmin (a protein which can bind six to eight Cu(II) atoms) and serum albumin appear to be the major carriers of copper through the bloodstream.

Bile is the major elimination pathway for liver copper as it accounts for approximately 80% of the copper leaving the liver. Pregnancy is associated with increased copper retention likely due to decreased biliary excretion resulting from the hormonal changes which typically occur. Urinary excretion and sweating are minor contributors to copper removal.

The use of topical medications containing copper compounds can increase dermal absorption of copper (Eldad et al., 1995). Components of topical medication such as salicylic acid or phenylbutazone facilitate the transport of copper through the skin.

A2-7.2 Toxicology
A2-7.2.1 Non-Cancer Effects

Inhalation exposure information is limited to studies on factory workers who have been exposed to significantly higher copper air concentrations than the general public. Copper dust is considered a respiratory irritant as factory workers experienced irritation of the mucosal membranes of the mouth, nose and eyes. Metal fume fever has also been observed in workers exposed to high concentrations of fine copper dust in air. Gastrointestinal effects such as nausea, anorexia and occasionally diarrhea were also experienced by factory workers and it is thought that the gastrointestinal effects are primarily due to swallowing a portion of the airborne copper (i.e., would be classified as an oral exposure).

Copper is rarely toxic unless very large amounts are ingested. The available toxicity data associated with oral consumption of copper are limited to ingestion of water with very high copper concentrations or suicide attempts involving copper sulphate. Chronic exposure to drinking water containing approximately 60 μg copper/kg-day (equals 4,200 μg copper/day for a 70 kg adult), resulted in nausea, vomiting and abdominal pain shortly after consumption of the water. The gastrointestinal difficulties stopped after an alternate water supply was found for the affected persons.

Chronic copper poisoning is very rare, since the capacity for healthy human livers to excrete copper is considerable. Any reports of chronic copper poisoning that do exist involve patients with liver disease.

Developmental effects have not been observed in children of mothers with Wilson's Disease (a metabolic disorder which causes accumulation of copper in the liver) or healthy humans. Developmental toxicity has been found in mice, mink and hamsters who were fed a high copper diet or were injected with copper. Reproductive effects have not been observed in human populations exposed to high copper levels. Copper containing intrauterine devices are used as a method of birth control and animal studies have shown that the copper wires contained within these devices are the contraceptive agent.

Dermal exposure to copper can result in allergic contact dermatitis.

A2-7.2.2 Cancer Effects

The United States Environmental Protection Agency has classified copper and copper compounds in Group D which indicates that they are substances for which inadequate data are available to make a carcinogenicity assessment. Specifically, for copper and copper compounds there are no human carcinogenicity data, animal bioassay data is inadequate and mutagenicity tests are equivocal (US EPA IRIS, 1998 - carcinogenicity assessment last revised 1991).

A2-7.2.3 Susceptible Populations

Infants and children under one year old are unusually susceptible to copper toxicity because they have not developed the homeostatic mechanism to remove copper from the body. Wilson's Disease is a genetic disorder associated with impaired transport of copper from the liver to the bile, thereby resulting in increased copper concentrations in the liver as they are not able to maintain homeostasis. Another genetic condition which increases the susceptibility to copper toxicity is a deficiency in the enzyme glucose-6-phosphate dehydrogenase. Individuals with liver disease are also susceptible to copper toxicity because of the critical role the liver plays in eliminating copper from the body.

A2-7.3 Current Exposure Limits
A2-7.3.1 Oral Exposure Limits

As copper is considered an essential element for humans there are two types of exposure limits that are considered: (a) the minimal daily intake so that a person will not suffer from copper deficiency; and (b) the maximal permissible daily intakes so that a person will not suffer from copper toxicity.

The World Health Organization (WHO, 1998) has determined the minimal daily copper intake for adults to be 20 µg copper/kg-day which is equivalent to 1,400 µg copper per day for the average

70 kg adult. For children, the World Health Organization concluded that the minimal daily copper intake should be 50 µg copper/kg-day (equivalent to 750 µg copper per day for a 15 kg child). The minimal daily copper intake was determined as the amount of copper needed for a child or adult to function properly while accounting for variables such as differences in copper absorption, retention and storage.

The Recommended Dietary Allowance (RDA) for US adults is 900 µg copper/day or about 13 µg/kg-day (IOM, 2001). This RDA is a combination of indicators, including plasma copper and ceruloplasmin concentrations, erythrocyte superoxide dismutase activity and platelet copper concentration in controlled human depletion/repletion studies.

The US Reference Daily Intake (a term which replaces "US RDA") for copper is 2,000 µg/day or about 30 µg/kg/day for adults (US FDA, 1999).

The tolerable upper intake level for US adults is 10,000 µg/day or about 140 µg/kg/day, and is based on protection from liver damage (IOM, 2001).

A2-7.3.2 Inhalation Exposure Limits

A chronic non-cancer Reference Exposure Level (REL) of 2.4 µg/m^3 is listed for copper compounds in the California Air Pollution Control Officers Association Air Toxics "Hot Spots" Program, Revised 1992 Risk Assessment Guidelines. This REL is based on respiratory effects (CAPCOA, 1993, CARB, 1998). The United States Environmental Protection Agency (US EPA) has not established a Reference Concentration (R$_f$C) for copper compounds (US EPA IRIS, 1998 - inhalation R$_f$C assessment last revised 1991).

A2-7.3.3 Selection of Exposure Limits

The estimates of the carcinogenic potencies of inhaled copper and non-cancer effects of ingested copper, developed by the Institute of Medicine (IOM) and the California Air Resource Board (CARB) will be used to assess potential human health risks associated with exposure to copper at this site. The selected exposure limits and the health effects upon which they are based are summarized below (Table A2-13).

Table A2-13: Selected Exposure Limits for Copper

Route of Exposure	Exposure Limit	Toxicological Basis	Source Agency
Non-Cancer Effects			
Ingestion	140 µg/kg-day	liver damage	IOM, 2001
Inhalation	2.4 µg/m^3	chronic reference exposure limit - respiratory	CAPCOA, 1993, CARB, 1998
Cancer Effects			
Ingestion	N/A[1]	-	-
Inhalation	N/A	-	-

1. Not Applicable

A2-7.4 Copper References

ATSDR (Agency for Toxic Substances and Disease Registry). 1990. Toxicological Profile for Copper. U.S. Department of Health and Human Services - Public Health Service (CDROM version, 2000).

California Air Pollution Control Officers Association (CAPCOA). 1993. Air Toxics "Hot Spots" Program: Revised 1992 Risk Assessment Guidelines. Prepared by CAPCOA, the Office of Environmental Health Hazard Assessment and the California Air Resources Board. October, 1993.

CARB (California Air Resources Board). 1998. List of Toxic Air Contaminants - Compound Summary Table. http://www.arb.ca.gov/toxics/tac/factshts/copperco.pdf.

Ehrenkranz, R.A. 1989. Mineral needs of the very-low-birth weight infant. Semin. Perinatol. 13:142-159.

Eldad, A., M. Wisoki, H. Cohen, S. Breiterman, M. Chaouat, M.R. Wexler, H. Ben-Bassat, 1995. Phosphorous burns: Evaluation of various modalities for primary treatment. J. Burn Care Rehabil. 16:49-55.

Gollan, J.L., D.J. Deller, 973. Studies on the nature and excretion of biliary copper in man. Clinical Science. 44:9-15.

IOM (Institute of Medicine - Food and Nutrition Board). 2001. Dietary Reference Intakes for Vitamin A, Vitamin K, Arsenic, Boron, Chromium, Copper, Iodine, Iron, Manganese, Molybdenum, Nickel, Silicon, Vanadium, and Zinc. National Academy Press, Washington, D.C.

US EPA IRIS. 1998. Copper. Integrated Risk Information System (IRIS). Environmental Criteria and Assessment Office, Office of Health and Environmental Assessment, Cincinnati, OH.Online at http://www.epa.gov/iris/index.html. (Accessed October 17, 2001).

US FDA. 1999. U.S. Food and Drug Administration, Department of Health and Human Services. 21CFR104.20. Code of Federal Regulations, Title 21: Food and Drugs, Part 104: Nutritional Quality Guidelines for Foods, Subpart B: Fortification Policy. Online at http://vm.cfsan.fda.gov/~lrd/CF104-20.HTML. (Accessed October 18, 2001).

WHO (World Health Organization). 1998. Copper. Guidelines for drinking-water quality, 2nd ed. Addendum to Vol. 2 Health criteria and other supporting information. WHO, Geneva. pp. 31-46.

A2-8 Toxicological Profile for Lead

Lead is a bluish-white lustrous metal. It is very soft, highly malleable, ductile and a relatively poor conductor of electricity. It is very resistant to corrosion but tarnishes upon exposure to air. Lead pipes bearing the insignia of Roman emperors, used as drains from the baths, are still in service. Alloys include pewter and solder. Tetraethyl lead was used in some grades of petrol (gasoline).

A2-8.1 Pharmacokinetics

The absorption, distribution, metabolism, and elimination of lead has been extensively studied in both animals and humans. Available data can be used to quantify the uptake and disposition of lead in the human body for various populations of children and adults. Lead absorption is influenced by the route of exposure, chemical speciation, the physicochemical characteristics of the lead and exposure medium, and the age and physiological states of the exposed individual (e.g., fasting, nutritional calcium and iron status). There is a significant body of data available in the scientific literature evaluating lead absorption from soil (summarized in NEPI, 2000).

The primary sites for inorganic lead absorption are the gastrointestinal and respiratory tracts. The bioavailability of ingested soluble lead in adults may vary from less than 10% when ingested with a meal to 60 – 80% when ingested after a fast. Immediately following absorption, lead is widely distributed to blood plasma and soft tissues, then it redistributes and accumulates in bone (ATSDR, 1993).

Bone lead accounts for approximately 73% of the total body burden in children, increasing to 94% in adults due to changes in bone turnover rates with age. Transplacental transfer of lead has been demonstrated based on measurements of lead in umbilical cord blood in humans, as well as tissue concentrations in offspring of mice.

Lead that is not retained in the body is excreted principally by the kidney as salts or through biliary clearance into the gastrointestinal tract in the form of organometallic conjugates. Excretion rates measured in infants, children, and adults are highly variable, although available data suggest that the fraction of absorbed lead that is retained in humans decreases with age (ATSDR, 1993).

Dermal absorption of lead compounds is less significant than either oral or inhalation routes of exposure (ATSDR, 1993). Information on the dermal absorption of lead containing compounds is limited to a single study, which applied a lotion containing lead acetate to the forearms of male volunteers and reported a dermal absorption rate of approximately 0.06% over a 12 hour period (ATSDR, 1993).

The toxicokinetics of lead seem to be affected by the polymorphism of the enzyme delta-aminolevulinic acid dehydratase (ALAD) (Smith et al., 1995). ALAD is a polymorphic enzyme with two common alleles, ALAD*1 and ALAD*2 (Petrucci et al., 1982). Gerhardsson et al., (1999) investigated the mobilization of lead by use of the chelating agent

2-3-meso-dimercaptosuccinic acid in male lead smelter workers. Two workers with the ALAD*2 allele seemed to mobilize less chelatable lead in relation to their blood, plasma, and urine concentrations of lead than the other workers. Smith et al., (1995) suggest that ALAD*2 in circulating red blood cells bind lead more avidly than ALAD*1, thus increasing the blood lead level, but at the same time decreasing the amount of lead delivered to soft tissues.

A2-8.2 Toxicology
A2-8.2.1 Non-Cancer Effects

The potential for lead to impair neurobehavioural development in children is the subject of much concern. Acute inhalation and oral exposures to lead often results in central nervous system effects including; dullness, restlessness, irritability, poor attention span, headaches, muscle tremors, hallucination and loss of memory (Health Canada, 1992). Encephalopathy has been reported at very high lead exposure levels (100 µg lead/deciliter of blood in adults and 80 µg/dL in children) (Health Canada, 1992).

Chronic exposure to elevated levels of lead can result in a number of nervous system effects. Tiredness, sleeplessness, irritability, headaches, joint pain and gastrointestinal symptoms have all been reported (Health Canada, 1992). In adults, these effects are seen at blood lead levels of 50 - 80 µg/dL. Occupationally exposed persons have been found to suffer from muscle weakness, mood disruptions, and peripheral neuropathy when blood lead levels reached 40 - 60 µg/dL. At levels of 30 - 50 µg/dL, significant reductions in nerve conductive velocities were also reported (Health Canada, 1992). Renal disease has also been reported, but nephropathy has not been detected in adults or children whose blood lead levels were below 40 µg/dL (Health Canada, 1992).

There is substantial human evidence in both adults and children which demonstrates that both the central and peripheral nervous system are the primary targets of lead toxicity. Sub-encephalopathy, neurological and behavioral effects in adults and electrophysiological evidence of nervous system damage in children have been reported at blood lead levels as low as 30 µg/dL (Health Canada, 1992). A number of epidemiological studies have examined the effects of lead exposure in young children. The studies were able to demonstrate no clear threshold below which the detrimental effects of lead on child neurological development does not occur (Health Canada, 1992).

Epidemiological studies have indicated that non-cancer neurological effects may occur at very low exposure levels. Therefore, an exposure level based on these effects will provide against the possible carcinogenic effects of lead. Health Canada (1996) recommended a provisional tolerable daily intake (PTDI) for lead of 3.57 g/kg-day. This value was based on technical reports from annual meetings of the Joint FAO/WHO Expert Committee on Food Additives (WHO/ JECFA, 1993), and epidemiological studies associating lead exposure with neurological effects in infants and children. The WHO value was established to prevent increases in blood lead levels in children. Studies with young children have shown that daily exposures to lead in the 3 - 4 µg/kg-

day range do not alter the blood lead level in the children. Intakes at or above 5 µg/kg-day resulted in significant increases in blood lead levels (WHO / JECFA, 1993).

A2-8.2.2 Cancer Effects

Epidemiological studies of occupationally exposed adults were not able to demonstrate an increase in cancers among an exposed cohort compared to control. The International Agency for Research on Cancer (IARC, 1987), considers the overall evidence of lead carcinogenicity in humans to be inadequate. Animal studies have reported renal tumors in rats exposed to 1,000 µg lead salts/g in the diet. While exposures to lead acetate, subacetate and phosphate salts produced renal tumors in rats, equivalent exposures to other lead salts did not result in the production of renal tumors (Health Canada, 1992). Health Canada has classified lead as a Group IIIB (possibly carcinogenic to humans) compound based on a lack of adequate human data and limited evidence of carcinogenicity in animals.

The US EPA (US EPA IRIS, 1998 - carcinogenicity assessment last revised 1993) has classified lead as a probable human carcinogen based on sufficient animal evidence. However, the Carcinogen Assessment Group (US EPA IRIS, 1998) did not recommend derivation of a quantitative estimate of oral carcinogenic risk, due to a lack of understanding pertaining to the toxicological and pharmacokinetic characteristics of lead. In addition, the neurobehavioural effects of lead in children were considered to be the most relevant endpoint in determining an exposure limit.

Health Canada (1992) classified lead in Group IIIB (possibly carcinogenic to humans) by ingestion. Lead and inorganic lead compounds have been placed in Group 2B (possible human carcinogen) by ingestion under IARC (1987).

A2-8.2.3 Susceptible Populations

There is a very large database which documents the effects of acute and chronic lead exposure in adults and children. Extensive summaries of the human health effects of lead are available from a number of sources including Health Canada, the US EPA IRIS database and the ATSDR. These reviews show that infants, young children up to the age of six and pregnant women (developing fetuses) are the most susceptible (Health Canada, 1992).

A2-8.3 Current Exposure Limits
A2-8.3.1 Oral and Inhalation Exposure Limits

Health Canada (1996) recommended a provisional tolerable daily intake (PTDI) for lead of 3.57 µg/kg-day. This value was based on technical reports from annual meetings of the Joint FAO/WHO Expert Committee on Food Additives (JECFA) (WHO, 1993 - as cited in Health Canada, 1996), and epidemiological studies associating lead exposure with neurological effects in infants and children.

Based on a critical blood lead level of 100 µg/L for cognitive deficit, hearing impairment and disturbed vitamin D metabolism in children, the annual average air lead level should not exceed 0.5 µg/m^3 (WHO, 2000).

The Ontario Ministry of Environment and Energy recommended an IOC$_{pop}$ (intake of concern for populations) of 1.85 µg/kg-day which incorporated the population-based significance of the health effects and attempted to minimize the predicted number of children with individual blood lead levels of concern (MOEE, 1994). Sub-clinical neurobehavioural and developmental effects were the critical effects appearing at the lowest levels of exposure (MOEE, 1994). The IOC$_{pop}$ was based on an LOAEL (lowest observed adverse effects level) in infants and young children of 10 µg/dL, converted to an intake, with an applied uncertainty factor of two for the use of an LOAEL (MOEE, 1994).

A2-8.3.2 Selection of Exposure Limits

See discussion in Section 5.8 (Main document - Part B).

A2-8.4 Lead References

ATSDR. 1993. Agency for Toxic Substances and Disease Registry, Toxicological Profile for Lead.

Gerhardsson, L., J. Börjesson, S. Mattsson, A. Schütz, and S. Skerfving, 1999. Chelated lead in relation to lead in bone and ALAD genotype. Environ. Research Section A. 80:389-398.

Health Canada. 1992. Guidelines for Canadian Drinking Water Quality - Supporting Document for Lead.

IARC (International Agency for Research on Cancer). 1987. Monographs on the evaluation of the carcinogenic risk of chemicals to humans. 7:230-232.

MOEE. 1994. Scientific Criteria Document for Multimedia Environmental Standards Development - Lead. pp. 332. ISBN 0-7778-2529-5.

NEPI. 2000. Assessing the Bioavailability of Metals in Soil for Use in Human Health Risk Assessments. Bioavailability Policy Report Phase II: Metals Task Force Report. National Environmental Policy Institute (NEPI). Summer 2000.

Petrucci, R., A. Leonardi, and G. Battistuzzi, 1982. The genetic polymorphism of delta-aminolevulinate dehydratase in Italy. Hum. Genet. 60:289-290.

Smith, C.M., X. Wang, H. Hu, and K.T. Kelsey, 1995. A polymorphism in the delta-aminolevulinic acid dehydratase gene may modify the pharmacokinetics and toxicity of lead. Environ. Health Perspect. 103:248-253.

US EPA. 1996. Urban Soil Lead Abatement Demonstration Project Volume 1: 600/p93/001aF.

US EPA IRIS. 1998. Lead and compounds (inorganic). Integrated Risk Information System (IRIS). Environmental Criteria and Assessment Office, Office of Health and Environmental Assessment, Cincinnati, OH. Online at http://www.epa.gov/iris/index.html. (Accessed October 17, 2001).

WHO (World Health Organization - JECFA). 1993. Evaluation of certain food additives and contaminants: Forty-first report of the Joint FAO/WHO Expert Committee on Food Additives (JECFA). WHO, Geneva (WHO Technical Report Series, No. 837).

WHO (World Health Organization) 2000. WHO Regional Office for Europe, Copenhagen. Air Quality Guidelines for Europe, Second Edition. ISBN 92-890-1358-3.

A2-9 Toxicological Profile for Nickel

Pure nickel is a hard, silvery-white metal, which has properties that make it very desirable for combining with other metals to form mixtures called alloys. Some of the metals that nickel can be alloyed with are iron, copper, chromium and zinc. These alloys are used in making metal coins and jewelry and in industry for making items such as valves and heat exchangers. Most nickel is used to make stainless steel. Compounds of nickel combined with many other elements, including chlorine, sulfur and oxygen exist. Many of these compounds dissolve fairly easily in water and have a characteristic green color. Nickel and its compounds have no characteristic odor or taste. Nickel compounds are used for nickel plating, to color ceramics, to make some batteries, and as substances known as catalysts that increase the rate of chemical reactions (ATSDR, 1997).

The physiological role of nickel in animals and humans has not yet been determined. It is believed, based on plants and microorganisms, that nickel is involved as a cofactor in metalloenzymes/proteins or as a cofactor which facilitates iron absorption in the intestine (Nielsen, 1985). Nickel may also affect endocrine function regulating prolactin levels. Nickel deficiency has not been observed in humans, but has been induced in animals, indicating that nickel is an essential element for animals (Schnegg and Kirchgessner, 1975).

An important issue relating to nickel toxicity is its speciation. Its form (metallic, salt, oxide, etc.) and solubility strongly influence its toxicology. The solubility (in water) of different nickel compounds ranges from the highly soluble nickel salts (nickel chloride - 642 g/L, nickel sulphate - 293 g/L) down to the insoluble nickel oxide (1.1 mg/L) and the sparingly soluble nickel subsulphide (517 mg/L) (ATSDR, 1997). Some of the more insoluble nickel compounds (i.e., nickel oxide, nickel subsulfide) may have higher solubilities in biological fluids (Yamada et al., 1993; Ishimatsu et al., 1995; Oller et al., 1997). The predominant nickel species in Rodney Street soils is the relatively insoluble nickel oxide (Results section of Part A).

The toxicity of nickel can be classified into four separate categories: (1) non-cancer respiratory and other disorders, due to the inhalation or ingestion of nickel compounds; (2) cancer, due to inhalation of nickel compounds; (3) allergy, a hypersensitivity to nickel manifested by contact dermatitis and asthma; and (4) iatrogenic poisoning which may have occurred in the past in patients undergoing hemodialysis, corrosion of stainless steel prostheses, and nickel-contaminated medication, or medication such as disulfiram that caused increased nickel concentration in the blood (not discussed).

A2-9.1 Pharmacokinetics
A2-9.1.1 Inhalation Exposure

Following inhalation exposure, nickel may deposit in the lungs depending on the size of the particle inhaled. Larger particles (5 - 30 μm) tend to accumulate in the upper respiratory tract while smaller particles are deposited in the lower respiratory system (see section A2-9.2.1.1). Absorption of nickel compounds deposited in the lung into the blood stream depends upon their

form and solubility. Soluble nickel compounds such as nickel chloride and nickel sulphate are absorbed readily (up to 100%) from the respiratory tract, while almost none of the less soluble nickel compounds such as nickel oxide and nickel subsulphide (as demonstrated by urinary nickel levels in exposed workers (ATSDR, 1997)) are absorbed. Inhaled nickel that is absorbed is excreted through the urine. Studies conducted on nickel workers show that nickel urinary excretion increased towards the end of the shift and also towards the end of the work week, indicating that one fraction is removed quickly, but that there was also a fraction which was removed more slowly (Ghezzi et al., 1989; Tola et al., 1979; as cited in TERA, 1999).

No reliable estimates are, however, found in the literature for retention and uptake of nickel from solely nickel oxide inhalation exposure in humans. Recently, Yu et al., (2001) have developed a dosimetry model for inhaled nickel compounds using occupational exposure data and information from studies of rats inhaling nickel.

Occupational exposure to nickel results in higher nickel lung burdens than the general population. Workers exposed to insoluble forms of nickel (such as nickel oxide and nickel sulphide) have higher nickel levels in the nasal mucosa than those workers exposed to more soluble forms of nickel (this may be related to larger inhalable dust particles being trapped in the upper respiratory tract). Less soluble nickel compounds, therefore, appear to remain in the nasal passage following inhalation exposure. Serum nickel levels are higher in workers exposed to soluble nickel compounds in comparison to those exposed to insoluble nickel compounds (Torjussen and Andersen, 1979, as cited in ATSDR, 1997). Nickel sensitized individuals had similar nickel levels in blood, urine and hair relative to non-sensitive individuals (Spruit and Bongaarts, 1977, as cited in ATSDR, 1997).

Pulmonary exposure to green nickel oxide in rats resulted in nickel excretion in the feces, but not in the urine, indicating that the primary removal mechanism of nickel oxide involved clearance from the lungs rather than by dissolution-absorption processes (Benson et al., 1994 as cited in ATSDR, 1997). The observed excretion could also reflect mucociliary clearance (being brought up in mucus and then being swallowed), in addition to macrophage clearance. Benson et al., (1994) also found that nickel subsulphide is cleared relatively rapidly (half life of four days) from the lungs of rats. They concluded that nickel subsulphide is relatively insoluble in water, but dissolves rapidly in lung fluid.

A2-9.1.2 Oral Exposure

Studies examining the absorption of nickel by humans found that nickel sulphate was up to 40 times more bioavailable if administered in water than in food (Sunderman et al., 1989). The bioavailability of nickel also increased when administered in a soft drink, but not when given in milk, coffee, tea or orange juice (Solomons et al., 1982). Serum nickel levels were found to be elevated in subjects who had fasted prior to the administration of nickel in drinking water, but this was not the case for those who were administered nickel in food. Food tends to decrease the bioavailability of nickel. Some nickel sensitive individuals were found to have decreasing nickel

serum concentrations and increasing nickel urinary concentrations with increased administered nickel concentrations (Santucci et al., 1994). This may be an indication that some nickel sensitive individuals can decrease nickel absorption in response to increased nickel intake. In non-occupationally exposed people, nickel concentrations tend to be highest in lungs, thyroid and adrenal glands, kidney, heart and liver (Rezuke et al., 1987, as cited in ATSDR, 1997). The total amount of nickel estimated to be present in the human body is about six mg for a 70 kg adult (Sumino et al., 1975, as cited in ATSDR, 1997).

Quantitative absorption data for unspecified forms of soluble nickel are as follows: 1 - 27% of ingested nickel is absorbed (depending on whether food is consumed); approximately 1 - 6% of nickel administered with food or during a meal is absorbed; 12 - 27% of nickel absorbed after a fast (data from Diamond et al., 1998, as cited in *TERA*, 1999).

Bioavailability and bioaccessibility of nickel is further discussed in Appendix 5.

Nickel metabolism occurs via a series of nickel exchange reactions (Sarkar, 1984, as cited in ATSDR, 1997). In human blood, nickel binds to a blood protein called albumin. Nickel competes with copper for a binding site on the albumin (Hendel and Sunderman, 1972, as cited in ATSDR, 1997). Nickel is then transferred from the albumin to L-histidine, an amino acid. The nickel-histidine complex has a low molecular weight and can easily cross biological membranes (Sarkar, 1984, as cited in ATSDR, 1997). Nickel is also tightly bound to a nickeloplasmin human blood which is not available for exchange and hence not transported across biological membranes (Sunderman, 1986, as cited in ATSDR, 1997).

Most ingested nickel is excreted via feces, although the nickel absorbed by the gastrointestinal tract is excreted in the urine. In comparison, studies of nickel doses administered with food or water, 26% of the dose given in water was eliminated in the urine and 76% in the feces by the fourth day following administration (Sunderman et al., 1989). In contrast, 2% of the nickel dose administered in food was eliminated in the urine and 102% was eliminated in the feces during the same time period. Nickel can also be eliminated through hair, sweat, milk and skin.

No reliable estimates are, however, found in the literature for retention and uptake of nickel from nickel oxide ingestion exposure.

A2-9.1.3 Dermal Exposure

Studies of the dermal uptake of nickel in humans have been summarized in Appendix 7.

A2-9.2 Toxicology
A2-9.2.1 Inhalation Exposure

In two year inhalation studies of nickel oxide, nickel subsulfide, and nickel sulfate in rats and mice, respiratory lesions observed for all compounds included increased lung weights, focal

alveolar/bronchiolar hyperplasia, inflammation and/or fibrosis of the lung, and lymphoid hyperplasia of the lung-associated lymph nodes (Dunnick et al., 1995; NTP 1996a, 1996b, 1996c). The investigators noted that qualitatively the inflammatory responses in the lungs were similar with all three compounds, however, the effects were more severe after exposure to nickel oxide and nickel subsulfide. In addition to lung effects, atrophy of the olfactory epithelium was also observed after exposure to nickel sulfate. Compared to rats, mice were more resistant to the development of lung lesions following nickel exposure.

The only data available for chronic nickel inhalation exposure for humans is limited to occupational data. One of the limitations associated with the epidemiological data available is that the workers were exposed to several different forms of nickel as well as other metals and irritant gases at the same time. As a result, the observed effects can not be attributed to a particular type of nickel. Other lifestyle factors, such as smoking, which affect disease outcomes, are also not always accounted for, thereby limiting the conclusions that can be drawn. Cancer effects related to inhalation exposure are further discussed in section A2-9.2.3.

One death has been reported as the result of exposure to very high metallic nickel concentrations (382 mg/m^3) of a small particle size (Sunderman, 1993, as cited in ATSDR, 1997). Workers who were chronically exposed to nickel oxide or metallic nickel at concentrations greater than 0.04 mg/m^3 had a greater incidence of death from respiratory disease (Cornell and Landis, 1984; Polednak, 1981, as cited in ATSDR, 1997). Other respiratory effects found included chronic bronchitis, emphysema, and reduced vital capacity. These workers were also exposed to other metals, so it can not be concluded that nickel is the sole causative agent of the effects observed. Asthma from primary irritation and as the result of dermal sensitization has also been documented among nickel workers (Dolovich et al., 1984; Novey et al., 1983; Shirakawa et al., 1990; as cited in ATSDR, 1997). Increased incidence of cardiovascular related deaths has not been found in nickel workers.

Nickel refinery workers with elevated urinary nickel concentrations also showed a significant increase in urinary β_2-microglobulin levels, which is indicative of tubular dysfunction in the kidneys (ATSDR, 1997). However, marked differences are seen between the results using single urine samples ("spot samples"), and sampling conducted over a 24 hour period *(TERA*, 1999). Although male and female workers were exposed to the same average nickel (nickel chloride and nickel sulphide) air concentrations, the women had twice the nickel urinary concentrations of the men (Sunderman and Horak, 1981, as cited in ATSDR, 1997). A study of nickel production workers has found significant increases in levels of immunoglobulin G (IgG), IgA, and IgM as well as a significant decrease in IgE. Serum proteins involved in cell-mediated immunity also increased, suggesting stimulation of the immune system by nickel (Bencko et al., 1983; 1986; as cited in ATSDR, 1997). The *TERA* (1999) report concluded that "the overall epidemiological database regarding potential kidney effects of inhalation exposure to soluble nickel is weak. However, the available data do provide suggestive evidence that the kidney can be affected under exposure conditions below those causing acute toxicity."

Studies show that pregnant female workers at a nickel refining plant in the Kola region in Russia had a 15.9% increase in spontaneous abortions in comparison with a control population of pregnant female construction workers (who were not occupationally exposed to nickel) who had a spontaneous abortion rate of 8.5% (Chashschin et al., 1994). The Russian metal refinery workers were exposed to nickel sulphate concentrations of approximately 0.08 to 0.196 mg nickel/m^3 and corresponding urinary nickel concentrations were 3.2 to 22.6 µg/L. Nickel urinary concentrations in persons not occupationally exposed ranged from <0.1 to 13.3 µg/L. Heavy lifting and heat stress are also associated with nickel refining. A preliminary study of pregnant Russian nickel refinery workers also indicated that babies born to these women had a 16.9% increase in development effects (primarily cardiovascular and musculoskeletal defects), relative to the children of construction workers who had a 5.8% increase in developmental effects. It is not clear whether the fact that the Russian workers also were exposed to heavy lifting and heat stress contributed to the observed abortions. No indications of fetal toxicity (birth weight of first child) in the general population in nickel smelter cities in the Kola region in Russia (Nikel and Zapoljarnij) were found in a large comparative study of pollution and health in the Norwegian-Russian border area. Further studies are in progress (Smith-Sivertsen et al., 1997; Odland, 1999).

A significant increase of gaps in the chromosomes were found in white blood cells of nickel workers who were exposed to nickel monosulphide and nickel subsulphide. Breakage or exchange of the chromosomes was not observed. The study did not find any correlation between the incidence of the chromosome gaps, blood nickel concentration, duration of nickel exposure, or age of workers (Waksvik and Boysen, 1982, as cited in ATSDR, 1997).

It should be noted that the effects discussed in these paragraphs require absorption from the lungs and systemic distribution of nickel, which would be unlikely to occur following inhalation exposure to nickel oxide alone.

A2-9.2.1.1 An Examination of Nickel on Airborne Particulates in Occupational and Environmental Settings

In order to characterize the potential risk of lung cancer due to airborne nickel in ambient air, a number of the exposure parameters and assumptions relating to the development of the unit risk factor and the characteristics of the nickel in the ambient air being inhaled need to be examined. Airborne nickel compounds are typically associated with suspended particles. The potential risk of inhaling particles is determined by several parameters including:

particle size characteristics;

chemical composition or speciation; and

mass concentration.

In addition, the type of breathing patterns and duration of exposure of the receptor will influence

the intake or dose of nickel to the lungs (Casarett and Doull, 1991; Snipes et al., 1997; Health Canada, 1999; Yu et al., 2001).

The unit risk factors available from US EPA (1986), WHO (2000) and Health Canada (1996) to characterize lung cancer risk are all based on extrapolations from lung cancer incidence in nickel refinery workers and various assumptions and estimates of airborne nickel in the workplace of these workers. Actual measurements of the airborne nickel in these workplaces during the period when the workers received the exposure resulting in lung cancer are limited because this period predates the use of appropriate technology to monitor workplace hygiene and exposures.

A2-9.2.1.2 Particle Size Characteristics and Air Sampler Design

Information about particle size characteristics is important for understanding how suspended particles behave in the human respiratory tract and predicting lung exposures. Information on the design of air sampling devices used in the workplace and the environment can be used to evaluate and compare inhalation estimates from different sources and evaluate the relevance of estimates based on previous definitions of inhalable and respirable fractions and historical methods of air sampling. For example, much workplace air sampling is based on personal air samplers which are worn on the worker close to the breathing zone. Stationary monitoring of particles in a particular work area is not considered representative of the inhalation exposure of a worker moving about the workplace and is only measured sporadically if at all. By contrast, environmental monitoring mainly involves air sampling at fixed sites, usually related to known emission sources. The use of personal air samplers to monitor environmental air levels as people move around during the day has been implemented for short periods on small populations in Ontario at specific locations (Hamilton (HAQI, 1997), Windsor (MOEE, 1994)).

The behavior of suspended particles in the human respiratory tract is well studied in both occupational and environmental situations (Casarett and Doull, 1991; Snipes et al., 1997; Health Canada 1999; Yu et al., 2001). The size and aerodynamic properties of suspended particles influences how far a particle penetrates the respiratory tract, where it deposits, and its retention behavior. The human respiratory tract can be divided into the nasopharyngeal region (nose, mouth and throat portion), the tracheobronchial region where the windpipe, and the major conducting airways of the lung connect with the pulmonary region of the lung where gas exchange takes place (here air passages are tiny and end in little air sacs or alveoli). Only part of the total quantity of suspended particles present in the breathing zone of the receptor is inhaled. Particles with an aerodynamic diameter of 5 - 30 µm are largely deposited in the nose, mouth and throat, the majority of particles with aerodynamic diameters of 1 - 5 µm are inhaled past this region and are deposited in the tracheobronchial region and particles < 0.5 µm are inhaled (or respired) right into the alveoli in the deepest parts of the lung (Casarett and Doull, 1991). The patterns of deposition of different sized particles reflect the combination of particle size, narrowing air passages, reduced air velocity and changes in airflow direction as particles penetrate the different regions of the respiratory tract (Snipes et al., 1997; Yu et al., 2001). An important factor that can influence this pattern of deposition is the pattern of breathing. During quiet breathing, most breathing occurs

through the nose (nasal breathing) and the inflation of the lung is small. Consequently, the nasal passages scavenge larger particles and a large portion of the inhaled particles are exhaled. During exercise, larger and larger volumes of air are inhaled and at some stage, the person switches over to mainly mouth breathing and particles can penetrate deeper into the lower regions of the lungs.

Suspended particles can be classified into various fractions depending on particle size and degree of penetration into the respiratory tract. In the workplace, air sampling is mainly indoors and personal, i.e., the air sampler is attached to the worker. For particle monitoring in the workplace, there is a recently accepted international definition of health-related sampling criteria based on progressively finer sized particle fractions: inhalable, thoracic or respirable (ACGIH, 1998). The inhalable fraction represents particles that enter the mouth and nose (the particle size (aerodynamic diameter) has a 50% cut-point or particle size corresponding to 50% sampling efficiency of 100 μm). The thoracic fraction represents inhalable particles that pass into the trachea and the larger upper airways of the lung (the 50% cut-point is 10 μm). The respirable fraction is the portion of inhalable particles that enter the deepest part of the lung (the 50% cut-point is 4 μm). These new definitions of sampling criteria are being used to implement new designs for air monitoring sampling devices for the workplace, and, to derive new workplace exposure criteria based on these new sampling criteria. For example, ACGIH adopted new TLVs (threshold limit values) for nickel compounds in 1998 based on conversion factors for the relative efficiencies of specific air samplers. This resulted in the old TLV for metallic nickel, insoluble nickel and nickel subsulphide changing from 1 mg/m^3 to 1.5 mg/m^3 for metallic nickel, 0.2 mg/m^3 for insoluble nickel and 0.1 mg/m^3 for nickel subsulphide, respectively. The TLVs for soluble nickel and nickel carbonyl were unchanged.

The older "inspirable" or "total" fraction had a 50% cut-point of less than 100 μm and the "respirable" fraction had a 50% cut-point range of 3.5 to 5 μm. Recent studies show that the new "inhalable" personal monitors tend to collect about twice the particle mass collected by the old "total" or "inspirable" personal air samplers (Werner et al., 1999).

By contrast, outdoor ambient air monitoring uses larger air pumping devices (hi-vol and lo-vol) and larger area filters than personal samplers and are subject to different conditions than indoors, e.g., wind. Total suspended particulate (TSP) filters have a poorly defined cut-point ranging from 50 μm down to 22 μm depending on wind speed and may retain from submicron up to 50 μm particles. PM$_{10}$ (particulate matter 10 μm or less in diameter) filters have a 10 μm cut point and may cover the submicron to 10 μm particles and addresses the thoracic fraction particles. PM$_{2.5}$ filters have a 2.5 μm cut-point and cover the submicron to 2.5 μm particles and are meant to address the issue of fine, submicron particles which penetrate down to the alveoli (Health Canada, 1999).

Outdoor air monitoring is complicated by the occurrence of bi-modal and tri-modal particle size distributions in environmental aerosols. Environmental aerosols tend to be bimodal with a fine, submicron mode distributed between 0.1 μm and 1 μm and a coarse mode distributed from 1 μm out to 100 μm with a maximum in the 10 μm to 30 μm range. The fine mode represents fine,

carbonaceous particles from combustion sources and the coarse mode represents crustal particles eroded from surface material. A third intermodal distribution with a maximum between 1 µm and 5 µm occurs but it is not clear whether this is artefactual or real (Snipes et al., 1997, Health Canada, 1999).

To summarize, in terms of how to interpret occupational and environmental air sampling data, the new IOM "inhalable" personal sampler collects about twice the particle mass collected in the old "total" personal sampler. This difference mainly relates to improved collection of coarser particle sizes that may deposit in the nose, mouth or throat. The TSP sampler shares some characteristics (particle size and mass range) with the old "total" personal sampler. The PM_{10} sampler collects about half the mass collected in the TSP sampler and addresses the particle size range inhaled into the thoracic and respirable regions of the respiratory tract. The occupational "respirable" sampler would be expected to sample a slightly larger range of respirable particles than the $PM_{2.5}$ sampler. The particle mass collected by "respirable" and $PM_{2.5}$ samplers would normally be expected to be less than the larger particle size range samplers unless the "respirable" particle fraction is increased by some process.

A2-9.2.1.3 Particle Size Characteristics of Nickel Refinery Dust

Information on the particle size distribution and nickel speciation of the aerosols in the nickel refineries associated with increased lung cancer mortality used to derive inhalation unit risks is relatively unknown. A study of side-by-side sampling of workers wearing both the "total" aerosol sampler (37 mm closed face cassette) and an inhalable aerosol sampler (the IOM personal aerosol sampler) showed that the level of exposure based on inhalable aerosol consistently exceeds the that for "total" aerosol. The observed bias ranged up to three fold especially in workplaces with coarse aerosols (Werner et al., 1999).

Several studies have also evaluated the contribution of the "respirable" fraction to the inhalable or total fraction in nickel refineries and suggest that the respirable fraction is < 10% (Thomassen et al., 1999; Werner et al., 1999). The overall implication is that workplace atmospheres contain a larger proportion of larger, coarse particles that may not penetrate beyond the nose, mouth or throat.

A2-9.2.1.4 Particle Size Characteristics of Airborne Particulate in Port Colborne

By comparison, the results of many years of sampling ambient outdoor air particles in Ontario typically show mass ratios of 100:40:24 for TSP, PM_{10} and $PM_{2.5}$, respectively (information from Neil Buonocore, Supervisor, Air, Pesticides & Environmental Planning, West Central Region, MOE). Extensive federal air monitoring across Canada yields similar ratios of 100:50:25 for TSP, PM_{10} and $PM_{2.5}$, respectively (Health Canada, 1999).

An added complication is that MOE data on airborne nickel is based on hi-vol TSP samplers whereas the Environment Canada air samplers use lo-vol PM_{10} samplers. In terms of lung cancer, the PM_{10} data is more appropriate than TSP data, as it better represents the respirable fraction that would enter the lungs. However, the workplace air monitoring data is the old "total" inhalable fraction and its respirable fraction may be <10% of the "total" inhalable fraction. Essentially, the lung cancer risk estimate of ambient air in Port Colborne with an assumed nickel speciation based on soils containing elevated levels of nickel and measured using hi-vol TSP samplers is being characterized using a unit risk for lung cancer based on estimates of airborne nickel levels of particular nickel speciation in very dusty workplaces using personal air sampling devices.

A2-9.2.1.5 Chemical Composition / Speciation in Nickel Refinery Dust

Nickel refinery dusts of the era when nickel workers were exposed to airborne nickel levels associated with respiratory cancer contained varying percentages of metallic, oxidic, sulfidic and soluble nickel (see Table 3 in Doll et al., 1990 - details of the specific plants, areas of the plant and nickel refining process involved can be obtained from the references in Doll et al., 1990).

A dust sample from the Port Colborne refinery taken in 1959 (Gilman and Yamashiro, 1985) is described as containing 58% nickel subsulphide (Ni_3S_2), 20% nickel sulphate ($NiSO_4,6H_2O$) and 6.3% nickel oxide (NiO). This dust was used in animal carcinogenicity testing (Gilman and Ruckerbauer, 1962). This represents an elemental nickel content of about 51% by weight. US EPA (1986) has based nickel speciation assumptions on the composition of these nickel refinery flue dusts.

Dust samples taken from the Clydach, UK refinery and believed to represent dusts from 1920 and 1929 contained 37.5% and 26.7% nickel by weight, respectively. X-ray powder diffraction analysis indicated that nickel oxide peaks were prominent in spectra from both samples (Draper et al., 1994).

Dusts from the Port Colborne refinery were analyzed by Inco in 1978. Three dusts from nickel refining processes contained nickel levels ranging from 38.6% (Cottrell precipitator), 42.9% (Tumblast stack dust), to 72.2% nickel (Submerged Combustion Evaporator stack emission (SCE)) by weight. X-ray diffraction microprobe analysis of the Tumblast stack dust showed that there were two major phases, a nickel-magnesium-silicon alloy (50% Ni, 20% Mg, 30% Si), and a nickel-magnesium alloy (80% Ni, 20% Mg). Other phases also contained iron. In the Cottrell

precipitator dust, the most prevalent compound was nickel oxide, followed by lead sulphate, nickel sulphate and copper sulphate. In the SCE stack emission sample, the only compound detected was nickel oxide. It is not known whether any of these processes vented directly to the outside or were a component of stack emissions.

Andersen et al., (1996) assumed that the nickel species occurred in respirable dust at the Kristiansand, Norway refinery in the same proportion as the material being handled in various work areas. Nickel species were divided into four categories: metallic nickel, oxidic nickel and sulphidic nickel (all grouped as insoluble), and, soluble nickel (nickel sulphate, nickel chloride, nickel carbonate and nickel hydroxide). Nickel oxide is cited as the most frequent form of nickel in the roasting and calcining areas of the refinery (Andersen et al., 1996), however, Grimsrud et al., (2000) indicate that relatively higher proportions of soluble nickel (4% to 40%) may have been present. The percentage of nickel subsulphide would be correspondingly lower (T. Norseth - personal communication).

Stationary monitoring at the Finnish nickel refinery where the process involves leaching of matte and electrowinning of nickel from solution, indicates most of the airborne nickel is in the form of dry crystalline salts (95% nickel sulphate), except at the leaching site where 11% was insoluble forms of nickel (Kiilunen et al., 1997).

Nickel speciation results at a Russian refinery using older refining processes showed that the predominant chemical form in electrorefining department workroom air was soluble nickel (55 - 99%); oxidic and sulfidic nickel constituted, respectively, <0.6 - 34% and 1 - 19% for various jobs, with very little in the metallic form. The amount of sulfidic was reduced substantially during matte roasting with a concomitant increase in the oxidic and metallic fractions. It was also noticeable that in the early stages of the roasting process, the amount of soluble nickel increased and that the workroom air concentration of soluble nickel was higher at the top of the roaster than in the electrolytic department (Thomassen et al., 1999).

In terms of speciation, it is clear that the assumed speciation of nickel on air particles in Port Colborne ambient air is different from the speciation of nickel in nickel refinery dust. North American nickel refinery dust appears to differ from the Norwegian nickel refinery dust in that nickel subsulphide may form up to 50% of the nickel species and nickel oxide is < 10%, whereas the percent of nickel subsulphide in the Norwegian nickel refinery dust is lower and the percent of nickel oxide is higher. The percent of soluble nickel in both nickel refinery dusts appears to be comparable.

A2-9.2.1.6 Chemical Composition / Speciation in Airborne Particulate in Port Colborne

No chemical speciation data are available for airborne nickel in Port Colborne, however, nickel speciation information available for selected Port Colborne soils is described in Part A, Results - Nickel Speciation (section 6.2).

In summary, all of the five laboratories which performed analysis of Rodney Street soils detected nickel oxide. Elemental or metallic nickel was detected by two laboratories. One laboratory measured nickel sulphide and traces of soluble nickel sulphate. These results indicate that a range of analytical techniques are required to characterize nickel species in soil. The overall results indicate that nickel oxide is the predominant form and ranges from 80% - 94% of the total nickel. Metallic nickel and nickel alloys with copper and cobalt were also observed. Low amounts of sulphidic nickel and soluble nickel would be expected given the "ageing" effects of soil breakdown processes and leaching by rainfall. On average, about 16% - 19% of nickel in Rodney Street community soils is acid-soluble (Appendix 5). The ministry assumed that airborne nickel in Port Colborne had similar speciation to the Rodney Street community soils.

There is almost no information on nickel speciation of airborne nickel in North America. A recent study has provided nickel species-specific measurements of air near a structural steel plant, a stainless steel plant and an urban background site in Dortmund, Germany. Air near the steel mill contained 42% soluble nickel vs 58% insoluble (sulfidic, metallic and oxidic) nickel. Oxidic nickel was 46% of the total airborne nickel. At the urban background site, soluble nickel was about 22% and the rest insoluble. Oxidic nickel was 51% of the total nickel in urban air (Fuchtjohann et al., 2001, cited in the European Commission, DG Environment, 2000). Fuchtjohann et al., 2001, state that the mean fractions of total nickel in air samples near the stainless steel plant were found to contain $36 \pm 20\%$ soluble, $6 \pm 4\%$ sulfidic, $11 \pm 15\%$ metallic and $48 \pm 18\%$ oxidic nickel.

A2-9.2.1.7 Mass Concentration in Nickel Refinery Dust

Estimates of mass concentration of total airborne nickel in nickel refineries are limited and generally come from monitoring periods (1970s and later) subsequent to the highest occupational exposures. The exposure estimates developed by Doll et al., (1990) have been widely used (Health Canada, 1996; Andersen et al., 1996). For the most part, workplace exposures were classified as "high", "low", etc. on the basis of job category and duration of employment. Occupational exposure to nickel has also been reviewed by Nieboer (1992). The critical workplace concentrations associated with excess respiratory cancer risk are 10 mg/m^3 for insoluble forms of nickel, and 1 mg/m^3 for soluble forms of nickel (Doll et al., 1990). The recent studies of workers exposed to a high percent of soluble nickel in workplace air suggest that the critical workplace concentration for soluble salts may be 0.5 mg/m^3 (Kiilunen et al., 1997, Anttila et al., 1998). It is controversial, however, whether the effects of this concentration are due to soluble nickel alone, or interactions with insoluble forms and/or smoking.

A description of environmental conditions in the Port Colborne sinter plant is found in the Appendix to Doll et al., (1990). Of interest is a description of dust testing at this facility in 1953 by the Ontario Department of Health, Division of Occupational Hygiene. Dust concentrations in the five samples ranged from 61 to 1075 mg/m^3. If the highest value is dropped, the average dust concentration was 156 ± 55 mg/m^3. If it is assumed that the percentage of elemental nickel in these dusts ranged from 27% to 51%, the airborne nickel concentration would have ranged from

42 to 80 mg/m^3. This estimate agrees with reported airborne nickel levels in a similar Ontario nickel refinery (Copper Cliff) over this time period (Doll et al., 1990). It is not known whether any of these processes vented directly to the outside or were a component of stack emissions.

The recent WHO air quality guideline for nickel used the exposure data of Doll et al., (1990) and updated epidemiology data from the Kristiansand, Norway cohort (Andersen et al., 1996; WHO, 2000). The exposure assessment is further discussed in Grimsrud et al., (2000) and involved several thousand personal measurements of total nickel in the breathing zone from 1973 to 1994, a period which overlaps the time span of the Andersen et al., (1996) cohort. Most of the personal sampling measurements were performed using a 37 mm filter cassette. Total nickel in this context may correspond with total nickel on TSP filters outdoors, however, the occupational measurements may address a nickel enriched atmosphere with a different particle size distribution than outdoors.

Workplace levels of airborne nickel comparable in magnitude to those reported or estimated for similar operations during the nineteen sixties and seventies in western Europe and North America have been measured in a Russian nickel refinery (Thomassen et al., 1999; Höflich et al., 2000).

A2-9.2.1.8 Mass Concentration in Airborne Particulate in Port Colborne

MOE monitored airborne particulate (total suspended particulate (TSP)) and airborne nickel in Port Colborne at three locations from 1973 to 1996. The locations of the monitors (Hi-vol samplers) and the periods they were operated are:

Station 27030 located at Killaly Street East and James Street (about 1400 m NE of the refinery) from 1973 to 1985 (Avg = 0.278 µg/m^3). At this station, the annual average air concentration of nickel recorded in 1985, the year after the refinery ceased full scale operation, was 0.088 µg/m^3. Station 27031 located at Killaly St East and Welland Avenue (about 1200m NW of the refinery) from 1973 to 1979. The annual average air concentration of nickel for 1979 was 0.169 µg/m^3. Station 27047 located at Davis Street and Fraser Street (about 600m N of the refinery) from 1973 to 1996. The annual average air concentration of nickel for 1995, the last year with a full record was 0.023 µg/m^3. This airborne nickel concentration represented only 0.036% of the airborne particulate.

A new air monitoring station with TSP and PM$_{10}$ samplers located on Rodney Street and just west of and adjacent to the Inco facility began operation in July 2001. The results of sampling into early October are summarized in Tables A1-2a, A1-2b and A1-3, Appendix 1. These short term results show averages of 0.013 and 0.0059 µg Ni/m^3 for the TSP and PM$_{10}$ samplers, respectively. The corresponding air particulate data averages are 51.8 and 22.8 µg/m^3. This represents airborne nickel concentrations that are 0.025% to 0.026% of the airborne particulate. These TSP-based nickel levels are lower than those measured in 1995.

Air monitoring data from other sites in Ontario are also summarized in Appendix 1. Since

Environment Canada's air monitoring network only reports data from lo-vol PM_{10} and $PM_{2.5}$ samplers, the federal air data cannot be compared with Ontario's TSP data directly. Table A1-2b compares Environment Canada's data for sites in Southwestern Ontario with MOE PM_{10} data from Port Colborne in the Summer of 2001.

It would appear that the best comparison is of TSP-based airborne nickel levels to the old "total" personal air sampling data from the nickel refinery workers for estimating potential lung cancer risk associated with breathing Port Colborne air. The use of PM_{10} and $PM_{2.5}$ data introduces a requirement to prorate older workplace exposure data so that "respirable" fractions are compared.

A2-9.2.2 Oral Exposure

One death has been reported due to the accidental consumption of an extremely high nickel sulphate concentration (570 mg nickel/kg) by a two year old (Daldrup et al., 1983, as cited in ATSDR, 1997). Gastrointestinal effects were reported in an incident where workers drank water from a fountain containing nickel sulphate and nickel chloride (Sunderman et al., 1988). Exposure doses ranged from 7.1 to 35.7 mg nickel/kg. Symptoms included nausea, abdominal pain, vomiting and diarrhea. Neurological effects were also observed in the affected workers.

Oral lethality tests of rats indicated that soluble nickel compounds were more toxic than insoluble nickel compounds. An oral lethal dose for 50% of the population (LD_{50}) for nickel sulphate in female rats was reported to be 39 mg/kg while oral LD_{50} values for insoluble nickel compounds were >3,930 and >3,665 mg/kg for nickel oxide and nickel subsulfide, respectively (Mastromatteo, 1986).

Decreased body weight has been observed in rats and mice given nickel chloride and nickel sulphate in drinking water (Schroeder et al., 1974). Ambrose et al., (1976) reported data on albino (Wistar) rats and beagle dogs exposed for two years to nickel sulphate in the diet at 100, 1,000 and 2,500 ppm. Non-cancer effects included decreased growth in dogs (mid and high doses) and rats (high dose), alterations in blood and urinary chemistry in high dose dogs, and changes in relative organ weights for mid and high dose female rats (heart and liver) and high dose dogs (kidney and liver). The No-Observable-Adverse Effects Level (NOAEL) was estimated to be 5 mg Ni/kg-day, based on the non-cancer changes in the rat. This study has been used as a supporting study for exposure limit development by US EPA IRIS (1998c - oral R/D assessment last revised 1996), WHO (1996), Health Canada (1996), and IOM (2001).

Increased albuminuria was observed in Wistar rats administered nickel sulphate in drinking water for six months (Vyskocil et al., 1994). Groups of male and female Wistar rats (n = 10) were given nickel (as sulphate) in the drinking water, at a concentration of 100 mg/L, for up to six months (Vyskocil et al., 1994). At six months, females exhibited a four fold increase in urinary excretion of albumin (p<0.05), as compared to age matched controls. Kidney weights in males were significantly increased. Absolute nickel intakes were estimated to range from 6.3 mg/kg/d to 8.4 mg/kg/d. Body weight gains and drinking water volumes consumed were comparable with those

of age-matched controls. These results were attributed to nickel induced changes in glomerular permeability, particularly in females. In males, some treatment-related exacerbation of the normal age-related nephropathy could not be ruled out.

On behalf of the US EPA Office of Water, Metal Finishers of Southern California and Health Canada, Toxicology Excellence for Risk Assessment (*TERA*, 1999) conducted a review of a number of key studies of oral exposure involving soluble nickel. As part of this review, *TERA* (1999) identified a LOAEL of 7.6 mg/kg/day for the Vyskocil et al., (1994) study.

ATSDR (1997), and US EPA IRIS (1998c - oral R/D assessment last revised 1996) discuss the 90 day study with nickel chloride in water (0, 5, 35 and 100 mg/kg/day) administered by gavage i.e., oral intubation) to both male and female CD rats conducted by the American Biogenics Corporation (ABC, 1988). The body weight and food consumption values were consistently lower than those of controls for the 35 and 100 mg/kg/day dosed males. The 5 mg/kg/day group did not show any significant clinical signs of toxicity. There was 100% mortality in the high-dose group; 6/30 males and 8/30 females died in the mid-dose group (35 mg/kg/day). Histopathologic evaluation indicated that deaths of 3/6 males and 5/8 females in the mid-dose group were due to gavage errors. At sacrifice, kidney, liver and spleen weights for 35 mg/kg/day treated males and right kidney weights for 35 mg/kg/day treated females were significantly lower than controls. Based on the results obtained in this study, US EPA (IRIS) considered the 5 mg/kg/day nickel dose was a NOAEL, whereas 35 mg/kg/day was a LOAEL for decreased body and organ weights. This study was considered a supporting study by US EPA IRIS (1998c - oral R/D assessment last revised 1996) for development of their R/D.

A three generation study, carried out by Ambrose et al., (1976), noted a higher incidence of stillborns in the first generation of albino (Wistar) rats fed 250, 500 or 1,000 ppm nickel in their diet (nickel sulphate) and depressed body weights of weanlings on the 1,000 ppm diet in all generations. A higher incidence of stillborns was not observed in subsequent generations (Ambrose et al., 1976). Both parental and F_1 generations were treated for 11 weeks before mating. The study had some limitations including small sample size and use of pups rather than litters as the unit for comparison. No statistical analysis of the results was presented and a NOAEL or LOAEL has not been defined for this study.

ATSDR (1997), US EPA IRIS (1998c - oral R/D assessment last revised 1996), Velazquez and Poirier (1994) and WHO (1998) discuss a multi generation study using male and female CD rats exposed to nickel chloride in the drinking water at nickel concentrations of 50, 250 or 500 ppm (equivalent to 7.3, 30.8 or 51.6 mg nickel/kg/day) conducted by Research Triangle Institute (RTI, 1988). Animals were exposed to nickel in drinking water for 90 days before breeding. The highest exposure produced maternal toxicity characterized by a decrease in body weight and decreased absolute and relative liver weights of the dams. A decrease in the number of live pups per litter, an increase in pup mortality, and a decrease in the average body weight of the pups was noted for the $F1_a$ and $F1_b$ generations of the 500 ppm test group. Both the 50 and 250 ppm exposures have been considered to be a NOAEL (ATSDR, 1997, WHO, 1998, Velazquez and Poirier, 1994). However,

US EPA IRIS (1998c - oral R$_f$D assessment last revised 1996) did not consider RTI (1988) to be a supporting study in developing their R$_f$D. Decreased food and water intake were observed in the exposed animals. For example, the low dose animals ranged from 1.75 mg Ni/kg/day to 11.78 mg Ni/kg/day as calculated on a weekly basis. Also, the room temperature was up to 6°C higher than normal at certain times during gestation and the early postnatal days. Increased room temperatures have been reported to affect reproductive performance (National Research Council, 1996, Wolfensohn and Lloyd, 1998).

Smith et al., (1993) also studied the reproductive and fetotoxic effects of nickel. Four groups of 34 female Long-Evans rats were given drinking water containing nickel chloride in the following concentrations of nickel: 0, 10, 50 or 250 ppm (0, 1.3, 6.8 or 31.6 mg/kg/day) for 11 weeks prior to mating and during two successive gestation periods (G1, G2) and lactation periods (L1, L2). Maternal body weight gain was reduced during G1 in mid and high-dose females. The reproductive performance of the exposed rats was not affected. Pup birth weight was unaltered by treatment, and weight gain was reduced only in male pups exposed to 50 ppm nickel during L1. The most significant toxicological finding was the increased incidence of perinatal mortality. The proportion of dead pups per litter was elevated at the high dose in L1 and at 10 and 250 ppm in L2. However, at the same time, the perinatal mortality of the L2 controls was halved. The authors (Smith et al., 1993) consider the lowest dose (1.3 mg/kg/day) to be a LOAEL. While the perinatal mortality reported in this study is consistent with other reproductive studies on nickel, US EPA IRIS (1998c - oral R$_f$D assessment last revised 1996) considered it hard to define a NOAEL and LOAEL because of the absence of a clear dose-response trend at the lower doses. Other agencies (Health Canada, 1996, WHO, 1998) consider 1.3 mg/kg/day to be a LOAEL.

Since the *TERA* (1999) review, an unpublished two generation reproduction study of orally administered nickel sulfate hexahydrate using Sprague Dawley rats was conducted at Springborn Laboratories in 2000 for the Nickel Producers Environmental Research Association (NiPERA) (Springborn, 2000). The test protocols were compliant with the OECD 416 test guidelines which are harmonized with current US EPA reproductive test guidelines (US EPA OPPTS, 2001). The range finding and definitive studies for this study were conducted using gavage (i.e., oral intubation) as the route of exposure, due to palatability problems with nickel in drinking water and bioavailability problems with nickel in food. Some of the problems with delivering a precise dose to the animals is reduced, though a bolus effect may be involved. The range finding study was designed in two parts. The first part of the range finding studies was a dose-response probe utilizing small numbers of animals and nickel sulfate hexahydrate exposures of 0, 5, 15, 25, 50, 75 and 150 mg/kg/day. Lethality was observed at the 150 mg/kg/day exposure level.

The second part of the range finding study (i.e., a one generation reproductive toxicity study) utilized nickel sulfate hexahydrate exposures of 0, 10, 20, 30, 50 and 75 mg/kg/day. These doses had no effect on parental survival, growth, mating behavior, copulation, fertility, implantation or gestation length. However, evaluation of post-implantation/perinatal lethality among the offspring of the treated parental rats (i.e., number of pups conceived minus the number of live pups at birth) showed statistically significant increases at the 30 to 75 mg/kg/day exposures and questionable

increases at the 10 and 20 mg/kg/day levels.

Based upon the results of the one generation study, nickel sulfate hexahydrate exposure levels of 1, 2.5, 5.0 and 10 mg/kg/day (equivalent to 0.22, 0.56, 1.12 and 2.23 mg Ni/kg/day) were administered by gavage to five groups of male and female rats in the definitive two generation study. Males of the parental (F_0) generation were dosed during growth and for at least one complete spermatogenic cycle in order to elicit any possible adverse effects on spermatogenesis by the test substance. Females of the F_0 generation were dosed during growth and for several complete estrous cycles in order to elicit any possible adverse effects on estrous by the test substance. The test substance was administered to F_0 animals during mating, pregnancy, and through the weaning of their first generation (F_1) offspring. At weaning, the administration of the substance was continued to F_1 offspring during their growth into adulthood, mating and production of an F_2 generation, and up until the F_2 generation was weaned.

The results from the two generation study indicate that the highest dose selected (10 mg/kg/day or 2.2 mg Ni/kg/day) was a NOEL for adult and offspring rats for all the endpoints studied, including the variable of post-implantation/perinatal lethality. This study is under review by US EPA.

The selection of studies of oral exposures to soluble nickel salts for exposure limits is further discussed in section A2-9.3.2.1.

A2-9.2.3 Cancer Effects

Extensive reviews of the toxicology of nickel and nickel compounds, including animal carcinogenicity and human epidemiological data, have been published (US EPA, 1986, IARC, 1990, Doll et al., 1990, CEPA, 1994, Hughes et al., 1994, *TERA*, 1999). The studies reviewed included human exposures associated with nickel mining, smelting, refining and high nickel alloy manufacture. The reviews also indicated that different classes of nickel compounds have different carcinogenic potencies.

A2-9.2.3.1 Cancer Potential (Oral)

Increased tumourigenesis in mice, rats and dogs has not been associated with nickel compounds ingested in the diet or in drinking water (Schroeder et al., 1964; Schroeder and Mitchener, 1975; Ambrose et al., 1976, *TERA*, 1999).

Intrarenal injection of nickel subsulphide was reported to result in an increased incidence of renal tumours in male Fischer 344/NC rats (Higinbotham et al., 1992). Intrarenal administration of nickel subsulphide to male Fischer 344 rats was associated with an increase in kidney tumours (Sunderman et al., 1990). Nickel acetate was reported to induce a significant increase in lung tumours in rats following a series of intraperitoneal injections (Stoner et al., 1976).

A2-9.2.3.2 Cancer Potential (Inhalation)

Numerous carcinogenicity experiments have been conducted with nickel compounds, administered via injection, inhalation or ingestion (reviewed by ATSDR, 1997 and *TERA*, 1999). Recent chronic inhalation studies have clearly indicated that different nickel compounds have different carcinogenic potentials and different animal species show different carcinogenic responses to various nickel compounds (NTP, 1996a; b; c).

Inhalation studies of the effects of nickel oxide concentrations of up to $42mg/m^3$ on hamsters for a life-time did not show nickel induced carcinogenicity (Wehner et al., 1975 as cited in CEPA, 1994). However, rats exposed to 5 or 15 mg or nickel oxide via intratracheal instillation, demonstrated an increase in lung tumours (Pott et al., 1987, as cited in CEPA, 1994). Nickel oxide compounds also caused an increased incidence of tumours at the site of injection in various experimental animals (IARC, 1990).

A number of inhalation studies of nickel carcinogenesis in rats and mice have yielded positive results. Ottolenghi et al., (1974) exposed Fischer 344 rats to 0.97 mg nickel sulphide/m^3 for 78 weeks. An increased incidence of lung tumours was observed during treatment and during a 30 week observation period. Sunderman et al., (1957; 1959) also observed increased incidences of lung tumours in rats exposed to nickel carbonyl for up to 52 weeks.

The most recent chronic inhalation studies included up to two year inhalation exposures to nickel subsulphide, nickel sulphate hexahydrate and nickel oxide (NTP, 1996a,b,c).

In the nickel subsulphide inhalation study, rats were treated with 0, 0.11 or 0.73 mg Ni/m^3 and mice with 0, 0.44 or 0.88 mg Ni/m^3, 6 hours/day, 5 days/week for 104 weeks. NTP concluded that there was an increased incidence of alveolar/bronchiolar adenoma or carcinoma or squamous cell carcinoma in male and female rats, benign or malignant pheochromocytoma in males and benign pheochromocytoma in female rats. NTP concluded that there was no evidence of carcinogenic activity in mice and clear evidence of carcinogenic activity in male and female rats (NTP, 1996b).

In the nickel sulphate hexahydrate study, rats were exposed to 0, 0.03, 0.06 or 0.11 mg Ni/m^3, and mice were exposed to 0, 0.06, 0.11 or 0.22 mg Ni/m^3, by inhalation of a form of nickel sulphate hexahydrate aerosol for 104 weeks. NTP concluded that there was no evidence of carcinogenic activity (NTP, 1996c).

As summarized in a recent review (*TERA*, 1999), it has been suggested that the negative evidence from the NTP bioassays cannot be considered definitive. For example, as noted in that document, one line of reasoning is that insufficiently high concentrations were tested, although NTP did consider the testing to be adequate, and additional analysis showed that the lung tissue dose in the animal bioassay was comparable to that under occupational conditions, once particle size is taken into account.

In the nickel oxide inhalation study, rats were treated with 0, 0.5, 1.0 or 2.0 mg Ni/m^3 and mice with 0, 1.0, 2.0 or 3.9 mg Ni/m^3 for 104 weeks. NTP concluded that there was some evidence of an increased incidence of alveolar/bronchiolar adenoma or carcinoma or squamous cell carcinoma, and benign or malignant pheochromocytoma in rats. NTP concluded that there was no evidence of carcinogenic activity in male mice but equivocal evidence of alveolar/bronchiolar adenoma or carcinoma in female mice (NTP, 1996a).

The most complete analysis of pre-1990 epidemiology studies of nickel workers was carried out by the International Committee on Nickel Carcinogenesis in Man (ICNCM) (Doll et al., 1990). This study examined ten cohorts involving workers mining, smelting and refining nickel as well as workers in nickel related industries (metallic nickel powder and nickel alloy) (Inco, Clydach, Wales, Falconbridge, Ontario, Huntington, W. Va., Inco, Copper Cliff and Coniston, Sudbury, Ontario, Inco, Port Colborne, Ontario, Falconbridge, Kristiansand, Norway, Oak Ridge, Tenn., Outokumpu Oy, Finland, Societe le Nickel, New Caledonia and Henry Wiggin Alloy Co., Hereford, England). This report concluded that more than one form of nickel gives rise to lung and nasal cancer. While much of the respiratory cancer risk seen in nickel refinery workers could be attributed to mixtures of oxidic and sulfidic nickel at concentrations greater than 10 mg/m^3, there was evidence that oxidic nickel and soluble nickel were also associated with lung and nasal cancer risks. Respiratory cancer risks were related to soluble nickel exposures at concentrations in excess of 1 mg/m^3. Soluble nickel exposures may enhance risks associated with exposure to less soluble forms of nickel. There was no evidence that metallic nickel was associated with increased lung and nasal cancer risks.

The epidemiological studies reviewed by IARC (1990) and Doll et al., (1990) have several limitations. The principal limitation was the limited data related to concentrations of specific nickel species in the air within the facilities that were studied. Consequently, it was not possible to establish dose-response relationships for specific nickel species. Doll et al., (1990) noted that the conclusions of many of the epidemiological studies (with respect to lung tumours) were confounded by a lack of information about the smoking habits of the workers. It should be noted that several epidemiology studies (including updates of those in the Doll report) have been published since the completion of the Doll report *(TERA*, 1999).

Update reports for the Clydach, Wales refinery cohorts have been reported (Easton et al., 1992; Draper et al., 1994). Easton et al., (1992) confirm that cancer risks are largely confined to workers employed in the 1930s and earlier. The results suggest that nasal cancer risk is due to soluble nickel exposure and at least one other insoluble nickel species, possibly nickel oxide, contributes to this risk. Draper et al., (1994) suggests that the cessation of leaching the nickel matte with an arsenic containing sulfuric acid in the 1920s is associated with the cessation of the occurrence of the sinus/nasal cancers.

Julian and Muir (1996) examined cancer incidence in Ontario men employed by Inco and Falconbridge (also examined by Doll et al., 1990). Their study is further discussed in ODP (1997). An excess of laryngeal cancer was reported for employees not exposed to the sintering process.

Underground miners were also shown to have a significant lung and nasal cancer excess for miners with greater than 25 years exposure. The prevalence of smoking among this cohort may have contributed to this excess lung cancer.

A morbidity update of the Kristiansand, Norway cohort (reviewed by Doll et al., 1990) was reported by Andersen (1992) and Andersen et al., (1996). Their analysis showed an excess risk of lung cancer in association with exposure to soluble forms of nickel. The study suggested a multiplicative effect of smoking and nickel exposure.

Pang et al., (1996) reported on the mortality of a small cohort of English nickel platers with no exposure to chromium. These workers were principally exposed to soluble nickel salts. No risk of occupational lung cancer was discerned. However, there was weak evidence that nickel plating is associated with an excess risk of stomach cancer.

The cohort of US high alloy nickel workers examined by Doll et al., (1990) was expanded and updated by Arena et al., (1998). An overall significant 13% increased risk for lung cancer was noted when compared to the total US population. However, no significant excess was identified when local populations were used as a comparison.

Anttila et al., (1998) updated cancer incidence among workers at the Finnish smelter and refinery examined by Doll et al., (1990). Nickel compounds were statistically significant in smelter workers with a latency of greater than 20 years. Refinery workers exposed to nickel sulfate levels less than 0.5 mg/m^3 showed an increased risk for nasal cancer and an excess risk for stomach and lung cancer.

The epidemiology studies of nickel process workers published since Doll et al., (1990) generally support the conclusions of the ICNCM report and expand on the association of occupational exposure to elevated levels of nickel compounds and respiratory cancer. The role of soluble nickel species as a contributor to risks associated with exposure to insoluble nickel compounds is confirmed (Easton et al., 1992; Anttila et al., 1998). The association of soluble nickel exposure with excess stomach cancer risk is notable (Pang et al., 1996, Anttila et al., 1998). Further support for a multiplicative role for smoking and nickel exposure leading to excess lung cancer risk is also notable (Andersen et al., 1996; Julian and Muir, 1996; ODP, 1997). It should be noted also that nickel smelter and refinery workers had concomitant exposures to copper, cobalt, arsenic, lead, sulphuric acid mist and other substances depending on the type of nickel product being processed. The role of these other substances in nickel-related cancer incidence has not been addressed.

A2-9.2.4 Contact Dermatitis

Contact or allergic dermatitis is an allergy to nickel following some sensitizing exposure in individuals who are nickel sensitive. A relationship between specific human lymphocyte antigens and nickel sensitivity has been observed (Mozzanica et al., 1990). Nickel dermatitis is the most commonly observed adverse effect of nickel. Asthma may occur in a small number of sensitized

individuals (Dolovich et al., 1984; Novey et al., 1983; Shirakawa et al., 1990; as cited in ATSDR, 1997). This allergic response is manifested in two main ways. Sensitized individuals can react to nickel in subsequent dermal exposures, or, following oral exposures to nickel (Keczkes et al., 1982).

A2-9.2.4.1 Dermal Exposure

In non-occupational exposures, the primary exposure to nickel as a sensitizing event in non-sensitized individuals (sensitization) occurs primarily as a result of prolonged skin contact with nickel-containing metal objects (jewelry, coins, dental braces, stainless steel and metal fastenings on clothes) (Menne and Maibach, 1989; Menne et al., 1989; Wilkinson and Wilkinson, 1989) or when metal objects are inserted into body parts (ear piercing, orthodontics and orthopaedic devices) (Dotterud and Falk, 1994; Larsson-Stymne and Widstrom, 1985; Meijer et al., 1995; van Hoogstraten et al., 1991). Contact dermatitis may also result from occupational exposure (Liden, 1994). Once an individual has been sensitized to nickel, subsequent exposure (inhalation, ingestion or dermal contact) to low levels of nickel may cause a reaction (elicitation) (Keczkes et al., 1982). However, continued oral exposure to nickel has also been shown to desensitize some individuals and prevent sensitization in other cases (see later discussion). The amount of allergen required to induce sensitization in a non-sensitized person generally is greater than that required to elicit a response in a previously sensitized individual (Menne, 1994).

Studies of Norwegian populations have indicated a prevalence range of 8 to 11% in females and approximately 1 to 2% in males (Nielsen and Menne,1993; Peltonen, 1979 as cited in Smith-Sivertsen et al., 1999). As studies have indicated that the most important known risk factor associated with nickel allergy is ear piercing (Nielsen and Menne, 1993), it is likely that gender differences in dermatitis prevalence can be explained primarily by differences in ear piercing habits and use of jewelry (Smith-Sivertsen et al., 1999).

Smith-Sivertsen et al., (1999) conducted a large study of Norwegian populations to evaluate the importance of local nickel pollution, as well as ear piercing and atopic dermatitis, on the prevalence of nickel allergy. A total of 1,767 adults were randomly selected from two different cross-sectional studies: i) in the town of Sør-Varanger, which is situated near a nickel smelter in the Russian city of Nikel; and ii) in the town of Tromsø, which has no nearby nickel-polluting industries. Results of the study indicated that nickel allergy was very common among adults in both of the north-Norwegian study populations, and that despite the geographical closeness to the Russian nickel industry, inhabitants of Sør-Varanger did not have a higher prevalence than those living in the city of Tromsø. Interestingly, ear piercing in women demonstrated a more than three fold increased risk of being sensitized to nickel, and increased further with increasing number of holes in the ear lobes. Based upon comparisons of urinary nickel concentrations in each of the study populations, the authors concluded that a pollution-dependent increased risk of nickel allergy was unlikely, and that intimate skin contact with any object that releases nickel (e.g., ear rings or jewelry) constitutes a much larger risk of primary sensitization or elicitation of dermatitis in individuals already nickel-sensitized (Smith-Sivertsen et al., 1999).

Skin contact with nickel appears to be facilitated by the release of nickel as a soluble form into sweat. Nickel must be in a soluble form in order to be absorbed into the skin through the horny layer. Sweat and friction facilitate this process. Normal sweat has a slightly acidic to neutral pH, and contains amino acids and salts containing chloride (ICRP, 1984, Casarett and Doull, 1991). The development of contact dermatitis after this type of contact with nickel occurs through a series of cellular skin responses, initiating the allergic reaction to nickel, and manifesting as contact dermatitis.

Dermal patch studies in sensitive individuals show a dose response relationship between the amount of nickel applied and the severity of the test response (Emmet et al., 1988; Eun and Marks, 1990). Most patch testing is done with nickel sulphate because it is less irritating, however, nickel chloride may be more biologically relevant (Menne, 1994) and has been shown to pass more rapidly through the upper layers of the skin (Fullerton et al., 1986). Studies of skin reactions with nickel chloride in sensitive individuals indicate that the NOAEL aqueous nickel concentration is about 300 ppm (Eun and Marks, 1990).

Other studies with nickel alloys tested for their release of nickel into synthetic sweat indicated weak reactivity at release rates $< 0.5 \ \mu g/cm^2/week$ (or less than $0.07 \ \mu g/cm^2/day$) (Menne et al., 1987). This information forms the basis for the 1994 European Union Directive forbidding the use of nickel in products placed in direct contact with the skin and to restrict release of nickel to less than $0.5 \ \mu g/cm^2/week$ during normal use for up to two years.

The setting of soil remediation criteria on the basis of protecting against elicitation of allergic contact dermatitis to trivalent and hexavalent chromium in sensitized individuals has been proposed by New Jersey and Massachusetts (NJDEPE, 1998, MADEP, 1998, Horowitz and Finley, 1994). However, while soil remediation criteria for residential soils for trivalent chromium on this basis were used until 1998, it was determined that allergic contact dermatitis was not a relevant endpoint for trivalent chromium due to the insolubility of trivalent chromium compounds typically found in the environment. MADEP (1998) has attempted to apply this approach to develop soil clean-up criteria for hexavalent chromium based on dermal elicitation of allergic contact dematitis by soil. Due to a concern that hexavalent chromium was a more potent inducer of allergic contact dermatitis by the oral or inhalation route, it was decided that a soil remediation criterion for hexavalent chromium based on the dermal route alone was not appropriate at the present time. Some calculations based on this approach are provided to place the risk of elicitation of allergic contact dermatitis by soil borne nickel in the Rodney Street community in context. According to Horowitz and Findley (1994), the following information is required:

a) dose response data from human patch testing studies expressed as mass allergen per unit area of skin;

b) accurate estimates of soil adherence to skin on a soil mass per unit area of skin; and

c) accurate estimates of the degree to which the allergen leaches from soil into human sweat.

This method does not address contributions to allergic responses from oral challenge, nor does it address cross-sensitization from other metals or substances.

Application of this approach to the situation on the Rodney Street community may be addressed using:

a) the EU limit of <0.07 ug Ni/cm^2/day. This is well below the minimum eliciting concentration for non-occluded nickel exposure (1.5 ug/cm^2) reported by Menne (1994);

b) the soil adherence value of 0.2 mg soil/cm^2. US EPA (2001) considers use of this soil adherence factor, which is the 50th percentile for children playing in wet soil as a recommended weighted adherence factor for a child resident (<1 to <6 years old); and

c) the soluble nickel speciation data from the Lakefield testing (Part A, Table 10). The soluble nickel leaching approach used by Lakefield extracted the soil samples in hot water for 30 min. Soil pH (Part A, Table 9) ranged from pH 6.85 to pH 7.75. However, while the pH of the hot water extracts was not reported, it is not expected to be much below pH 7. Hot water was used instead of the 0.1 M ammonium citrate (pH 4.4) used by Zatka et al., (1992), a fluid that is similar to artificial sweat solutions used in other studies because it gave a more accurate extraction of soluble nickel salts. Freshly perspired sweat has a neutral pH, however, it becomes slightly acidic (pH 4 to pH6) depending on the presence of resident skin bacteria. The mean soluble nickel value in hot water is 0.37% (range = 0.26% to 0.68%). Tests to measure plant-extractable nickel in Port Colborne soils averaged 0.22% for pH neutral mineral soils (Part A, section 6.1.3).

Inco has provided information to suggest that at pH 7 without the glycine buffer used by Exponent in their bioaccessibility studies (Appendix 5), nickel extractability is 1.5 %.

A nickel concentration in soil equivalent to the EU limit may be derived as follows:

(mg nickel/cm^2 ÷ mg soil/cm^2) X 10^6 mg soil/kg soil   = mg allergen/kg soil

therefore:

(0.00007 mg Ni/cm^2 ÷ 0.2 mg soil/cm^2) X 10^6 mg soil/kg soil = 350 mg Ni/kg soil

Adjusting this value for sweat extractability at 1.5%, 350/0.015 = 23,333 mg Ni/kg soil (which is 23,333 µg Ni/g soil). This derivation suggests that for the Rodney Street community soils with their low soluble nickel content, development of allergic contact dermatitis to nickel is unlikely below the soil intervention level of 8,000 µg Ni/g soil. Intimate skin contact with jewelry containing nickel may be a greater risk for elicitation of dermatitis in nickel-sensitized individuals (Smith-Sivertsen et al., 1999). There is some uncertainty in the calculation of a soil nickel concentration that might be associated with elicitation of an allergic contact dermatitis response.

The uncertainty is mainly related to the nickel leachability into human sweat. Use of the EU limit which is based on studies of nickel elicitation of allergic contact dermatitis in occluded skin studies overestimates the threshold for non-occluded allergic contact dermatitis. In the case of the Rodney Street soils where the nickel is mostly insoluble, some of the uncertainty regarding nickel leachability may be reduced.

A2-9.2.4.2 Oral Exposure

Flare up of skin reactions to nickel can occur in sensitized people following single oral exposures to nickel. The issue of allergic dermatitis following ingestion of nickel-containing food items has been reviewed (US FDA, 1993). In studies where nickel (mainly in a soluble form such as the sulphate) was administered orally to human subjects with chronic nickel dermatitis or eczema, single doses of 2,500 µg to 5,600 µg nickel produced aggravated reactions (Cronin et al., 1980; Kaaber et al., 1978; Gawkroder et al., 1986; Veien et al., 1983). One double blind study showed that a single 2,500 µg dose of orally administered nickel was sufficient to aggravate the chronic nickel dermatitis in 17 of the 28 patients tested (Veien et al., 1983). Other, less reliable studies suggest that as little as 600 µg or 1,250 µg of ingested nickel may exacerbate the skin conditions in patients with long standing (10 - 17 years) nickel sensitivity. In one double blind study (Jordan and King, 1979), one of the ten nickel hypersensitive patients tested consistently reacted to a 500 µg oral nickel challenge. Thus, oral nickel exposure of as little as 500 µg/day in addition to the diet may produce adverse reactions in some nickel hypersensitive persons. The lowest single dose resulting in dermatitis in sensitized individuals is about 0.009 mg/kg/day (Cronin et al., 1980). Other studies showed that a low incidence of allergic dermatitis responses occurred in the 0.02 to 0.04 mg/kg/day range (Burrows et al., 1981; Gawkroder et al., 1986; Kaaber et al., 1978; Menne and Maibach, 1987). This situation is complicated by studies that show that low nickel diets can prevent induction of nickel sensitivity (van Hoogstraten et al., 1991).

Oral hyposensitization (or de-sensitization) to nickel after various oral dose regimes is discussed in WHO (1998). This response was reported after six weekly doses of 5 mg of nickel in a capsule (Sjöwall et al., 1978) and 0.1 ng of nickel sulphate daily for three years (Panzani et al., 1995). Cutaneous lesions were improved in eight patients with contact allergy to nickel after oral exposure to 5 mg of nickel weekly for eight weeks (Bagot et al., 1995). Nickel in water (as nickel sulphate) was given to 25 nickel-sensitive women in daily doses of 0.01–0.04 mg/kg of body weight per day for three months after they had been challenged once with 2.24 mg of nickel (Santucci et al., 1988). In 18 women, flares occurred after the challenge dose, whereas only three out of 17 subjects had symptoms during the prolonged exposure period. Later, Santucci and co-workers (1994) gave increasing oral doses of nickel in water (0.01 – 0.03 mg of nickel per kg of body weight per day) to eight nickel-sensitive women for up to 178 days. A significant improvement in hand eczema was observed in all subjects after one month.

ATSDR (1997) also discusses the same studies of contact dermatitis following oral exposure and indicates that setting of oral exposure limits for nickel is complicated by the presence of sensitized individuals in the general population. Some of the induced reactions at very low levels of

exposure indicate that individual characteristics may be at play. Given the literature showing both elicitation and de-sensitization of contact dermatitis following oral supplementation of ambient nickel levels in the diet and/or drinking water, it is not possible to set oral exposure limits based on contact dermatitis reactivation at this time.

A2-9.2.5 Susceptible Populations

Populations which are unusually susceptible to nickel are those people already sensitive to nickel due to prolonged contact with nickel. Subsequent exposures may result in an allergic reaction. A greater number of women tend to be sensitized to nickel than men and this is believed to be related to the fact that woman tend to wear more metal jewelry than men. Further study is required to determine whether there is indeed a gender difference in nickel sensitivity. Persons with kidney dysfunction are also likely to be more susceptible to nickel as the primary route of nickel elimination is via the urine.

A2-9.3 Current Exposure Limits

It should be noted that the Health Canada (1996) exposure limits for nickel compounds cited below are based on toxicological literature reviewed up to 1993. Several key studies have been published since 1993 (*TERA*, 1999, WHO, 1998, WHO, 2000 and IOM, 2001).

A2-9.3.1 Nickel Refinery Dusts and Nickel Subsulphide
A2-9.3.1.1 Inhalation

The inhalation exposure limits for the three majors agencies and their derivation is described below. Neither US EPA, Health Canada nor WHO state their limits as $\mu g\ Ni/m^3$ or risk per μg Ni/m^3. Doll et al., (1990) does specific airborne Ni levels as such. It may be reasonably assumed that the nickel refinery worker personal air samplers were analyzed using a total Ni approach and that nickel speciation was imposed retroactively. The implication is that the analytical data is total Ni and no further conversion is needed.

A2-9.3.1.2 US EPA

Nickel refinery dusts and nickel subsulphide are both classified by the US EPA as group A: human carcinogens (US EPA, 1998a; 1998b - carcinogenic assessments last revised 1991). Only inhalation unit risk values for these substances are available. The US EPA quantitative inhalation risk estimates are based on epidemiological studies of four nickel refineries (Huntington, W. Va., Inco, Copper Cliff, Ontario, Inco, Clydach, Wales and Falconbridge, Kristiansand, Norway) (US EPA, 1986). These cohorts were selected from a review of 18 industrial facilities involving workers mining, smelting, and refining nickel as well as workers in nickel related industries (metallic nickel powder, nickel alloy, electroplating, nickel-cadmium battery and stainless steel welding).

A cohort of employees of a nickel refinery in West Virginia who experienced a minimum one year exposure to nickel refinery dusts (containing nickel subsulphide, sulphate and oxide or only nickel oxide) did not show an increased incidence of lung cancer above expected rates (Enterline and Marsh, 1982). Chovil et al., (1981) studied a cohort of nickel refinery workers in Ontario, and observed a dose-related trend for the relationship between weighted exposure in years to the incidence of lung cancer. Similarly, a cohort of Welsh nickel refinery workers had elevated risks of cancer compared to the national average. Increased rates of nasal cancer were observed in men employed prior to 1920, while this rate was less than the national average for those starting work between 1920 and 1925, and equaled the expected value for those employed after 1925 (Doll et al., 1977). A significantly increased lung cancer related mortality was observed in employees starting prior to 1925 but not in those starting between the years 1930 to 1944. Pedersen et al., (1973) conducted a study of men employed at a nickel refinery in Norway, and reported an elevated occurrence of respiratory cancer for nickel exposed workers compared to expected values, and for workers involved in nickel processing steps compared to non-processing employees.

Two dose-response models (the excess additive risk model and the multiplicative or relative risk model) were evaluated. Two main assumptions were necessary:

1. For particulates such as nickel subsulphide and nickel refinery dust with relatively long lung clearance times, the cancer response was some function of a cumulative dose or exposure;

2. The magnitude of the response is a linear function of the cumulative dose.

The estimates of incremental unit risks for lung cancer due to exposure to 1 μg Ni/m^3 as nickel refinery dust for a life-time using either model ranged from 1.1 X 10^{-5} to 4.6 X 10^{-4}. The mid point of this range is 2.4 X 10^{-4}. Since nickel subsulphide was believed to be a major component of nickel refinery dust (roughly 50%) and the animal evidence at that time showed nickel subsulphide to be the most carcinogenic compound tested, US EPA recommended that the nickel refinery dust unit incremental risk estimate multiplied by two could be used for nickel subsulphide.

For nickel subsulphide, the inhalation unit risk is 4.8 x 10^{-4} (μg/m^3)$^{-1}$ (US EPA, 1998b - carcinogenic assessment last revised 1991).

A2-9.3.1.3 Health Canada

Health Canada (1996) has classified oxidic, sulphidic and soluble nickel as Group I (Carcinogenic to Humans). This group includes nickel oxide, nickel copper oxide, nickel silicate oxides and complex oxides, nickel sulphide, nickel sulphate and nickel chloride. This classification is based on the studies of Doll et al., (1990) and the International Agency for Research in Cancer (IARC) evaluation (IARC, 1990).

Health Canada (1996) has developed an inhalation tumorigenic concentration (TC_{05}) of 0.04 mg/m^3 for combined oxidic, sulphidic and soluble nickel. This TC_{05} is based on the studies of nickel refinery workers and workers exposed to metallic nickel powder or nickel alloys (Doll et al., 1990) (see section A2-9.2.3). Health Canada selected two large cohorts of exposed workers from Doll et al., (1990) for whom exposure information was available (Inco, Copper Cliff, Ontario, and Falconbridge, Kristiansand, Norway). The TD_{05} for lung cancer mortality for soluble nickel is 0.07 mg/m^3 (CEPA, 1994, Hughes et al., 1994, Health Canada, 1996).

Health Canada (1996) reports a non-cancer tolerable inhalation concentration of 0.018 µg/m^3 for nickel subsulphide.

A2-9.3.1.4 WHO

WHO (1987) developed an incremental unit risk of 4×10^{-4} per µg/m^3 for nickel subsulphide based on three epidemiological data sets for nickel refinery workers: the Kristiansand, Norway data (Magnus et al., 1982, the Copper Cliff, Ontario data (Chovil et al., 1981), and the Clydach, Wales data (Doll et al., 1977). The incremental unit risks for lung cancer calculated from each data set ranged from 1.5×10^{-4} to 5.9×10^{-4} per µg/m^3. The geometric mean of these three risk estimates was 4×10^{-4} per µg/m^3.

WHO (2000) reassessed this unit risk for lung cancer using updated epidemiology data from workers at the Kristiansand, Norway refinery first employed between 1968 and 1972 and followed through to 1987 (Andersen, 1992, Andersen et al., 1996). An incremental life-time unit risk of 3.8×10^{-4} per µg/m^3 was calculated using an estimated risk of 1.9 for this group and an exposure estimate of 2.5 µg/m^3 resulting in a life-time exposure of 155 µg/m^3. The WHO air quality guideline does not specify what form(s) of airborne nickel are addressed.

A2-9.3.1.5 Discussion

It is interesting to note that the lung cancer based inhalation exposure limits based on nickel mining, smelting and refining operations in North America, Wales and Norway, which were included in the WHO's, Health Canada's and the US EPA's derivation of quantitative risk estimates result in cancer potency estimates that are approximately five fold different. These differences in inhalation cancer potencies can be attributed to the use of different epidemiology data from the cohorts selected, and mathematical extrapolation procedures. The US EPA used the mid point of the range of maximum likelihood estimates (MLE) of a relative risk model from two risk models and for workplace data sets (US EPA, 1986).

WHO (2000) does not specify a confidence interval for their linear extrapolation from a single workplace data set. Health Canada also does not specify a confidence interval and used a model based on an exponential function of the cumulative exposure to total nickel from three Canadian workplace data sets and the Norwegian data (CEPA, 1994; Hughes et al., 1994).

In a paper describing Health Canada's approach to characterizing carcinogenic potency and development of the TD_{05}, it is stated: "any model that fits the empirical data well is likely to provide a reasonable estimate of the potency; choice of the model may not be critical since estimation is within the observed range, thereby avoiding the numerous uncertainties associated with low dose extrapolation" (Meek et al., 1994). A footnote in the same paper states, "the TD_{05} is not based on the confidence limit but rather, is computed directly from the curve. This was considered to be appropriate in view of the stability of the data in the experimental range and to avoid unnecessary conservative assumptions. Also, use of a point estimate or confidence limit does not affect the relative magnitude of the potency estimates for different compounds". Extrapolation of the TC_{05} to negligible risk levels (10^{-5} to 10^{-6}) is assumed to be strictly linear (Health Canada, 1996), whereas the US EPA unit cancer risk used a different mathematical extrapolation.

A2-9.3.1.6 Ingestion

No ingestion exposure limits for nickel refinery dusts and nickel subsulphide were located.

A2-9.3.2 Nickel Soluble Salts
A2-9.3.2.1 Inhalation

For the inhalation route, Health Canada (1996) recommends a tolerable inhalation concentration (non-cancer effects) of 0.0035 $\mu g/m^3$ for nickel sulphate. The TC was based on a subchronic study of lung and nasal lesions in rats and mice observed by Dunnick et al., (1989). *TERA* (1999) have developed an inhalation R/C of 0.2 $\mu g/m^3$ based on a benchmark concentration (BMC_{10}) for lung fibrosis in rats.

Health Canada developed a TC_{05} for lung cancer mortality for soluble nickel (mainly nickel sulphate and nickel chloride) of 0.07 mg/m^3. This TC_{05} is based on data from a cohort in Norway (Doll et al., 1990).

ATSDR (1997) has developed a chronic MRL for inhalation exposure of 2 x 10^{-4} mg/m^3 (0.2 $\mu g/m^3$) based on a study of nickel sulphate hexahydrate inhalation in rats (NTP,1996c).

A2-9.3.2.2 Ingestion

The US EPA (US EPA IRIS, 1998c - oral R/D assessment last revised 1996) recommended an oral R/D of 20 $\mu g/kg/day$ for soluble salts of nickel based on decreased body and organ weight data in two year dietary study in rats (Ambrose et al., 1976). This R/D was originally placed on IRIS in 1988 and may not necessarily protect the already sensitized individual.

For nickel sulphate, Health Canada (1996) derived a TDI of 50 $\mu g/kg/day$, based on the NOAEL from the Ambrose et al., (1976) two year dietary study in rats.

WHO (1998) re-evaluated and maintained their drinking water quality guideline for nickel of 0.02 mg/L, but state that this value is considered provisional owing to uncertainties about the effect level for perinatal mortality (in rats). WHO (1998) refers to a recent, well conducted study on rats exposed to nickel chloride in drinking water with a LOAEL for litter mortality of 1.3 mg nickel/kg body weight/day in the second litter (Smith et al., 1993). However, Smith et al., (1993) is a one generation, two litter study not a two generation study.

Earlier descriptions of this WHO drinking water quality guideline refer to a TDI of 5 μg nickel/kg/day (WHO, 1993, 1996). This TDI is described as being based on a dietary study in rats fed nickel chloride that showed a NOAEL of 5 mg/kg/day for altered organ-to-body-weight ratios (Ambrose et al., 1976) and was derived using an overall uncertainty factor of 1,000. This uncertainty factor was made up of 100 for inter and intraspecies variation. An additional factor of ten was to compensate for the lack of adequate studies on long term toxicity and reproductive effects, the lack of data on carcinogenicity by the oral route, and a much higher intestinal absorption when taken on an empty stomach in drinking water than when taken together with food. The earlier derivation of the WHO drinking water quality guideline allocated 10% of this TDI to drinking water exposure to derive 0.02 mg/L. In addition, it was stated that this drinking water guideline should provide sufficient protection for nickel-sensitive individuals (WHO, 1993, 1996).

It is of note that this TDI is not referenced in the 1998 WHO re-evaluation of its drinking water quality guideline, nor, is there any further description of this TDI elsewhere in the WHO literature. Since the 1998 WHO re-evaluation of its drinking water quality guideline for nickel discusses different animal studies than the 1993 and 1996 evaluations, the status of this TDI is unclear.

ATSDR (1997) did not determine oral MRLs for nickel because the protection of sensitized individuals and application of uncertainty factors to the LOAEL for contact dermatitis (0.009 mg/kg/day, Cronin et al., 1980) would result in an MRL which would bring the dose below normal dietary intake (about 0.002 mg/kg/day in the US).

Health Canada (1996) developed a TDI for soluble nickel of 1.3 μg Ni/kg body weight/day. This is based on a LOAEL of 1.3 mg/kg/day for an increased proportion of dead pups per litter in a one generation, two litter study of rats administered nickel chloride in drinking water (George et al., 1989; Smith et al., 1993). This TDI was derived using an overall 1,000-fold uncertainty factor which was based on ten fold for intraspecies variation, ten fold for interspecies variation and ten fold because a LOAEL rather than a NOAEL was observed.

The US National Academies' Institute of Medicine report on Dietary Reference Intakes (DRI) (IOM, 2001) did not have enough data to define whether there was a beneficial role for nickel and therefore did not establish recommended intake levels. However, based on adverse effect levels in animal studies, tolerable upper intake levels were set for nickel at 1 mg/day for adults and 0.2 mg/day for one to three year old children. DRIs expand on Recommended Dietary Allowances

(RDAs) in the US and Recommended Nutrient Intakes in Canada. This tolerable upper intake level (UL) for nickel represents about 17 µg Ni/kg/day for a 61 kg female adult. The UL for one-three year old children represents about 15 µg Ni/kg/day for a 13.3 kg US toddler. The UL applies to soluble nickel salt intake in excess of nickel consumed in a normal diet. The mean dietary nickel consumption of US infants and young children is cited as 69 to 90 µg/day based on a 1984 US FDA Total Diet Study (IOM, 2001), and as 190 to 251 µg/day for Canadian children (Dabeka and McKenzie, 1995). For adults US dietary nickel intakes are cited as 74 to 100µg/day and 207 to 406 µg/day for Canadians. The median supplemental intake for adult men and women was approximately 5 µg/day.

IOM (2001) used oral subchronic and chronic rat studies (ABC, 1988, Ambrose et al., 1976) to identify a NOAEL of 5 mg/kg/day for soluble nickel salts. An overall uncertainty factor (UF) of 300 was used, which incorporated a UF of ten for rat to human extrapolation, a second UF of ten for variation in human population, including uncertainties in nickel hypersensitivity in sensitive individuals, and a third UF of three because of uncertainties raised by studies of reproductive effects (in animals) and whether reproductive effects may occur at levels lower than the NOAEL (IOM, 2001).

IOM (2001) identified three animal reproduction studies (Schroeder and Mitchener, 1971, RTI, 1988, and Smith et al., 1993). The lowest LOAEL identified was 1.3 µg/kg/day (Smith et al., 1993) based on the number of dead pups in a litter. All of the reproduction studies were considered flawed or difficult to interpret due to their statistical design, and limitations in methods and data reporting, and not suitable for use in establishing a UL.

The ingestion exposure limits for soluble forms of nickel are summarized in Table A2-14. Refer to Section A2-9.2.2 for further information on the toxicity studies cited in this Table. The derivation and application of uncertainty factors to toxicity endpoints is further discussed in Section A2-1.

Table A2-14: Survey of the Rationales for Soluble Nickel Exposure Limits Developed by Major Agencies

RfD (µg/kg/day)	Study Selected	NOAEL/LOAEL/UF	Agency
50 (nickel sulphate)	Based on decreased body and organ weight data in two year dietary nickel sulphate study in Wistar rats (Ambrose et al., 1976)	NOAEL = 5 mg Ni/kg/day UF = 100 X10 for interspecies variation X10 for intraspecies variation	Health Canada (1996)
20 (soluble salts) Total nickel intake	Based on 1) decreased body and organ weight data in two year dietary nickel sulphate study in Wistar rats (Ambrose et al., 1976), and, 2) haematological effects in a 90 day study of nickel chloride in drinking water administered by gavage to CD rats (ABC, 1988)	NOAEL = 5 mg Ni/kg/day UF = 300 X10 for interspecies variation X10 to protect sensitive populations X3 for inadequacies in reproductive studies	US EPA IRIS (1998c)
Tolerable upper intake level intake in excess of the normal US diet (soluble salts) 17 for adult female (61 kg) 15 for 1-3 yrs US toddler = 13.3 kg	Based on 1) decreased body and organ weight data in two year dietary nickel sulphate study in Wistar rats (Ambrose et al., 1976), and, 2) haematological effects in a 90 day study of nickel chloride in drinking water administered by gavage to CD rats (ABC, 1988)	NOAEL = 5 mg Ni/kg/day UF = 300 X10 for interspecies variation X10 to protect sensitive populations X3 for inadequacies in reproductive studies (in animals) and whether reproductive effects may occur at levels lower than the NOAEL	(IOM, 2001)
5 (nickel sulphate) Total nickel intake	Based on decreased body and organ weight data in two year dietary nickel sulphate study in Wistar rats (Ambrose et al., 1976)	NOAEL = 5 mg Ni/kg/day UF = 1,000 X10 for interspecies variation X10 for intraspecies variation X10 for insufficient studies for long term toxicity and reproductive effects, lack of data on carcinogenicity by the oral route, and higher intestinal absorption when taken in drinking water on an empty stomach	WHO (1996)

Table A2-14: Survey of the Rationales for Soluble Nickel Exposure Limits Developed by Major Agencies

RfD (µg/kg/day)	Study Selected	NOAEL/LOAEL/UF	Agency
? = not specified (studies involving nickel sulphate and nickel chloride) Total Nickel intake and Nickel intake in addition to diet	Based on 1) decreased body and organ weight data in two year dietary nickel sulphate study in Wistar rats (Ambrose et al., 1976); 2) mainly haematological changes in several subchronic studies in rats, and, a 90 day study of rats administered nickel chloride in drinking water (RTI, 1988 described in Velazquez and Poirier, 1994, ATSDR, 1997), and 3) increased pup mortality in a one generation, two litter reproduction study of female Long Evans rats treated with nickel chloride in drinking water for 11 weeks prior to and during two successive gestational periods (Smith et al., 1993)	1) NOAEL = 5 mg Ni/kg/day 2) NOAEL = 10, 10 and 5 mg Ni/kg/day 3) LOAEL = 1.3 mg Ni/kg/day UF not stated, probably 1,000	WHO (1998)
1.3 (nickel chloride)	Increased pup mortality in a 1-generation, 2 litter reproduction study of female Long Evans rats treated with nickel chloride in drinking water for 11 weeks prior to and during two successive gestational periods (Smith et al., 1993)	LOAEL = 1.3 mg Ni/kg/day UF = 1,000 X10 for interspecies variation; X10 for intraspecies variation; X10 for LOAEL rather than NOAEL	Health Canada (1996)

Studies selected for review

RfD (µg/kg/day)	Study Selected	NOAEL/LOAEL/UF	Agency
Eight (nickel soluble salts) Nickel intake in addition to diet	Based on increased albuminuria in Wistar rats administered nickel sulphate in drinking water for six mo. (Vyskocil et al., 1994)	LOAEL = 7.6 mg Ni/kg/day UF = 1,000 X10 for interspecies variation X10 for intraspecies variation X10 for subchronic to chronic extrapolation, insufficient database and use of a LOAEL	TERA (1999) sponsored by US EPA, Health Canada and the Metal Finishers of Southern California

Table A2-14: Survey of the Rationales for Soluble Nickel Exposure Limits Developed by Major Agencies

RfD (µg/kg/day)	Study Selected	NOAEL/LOAEL/UF	Agency
22 (nickel sulphate)	Based on a NOAEL for parental survival, growth, mating behavior, fertility, implantation, gestation length, and post-implantation/perinatal lethality in a two generation reproduction study of Sprague Dawley rats administered nickel sulphate by gavage (Springborn, 2000)	NOAEL = 2.2 mg Ni/kg/day UF = 100 X10 for interspecies variation X10 for intraspecies variation	Springborn (2000) sponsored by NiPERA and under review by US EPA

A2-9.3.3 Nickel Oxide
A2-9.3.3.1 Inhalation

A tolerable inhalation concentration (TC_{05}) (non-cancer effects) of 0.02 $\mu g/m^3$ based on changes in alveolar macrophages, granulocytes and lymphocytes in rat lungs (Spiegelberg et al., 1984) has been developed (Health Canada, 1996).

Health Canada (1996) has classified oxidic nickel (including nickel oxide, nickel copper oxide, nickel silicate oxides and complex oxides as Group I (Carcinogenic to Humans). This classification is based on the studies of Doll et al., (1990) and the International Agency for Research in Cancer (IARC) evaluation (IARC, 1990).

It should be clarified that all toxicological information regarding the carcinogenicity of nickel oxide, either as a component of nickel refinery dusts or as a pure compound administered to rats and mice is only by the inhalation route. In addition, while nickel oxide has carcinogenic potential when inhaled (based on human and animal studies), there are no published inhalation unit cancer risks by which to assess its potency.

A2-9.3.3.2 Ingestion

There is no information regarding its carcinogenicity via the ingestion route in humans or animals.

A2-9.3.4 Metallic Nickel
A2-9.3.4.1 Inhalation

Health Canada (1996) classified metallic nickel as Group IV (unclassifiable with respect to carcinogenicity in humans). Health Canada (1996) reports a provisional non-cancer tolerable concentration (inhalation) of 0.018 $\mu g/m^3$.

A2-9.3.4.2 Ingestion

No ingestion exposure limits for metallic nickel were located.

A2-9.3.5 Dermal Exposure Limits

Contact dermatitis caused by nickel exposure is discussed in section A2-9.2.4, above.

The European Directive 94/27/EC (European Union, 1994) of June 30, 1994 addresses the issue of (metallic) nickel in certain objects (mainly jewelry, but including clothes fasteners) coming into direct and prolonged contact with the skin and causing sensitization and allergic responses in humans. This directive requires that:

(1) jewelry that pierces the skin must contain less than 0.05% nickel (500 μg/g) on a mass basis;
(2) products coming into direct and prolonged contact with the skin are restricted to release nickel at a rate of less than 0.5 μg/cm^2/week; and
(3) products listed in (2) with a non-nickel coating must also ensure that the nickel release rate not exceed 0.5 μg/cm^2/week for at least two years normal usage of the product.

This directive is not directly applicable to the situation where soil nickel gets on the skin.

A2-9.3.6 Selection of Exposure Limits
A2-9.3.6.1 Inhalation

To assess the potential for cancer effects related to inhalation of nickel oxide, the annual average ambient air concentration (TSP sampler) from MOE monitoring station 27047 (at Davis and Fraser Streets) data was compared to inhalation life-time cancer risk factors from various agencies (tabulated in Table A2-15). These inhalation unit risks may not be based on respirable nickel compounds since total airborne nickel was measured in the workplace. TSP sampler data is the most comparable data for use with workplace exposure data (section A2-9.2.1.1).

A2-9.3.6.2 Ingestion

For the purposes of this risk assessment, the US EPA R$_f$D for soluble nickel was selected to assess potential non-cancer effects from estimated nickel intakes from all exposure routes. This is essentially the same exposure limit as the tolerable upper intake level for soluble nickel recently derived by IOM (2001) (section A2-9.3.2).

Animal studies show that the absorption of orally administered nickel compounds is closely related to the solubility of the compound (Haber et al., 2000). The only studies indicating a lower bioavailability and corresponding lower toxicity of insoluble forms of nickel are Mastromatteo (1986), Ishimatsu et al., (1995) and Griffin et al., (1990). These studies are cited and described in section A2-9.2.2 and in Appendix 5. There is some suggestion that some insoluble nickel forms, e.g., nickel subsulfide, have limited solubility in body fluids, however, this appears to be in the context of inhaled insoluble nickel forms being taken into the lung tissue by phagocytosis (Yamada et al., 1993). Even though the predominant form of nickel in Rodney Street community soils is the insoluble nickel oxide, it is anticipated that the main way that nickel is absorbed is by diffusion as the soluble nickel (II) ion moiety which mainly occurs under acidic conditions. As the acidic stomach contents empty into the small intestine, the first few centimeters of the small intestine constitute the optimum location for metal absorption. As the pH becomes alkaline, the metal ions become less available due to formation of less soluble compounds and will pass through to the colon without being absorbed. Since the scientific evidence suggests that it is likely that only soluble nickel forms are absorbed from the ingested soil, the use of a soluble nickel R$_f$D is appropriate.

This preferential uptake of soluble nickel forms from the digestive and respiratory systems with subsequent excretion in the urine is well documented for humans and animals (section A2-9.1). There is difference of opinion on the suitability of some reproductive studies involving nickel chloride administered in drinking water for use in deriving exposure limits (section A2-9.2.2). Recent literature suggests that the animal reproduction study and the uncertainty factors used to derive the Health Canada TD_{05} for nickel chloride and the WHO TDI should be reviewed.

As stated in chapter 3 and section A2-1, the development of new exposure limits was not considered. Each toxicity study used to support exposure limits (R/D, TDI, etc.), has been subjected to detailed evaluation and review by the agency involved and some discussion of the reliability, strengths and weaknesses and confidence rating of the studies can be found (US EPA, 1998c - oral R/D assessment last revised 1996, CEPA, 1994, Hughes et al., 1994, WHO, 1996, 1998, IOM, 2001). For the chronic toxicity and reproductive toxicity studies selected to support exposure limits, the NOAEL/LOAEL range is 1.3 mg/kg/day (LOAEL, Smith et al., 1993) to 5 mg/kg/day (NOAEL, Ambrose et al., 1976, ABC, 1988) (Table A2-14). Other assessments have considered basing R/Ds on values ranging from 2.2 mg/kg/day (NOAEL, Springborn, 2000) to 7.6 mg/kg/day (LOAEL, *TERA*, 1999 and Vyskocil et al., 1994) (section A2-9.2.2). In conjunction with UFs of ten fold for inter and intra species extrapolation, additional UFs have been used to account for other shortcomings or deficiencies in the supporting study selected (this is discussed in section A2-1).

For the studies selected to develop ingestion exposure limits for soluble nickel, the NOAEL for chronic toxicity of 5 mg/kg/day is well established (Ambrose et al., 1976). This NOAEL value was recently endorsed by IOM (2001). Lack of a defined NOAEL for reproductive toxicity is a data gap which has required the use of an additional UF (uncertainty factor) due to uncertainty about the NOAEL for this endpoint (WHO, 1996, Health Canada, 1996). The new unpublished study of Springborn (2000) is currently under review by US EPA as part of a re-assessment of the 1988 R/D for soluble nickel ingestion on IRIS (Ambika Bathija - personal communication). Springborn (2000) demonstrates a clear NOAEL for reproductive toxicity. This suggests that a UF to account for this data gap may no longer be necessary or that the magnitude of the UF adjusting for lack of this information can be revised resulting a smaller overall UF. Consequently, the NOAEL and UFs used in the US EPA R/D for soluble nickel continue to provide an adequate margin of safety to protect human health.

Other considerations include:

Other oral exposure limits below this value are directed towards intake, e.g., drinking water intakes, and may be unsuited for total exposure considerations.

Any exposure limit for soluble nickel has to consider the oral hypersensitivity of some nickel sensitized people. This may be offset by other studies showing that nickel supplements can lead to hyposensitization of other sensitized people (section A2-9.2.4).

Table A2-15: Selected Exposure Limits for Nickel Compounds

Route of Exposure	Exposure Limit	Toxicological Basis	Source Agency
Non-Cancer Effects			
Ingestion	20 µg Ni/kg-day	reproductive effects and decreased body and organ weight in rats	US EPA, 1998c, IOM, 2001
Inhalation	-	-	-
Dermal Contact	-	-	-
Cancer Effects			
Ingestion	N.A.[1]	-	-
Inhalation[2]	2.4×10^{-4} $(\mu g/m^3)^{-1}$; 3.8×10^{-4} $(\mu g/m^3)^{-1}$ and TC_{05} $(0.04 \ mg/m^3)$	lung cancer in nickel refinery workers	US EPA, 1998a; WHO, 2000 and Health Canada, 1996
Dermal Contact	N.A.	-	-

1. Not Applicable.
2. Neither US EPA, Health Canada nor WHO state their limits as µg Ni/m³ or risk per µg Ni/m³. Doll et al., (1990) does specify airborne Ni levels as such. It may be reasonably assumed that the nickel refinery worker personal air samplers were analyzed using a total Ni approach and that speciation was imposed retroactively. The implication is that the analytical data is total Ni and no further conversion is needed.

A2-9.4 Nickel References

ACGIH. 1998. Particle size selective sampling for particulate air contaminants. American Conference of Governmental Industrial Hygienists, Cincinnati, Ohio, USA.

Ambrose, A.M., P.S. Larson, J.F. Borzelleca, and G.R. Hennigar, 1976. Long term toxicologic assessment of nickel in rats and dogs. J Food Sci Technol. 13:181-187.

American Biogenics Corporation (ABC). 1988. Ninety day gavage study in albino rats using nickel. Final report submitted U.S. Environmental Protection Agency, Office of Solid Waste. Submitted by Research Triangle Institute and American Biogenics Corporation.

Andersen. A. 1992. Recent follow-up of nickel refinery workers in Norway and respiratory cancer. In: Nieboer, E. and Nriagu, J.O., ed. Nickel and human health: Current perspectives. New York, Wiley, pp. 621-628.

Andersen A., S.R. Berge, A. Engeland, T. Norseth. 1996. Exposure to nickel compounds and smoking in relation to incidence of lung and nasal cancer among nickel refinery workers, Occup. Environ. Med. 53:708-713.

Anttila, A., E. Pukkala, A. Aitio, T. Rantanen and S. Karjalainen. 1998. Update of cancer incidence among workers at a copper/nickel smelter and nickel refinery. Int. J. Occup. Environ. Health. 71(4):245-250.

Arena, V.C, N.B. Sussman, C.K. Redmond, J.P. Constantino and J.M. Trauth. 1998. Using alternative comparison populations to assess occupation-related mortality risk. Results for the high nickel alloys cohort. J. Occup. Environ. Medicine. 40(10):907-916.

ATSDR (Agency for Toxic Substances and Disease Registry). 1997. U.S. Department of Health and Human Services. Toxicological Profile for Nickel. Atlanta, Georgia, USA.

Bagot M., D. Charue, M.L. Flechet, N. Terki, A. Toma, J. Revuz, 1995. Oral desensitization in nickel allergy induces a decrease in nickel-specific T cells. European J. Dermatology. 5:614-8.

Bencko, V., V. Wagner, M. Wagnerova, E. Reichrtova. 1983. Immuno-biochemical findings in groups of individuals occupationally and non-occupationally exposed to emissions containing nickel and cobalt. J. Hyg. Epidemiol. Microbiol. Immunol. 27:387-394.

Bencko, V., V. Wagner, M. Wagnerova, V. Zavázal. 1986. Human exposure to nickel and cobalt: Biological monitoring and immunobiological response. Environ. Res. 40:399-410.

Benson, J.M., E.B. Barr, W.E. Bechtold, Y.S. Cheng, J.K. Dunnick, W.E. Eastin, C.H. Hobbs, C.H. Kennedy, K.R. Maples, 1994. Fate of inhaled nickel oxide and nickel subsulfide in F344/N rats. Inhalat. Toxicol. 6:167-183.

Burrows, D., S. Creswell and J.D. Merrett. 1981. Nickel, hands and hip prostheses. Br. J. Dermatol. 105: 437-444.

Casarett and Doull's Toxicology, 4th Ed. 1991. Eds. Amdur, M.O., J. Doull and C.D. Klaassen. Pergamon Press.

CEPA (Canadian Environmental Protection Act). 1994. Nickel and its compounds. Priority substances list assessment report. Government of Canada: Environment Canada, Health Canada. ISBN 0-662-22340-3.

Chashschin, V.P., G.P. Artunina, T. Norseth, 1994. Congenital defects, abortion and other health effects in nickel refinery workers. Sci. Total Environ. 148:287-291.

Chovil, A., R.B. Sutherland, and M. Halliday, 1981. Respiratory cancer in a cohort of nickel sinter plant workers. Brit J Ind Med. 38:327-333.

Cornell, R.G. and J.R. Landis. 1984. Mortality patterns among nickel/chromium alloy foundry workers. In: Sunderman, F.W., Jr., A. Aitio, A. Berlin, eds. Nickel in the human environment. IARC scientific publication no. 53. Lyon, France: International Agency for Research on Cancer. 87-93.

Cronin, E., A.D. DiMichiel, and S.S. Brown, 1980. In: Nickel Toxicology. Brown, S.S. and Sunderman, W.R., Eds., Academic Press, NY, 149.

Dabeka, R.W and A.D. McKenzie. 1995. Survey of lead, cadmium, fluoride, nickel and cobalt in food composites and estimation of dietary intakes of these elements by Canadians in 1986-1988. J.A.O.A.C. 78: 897-909.

Daldrup, T., K. Haarhoff, S.C. Szathmary. 1983. Toedliche nickel sulfaye-intoxikation. Berichte zur Serichtlichen Medizin. 41:141-144.

Diamond, G.L., P.E. Goodrum, S.P. Felter, W.L. Ruoff, 1998. Gastrointestinal absorption of metals. Drug Chem. Toxicol. 21:223-251.

Doll, R., J.D. Matthews, and L.G. Morgan, 1977. Cancers of the lung and nasal sinuses in nickel workers: A reassessment of the period of risk. Brit. J. Ind Med. 34:102-105.

Doll, R., A. Anderson, W.C. Copper, I. Cosmatos, D.L. Cragle, D. Easton, P. Enterline, M. Goldberg, L. Metcalfe, T. Norseth, J. Peto, J-P. Rigaut, R. Roberts, S. Seilkop, H. Shannon, F. Speizer, F.W. Sunderman Jr. P. Thornhill, J.S. Warner, J. Weglo, and M. Wright, 1990. Report of the international committee on nickel carcinogenesis in man. Scand J Work Environ Health. 16:1-82.

Dolovich, J., S.L. Evans, E. Nieboer. 1984. Occupational asthma from nickel sensitivity: I. Human serum albumin in the antigenic determinant. Br. J. Ind. Med. 41:51-55.

Dotterud, L.K., E.S. Falk, 1994. Metal allergy in north Norwegian schoolchildren and its relationship with ear piercing and atopy. Contact Dermatitis. 31:308-313.

Draper, M.H., J.H. Duffus, P. John, L.P. Metcalfe, L.G. Morgan, M..V. Park and M.I. Weitzner. 1994. Characterization of historical samples of nickel refinery dusts from the Clydach refinery. Exp. Toxic. Pathol. 46:111.

Dunnick, J.K., M.R. Elwell, J.M. Benson, C.H. Hobbs, F.F. Hahn, P.J. Haly, Y.S. Cheng, and A.F. Eidson, 1989. Lung toxicity after 13-week inhalation exposure to nickel oxide, nickel subsulfide, or nickel sulphate hexahydrate in F344/N rats and B6C3F1 mice. Fund. Appl. Toxicol. 12(3):584-594.

Dunnick, J.K., M. Elwell, A.E. Radovsky, J. M. Benson, F.F. Hahn, K.J. Nikula, E.B. Barr, C.H. Hobbs. 1995. Comparative carcinogenic effects of nickel subsulfide, nickel oxide, and nickel sulfate hexahydrate chronic exposures in the lung. Cancer Res. 55:5251-5256.

Easton, D.F., J. Peto, L.G. Morgan, L.P. Metcalfe, V. Usher and R. Doll. 1992. Respiratory cancer in Welsh nickel refiners: Which nickel compounds are responsible? In: Nieboer, E. and Nriagu, J.O., ed. Nickel and human health: Current perspectives. New York, Wiley, pp. 621-628.

Emmett E.A., T.H. Risby, L. Jiang, S.K. Ng, S. Feinman. 1988. Allergic contact dermatitis to nickel: Bioavailability from consumer products and provocation threshold. J. Am. Acad. Dermatol. 19(2): 314-322.

Enterline, P.E., and G.M. Marsh, 1982. Mortality among workers in a nickel refinery and alloy manufacturing plant in West Virginia. J. Nat. Cancer Inst. 68(6):925-933.

Eun H.C., R. Marks. 1990. Dose-response relationships for topically applied antigens. Br. J. Dermatol. 122: 491- 499.

European Commission, DG Environment. 2000. Ambient air pollution by As, Cd and Ni compounds. Final Position Paper of the Working Group on Arsenic, Cadmium and Nickel. http://europa.eu.int/comm/environment/air/pp_as_cd_ni.pdf (Accessed October 18, 2001).

European Union. 1994. European Parliament and Council Directive 94/27/EC of 30 June, 1994. http://europa.eu.int/eur-lex/en/lif/dat/1994/en_394L0027.html. (Accessed October 18, 2001).

Fuchtjohann, L., N. Jakubowski, D. Gladtke, D. Klockow and J.A.C. Brockaert. 2001. Speciation of nickel in airborne particulate matter by means of a micro flow system and determination by graphite furnace atomic absorption spectroscopy and inductively coupled spectrometry. J. Environ. Monit. 3:681-687.

Fullerton, A., J.R. Andersen, A. Hoelgaard, T. Menne. 1986. Permeation of nickel salts through human skin *in vitro*. Contact Dermatitis. 15:173-177.

Gawkrodger, D.J., S.W. Cook, G.S. Fell, J.A. Hunter, 1986. Nickel dermatitis: The reaction to oral nickel challenge. Br. J. Dermatol. 115:33.

George, E.L., J.A. Stober, G.L. Kimmel, M.K. Smith, 1989. The developmental effects of nickel chloride in drinking water. Toxicologist. 9(1):272.

Ghezzi, I., A. Baldasseroni, G. Sesana, et al., 1989. Behavior of urinary nickel in low-level occupational exposure. Med. Lav. 80:244-250.

Gilman, J.P.W. and G.M. Ruckerbauer. 1962. Metal carcinogenesis. I. Observations on the carcinogenicity of a refinery dust, cobalt oxide, and colloidal thorium dioxide. Cancer Res. 22: 152-157.

Gilman, J.P.W. and S. Yamashiro. 1985. Muscle Tumorigenesis by Nickel Compounds. In: Progress in Nickel Toxicology: Proceedings of the Third International Conference on Nickel Metabolism and Toxicology, September 1984, Paris, France. S.S. Brown and F.W. Sunderman, Jr. Ed., Blackwell Scientific Publication, Oxford, United Kingdom. pp. 9-22.

Griffin, S.R., R. Rubenstein, S. Irene, C. DeRosa, and H. Choudhury, 1990. Bioavailability in rats of metals adsorbed to soils. U.S. Environmental Protection Agency, Washington, D.C., Hazelton Laboratories, America, Inc. Poster presented at the Society of Toxicology 29th Annual Meeting, Miami Beach, FL, February 12-16. Poster paper No. 623.

Grimsrud., T.K., S.R. Berge, F. Resmann, T. Norseth, and A. Andersen. 2000. Assessment of historical exposures in a nickel refinery in Norway. Scand. J. Work Environ. Health 26(4): 338-345.

HAQI (Hamilton-Wentworth Air Quality Initiative). 1997. Human health risk assessment for priority pollutants. Report of the Human Health Working Group. December 1997.

Health Canada. 1996. Health-Based Tolerable Daily Intakes/Concentrations and Tumorigenic Doses/Concentrations for Priority Substances. ISBN 0-662-24858-9.

Health Canada. 1999. National ambient air quality objectives for particulate matter. Part 1: Science assessment document. A report of the CEPA/FPAC Working Group on air quality objectives and guidelines. Cat.No. H46-2/98-220-1E. (Available online at http://www.hc-sc.gc.ca/ehp/ehd/catalogue/bch_pubs/99ehd220-1.htm). (Accessed October 17, 2001.)

Hendel, R.C. and F.W. Sunderman, Jr., 1972. Species variations in the proportions of ultrafiltrable and protein-bound serum nickel. Res. Commun. Chem. Pathol. Pharmacol. 4:141-146.

Higinbotham, K.G., J.M. Rice, B.A. Diwan, K.S. Kasprzak, C.D. Reed, A.O. Perantoni, 1992. GGT to GTT transversions in codon 12 of the K-ras oncogene in rat renal sarcomas induced with nickel subsulphide or nickel subsulphide/iron are consistent with oxidative damage to DNA. Cancer Res. 52:4747-4751.

Höflich, B.L.W., M. Wentzel, H.M. Ortner, S. Weinbruch, A. Skogstad, S. Hetland, Y. Thomassen, V.P. Chaschin, E. Nieboer. 2000. Chemical composition of individual aerosol particles from working areas in a nickel refinery. J. Environ. Monit. 2:213-217.

Horowitz, S.B., and B.L. Finley. 1994. Setting health protective soil concentrations for dermal contact allergens: A proposed methodology. Regul. Toxicol. Pharmacol. 19:31-47.

Hughes, K., M.E. Meek, P.K.L. Chan, J. Shedden, S. Bartlett, and L.J. Seed, 1994. Nickel and its compounds: Evaluation of risks to health from environmental exposure in Canada. Environ. Carcino. Ecotox. Revs. C12:417-433.

IARC (International Agency for Research on Cancer). 1990. Nickel and nickel compounds. Monographs on the evaluation of the carcinogenic risk of chemicals to humans. 49:257-445.

International Commission on Radiological Protection (ICRP). 1984. Report of the Task Group on Reference Man (ICRP Publication No. 23). Pergamon Press.

IOM (Institute of Medicine - Food and Nutrition Board). 2001. Dietary Reference Intakes for Vitamin A, Vitamin K, Arsenic, Boron, Chromium, Copper, Iodine, Iron, Manganese, Molybdenum, Nickel, Silicon, Vanadium, and Zinc. National Academy Press, Washington, D.C.

Ishimatsu, S., T. Kawamoto, K. Matsuno, & Y. Kodama, 1995. Distribution of various nickel compounds in rat organs after oral administration. Biol. Trace Element Res. 49:43-52.

Jordan, W.P. and S.E. King, 1979. Nickel feeding in nickel-sensitive patients with hand eczema. J. Am. Acad. Dermatol.1:508.

Julian, J.A., and D.C.F. Muir. 1996. Report to the Occupational Disease Panel of the Province of Ontario: "A study of cancer incidence in Ontario nickel workers". January 15, 1996.

Kaaber, K., N.K. Veien, and J.C. Tjell, 1978. Low nickel diet in the treatment of patients with chronic nickel dermatitis. Br. J. Dermatol. 98:197.

Keczkes, K., A.M. Basheer, E.H. Wyatt. 1982. The persistence of allergic contact sensitivity: A 10 year follow-up in 100 patients. Br. J. Dermatol. 107:461-465.

Kiilunen, M., A. Atio, and A. Tossavainen. 1997. Occupational exposure to nickel salts in electrolytic plating. Ann. Occup. Hyg. 41:189-200.

Larsson-Stymne, B.and L. Widstrom, 1985. Ear piercing-A cause of nickel allergy in schoolgirls? Contact Dermatitis. 13(5):289-293.

Liden, C. 1994. Occupational contact dermatitis due to nickel allergy. Sci. Tot. Environ. 148:283-285.

Magnus, K., A. Andersen, A. Hogetveit. 1984. Cancer of respiratory organs among workers at a nickel refinery in Norway. Int. J. Cancer. 30:681-685.

Massachusetts Department of Environmental Protection (MADEP). 1998. Evaluation of the risk assessment methodologies based on the hexavalent chromium [Cr(VI)]-elicited allergic contact dermatitis (ACD) in sensitized population and MADEP's recommendations. http://www.state.ma.us/dep/ors/files/crviderm.doc (Accessed October 17, 2001).

Mastromatteo, E. 1986. Yant memorial lecture: Nickel. Am. Ind. Hyg. Assoc. J. 47:589-601.

Meek, M.E., R. Newhook, R.G. Liteplo, and V.C. Armstrong, 1994. Approach to assessment of risk to human health for priority substances under the Canadian Environmental Protection Act. Environ. Carcino. Ecotox. Revs. C12:105-134.

Meijer, C., M. Bredberg, T. Fischer, L. Widström, 1995. Ear piercing, and nickel and cobalt sensitization, in 520 young Swedish men doing compulsory military service. Contact Dermatitis. 32(3):147-149.

Menne, T., F. Brandup, K. Thestrup-Pedersen, N.K. Veien, J.R. Andersen, F. Yding, G. Valeur, 1987. Patch test sensitivity to nickel alloys. Contact Dermatitis. 16:255-259.

Menne, T., and H.I. Maibach, 1987. Systemic contact allergy reactions. Semin. Dermatol. 6:108-118.

Menne, T., and H.I. Maibach, 1989. Nickel allergic contact dermatitis: A review. J. Am. Coll. Toxicol. 8:1271-1273.

Menne, T., J. Christophersen, A. Green, 1989. Epidemiology of nickel dermatitis. In: Maibach, H.I., Menne, T., eds. 1989. Nickel and the Skin: Immunology and toxicology. Boca Raton, FL: CRC Press, Inc. pp. 109 -115.

Menne, T. 1994. Quantitative aspects of nickel dermatitis: Sensitization and eliciting threshold concentrations. Sci. Tot. Environ.148:275-281.

MOEE, 1994. Windsor air quality study: Personal exposure survey results. Ontario Ministry of Environment and Energy. Fall 1994.

Mozzanica, N., L. Rizzolo, G. Veneroni, R. Diotti, S. Hepeisen, A.F. Finzi, 1990. HLA-A, B, C and DR antigens in nickel contact sensitivity. Br. J. Dermatol. 122(3):309-313.

New Jersey Department of Environmental Protection (NJDEP). 1998. Summary of the basis and background of the soil cleanup criteria for trivalent and hexavalent chromium. http://www.state.nj.us/dep/srp/chrome/b_b_sum.pdf (Accessed March 5, 2002).

Nieboer, E. 1992. Occupational exposures to nickel. In: Nieboer, E. and Nriagu, J. O., ed. Nickel and human health: Current perspectives. New York, Wiley, pp. 37-47.

Nielsen, F.H. 1985. The importance of diet composition in ultratrace element research. J. Nutr. 115:1239-1247.

Nielsen, N.H. and T. Menne. 1993. Nickel sensitization and ear piercing in an unselected Danish population. Contact Dermatitis 29:16-21.

Novey, H.S., M. Habib, I.D. Wells. 1983. Asthma and IgE antibodies induced by chromium and nickel salts. J. Allergy Clin. Immunol. 72:407-412.

National Research Council. 1996. Guide for the Care and Use of Laboratory Animals. Institute of Laboratory Animal Resources, Commission on Life Sciences, National Research Council, National Academy Press, Washington, D.C.

NTP. 1996a. Toxicology and Carcinogenesis Studies of Nickel Oxide in F344/N Rats and B6C3F1 Mice (CAS No.1313-99-1). US Department of Health and Human Services. National Toxicology Program. Technical Report Series. No. 451.

NTP. 1996b. Toxicology and Carcinogenesis Studies of Nickel Subsulfide in F344/N Rats and B6C3F1 Mice (CAS NO. 12035-72-2). US Department of Health and Human Services. National Toxicology Program. Technical Report Series. No. 453.

NTP. 1996c. Toxicology and Carcinogenesis Studies of Nickel Sulfate Hexahydrate in F344/N Rats and B6C3F1 Mice (CAS No. 10101-97-0). US Department of Health and Human Services. National Toxicology Program. Technical Report Series. No. 454.

Odland, J.O. 1999. Environmental and occupational exposure, life-style factors and pregnancy outcome in Arctic and Subarctic populations of Norway and Russia. Institute of Community Medicine, University of Tromso, Norway. ISBN 82-90262-57-4. 2000.

Oller, A.R., M. Costa, and G. Oberdorster, 1997. Carcinogenicity assessment of selected nickel compounds. Toxicol. Appl. Pharmacol. 143:152-166.

Ontario Disease Panel (ODP). 1997. Report to the Workers' Compensation Board on Cancer of the Larynx in Workers in Primary Nickel Production. Ontario Disease Panel Report No. 19, July, 1977. ISBN 0-7778-6413-4.

Ottolenghi, A.D., J.K. Haseman, W.W. Payne, H.L. Falk, and H.N. MacFarland, 1974. Inhalation studies of nickel sulfide in pulmonary carcinogenesis of rats. J. Nat Cancer Inst. 54(5):1165-1170.

Pang, D., D.C.L. Burges and T. Sorahan. 1996. Mortality study of nickel platers with special reference to cancers of the stomach and lung. Occup. Environ. Medicine. 53: 714-717.

Panzani, R.C., D. Schiavino, E. Nucera, S. Pellegrino, G. Fais, G. Schinco, G. Patriarca, 1995. Oral hyposensitization to nickel allergy: Preliminary clinical results. International archives of allergy and applied immunology: 107:251-254.

Pedersen, E., A.C. Hogetveit and A. Andersen. 1973. Cancer of respiratory organs among workers at a nickel refinery in Norway. Int. J. Cancer. 12: 32-41.

Peltonen, L. 1979. Nickel sensitivity in the general population. Contact Dermatits. 5:27-32.

Polednak, A.P. 1981. Mortality among welders, including a group exposed to nickel oxides. Arch. Environ. Health. 36:235-242.

Pott, F., U. Ziem, F.J. Reiffer, F. Huth, H. Ernst, and U. Mohr. 1987. Carcinogenicity studies on fibres, metal compounds and some other dusts in rats. Exp. Pathol. 32:129-152.

Rezuke, W.N., J.A. Knight, and F.W. Sunderman, Jr., 1987. Reference values for nickel concentrations in human tissues and bile. Am. J. Ind. Med. 11:419-426.

RTI (Research Triangle Institute). 1988. Two generation reproduction and fertility study of nickel chloride administered to CD rats in the drinking water. Final study report (Vol I to III). Report to Office of Solid Waste Management, US Environmental Protection Agency by Research Triangle Institute.

Santucci, B., A. Cristaudo, C. Cannistraci, M. Picardo, 1988. Nickel sensitivity: Effects of prolonged oral intake of the element. Contact dermatitis: 19:202-205.

Santucci, B., F. Manna, C. Cannistraci, A. Cristaudo, R. Capparella, A. Bolasco, M. Picardo, 1994. Serum and urine concentrations in nickel - sensitive patients after prolonged oral administration. Contact Dermatitis. 30:97-101.

Sarkar, B. 1984. Nickel metabolism. In: Sunderman, F.W. Jr., A. Aitio, A. Berlin, eds. Nickel in the human environment. IARC scientific publication no. 53. Lyon, France, March 1983: International Agency for Research on Cancer. 367-384.

Schnegg, A. and M. Kirchgessner. 1975. Changes in hemoglobin content, erythrocyte count and hemocrit in nickel deficiency. Nutr. Metab. 19:268-278.

Schroeder, H.A., J.J. Balassa, and W.H. Vinton, 1964. Chromium, lead, cadmium, nickel and titanium in mice: Effect on mortality, tumours and tissue levels. J Nutr. 83:239-250.

Schroeder, H.A., and M. Mitchener, 1971. Toxic effects of tract elements on the reproduction of mice and rats. Arch. Environ. Health. 23:102.

Schroeder, H.A., M. Mitchener, and A.P. Nason, 1974. Life-term effects of nickel in rats: Survival, tumors, interactions with trace elements and tissue levels. J. Nutr. 104:239-243.

Schroeder, H.A., and M. Mitchener, 1975. Life-term effects of mercury, methyl mercury, and nine other trace metals on mice. J. Nutr. 105:452-458.

Shirakawa, T., Y. Kusaka, N. Fujimura, M. Kato, S. Heki, K. Morimoto, 1990. Hard metal asthma - cross immunological and respiratory activity between cobalt and nickel. Thorax. 45:267-271.

Sjöwall, P., O.B. Christensen, H. Möller, 1978. Oral hyposensitization in nickel allergy. Journal of the American Academy of Dermatology. 17:774-778.

Smith, M.K., E.L. George, J.A. Stober, H.A. Feng, and G.L. Kimmel, 1993. Perinatal toxicity associated with nickel chloride exposure. Environ. Res. 61:200-211.

Smith-Sivertsen, T., V. Tchachtchine, E. Lund, T. Norseth and V. Bykov. 1997. The Norwegian - Russian Health Study 1994/95: A cross-sectional study of pollution and health in the border area. University of Tromso, Norway, Kola Research Laboratory for Occupational Health, Kirovsk, Russia, National Institute of Occupational Health, Oslo, Norway. ISBN 82-90262-48-5.

Smith-Sivertsen, T., L.K. Dotterud, and E. Lund. 1999. Nickel allergy and its relationship with local nickel pollution, ear piercing, and atopic dermatitis: A population-based study from Norway. J. Am. Acad. Dermatol. 40:726-35.

Snipes, M.B., A.C. James, and A.M. Jarabek. 1997. The 1994 ICRP66 human respiratory tract dosimetry model as a tool for predicting lung burdens from exposures to environmental aerosols. Appl. Occup. Environ. Hyg. 12(8):547-554.

Solomons, N.W., F. Viteri, T.R. Shuler, F.H. Nielsen, 1982. Bioavailability of nickel in man: Effects of food and chemically defined dietary constituents on the absorption of inorganic nickel. J. Nutr. 112:39-50.

Spiegelberg, T., W. Kordel, and D. Hochrainer. 1984. Effects of NiOH inhalation on alveolar macrophages and the humoral immune systems of rats. Ecotox. Environ. Safety. 8:516-525.

Springborn Laboratories Inc. 2000. An oral (gavage) two generation reproduction toxicity study in Sprague Dawley rats with nickel sulfate hexahydrate. Final report submitted to NiPERA Inc., Durham, NC 27713.

Spruit, D. and P.J.M. Bongaarts. 1977. Nickel content of plasma, urine and hair in contact dermatitis. Dermatologica. 154:291-300.

Stoner, G.D., M.B. Shimkin, M.C. Troxell, T.L. Thompson, and L.S. Terry, 1976. Test for carcinogenicity of metallic compounds by the pulmonary tumor response in strain A mice. Cancer Res. 36:1744-1747.

Sumino, K., K. Hayakawa, T. Shibata, S. Kitamura, 1975. Heavy metals in normal Japanese tissues: Amounts of 15 heavy metals in 30 subjects. Arch. Environ. Health. 30:487-494.

Sunderman, F.W., Jr. 1986. Sources of exposure and biological effects of nickel. In: O'Neil, I.K., P. Schuller, L. Fishbein, eds. Environmental carcinogens selected methods of analysis. Volume 8: Some metals: As, Be, Cd, Cr, Ni, Pb, Se, Zn. IARC scientific publication no. 71. Lyon, France: International Agency for Research on Cancer, 79-92.

Sunderman, F.W., Jr. 1989. Mechanisms of nickel carcinogenesis. Scand. J. Work Environ. Health. 15:1-12.

Sunderman, F.W., Jr. 1993. Biological monitoring of nickel in humans. Scand. J. Work Environ. Health. 19 (Suppl. 1):34-38.

Sunderman, F.W., J.F. Kincaid, A.J. Donnelly, and B. West, 1957. Nickel poisoning IV. Chronic exposure of rats to nickel carbonyl: A report after one year of observation. Arch Ind Health. 16:480-485.

Sunderman, F.W., A.J. Donnelly, B. West, and J.F. Kincaid, 1959. Nickel poisoning IX. Carcinogenesis in rats exposed to Nickel Carbonyl. Arch. Ind. Health. 20:36-41.

Sunderman, F.W., Jr. and E. Horak. 1981. Biochemical indices of nephrotoxicity, exemplified by studies of nickel nephropathy. In: Brown, S.S. and D.S. Davies, eds. Organ-directed toxicity: Chemical indices and mechanisms. London, UK: Pergamon Press, 52-64.

Sunderman, F.W., Jr., B. Dingle, S.M. Hopfer, and T. Swift. 1988. Acute nickel toxicity in electroplating workers who accidently ingested a solution of nickel sulfate and nickel chloride. Am. J. Ind. Med. 14:257-266.

Sunderman, F.W., Jr., S.M. Hopfer, K.R. Sweeney, A.H. Marcus, B.M. Most, and J. Creason. 1989. Nickel absorption and kinetics in human volunteers. Proc. Soc. Exp. Biol. Med. 191:5-11.

Sunderman, F.W. Jr., S.M. Hopfer, and J.A. Knight, 1990. Carcinogenesis bioassays of nickel oxides and nickel-copper oxides by intramuscular administration to male Fischer-344 rats.15th International Cancer Congress, Hamburg, Germany, August 16-22, 1990. J Cancer Res. Clin. Oncol. 116 (Suppl. Part 1). 96.

TERA (Toxicology Excellence for Risk Assessment). 1999. Toxicological Review of Soluble Nickel Salts. [http://www.tera.org/vera/nickel%20doc%20page.htm]. (Accessed October 18, 2001).

Thomassen, Y., E. Nieboer, D. Ellingsen, S. Hetland, T. Norseth, J.O. Odland, N. Romanova, S. Chernova and V.P. Tchachtchine. 1999. Characterization of workers' exposure in a Russian nickel refinery. J. Environ. Monit. 1:15-22.

Tola, S., J. Kilpio, and M. Virtamo. 1979. Urinary and plasma concentrations of nickel as indicators of exposure to nickel in an electroplating shop. J. Occup. Med. 21:184-188.

Torjussen, W. and I. Andersen. 1979. Nickel concentrations in nasal mucosa, plasma and urine in active and retired nickel workers. Ann. Clin. Lab. Sci. 9:289-298.

US EPA. 1986. Health Assessment Document for Nickel and Nickel Compounds, Final Report. PB86-232212. EPA/6008-83/012FF.

US EPA. 2001. Risk assessment guidance for Superfund. Volume 1: Human health evaluation manual (Part E, Supplemental guidance for dermal risk assessment). Interim. Review Draft for Public comment. EPA/540/R/99/005.

US EPA IRIS. 1998a. Nickel refinery dust. Integrated Risk Information System (IRIS). Environmental Criteria and Assessment Office, Office of Health and Environmental Assessment, Cincinnati, OH.Online at http://www.epa.gov/iris/index.html. (Accessed October 17, 2001).

US EPA IRIS. 1998b. Nickel subsulfide. Integrated Risk Information System (IRIS). Environmental Criteria and Assessment Office, Office of Health and Environmental Assessment, Cincinnati, OH.Online at http://www.epa.gov/iris/index.html. (Accessed October 17, 2001).

US EPA IRIS. 1998c. Nickel, soluble salts. Integrated Risk Information System (IRIS). Environmental Criteria and Assessment Office, Office of Health and Environmental Assessment, Cincinnati, OH.Online at http://www.epa.gov/iris/index.html. (Accessed October 17, 2001).

US EPA OPPTS. 2001. US EPA Office of Prevention, Pesticides and Toxic Substances (OPPTS) Series 870 Human Health Harmonized Test Guidelines online at http://www.epa.gov/docs/OPPTS_Harmonized/870_Health_Effects_Test_Guidelines/ Last Revision: August 22, 2001. (Accessed March 5, 2002).

US FDA. 1993. Guidance document for nickel in shellfish. Center for Food Safety and Applied Nutrition, US Food and Drug Administration, Washington, D.C.

Veien, N.K., T. Hattel, O. Justesen, & A. Norholm, 1983. Oral challenge with metal salts. Various types of eczema. Contact Dermatitis. 9:407-410.

Velazquez, S.F., and K.A. Poirier. 1994. Problematic risk assessments for drinking water contaminants: Selenium, aldicarb and nickel. In Wang, R.G.M., ed. Water contamination and health. Integration of exposure assessment, toxicology and risk assessment. Dekker, N.Y., Environmental Science and Pollution Control Series, Vol. 9 pp. 467-495.

van Hoojstraten, I.M., K.E. Andersen, B.M. Von Blomberg, D. Boden, D.P. Bruynzeel, D. Burrows, J.G. Camarasa, A. Dooms-Goossens, G. Kraal, A. Lahti, 1991. Reduced frequency of nickel allergy upon oral nickel contact at an early age. Clin. Exp. Immunol. 85:441-445.

Vyskocil, A., C. Viau and M. Cizkova. 1994. Chronic nephrotoxicity of soluble nickel in rats. Human & Experimental Toxicology. 13:689-693.

Waksvik, H. and M. Boysen. 1982. Cytogenic analysis of lymphocytes from workers in a nickel refinery. Mutat Res. 103:185-190.

Wehner, A.P., R.H. Busch, R.J. Olson, and D.K. Craig. 1975. Chronic inhalation of nickel oxide and cigarette smoke by hamsters. Am. Ind. Hyg. Assoc. J. 36:801-809.

Werner, M.A., J.H. Vincent, Y. Thomassen, S. Hetland, and S. Berge. 1999. Inhalable and "total" metal and metal compound aerosol exposures for nickel refinery workers. Occup. Hyg. 5: 93-109.

Wilkinson, D.S. and J.D. Wilkinson, 1989. Nickel allergy and hand eczema. In: Nickel and the Skin: Immunology and Toxicology. Maibach, H.I., Menne, T. (eds) Boca Raton, Florida, USA: CRC Press, 1989:133.

Wolfensohn, S.E. and M.H. Lloyd. 1998. Handbook of Laboratory Animal Management and Welfare. 2nd Edition. Blackwell Science Limited, Oxford, pp.194.

World Health Organization (WHO). 1987. Air quality guidelines for Europe. WHO Regional Office for Europe, Copenhagen.

World Health Organization (WHO). 1993. Guidelines for drinking-water quality, 2nd ed. Vol. 1. Recommendations. Geneva, World Health Organization.

World Health Organization (WHO). 1996. Guidelines for drinking-water quality, 2nd ed. Addendum to Vol. 2. Health criteria and other supporting information. Geneva.

World Health Organization (WHO). 1998. Guidelines for drinking-water quality, 2nd ed. Addendum to Vol. 2 Health criteria and other supporting information. WHO, Geneva.

WHO. 2000. World Health Organization, Regional Office for Europe, Copenhagen. 2000. Air Quality Guidelines for Europe, Second Edition. ISBN 92-890-1358-3.

Yamada, M., S. Takahashi, H. Sato, T. Kondo, T. Kikuchi, K. Furuya, I. Tanaka, 1993. Solubility of nickel oxide particles in various solutions and rat alveolar macrophages. Biol. Trace Elem. Res. 36:89-98.

Yu, C.P., T.H. Hsieh, A.R. Oller, and G. Oberdorster, 2001. Evaluation of the human nickel retention model with workplace data. Regulatory Toxicol. Pharmacol. 33:165-172.

Zatka, V.J., J.S. Warner, D. Maskery. 1992. Chemical speciation of nickel in airborne dusts: Analytical method and results of an interlaboratory test program. Environ. Sci. Tech. 26:138-144.

Human Health Risk Assessment

Appendix 3

Detailed Estimates of Daily Intakes of Metals

Table of Contents
Human Health Risk Assessment - Appendix 3
Detailed Estimates of Daily Intakes of Metals

A3-1 Assessing Exposures to Metals

Each of the exposure pathways identified in Section 4.1 of the main report, that can contribute to the total daily metal exposures experienced by the residents of the Rodney Street community, is discussed below. The method of calculation is presented, identifying all of the receptors and site-specific parameters that are considered for each pathway. Exposures are assessed for all of the receptors identified in Section 4.1 of the main report, and were estimated using the receptor parameters listed in Table 4-3 of the main report and discussed in Appendix 6.

A3-1.1 Intake of Metals from Supermarket Food

Estimates of the daily dietary intakes of metals from supermarket foods are generally limited and the amount of information available varies widely between metals. The metals of concern in the Rodney Street community addressed in this exposure assessment include, antimony, beryllium, cadmium, cobalt, copper and nickel. Information regarding daily dietary intakes of these metals has been taken from Canadian and international regulatory agencies. Additional information has been taken from the available literature. For the purposes of assessing likely daily dietary metal intakes for the residents of the Rodney Street community, preference has been given to data generated from the Canadian population. It was felt that information from Canadian sources would provide the best reflection of likely dietary habits and metal intakes for residents of the Rodney Street community. The daily dietary intake of metals is discussed in detail in Appendix 4. A summary of the daily dietary intake of metals for all age groups is presented in Table A3-1.

Table A3-1: Estimated Daily Intakes of Metals from Supermarket Food

Receptor	Daily Intakes of Metals from Supermarket Food (µg/day)					
	Antimony	Beryllium	Cadmium	Cobalt	Copper	Nickel
Infant	1.3	4.8	5.08	4.18	518	109.2 (72.2-146.2)*
Toddler	2.3	8.6	10.6	7.0	822	190
Child	3.5	13.2	16.8	10.0	1230	251
Teen	4.0	15.0	17.3	12.0	1520	313
Adult	3.4	12.7	14.8	10.0	1430	307
Reference	FSA, 1997	Vaessen & Szteke, 2000	CEPA, 1994	Dabeka & McKenzie, 1995	CCME, 1997	Dabeka, 1989; Dabeka & McKenzie, 1995

*see table A4-4.

A3-1.2 Intake of Metals from Drinking Water

Daily intakes of metals from drinking water are dependent on the amount of drinking water consumed on a daily basis and the level of metals present in the drinking water. The estimated intakes of metals from drinking water for the Rodney Street community has been calculated as

shown in equation A3-1. Estimates of the intake of antimony, beryllium, cadmium, cobalt, copper and nickel from the consumption of drinking water for all age groups are shown in Table A3-2.

EQ A3-1:

$$Intake_{dw} = IR_{dw} * C_{dw}$$

Where:	$Intake_{dw}$	$=$	Intake from drinking water	µg/day
	IR_{dw}	$=$	Ingestion rate of drinking water	L/day
	C_{dw}	$=$	Metal concentration in drinking water	µg/L

The intake estimates are based on the highest level of each metal reported by the monitoring of drinking water taken from the municipal system at Charlotte Street. Although water quality was also measured at the treatment plant, the data from within the distribution system was felt to be more representative of the water quality in the Rodney Street community. The data in Table A3-2 shows that for most metals, daily intakes from drinking water are generally less than 1 µg/day. The most notable exception to this is copper, where intakes from drinking water range between 13 µg/day for infants and 66 µg/day for adults. For infants and toddlers, intakes of nickel from drinking water are below 1 µg/day, but intakes for children, teens and adults are greater than 1 µg/day. These values will be used in conjunction with intakes from other sources to provide estimates of total daily exposure for people in all age groups.

Table A3-2: Estimated Metal Intakes from Drinking Water

Metal	Receptor	C_{dw} (µg/L)	IR_{dw} (L/day)	$Intake_{dw}$ (µg/day)
Antimony	0 - 6 months	0.97	0.3	0.29
	7 months - <5 years	0.97	0.6	0.58
	5 - <12 years	0.97	0.8	0.78
	12 - <20 years	0.97	1	0.97
	20 + years	0.97	1.5	1.5
Beryllium	0 - 6 months	0.20	0.3	0.06
	7 months - <5 years	0.20	0.6	0.12
	5 - <12 years	0.20	0.8	0.16
	12 - <20 years	0.20	1	0.20
	20 + years	0.20	1.5	0.30
Cadmium	0 - 6 months	0.083	0.3	0.025
	7 months - <5 years	0.083	0.6	0.050
	5 - <12 years	0.083	0.8	0.066
	12 - <20 years	0.083	1	0.083
	20 + years	0.083	1.5	0.12
Cobalt	0 - 6 months	0.040	0.3	0.012
	7 months - <5 years	0.040	0.6	0.024
	5 - <12 years	0.040	0.8	0.032
	12 - <20 years	0.040	1	0.040
	20 + years	0.040	1.5	0.060
Copper	0 - 6 months	44	0.3	13
	7 months - <5 years	44	0.6	26
	5 - <12 years	44	0.8	35
	12 - <20 years	44	1	44
	20 + years	44	1.5	66
Nickel	0 - 6 months	1.3	0.3	0.39
	7 months - <5 years	1.3	0.6	0.78
	5 - <12 years	1.3	0.8	1.0
	12 - <20 years	1.3	1	1.3
	20 + years	1.3	1.5	2.0

A3-1.3 Intake of Metals from Ambient Air

Unlike other environmental media, such as soil or water, air quality may fluctuate from day to day or hour to hour, and exposure levels are also influenced by changes in meteorological conditions. To protect the general population against contaminants in outdoor air, on a continuous basis, time periods such as 24 hours or annual are used. These prescribed time periods are referred to as "averaging times" and are an important aspect of controlling air quality. This also has significance from a toxicological perspective since the dose of a chemical, which is time dependent, is a major determinant of toxicological effects. One consideration in establishing averaging time is to limit exposure peaks for airborne chemicals, which could occur within a long averaging period, such as a year.

Averaging time can be used to ensure protection against the different effects of airborne chemicals by ensuring that exposure limits for specific effects, acute or chronic, are not exceeded. Short term acute effects are normally based on a one hour (or less) exposure period while longer term chronic effects are based on a 24 hour or annual averaging time. Averaging times also provide useful benchmarks to monitor ambient air quality.

The annual arithmetic mean is a relatively stable measure of air quality that reflects the total cumulative dose of airborne substances to which an individual or population is exposed. Short-term peaks have an influence on the arithmetic mean that is proportional to their frequency, magnitude and duration, and thus, their contribution to cumulative exposure and risk. As a result, the annual arithmetic mean form of an annual standard provides protection across a wide range of the air quality distribution contributing to exposure and risk, in contrast to other forms, such as the geometric mean that de-emphasize the effects of short-term peak concentrations.

The time taken for chemical exposure to cause adverse health effects varies among chemicals and even a single chemical can cause different effects at different doses. Chemicals such as sulphur dioxide, may trigger an effect within 15 minutes, or less, of exposure. Others, such as the carcinogenic chemicals, may have a longer term cumulative effect, which may not clinically manifest for several years. The times over which concentrations should be averaged to reflect the time frame during which their effects become apparent varies, and averaging times are often set to reflect this.

Air monitoring data is usually collected on air samplers over relatively short time periods, e.g., one to two days, and the results integrate the chemical concentration over the volume of air filtered and the time period the sampler was running. A single air sample would result in the air concentration over a daily time period. In the course of a year, if sufficient "daily" samples are taken, an annual average air concentration can be calculated. This way a picture of the peak levels and the overall average concentration in the air over the year can be constructed.

In Ontario, the Ministry of the Environment (MOE) has established air quality standards (air standards) including ambient air quality criteria (AAQC) and point of impingement (POI) standards. Ambient air quality criteria are established to protect human health and the environment (terrestrial vegetation and wildlife), and to prevent aesthetic impacts such as odor, soiling of property and visibility. The AAQC are used to assess the quality of the ambient environment, while POI standards are used to evaluate the impacts of airborne emissions.

AAQC are developed to protect the most sensitive sub-populations, with consideration of the most sensitive adverse effect(s) induced by the exposure to a pollutant. These criteria are derived from the most reliable and up-to-date scientific, toxicological and epidemiological information obtained from peer reviewed literature or from the studies from which other environmental protection agencies derive their respective air guidelines. In some occasions, AAQC can be derived based on the scientific rationale of occupational exposure limits. However, occupational

exposure limits are developed to protect the safety of workers and their derivation may include science/policy decisions which may not be directly applicable to environmental health decision making.

Cancer risk is in general considered the likelihood of developing this disease as a result of a life-time exposure of an individual to low doses of a carcinogenic substance. Most of the cancer-based guidelines are derived from cancer slope factors which are mathematical estimates of additional or excess risk. Cancer risk-specific exposure concentrations can be derived from the slope factors. Cancer risk can also be expressed as the probability of developing this disease at a risk-specific exposure concentration, such as one case in a population of one million, one-hundred thousand or ten thousand. In line with risk levels considered for other MOE environmental programmes, the ministry has adopted the risk level of one in a million to derive an annual average AAQC for carcinogens. Corresponding air standards for other averaging times such as the 24 hour average AAQC and the half hour POI can be derived mathematically based on a power law equation and the scaling factors as follows:

24 hour average AAQC = annual AAQC x 5
half hour POI limit   = annual AAQC x 15

It should be noted that the cancer-based AAQC or corresponding POI standards may be derived based on a risk level of greater than one in a million. This happens when the air standards are not immediately achievable because of implementation issues, which may include high background concentrations, technical feasibility, significant economic impacts, or allowing a reasonable time frame for compliance. The ministry is in the process of developing a risk management framework to resolve these potential implementation issues.

In the case of the risk assessment for the Rodney Street community, air monitoring data comes from several sources and locations. Local air sampling for nickel, lead, copper and total suspended particulates was obtained from the ministry's sampling station at Davis Street and Fraser Street, which operated from 1992 to 1996, and air sampling done during the summer of 2000 near schoolyards in Port Colborne by Jacques Whitford Environmental Limited (JWEL, 2000a). The ministry's sampling station was about 600m north of Rodney Street. Prevailing winds in the general Port Colborne area are from the west and southwest. These sectors account for about 45-50% of winds. The other sectors occur less and fairly evenly, about 5-15% each (Frank Dobroff, MOE, personal communication). While the Davis and Fraser location may be deemed slightly upwind of the Rodney Street community, inspection of the nickel concentration in soil maps in the ministry's Phytotoxicology Soil Investigation Reports (MOE, 1999; MOE, 2000) indicate that it is located in an area where nickel levels in surface soils range up to 1,000 $\mu g/g$, and depending on wind direction would sample air particulates representative of the area just north of Rodney Street. The air monitoring performed at Port Colborne schools in the summer of 2000 (JWEL, 2000a) was only collected for the portion of the year that dust levels would normally be higher and may not be representative of long term average levels in the

community. However, in all cases where air monitoring data exists for arsenic, cobalt, copper, nickel, and TSP, the maximum and average air concentrations for each metal from the JWEL (2000a) air monitoring are less than or comparable with either the MOE or Environment Canada information.

Air concentrations of other metals not sampled extensively in Port Colborne (antimony, arsenic, cadmium, cobalt, lead) were taken from Environment Canada's National Air Pollution Surveillance (NAPS) air monitoring program for Ontario for 1995-1999 (Tom Dann, Environment Canada, personal communication). Environment Canada air monitoring data comes from nine sites spread across Ontario, six of which are in Hamilton, Toronto and Windsor. In general, the Environment Canada air monitoring data for the same chemicals sampled by MOE at Davis and Fraser (the maximum and annual average air concentrations) was lower. In the absence of more suitable air quality data for chemicals not sampled extensively in Port Colborne, Environment Canada air monitoring data was used.

A summary of the metal levels in air, used in the current assessment is provided in Table A3-3. The recent 2001 MOE air monitoring data for the Rodney Street location (Table A1-3) is not annualized, however, it supports the Table A3-3 values based on Environment Canada air monitoring data.

Table A3-3: Levels of Metals in Ambient Air in Port Colborne

	Metal Concentration in Air in Port Colborne ($\mu g/m^3$)					
	Antimony	Beryllium	Cadmium	Cobalt	Copper	Nickel
Short term maximum	0.0115	n/a	0.0067	0.017	0.56	0.69
Annual average (highest)	0.0011	0.00012	0.0007	0.002	0.11	0.033

In the Rodney Street community, inhaled metals will be associated with particulate matter and will not be present as free metal. Therefore, there is a potential for the inhaled particulate matter to be cleared from the lungs, through mucocilliary transport, and swallowed. Material cleared from the lungs in this fashion will add to the total daily ingestion of metal. The amount of particulate delivered to the stomach by this process is difficult to predict with any accuracy. Therefore, to provide conservative estimates of the amount of metal ingested as a result of the clearance of inhaled particles, it has been assumed that all inhaled metal is cleared from the lung and passed to the stomach. This approach will overestimate the contribution that inhalation exposures make to the total daily intakes of metals.

For these estimates the highest annual average air concentration from the MOE or Environment Canada air monitoring data was used. This is a more conservative estimate for long term inhalation exposure. Inhalation R/C and unit risks are developed for life-time exposure not short term maximum air concentrations. Characterization of potential health risks from inhalation is

discussed in Section 5.0 of the Human Health Risk Assessment main report (Part B) (Risk Characterization). The estimated inhalation intake of each metal for each receptor based on the highest annual average level (Table A3-3) is shown in Table A3-4. These values are calculated as shown in equation A3-2.

Eq A3-2:

$$Intake_{air} = Intake_{airout} + Intake_{airin}$$

$$Intake_{airout} = (Time_{outsum} * IR_{air} * Cair_{out}) + (Time_{outwin} * IR_{air} * Cair_{out})$$

$$Intake_{airin} = (Time_{insum} * IR_{air} * Cair_{in}) + (Time_{inwin} * IR_{air} * Cair_{in})$$

Where:	$Intake_{air}$ =	Intake from air	μg/day
	$Intake_{airout}$ =	Intake from air while outdoors	μg/day
	$Intake_{airin}$ =	Intake from air while indoors	μg/day
	IR_{air} =	Inhalation rate	m³/day
	$Cair_{out}$ =	Outdoor air concentration (measured)	μg/m³
	$Cair_{in}$ =	Indoor air concentration	μg/m³
		(75% of outdoor air concentration based on Roberts et al. (1974))	
	$Time_{insum}$ =	Fraction of time spent indoors during summer; see Appendix 6	unitless
	$Time_{inwin}$ =	Fraction of time spent indoors during winter; see Appendix 6	unitless
	$Time_{outsum}$ =	Fraction of time spent outdoors during summer; see Appendix 6	unitless
	$Time_{outwin}$ =	Fraction of time spent outdoors during winter; see Appendix 6 unitless	

Table A3-4: Estimated Metal Intakes from Air

Metal	Receptor	C_{air} (µg/m³)	IR_{air} (m³/day)	Intake$_{air}$ (µg/day)
Antimony	0 - 6 months	0.0011	2.1	0.0018
	7 mo - <5 years	0.0011	9.3	0.0081
	5 - <12 years	0.0011	14.5	0.013
	12 - <20 years	0.0011	15.8	0.014
	20 + years	0.0011	15.8	0.014
Beryllium	0 - 6 months	0.00012	2.1	0.0002
	7 mo - <5 years	0.00012	9.3	0.0009
	5 - <12 years	0.00012	14.5	0.0014
	12 - <20 years	0.00012	15.8	0.0015
	20 + years	0.00012	15.8	0.0015
Cadmium	0 - 6 months	0.0007	2.1	0.0011
	7 mo - <5 years	0.0007	9.3	0.0051
	5 - <12 years	0.0007	14.5	0.0080
	12 - <20 years	0.0007	15.8	0.0087
	20 + years	0.0007	15.8	0.0086
Cobalt	0 - 6 months	0.002	2.1	0.0033
	7 mo - <5 years	0.002	9.3	0.015
	5 - <12 years	0.002	14.5	0.023
	12 - <20 years	0.002	15.8	0.025
	20 + years	0.002	15.8	0.025
Copper	0 - 6 months	0.112	2.1	0.18
	7 mo - <5 years	0.112	9.3	0.82
	5 - <12 years	0.112	14.5	1.28
	12 - <20 years	0.112	15.8	1.39
	20 + years	0.112	15.8	1.38
Nickel	0 - 6 months	0.033	2.1	0.053
	7 mo - <5 years	0.033	9.3	0.24
	5 - <12 years	0.033	14.5	0.37
	12 - <20 years	0.033	15.8	0.41
	20 + years	0.033	15.8	0.40

A3-1.4 Intake of Metals from Backyard Garden Produce

Eating produce grown in backyards where metal levels are above typical levels, represents a potential exposure pathway if the metals present in the soil are taken up into the plant. The exposures received by people eating such produce depends upon the concentration of the metals in the fruits and vegetables and the amount of fruits and vegetables consumed from backyard gardens. The current assessment has assumed that backyard garden produce is consumed on a daily basis throughout the year. The amount of backyard garden fruits and vegetables consumed on an annually averaged daily basis is discussed in detail in Appendix 6.

As part of the ongoing work in Port Colborne, samples of backyard produce have been collected by the MOE and JWEL from Rodney and Mitchell Streets. The levels of individual metals in the various types of produce tested are provided in Appendix 1 of this report. For the purposes of this assessment, backyard garden produce has been divided into three general categories in order to ensure consistency with food consumption rates provided in Appendix 6:

root vegetables	includes: beet root, carrot, onion and radish
other vegetables	includes: beet tops, celery, lettuce, peppers, rhubarb, squash, leeks and tomatoes
fruits	includes: pear, apple, cantaloupe, peach, plum, watermelon, and grapes

For nickel, additional data sources such as those compiled from journal articles, MOE reports, and other relevant studies and reports have also been considered. Studies using forms of nickel with highly plant extractable or forms of nickel more soluble ($NiCl_2$ or $NiSO_4$) than in the Rodney Street soils (predominantly NiO) were not considered. Studies investigating only low soil nickel concentrations were also not considered. Port Colborne data prior to 1984 were not considered since they would have been strongly influenced by atmospheric deposition of Inco emissions such that nickel concentrations in produce would have resulted from atmospherically deposited nickel. The following table (Table A3-5) provides data sources considered for this evaluation and the rationale for the selection of relevant data sets.

Table A3-5: Data Sources Considered for Vegetation Uptake of Nickel	
Data Source	**Reason for Exclusion**
MOE. 1977. Effect of Heavy Metal on the Growth of Lettuce, Celery, and Onion, Groetelaars Farm, Port Colborne.	atmospheric deposition
MOE. 1978a. Investigations of the Effects of Heavy Metals on Muck Farms East of International Nickel Company, Port Colborne, Ontario, 1976 - 1977.	atmospheric deposition
MOE. 1978b. Investigations of the Effects of Heavy Metals on Muck Farms East of International Nickel Company, Port Colborne, Ontario, 1976-1977. Ontario Ministry of the Environment, Air Resources Branch, Phytotoxicology Section.	atmospheric deposition
MOE. 1979. Phytotoxicology Complaint Investigation, J. Overholt, Port Colborne, 1979.	atmospheric deposition
OMAF. 1980. Report on Agronomic Problems Experienced by Three Growers Located Near Port Colborne.	atmospheric deposition

Table A3-5: Data Sources Considered for Vegetation Uptake of Nickel	
Data Source	**Reason for Exclusion**
MOE. 1980a. Investigations of Agronomic Problems Associated with Metal Toxicity on Muck Farms in the Vicinity of Inco, Port Colborne, 1976-1979.	atmospheric deposition
MOE. 1980b. Phytotoxicology Complaint Investigation, J. Overholt, Port Colborne, 1980.	atmospheric deposition
MOE. 1981. Effects of Heavy Metals and Root Knot Nematode on Celery Grown on Organic Soil in the Vicinity of International Nickel Company, Port Colborne, Ontario, 1980.	atmospheric deposition
MOE. 1983. Levels of Ni, Cu, and Co in Vegetable and Soil Samples Collected from Residential Gardens in the Vicinity of Inco Limited, Port Colborne.	atmospheric deposition
MOE. 1984a. Investigation of Alleged SO_2 Injury to Celery on Overholt Farm, Port Colborne, October 3, 1983.	atmospheric deposition
MOE. 1984b. Nickel Phytotoxicity on Celery and Lettuce Grown on Soil Contaminated by a Nickel Refinery.	atmospheric deposition
MOEE. 1994. Nickel Uptake into Vegetables Growing in Experimental Plots. Ecological Standards & Toxicology Section, MOEE, unpublished data.	low soil concentrations
Cantox. 1999. Deloro Village Exposure Assessment and Health Risk Characterization for Arsenic and Other Metals, Cantox Environmental Inc., December, 1999.	low soil concentrations
JWEL. 2001. 2001 Food Basket Collection. Unpublished data supplied by JWEL on October 4, 2001. Jacques Whitford Environmental Limited.	not excluded
JWEL. 2000b. Garden Soil Data - Rodney Street Area, Sampled from the 18th-22nd of September, 2000. Jacques Whitford Environmental Limited.	not excluded
MOE. 2001. Garden soil and plant data from Rodney Street, Sampled from November 2000 to January 2001.	not excluded
Biró, B., I. Köves-Péchy, and I. Kádár. 1998. Toxicity of Some Field Applied Heavy Metal Salts to the Rhizobial and Fungal Microsymbionts of Alfalfa and Red Clover. Agrokémia és Talajtan 42:265-276.	$NiSO_4$ (soluble form of nickel)
Braillier, S., R.B. Harrison, C.L. Henry, and X. Dongsen. 1996. Liming effects on availability of Cd, Cu, Ni and Zn in a soil amended with sewage sludge 16 years previously. Water, Air and Soil Pollution. 86:195-206.	low soil concentrations
Frank, R., K.I. Stonefield, P. Suda, and J.W. Potter. 1982. Impact of nickel contamination on the production of vegetables on an organic soil, Ontario, Canada, 1980-1981. Sci. Total Environ. 26:41-65.	atmospheric deposition

Table A3-5: Data Sources Considered for Vegetation Uptake of Nickel	
Data Source	**Reason for Exclusion**
Guo, Y., R. Schulz, and H. Marschner. 1995. Genotypic differences in uptake and distribution of cadmium and nickel in plants. Agnew. Bot. 69:42-48.	$NiSO_4$ (soluble form of nickel) & low soil concentrations
L'Huillier, L., and S. Edighoffer. 1996. Extractability of nickel and its concentration in cultivated plants in Ni rich ultramafic soils of New Caledonia. Plant and Soil. 186:255-264.	Soil with exceptionally high plant extractable nickel
Maclean, A.J. and A.J. Dekker. 1978. Availability of zinc, copper and nickel to plants grown in sewage treated soils. Can. J. Soil Sci. 58:381-389.	$NiSO_4$ (soluble form of nickel)
Sajwan, K.S., W.H. Ornes, T.V. Youngblood, and A.K. Alva. 1996. Uptake of soil applied cadmium, nickel, and selenium by bush beans. Water, Air, Soil Pollut. 91:209-217.	$NiCl_2$ (soluble form of nickel) & low soil concentrations
Sauerbeck, D. R., and A. Hein. 1991. The nickel uptake from different soils and its prediction by chemical extractions. Water, Air, Soil Pollut. 57-58: 861-871.	low soil concentrations
Traynor, M.F. and B.D. Knezek. 1973. Effects of nickel and cadmium contaminated soils on nutrient composition of corn plants. Proc. Annual Conf. on Trace Substances in the Environment. 7:82-87.	$NiCl_2$ (soluble form of nickel)
Vergnano Gambi, O., R. Gabbrielli, and L. Pancaro. 1982. Nickel, chromium and cobalt in plants from Italian serpentine areas. Acta Oecologica Oecologica Plantarum 3(17): 291-306.	low soil concentrations

Data sources were narrowed down to three relevant data sets (JWEL, 2000b, JWEL, 2001 and MOE, 2001 data) that were reflective of current conditions. An additional backyard produce nickel data set from Port Colborne was recently available from JWEL, but the subsequent tissue nickel fresh weight mean and 95th percentile concentrations in the three produce categories and in the pooled data set were not significantly different from the values already calculated for the original data. Laboratory analysis of vegetables typically report concentrations on a dry weight basis. This was the case for all of the JWEL (2000b) and MOE (2001) and some of the JWEL (2001) data discussed above. Since vegetables are not typically consumed in a dry state, these concentrations were converted to a fresh (or wet) weight (as consumed). To convert dry weight to fresh weight, dry weight tissue concentrations were multiplied by the appropriate dry weight to fresh weight conversion factor based on the typical moisture contents of vegetables. The USDA (1963), Baes et al., (1984) and US EPA (1997) have recommended dry weight to fresh weight conversion factors for several fruits and vegetables collected as part of the JWEL (2000b), MOE (2001) and JWEL (2001) surveys (Table A3-6). Where plant specific factors were not available, the conversion factor was based on a similar plant type. The majority of the JWEL (2001) data was provided on a fresh weight basis (conversion was not necessary for this data).

Table A3-6: Dry Weight to Fresh Weight Conversion Factors

Vegetables	Conversion Factor	Fruits	Conversion Factor
Basil	0.045	Apples	0.15
Beans	0.099	Cantaloupes & Melons	0.10
Beets	0.13	Grapes	0.19
Beet tops	0.091	Peaches	0.11
Broccoli	0.093	Pears	0.17
Cabbage	0.0468	Plums	0.21
Carrots	0.12	Watermelons	0.10
Celery	0.059		
Leeks	0.124		
Lettuce	0.045		
Onions	0.11		
Parsnips	0.21		
Peppers	0.066		
Radish/Horse Radish	0.0516		
Rhubarb	0.052		
Squash	0.057		
Tomatoes	0.065		
Zucchini	0.054		

A review of the available produce data indicated that the concentrations of metals in produce is not strongly affected by the levels of metals present in the soil and as such it was not possible to derive appropriate uptake factors for the Rodney Street community. As a result, upper bound produce concentrations measured in the area were assumed for all gardens in the assessment. With the exception of nickel, maximum values were selected due to limited data sets (see Appendix 1). For nickel, over 180 relevant plant samples were available and as a result it was considered more appropriate to select an upper bound plant concentration to represent all gardens in the assessment (Appendix 1 presents the data available at the time of this assessment). The 95th percentile concentration was selected based on the following rationale: (i) others have considered the 95th percentile of a non-normal distribution to be representative of an upper bound value (the data set appears to be log-normally distributed); (ii) the data set is highly skewed and maximum values would not be reflective of reasonable upper bound exposures; (iii) a typical diet would consist of a composite of the available produce types, while the maximum level is only reflective of a single plant type and garden location. No distinction was made for different produce types, rather the selected upper bound concentration was assumed to represent all plant types. These selected levels are reported in Table A3-7.

Table A3-7: Metal Levels in Backyard Produce in Port Colborne

Vegetable	Metal Concentrations in Vegetables (µg/g) (Fresh Weight)					
	Antimony	Beryllium	Cadmium	Cobalt	Copper	Nickel
Root Vegetables	0.021	0.007	0.063	0.048	1.92	2.44
Other Vegetables	0.021	0.007	0.063	0.048	1.92	2.44
Fruits	0.021	0.007	0.063	0.048	1.92	2.44

Daily intakes of metal from backyard produce are calculated as shown in equation A3-3. Estimates of daily metals intakes from backyard garden produce for all age groups are shown in Table A3-8.

Eq A3-3:

$$Intake_{veg} = \left(IR_{root} * C_{root} \right) + \left(IR_{other} * C_{other} \right)$$

Where: $Intake_{veg}$ = Intake from backyard garden produce   µg/day
IR_x = Yearly averaged daily intake of backyard root or other
vegetables (see Appendix 6)   g/day
C_x = Metal concentration in root/other vegetables on a fresh weight basis   µg/g

Table A3-8: Estimated Metal Intakes from Backyard Vegetables

Metal	Receptor	Root Vegetables			Other Vegetables			Fruits			Total (µg/day)
		C_{root} (µg/g)	IR_{root} (g/day)	$Intake_{veg}$ (µg/day)	C_{root} (µg/g)	IR_{other} (g/day)	$Intake_{veg}$ (µg/day)	C_{root} (µg/g)	IR_{fruit} (g/day)	$Intake_{veg}$ (µg/day)	
Antimony	0 - 6 months	0.021	6.05	0.127	0.021	5.25	0.11	0.021	3.96	0.08	0.32
	7 mo - <5 years	0.021	7.65	0.161	0.021	4.88	0.10	0.021	6.81	0.14	0.41
	5 - <12 years	0.021	11.7	0.25	0.021	7.14	0.15	0.021	7.80	0.16	0.56
	12 - <20 years	0.021	16.5	0.35	0.021	8.75	0.18	0.021	7.51	0.16	0.69
	20 + years	0.021	13.7	0.29	0.021	9.99	0.21	0.021	7.13	0.15	0.65
Beryllium	0 - 6 months	0.007	6.05	0.04	0.0070	5.25	0.037	0.0070	3.96	0.028	0.11
	7 mo - <5 years	0.007	7.65	0.05	0.0070	4.88	0.034	0.0070	6.81	0.048	0.14
	5 - <12 years	0.007	11.7	0.08	0.0070	7.14	0.050	0.0070	7.80	0.055	0.19
	12 - <20 years	0.007	16.5	0.12	0.0070	8.75	0.061	0.0070	7.51	0.053	0.23
	20 + years	0.007	13.7	0.10	0.0070	9.99	0.070	0.0070	7.13	0.050	0.22
Cadmium	0 - 6 months	0.063	6.05	0.38	0.063	5.25	0.33	0.063	3.96	0.25	0.96
	7 mo - <5 years	0.063	7.65	0.48	0.063	4.88	0.31	0.063	6.81	0.43	1.22
	5 - <12 years	0.063	11.7	0.74	0.063	7.14	0.45	0.063	7.80	0.49	1.7
	12 - <20 years	0.063	16.5	1.0	0.063	8.75	0.55	0.063	7.51	0.47	2.1
	20 + years	0.063	13.7	0.86	0.063	9.99	0.63	0.063	7.13	0.45	1.9
Cobalt	0 - 6 months	0.048	6.05	0.29	0.048	5.25	0.25	0.048	3.96	0.19	0.73
	7 mo - <5 years	0.048	7.65	0.37	0.048	4.88	0.23	0.048	6.81	0.33	0.9
	5 - <12 years	0.048	11.7	0.56	0.048	7.14	0.34	0.048	7.80	0.37	1.3
	12 - <20 years	0.048	16.5	0.8	0.048	8.75	0.42	0.048	7.51	0.36	1.6
	20 + years	0.048	13.7	0.66	0.048	9.99	0.5	0.048	7.13	0.3	1.5
Copper	0 - 6 months	1.92	6.05	12	1.92	5.25	10.1	1.92	3.96	7.6	29
	7 mo - <5 years	1.92	7.65	15	1.92	4.88	9.4	1.92	6.81	13.1	37
	5 - <12 years	1.92	11.7	22	1.92	7.14	14	1.92	7.80	15	51
	12 - <20 years	1.92	16.5	32	1.92	8.75	17	1.92	7.51	14	63
	20 + years	1.92	13.7	26	1.92	9.99	19	1.92	7.13	14	59
Nickel	0 - 6 months*										
	7 mo - <5 years	2.44	7.65	19	2.44	4.88	12	2.44	6.81	17	47
	5 - <12 years	2.44	11.7	29	2.44	7.14	17	2.44	7.80	19	65
	12 - <20 years	2.44	16.5	40	2.44	8.75	21	2.44	7.51	18	80
	20 + years	2.44	13.7	33	2.44	9.99	24	2.44	7.13	17	75

*While it is possible that the infant would consume some produce in the first six months of life, home garden produce was not considered since total diet estimates included produce and inclusion of home garden produce would over-estimate infant exposures. See section 5.6.1, Table A4-4 and footnotes for details of nickel intake in the infant diet.

Note: Due to rounding of values, total intakes may not add up to exactly the sum of the intakes for each row, in particular the final decimal place.

A3-1.5 Intake of Metals from Soil/Dust

The metals of concern in the Rodney Street community area of Port Colborne are generally tightly bound to soil particles and are present in forms that either have limited solubility in water or are largely insoluble. However, the solubility of these metals increases under acidic conditions. When ingested, metals that are insoluble in water at neutral pH (6.0 - 8.0) can be solubilized and removed from soil particles in the acidic environment of the stomach. The metals released from the soil in the stomach become accessible for uptake by the gut. Ingested metals that remain bound to soil particles in the gut are not available for absorption and are excreted in the feces. The daily intake of metal from ingested soil is a function of the amount of soil ingested, the level of metal contained in the soil and the amount of metal released from the soil particles under the acidic conditions of the stomach. Similarly, metals are bound to indoor dusts in much the same way as soil, since the source of some of the indoor dust is actually soil from outdoors. While the physical characteristics of dusts may be significantly different than those of soil (e.g., particle size and composition), current data does not allow for an adequate distinction between the exposures from these two media. As a result, the current assessment considers these as a combined media group. The estimated daily intake of metal from the ingestion of soil is calculated as shown in equation A3-4.

Eq A3-4:

$$Intake_{soil} = Intake_{soilout} + Intake_{soilin}$$

$$Intake_{soilout} = \left[\left(Csoil_{out} * Fract_{winter} * Fract_{out} * WF \right) + \left(Csoil_{out} * \left(1 - Fract_{winter} \right) * Fract_{out} \right) \right] * IR_{soil} * Bio_{soil}$$

$$Intake_{soilin} = \left[\left(Cdust_{in} * Fract_{winter} * \left(1 - Fract_{out} \right) \right) + \left(Cdust_{in} * \left(1 - Fract_{winter} \right) * \left(1 - Fract_{out} \right) \right) \right] * IR_{soil} * Bio_{soil}$$

Where:			
$Intake_{soil}$	=	Intake from soil/dust	µg/day
$Intake_{soilout}$	=	Intake from soil while outdoors	µg/day
$Intake_{soilin}$	=	Intake from dust while indoors	µg/day
IR_{soil}	=	Soil/dust ingestion rate	g/day
Bio_{soil}	=	Soil bioaccessibility factor	unitless
$Csoil_{out}$	=	Outdoor soil concentration (measured)	µg/g
WF	=	10% winter covering factor assumed*	
$Cdust_{in}$	=	Indoor dust concentration (0.39 indoor dust to outdoor soil concentration ratio; see Appendix 6)	µg/g
$Fract_{out}$	=	Fraction of daily soil ingestion rate allocated to outdoor exposure; see Appendix 6	unitless
$Fract_{winter}$	=	Fraction of year that is considered winter; see Appendix 6	unitless

A winter covering factor has been assumed for the Rodney Street community. The basis of this factor is the following: (i) between December and March 1963 to 1993, daily minimum temperatures average less than 0°C for Port Colborne (Environment Canada; Seasonal Forecasts, http://weatheroffice.ec.gc.ca/saisons/index_e.html) indicating that the ground is likely to remain frozen throughout these months reducing the accessibility of the soil for direct contact and

movement of dust into homes; (ii) between December and March, daily maximum temperatures average less than 5°C for Port Colborne (Environment Canada; Climate Normals); due to these colder temperatures, it is likely that children will not be 'playing' in the dirt with exposed skin; most children will wear mittens or gloves throughout these winter months (iii) between December and March, most precipitation is in the form of snow, as a result, the ground will tend to be covered and unaccessible throughout this time period. On the basis of these factors, it has been assumed that contact with the soil will be approximately 1/10 for the four winter months as compared with other months of the year.

The soil ingestion rates and activity patterns for each of the receptor age groups are listed in Table 4-3 of the main report. The highest reported level of each metal in the soil from the Rodney Street community was used to estimate the daily ingestion of metal from soil. The Bio_{soil} parameter is a measure of the amount of metal that is released from the soil under the acidic and neutral pH conditions of the stomach and small intestine. This represents the amount of metal that is considered to be bio-accessible, or available to the gut for uptake, from the soil. The amount of each metal released from the soil in the stomach has been estimated using a simulated stomach acid leach test and a bioaccessibility test. The test methodologies are discussed in detail in Appendix 5. The results of the stomach acid leach test and bioaccessibility test for each metal are also provided in Appendix 5. For each metal, the highest average reported result for the sieved only soil samples tested by Exponent (Table A5-17) was used as a relative bioavailability adjustment factor to estimate the amount of metal that would be comparable to the form of the metal used in the toxicity data used to derive the oral R/D. This was used to estimate the effective daily intake for each metal from soil. The estimated daily intake of each metal from the soil is shown in Table A3-9.

Table A3-9: Estimated Metal Intakes from Soil

Metal	Receptor	C_{soil} (µg/g)	IR_{soil} (g/day)	Bio_{soil}	Total (µg/day)
Antimony	0 - 6 months	91.1	0.035	0.32	0.54
	7 mo - <5 years	91.1	0.100	0.32	1.54
	5 - <12 years	91.1	0.100	0.32	1.54
	12 - <20 years	91.1	0.020	0.32	0.309
	20 + years	91.1	0.020	0.32	0.309
Beryllium	0 - 6 months	4.56	0.035	0.59	0.050
	7 mo - <5 years	4.56	0.100	0.59	0.144
	5 - <12 years	4.56	0.100	0.59	0.144
	12 - <20 years	4.56	0.020	0.59	0.0287
	20 + years	4.56	0.020	0.59	0.0287
Cadmium	0 - 6 months	35.3	0.035	0.76	0.498
	7 mo - <5 years	35.3	0.100	0.76	1.42
	5 - <12 years	35.3	0.100	0.76	1.42
	12 - <20 years	35.3	0.020	0.76	0.284
	20 + years	35.3	0.020	0.76	0.284
Cobalt	0 - 6 months	262	0.035	0.29	1.41
	7 mo - <5 years	262	0.100	0.29	4.02
	5 - <12 years	262	0.100	0.29	4.02
	12 - <20 years	262	0.020	0.29	0.805
	20 + years	262	0.020	0.29	0.805
Copper	0 - 6 months	2720	0.035	0.43	21.7
	7 mo - <5 years	2720	0.100	0.43	61.9
	5 - <12 years	2720	0.100	0.43	61.9
	12 - <20 years	2720	0.020	0.43	12.4
	20 + years	2720	0.020	0.43	12.4
Nickel	0 - 6 months	17000	0.035	0.19	59.9
	7 mo - <5 years	17000	0.100	0.19	171
	5 - <12 years	17000	0.100	0.19	171
	12 - <20 years	17000	0.020	0.19	34.2
	20 + years	17000	0.020	0.19	34.2

A3-1.6 Intake of Metals Through Dermal Contact with Soil/Dust

Daily contact with metals through soil present on the skin represents a potential route of exposure. However, the insoluble nature of most metals in soil limits their bio-accessability for uptake into and through the skin. Where data is available, it shows that dermal uptake of metals is low (Paustenbach, 2000). The rate at which a metal is taken up into the outer layers of the skin is referred to as the *dermal uptake coefficient* (DUC). For the purposes of the current exposure assessment, the dermal uptake coefficients have been used to represent the amount of metal delivered to the skin surface from the soil that would be accessible for uptake. This is considered to be the *delivered dose* and has been considered to be equivalent to the dermal intake. A detailed

discussion of the derivation of the DUC values for each of the metals is provided in Appendix 7. The delivered dose, is calculated as shown in Eq A3-5. These values have been used in conjunction with the estimates of intake from other sources to provide an estimate of the total daily dose for each age group for each metal (Table A3-10).

Eq A3-5:

$$Intake_{dermal} = Intake_{dermout} + Intake_{der\,min}$$

$$Intake_{dermout} = \left[\left(Csoil_{out} * Time_{outwin} * SA_{win} * WF \right) + \left(Csoil_{out} * Time_{outsum} * SA_{sum} \right) \right] * A_{soil} * EF * DUC_{soil}$$

$$Intake_{der\,min} = \left[\left(Cdust_{in} * Time_{inwin} * SA_{win} * WF \right) + \left(Cdust_{in} * Time_{insum} * SA_{sum} \right) \right] * A_{soil} * EF * DUC_{soil}$$

Where:				
$Intake_{dermal}$	=	Dermal intake from soil/dust		µg/day
$Intake_{dermout}$	=	Dermal intake from soil/dust while outdoors		µg/day
$Intake_{dermin}$	=	Dermal intake from dust while indoors		µg/day
A_{soil}	=	Soil adhesion to skin		mg/cm^2/event
EF	=	Exposure Frequency (1 event/day)		event/day
DUC_{soil}	=	Dermal uptake coefficient		unitless
$Csoil_{out}$	=	Outdoor summer soil concentration (measured)		µg/g
WF	=	10% winter covering factor assumed (see above)		
$Cdust_{in}$	=	Indoor summer dust concentration (0.39 indoor dust to outdoor soil concentration ratio; see Appendix 6)		µg/g
SA_{win}	=	Exposed surface area during winter months; see Appendix 6		m^2
SA_{sum}	=	Exposed surface area during summer months; see Appendix 6		m^2
$Time_{insum}$	=	Fraction of time spent indoors during summer; see Appendix 6		unitless
$Time_{inwin}$	=	Fraction of time spent indoors during winter; see Appendix 6		unitless
$Time_{outsum}$	=	Fraction of time spent outdoors during summer; see Appendix 6		unitless
$Time_{outwin}$	=	Fraction of time spent outdoors during winter; see Appendix 6 unitless		

Table A3-10: Estimated Metal Intakes from Dermal Contact with Soil

Metal	Receptor	C_{soil} (μg/g)	A_{soil} (mg/cm^2/event)	DUC_{soil}	Intake$_{dermal}$ (μg/day)
Antimony	0 - 6 months	91.1	0.2	0.010	0.098
	7 mo - <5 years	91.1	0.2	0.010	0.167
	5 - <12 years	91.1	0.2	0.010	0.276
	12 - <20 years	91.1	0.07	0.010	0.146
	20 + years	91.1	0.07	0.010	0.156
Beryllium	0 - 6 months	4.56	0.2	0.010	0.00495
	7 mo - <5 years	4.56	0.2	0.010	0.00845
	5 - <12 years	4.56	0.2	0.010	0.0139
	12 - <20 years	4.56	0.07	0.010	0.00739
	20 + years	4.56	0.07	0.010	0.00789
Cadmium	0 - 6 months	35.3	0.2	0.010	0.038
	7 mo - <5 years	35.3	0.2	0.010	0.0649
	5 - <12 years	35.3	0.2	0.010	0.107
	12 - <20 years	35.3	0.07	0.010	0.0568
	20 + years	35.3	0.07	0.010	0.0606
Cobalt	0 - 6 months	262	0.2	0.004	0.0113
	7 mo - <5 years	262	0.2	0.004	0.0192
	5 - <12 years	262	0.2	0.004	0.0317
	12 - <20 years	262	0.07	0.004	0.0168
	20 + years	262	0.07	0.004	0.018
Copper	0 - 6 months	2720	0.2	0.010	2.93
	7 mo - <5 years	2720	0.2	0.010	4.99
	5 - <12 years	2720	0.2	0.010	8.23
	12 - <20 years	2720	0.07	0.010	4.37
	20 + years	2720	0.07	0.010	4.66
Nickel	0 - 6 months	17000	0.2	0.00038	0.695
	7 mo - <5 years	17000	0.2	0.00038	1.19
	5 - <12 years	17000	0.2	0.00038	1.96
	12 - <20 years	17000	0.07	0.00038	1.04
	20 + years	17000	0.07	0.00038	1.11

A3-2 Summary

For each receptor age group, daily metal intakes have been estimated for each of the pathways of concern. For each metal, intakes from all exposure pathways must be combined for each receptor in order to estimate the total daily dose received by each receptor age group. This summation of exposures is presented in Section 4.4 of the Human Health Risk Assessment main report (Part B).

A3-3 References

Baes, C.F., R.D. Sharp, A.L. Sjoreen, and R.W. Shor. 1984. Review and Analysis of Parameters and Assessing Transport of Environmentally Released Radionuclides Through Agriculture, Oak Ridge National Laboratory, Oak Ridge, TN.

Biró, B., I. Köves-Péchy, and I. Kádár. 1998. Toxicity of Some Field Applied Heavy Metal Salts to the Rhizobial and Fungal Microsymbionts of Alfalfa and Red Clover. Agrokémia és Talajtan 42:265-276.

Braillier, S., R.B. Harrison, C.L. Henry, and X. Dongsen. 1996. Liming effects on availability of Cd, Cu, Ni and Zn in a soil amended with sewage sludge 16 years previously. Water, Air and Soil Pollution. 86:195-206.

Cantox. 1999. Deloro Village Exposure Assessment and Health Risk Characterization for Arsenic and Other Metals, Cantox Environmental Inc., December, 1999.

CCME (Canadian Council of Ministers of the Environment). 1997. Canadian Soil Quality Guidelines for Copper. CCME Subcommittee on Environmental Quality Criteria for Contaminated Sites. ISBN 0-662-25520-8.

CEPA (Canadian Environmental Protection Act). 1994. Cadmium and its compounds. Priority substances list assessment report. Government of Canada: Environment Canada, Health Canada. ISBN 0-662-22046-3.

Dabeka, R.W. 1989. Survey of lead, cadmium, cobalt and nickel in infant formulas and evaporated milks and estimation of dietary intakes of the elements by infants 0-12 months old. Sci. Total Environ. 89:279-89.

Dabeka, R.W and A.D. McKenzie. 1995. Survey of lead, cadmium, fluoride, nickel and cobalt in food composites and estimation of dietary intakes of these elements by Canadians in 1986-1988. J.A.O.A.C. 78: 897-909.

Frank, R., K.I. Stonefield, P. Suda, and J.W. Potter. 1982. Impact of nickel contamination on the production of vegetables on an organic soil, Ontario, Canada, 1980-1981. Sci. Total Environ. 26:41-65.

FSA (Food Standards Agency). 1997. Food Safety Information Bulletin, Bulletin Number 90, November, 1997, Department of Health; United Kingdom.

Guo, Y., R. Schulz, and H. Marschner. 1995. Genotypic differences in uptake and distribution of cadmium and nickel in plants. Agnew. Bot. 69:42-48.

JWEL. 2001. 2001 Food Basket Collection. Unpublished data supplied by Jacques Whitford Environmental Limited on October 4, 2001.

JWEL. 2000a. Document in Preparation. Jacques Whitford Environmental Limited.

JWEL. 2000b. Garden Soil Data - Rodney Street Area, Sampled from the 18th-22nd of September, 2000. Jacques Whitford Environmental Limited.

L'Huillier, L., and S. Edighoffer. 1996. Extractability of nickel and its concentration in cultivated plants in Ni rich ultramafic soils of New Caledonia. Plant and Soil. 186:255-264.

Maclean, A.J. and A.J. Dekker. 1978. Availability of zinc, copper and nickel to plants grown in sewage-treated soils. Can. J. Soil Sci. 58:381-389.

MOE. 1977. Effect of Heavy Metal on the Growth of Lettuce, Celery, and Onion, Groetelaars Farm, Port Colborne.

MOE. 1978a. Investigations of the Effects of Heavy Metals on Muck Farms East of International Nickel Company, Port Colborne, Ontario, 1976 - 1977.

MOE. 1978b. Investigations of the Effects of Heavy Metals on Muck Farms East of International Nickel Company, Port Colborne, Ontario, 1976-1977. Ontario Ministry of the Environment, Air Resources Branch, Phytotoxicology Section.

MOE. 1979. Phytotoxicology Complaint Investigation, J. Overholt, Port Colborne, 1979.

MOE. 1980a. Investigations of Agronomic Problems Associated with Metal Toxicity on Muck Farms in the Vicinity of Inco, Port Colborne, 1976-1979.

MOE. 1980b. Phytotoxicology Complaint Investigation, J. Overholt, Port Colborne, 1980.

MOE. 1981. Effects of Heavy Metals and Root Knot Nematode on Celery Grown on Organic Soil in the Vicinity of International Nickel Company, Port Colborne, Ontario, 1980.

MOE. 1983. Levels of Ni, Cu, and Co in Vegetable and Soil Samples Collected from Residential Gardens in the Vicinity of Inco Limited, Port Colborne.

MOE. 1984a. Investigation of Alleged SO$_2$ Injury to Celery on Overholt Farm, Port Colborne, October 3, 1983.

MOE. 1984b. Nickel Phytotoxicity on Celery and Lettuce Grown on Soil Contaminated by a Nickel Refinery.

MOEE. 1994. Nickel Uptake into Vegetables Growing in Experimental Plots. Ecological Standards & Toxicology Section, MOEE, unpublished data.

MOE. 1999. Phytotoxicology Soil Investigation: Inco - Port Colborne (1998). Ontario Ministry of the Environment. Report No. SDB-031-3511-1999. ISBN 0-7778-9260-X.

MOE. 2000. Phytotoxicology Soil Investigation: Inco - Port Colborne (1999). Phytotoxicology and Soil Standards Section, Standards Development Branch, Ontario Ministry of the Environment. Report No. SDB-031-3511-2000.

MOE. 2001. Garden soil and plant data from Rodney Street, Sampled from November 2000 to January 2001.

OMAF. 1980. Report on Agronomic Problems Experienced by Three Growers Located Near Port Colborne.

Paustenbach, D.J. 2000. The Practice of Exposure Assessment: A State-of-the-Art Review. J. Toxicol. Environ. Health, Part B. 3:179-291.

Roberts, T.M., T.C. Hutchinson, J. Paciga, A. Chattopadhyay, R.E. Jervis, J. VanLoon, and D.K. Parkinson. 1974. Lead contamination around secondary smelters: estimation of dispersal and accumulation in humans. Science 186:1120.

Sajwan, K.S., W.H. Ornes, T.V. Youngblood, and A.K. Alva. 1996. Uptake of soil applied cadmium, nickel, and selenium by bush beans. Water, Air, Soil Pollut. 91:209-217.

Sauerbeck, D.R., and A. Hein. 1991. The nickel uptake from different soils and its prediction by chemical extractions. Water, Air, Soil Pollut. 57-58: 861-871.

Traynor, M.F. and B.D. Knezek. 1973. Effects of nickel and cadmium contaminated soils on nutrient composition of corn plants. Proc. Annual Conf. on Trace Substances in the Environment 7:82-87.

USDA. 1963. Agricultural Handbook No. 8., published by the United States Department of Agriculture (revised 1963).

US EPA. 1997. Exposure Factors Handbook. US Environmental Protection Agency. EPA/600/P-95/002Fa.

Vaessen, H.A.M.G., and B. Szteke. 2000. Beryllium in food and drinking water - a summary of available knowledge. Food Additives and Contaminants. 17(2): 149-159.

Vergnano Gambi, O., R. Gabbrielli, and L. Pancaro. 1982. Nickel, chromium and cobalt in plants from Italian serpentine areas. Acta Oecologica Oecologica Plantarum. 3(17): 291-306.

Human Health Risk Assessment

Appendix 4

Estimating Daily Intakes of Metals from Supermarket Food

Table of Contents
Human Health Risk Assessment - Appendix 4
Estimating Daily Intakes of Metals from Supermarket Food

A4-1 Estimating Metal Intakes from Supermarket Food

Estimates of the dietary intakes of metals from supermarket food by the general Canadian population are limited. Daily dietary intake estimates for cadmium, cobalt, copper and nickel have been published by CEPA (Health Canada and Environment Canada), CCME and others (see Table A4-1). Information on dietary intakes for all age groups is not available for all metals. For example, information on dietary intakes of antimony and beryllium are limited to single estimates of daily intake by the general population (FSA, 1997; Vaessen and Szteke, 2000).

A4-2 Estimating Dietary Intakes of Metals for All Age Groups

The lack of dietary intakes for all age groups used in this assessment required that intake estimates for specific age groups be derived from available information. The shaded areas in Table A4-1 indicate where such derivations have been necessary. Cadmium intake rates were recalculated from CEPA (1994a and 1994b) values using current receptor characteristic values to obtain units in µg/day.

Table A4-1: Estimated Total Daily Intakes of Metals from Supermarket Food

Receptor	Daily Intakes of Metals from Supermarket Food (µg/day)					
	Antimony	Beryllium	Cadmium	Cobalt	Copper	Nickel
Infant	1.3	4.8	5.08	4.18	518	109.2 (72.2-146.2)*
Toddler	2.3	8.6	10.6	7.0	822	190
Child	3.5	13.2	16.8	10.0	1230	251
Teen	4.0	15.0	17.3	12.0	1520	313
Adult	3.4	12.7	14.8	11.0	1430	307
Reference	FSA, 1997	Vaessen & Szteke, 2000	CEPA, 1994a	Dabeka & McKenzie, 1995	CCME, 1997	Dabeka, 1989; Dabeka & McKenzie, 1995

Shaded cells represent calculated values (see text for explanations).
*see table A4-4.

CEPA and CCME provide daily dietary intake estimates for cadmium, and copper for all age groups. The age groups examined by Dabeka and McKenzie (1995) differ slightly from those used by CEPA and CCME. Dabeka and McKenzie (1995) do not report intakes for children age 0 - 1 year, and their toddler age group includes children aged 1 - 4 years rather than the 7 months to less than 5 years used by CEPA and CCME. Dabeka and McKenzie (1995), CEPA and CCME use the same age groupings for children (5 - 11 years), teens (12 - 19 years) and adults (20+ years). For the purposes of this assessment the intake estimates provided by Dabeka and McKenzie (1995) for cobalt for toddlers, have been applied to the toddler (7 months to less than 5 years) used in this assessment. The nickel and cobalt intakes for infants, shown in Table A4-1 have been estimated based on the intakes reported for toddlers. The ratios were determined by

averaging the ratios of infant and toddler intakes for other metals as shown in equation A4-1.

Eq A4-1:

$$C_I = \left[\frac{\left(\dfrac{I_{As}}{T_{As}}\right) + \left(\dfrac{I_{Cd}}{T_{Cd}}\right) + \left(\dfrac{I_{Cu}}{T_{Cu}}\right) + \left(\dfrac{I_{Ni}}{T_{Ni}}\right)}{4} \right] = 0.598$$

Where: C_I = Correction factor for infant intake
　　　　　I_x = Infant intake for arsenic, cadmium, copper, and nickel
　　　　　T_x = Toddler intake for arsenic, cadmium, copper, and nickel

A4-3 Additional Information on Dietary Intakes for Antimony, Beryllium and Nickel

Dietary intake information for antimony and beryllium is limited to a single, general population estimate for each (FSA, 1997; Vaessen and Szteke, 2000). In order to develop likely total daily intakes from supermarket foods for all age groups of concern in this assessment, the single values have been used as a basis for estimating intakes in all age groups. A ratio process similar to the one outlined in Equation A4-1 was used. The estimated daily intakes of antimony and beryllium are 4 µg/day (FSA, 1997) and 15 µg/day (Vaessen and Szteke, 2000), respectively. A review of intake data for the other metals shows that the highest daily intakes of metals generally occurs in the "teen" age group for all metals considered (See Table A4-1). Therefore, the values reported for antimony and beryllium were assigned to this age group. Ratios for metal intakes between the teen age group and the other age groups were developed for arsenic, cadmium, cobalt and lead. The average of these values for each of the age groups was used to generate the intake estimates for antimony and beryllium for each of the age groups. The derivation of the ratios is shown in Table A4-2.

Table A4-2: Dietary Intake Ratios for Different Age Groups for Arsenic, Cadmium, Cobalt and Lead

Receptor	Daily Intakes of Metals from Supermarket Food (µg/day)								Averaging Ratio
	Arsenic[1]		Cadmium		Cobalt		Lead[2]		
	Intake	Ratio	Intake	Ratio	Intake	Ratio	Intake	Ratio	
Infant	19.70	0.28	5.08	0.29	4.18	0.35	8.97	0.37	0.32
Toddler	33.00	0.46	10.60	0.61	7.00	0.58	15.00	0.63	0.57
Child	62.50	0.87	16.80	0.97	10.00	0.83	20.00	0.83	0.88
Teen	71.60	1.00	17.30	1.00	12.00	1.00	24.00	1.00	1.00
Adult	42.40	0.59	14.80	0.86	10.50	0.88	25.70	1.07	0.85

[1] Dietary intake information for arsenic was taken from Yost et al., (1998).
[2] Information on lead in the diet comes from Dabeka and MacKenzie (1995).

Because nickel and copper can be added during food processing operations, it was felt that the levels of these metals would not provide a true reflection of trace metal levels in foods. Therefore nickel and copper were not considered in the development of the ratios used to estimate the daily intakes of the trace metals antimony and beryllium.

A4-3.1 Antimony

There is very limited data on dietary intakes of antimony in general/supermarket food. ATSDR (1990) estimated that the antimony concentration in the diet of a typical adult male was 9.3 µg/kg dry weight. The WHO used the information cited by the ATSDR to develop an estimate of the daily intake of antimony from food of 18 µg/day (WHO, 1996). Two studies that post date the work cited by ATSDR and the WHO have also examined dietary intakes of antimony (FSA, 1997 and Miahara et al., 1998). Miahara et al., (1998), examined antimony intakes in preschool children and the elderly in Brazil. Estimates of dietary intake ranged between 1.1 µg/day and 2.3 µg/day. The Food Standards Agency (1997) in Great Britain estimated dietary intakes of antimony in the British population. The study found a mean daily intake of 3 µg/day with a 97.5 percentile estimate of 4 µg/day. The study further noted that these values are approximately ten fold lower than the previous estimate of 29 µg/day that was based on a 1976 survey. The difference was attributed to a significant lowering of analytical detection limits between the time of the two studies (FSA, 1997). Although the WHO suggested a daily intake of 18 µg/day in 1996, this value was based on estimates developed before changes in analytical techniques allowed for better estimates of antimony levels in foods. As a result, the WHO value is likely to overestimate daily dietary intakes of antimony. For the purposes of this assessment, the upper estimate of 4 µg/day suggested by the FSA has been used to estimate dietary intakes of antimony for the residents of Rodney Street in Port Colborne. The upper FSA estimate (4 µg/day) was assumed to be a lifetime daily intake for a typical Rodney Street teen and was prorated to average estimates of supermarket food intake for other age classes as shown in Table A4-3.

A4-3.2 Beryllium

Information on the dietary exposure to beryllium is limited. Recently, a review of the worldwide literature on the occurrence of beryllium in food and drinking water and estimates of daily dietary exposure was sponsored by the Food Chemistry Commission of the International Union for Pure and Applied Chemistry (IUPAC) (Vaessen and Szteke, 2000). Beryllium levels in food were found to range from <1 to approximately 20 µg/kg fresh weight. In the US, the average beryllium concentration in drinking water is 0.2 µg/L. Estimates of beryllium intake from food consumption for the UK and the US ranged from 12 to 15 µg/day, however, these food intakes were considered to be rough estimates. The 15 µg/day estimate was assumed to be a life-time daily intake for a typical Rodney Street teen and was prorated to average estimates of supermarket food intake for other age classes as shown in Table A4-3.

Table A4-3: Sample Calculation of Estimated Dietary Intakes for Each Age Class Using Averaging Ratios

| Receptor | Averaging Ratio | Daily Intakes of Metals from Supermarket Food (µg/day) | | | |
| | | Antimony | | Beryllium | |
		Reported	Calculated	Reported	Calculated
Infant	0.323	-	1.3	-	4.8
Toddler	0.571	-	2.3	-	8.6
Child	0.878	-	3.5	-	13.2
Teen	1.00	4.0	4.0	15.0	15.0
Adult	0.848	-	3.4	-	12.7

A4-3.3 Nickel

Interpreting information from dietary intake studies requires assessing a whole range of information from levels of nickel in specific food items to how this information is integrated into overall population intakes by age class and averages and percentiles for each age class. Some agencies report just average intakes for the adult population, others indicate upper ranges of intake, as well, and sometimes just a range of intakes is reported. Consequently, the full range of intakes reported by various agencies is tabulated in Table A4-4.

Several studies have attempted to estimate the daily intake of nickel from supermarket or processed food in the Canadian and North American populations. Based on the US Food and Drug Administration's Total Diet Study of 1984, the mean nickel consumption of infants and young children was 69 to 90 µg/day (Pennington and Jones, 1987). Average daily dietary intake of nickel in the US has been reported as 168 µg/day (Myron et al., 1978; cited in ATSDR, 1997). A more recent review of dietary intake has included nickel intakes from dietary supplements and estimates that adults consume 76 to 105 µg/day of nickel from diet and supplements (IOM,

2001). The US dietary intake data formatted to match the Canadian age class groups is shown in Table A4-4.

A 1984 market basket survey of dietary nickel intake in England determined an intake of 154-166 µg/day (Smart and Sherlock, 1987). More recently, the results of the 1997 UK Total Diet Study were published (Ysart et al., 2000). The average dietary exposure for UK adults was 120 µg/day and the 97.5th percentile was 210 µg/day, similar to their 1994 survey. This information was prorated to Canadian age class intervals using the averaging ratios in Table A4-3 and is shown in Table A4-4.

CEPA, provides estimates of daily nickel intakes from food for the general Canadian population (CEPA, 1994b). These estimates are based on a survey of nickel concentrations in various foods conducted by National Health and Welfare, 1992 (unpublished, cited in CEPA, 1994b) and estimates of age specific food intakes derived from a Nutrition Canada, Environmental Health Directorate survey (CEPA, 1994c). More detailed information on dietary intakes of nickel by Canadians was reported in Dabeka and McKenzie (1995). The Canadian dietary intake of nickel for all ages, male and female, is 286 µg/day (Dabeka and McKenzie (1995)). It is not indicated whether the Canadian dietary intakes are averages or some upper range, however, inspection of the tables of nickel levels in various food categories indicates that the reported Canadian dietary intakes are average values.

Inspection of Table A4-4 shows that Canadian dietary nickel intakes are higher than US and UK estimates. Dabeka and McKenzie (1995) comment on this situation and indicate that the highest nickel intakes were for meat and poultry (about 40%), bakery goods and cereals (about 19%), soups (about 15%) and vegetables (about 11%). These data are felt to provide the best representation of likely nickel intakes from food for the Canadian population as a whole. Therefore these values have been used to represent the intake of nickel from non-home grown food sources for the residents of Rodney Street (Table A4-1).

The available research indicates that the use of stainless-steel cookware does not appreciably add to the amount of nickel or chromium ingested. A study by Accominotti et al., (1998) found that the levels of metals released during cooking were still less than the tolerable daily intake (TDI) recommended by the World Health Organization. This was supported by other studies which found that certain acidic fruits could leach metals. A study by Kuligowski and Halperin (1992) found that stainless is readily attacked by organic acids at cooking temperatures and mildly acidic pH. As a result, iron, chromium, and nickel did leach from the material into the food being prepared. Nickel was a major corrosion product from stainless steel utensils and the authors recommend that nickel-sensitive patients switch to utensils made from a material other than stainless steel. Studies have indicated that the vast majority of the nickel available to the food would be leached out of the cookware on the first use. However, this situation did not recur in subsequent usage, even after the cookware had been cleaned via abrasion (Flint and Packirisamy,

1997). The study by Dabeka and McKenzie (1995) did indicate that stainless steel cooking utensils (e.g., oven pan and roasting pan) appear to contribute to the higher levels of nickel in cooked steak, ground beef, port, lamb, and poultry. This may provide some rationale as to why the intake concentrations reported by Dabeka and his colleagues were higher than those presented in our jurisdictions.

Table A4-4: Estimated Daily Dietary Intake of Nickel for Various Countries

Source	Daily Intake 0 - 6 months (µg/day)	Daily Intake 1-4 year (µg/day)	Daily Intake 5-11year (µg/day)	Daily Intake 12-19 year (µg/day)	Daily Intake Adult (µg/day)
CEPA, 1994b	154	208	270	325	308
Dabeka, 1989; Dabeka and McKenzie, 1995	109.2 (72.2-146.2)*	190	251	313	307
US FDA Total Dietary Study (95th %ile) (IOM, 2001)	9(37) (reported)	81(153) (recalculated)	107(199) (recalculated)	125(250) (recalculated)	119(233) (recalculated)
UK Total Dietary Study 1997(97.5th %ile) (Ysart et al., 2000)	39(68) (calculated)	68(120) (calculated)	105(184) (reported)	120(210) (reported)	102(178) (calculated)

* the CEPA and Dabeka (1989) infant diet is based on dietary surveys taken in the 1970s. A comparison of the infants exposures under the following scenarios is provided:

1.   Diet A - assumes that the infant only consumes breast milk for six months;
2.   Diet B - assumes that the infant only consumes formula for six months; and
3.   Diet C - assumes that the infant consumes breast milk or formula only for the first three months, and this diet is then supplemented by vegetables, cereal and bread, and, fruit and fruit juices.

Diet A - Recent studies of nickel in human milk using the ICP-MS analytical method indicate low levels in European mothers. Biego et al., (1998) did not detect nickel in seventeen French breast milk samples at a reported detection limit of 2.9 µg/L. Krachler et al., (2000) analysed milk samples from 27 Austrian mothers and reported a median value of 0.79 µg/L (range - detection limit 0.13 µg/L to 6.35 µg/L). In a recent Canadian study, milk from 43 mothers living in Newfoundland was analysed during the first 12 weeks of breast feeding (Friel et al., 1999). Median values for nickel ranged from 0 to 28 µg/L. If an infant consumes 850 mL breast milk per day (Emmett et al., 2000), nickel intakes resulting from Diet A could range from 0.67 µg/day to 23.8 µg/day.

Diet B - Dabeka (1989) reported milk-based formulas as having half the nickel content of soy based formulas. For the zero - six month age group, reported nickel intakes from formula range from 35.7 µg/day (evaporated milk) to 74.7 µg/day (soy-based formula).

Diet C - The combination diet (Diet C) suggested above uses Nutrition Canada's values for vegetable, cereal and fruit intakes for this age group (33g, 34.4g and 75.7g/day, respectively). Using the overall nickel intake for each of these food groups based on Dabeka and McKenzie

(1995) and the Nutrition Canada food intake factors, this results in a nickel intake estimate of 143 µg/day. Diet A and three months of vegetable, cereal and fruit intakes suggests overall intakes of 72.2 µg/day to 95.3 µg/day over the first six months. Similarly, Diet B and three months of vegetable, cereal and fruit intakes suggests overall intakes of 107.2 µg/day to 146.2 µg/day over this period. The mid point of this range (72.2 to 146.2 µg/day) is 109.2 µg/day.

A4-4 References

Accominotti, M., M. Bost, P. Haudrechy, B. Mantout, P.J. Cunat, F. Comet, C. Mouterde, F. Plantard, P. Chambon, J.J. Vallon. 1998. Contribution to chromium and nickel enrichment during cooking of foods in stainless steel utensils. Contact Dermatitis. 38 (6): 305-10.

ATSDR (Agency for Toxic Substances and Disease Registry). 1990. Draft toxicological profile for antimony and compounds. Atlanta, GA, US Department of Health and Human Services, 1990.

ATSDR (Agency for Toxic Substances and Disease Registry). 1997. Toxicological Profile for Nickel. U.S. Department of Health and Human Services - Public Health Service (CDROM version, 2000).

Biego, G.H., M. Joyeux, P. Hartemann and G. Debry. 1998. Determination of mineral contents in different kinds of milk and estimation of dietary intake in infants. Food Additives Contam. 15 (7): 775-781.

CCME (Canadian Council of Ministers of the Environment). 1997. Canadian Soil Quality Guidelines for Copper. CCME Subcommittee on Environmental Quality Criteria for Contaminated Sites. ISBN 0-662-25520-8.

CEPA (Canadian Environmental Protection Act). 1994a. Cadmium and its compounds. Priority substances list assessment report. Government of Canada: Environment Canada, Health Canada. ISBN 0-662-22046-3.

CEPA (Canadian Environmental Protection Act). 1994b. Nickel and its compounds. Priority substances list assessment report. Government of Canada: Environment Canada, Health Canada. ISBN 0-662-22340-3.

CEPA (Canadian Environmental Protection Act). 1994c. Human Health Risk Assessment for Priority Substances. Health Canada. ISBN 0-662-22165-5.

Dabeka, R.W. 1989. Survey of lead, cadmium, cobalt and nickel in infant formulas and evaporated milks and estimation of dietary intakes of the elements by infants 0-12 months old. Sci. Total Environ. 89:279-289.

Dabeka, R.W and A.D. McKenzie. 1995. Survey of lead, cadmium, fluoride, nickel and cobalt in food composites and estimation of dietary intakes of these elements by Canadians in 1986-1988. J.A.O.A.C. 78: 897-909.

Emmett, P. K. North, S. Noble and the ALSPAC Study Team. 2000. Types of drinks consumed by infants at four and eight months of age: A descriptive study. Public Hlth. Nutr. 3: 211-217.

Flint, G.N. and S. Packirisamy. 1997. Purity of food cooked in stainless steel utensils. Food Addit Contam 14(2):115-26.

Food Standards Agency (FSA). 1997. Food Safety Information Bulletin, Bulletin Number 90, November, 1997, Department of Health; United Kingdom.

Friel, J.K., W.L. Andrews, S.E. Jackson, H.P. Longerich, C. Mercer, A. McDonald, B. Dawson and B. Sutradhar. 1999. Elemental composition of human milk from mothers of premature and full-term infants during the first three months of lactation. Biol. Trace Element Res. 67: 225-247.

Institute of Medicine - Food and Nutrition Board (IOM). 2001. Dietary Reference Intakes for Vitamin A, Vitamin K, Arsenic, Boron, Chromium, Copper, Iodine, Iron, Manganese, Molybdenum, Nickel, Silicon, Vanadium, and Zinc. National Academy Press, Washington, D.C.

Krachler, M., T. Prohaska, G. Koellensperger, E. Rossipal and G. Stingeder. 2000. Concentrations of selected trace elements in human milk and infant formulas determined by magnetic sector field inductively coupled plasma-mass spectrometry. Biol. Trace Element Res. 76: 97-111.

Kuligowski, J., and K.M. Halperin. 1992. Stainless steel cookware as a significant source of nickel, chromium, and iron. Arch Environ Contam Toxicol. 23(2) 211-5.

Miahara, V.A., M.B.A. Vasconcellos, M.B. Cordeiro, and S.M.F. Cozzolino. 1998. Estimate of toxic element intake in diets of pre-school children and elderly collected by duplicate portion sampling. Food Additives and Contaminants. 15: 782-788.

Myron, D.R., T.J. Zimmerman, T.R. Shuler, L.M. Klevay, D.E. Lee, F.H. Nielsen. 1978. Intake of nickel and vanadium by humans. A survey of selected diets. Am. J. Clin. Nutr. 31:527-531.

Pennington, J.A. and J.W. Jones. 1987. Molybdenum, nickel, cobalt, vanadium, and strontium in total diets. Research. 87: 1644-1650.

Smart, G.A, and J.C. Sherlock. 1987. Nickel in foods and the diet. Food Additives and Contaminants. 4: 61-71.

Vaessen, H.A.M.G., and B. Szteke. 2000. Beryllium in food and drinking water - a summary of available knowledge. Food Additives and Contaminants. 17(2): 149-159.

World Health Organization (WHO). 1996. Antimony. Guidelines for drinking-water quality, 2nd ed. Vol. 2 Health criteria and other supporting information. WHO, Geneva. pp. 147-156.

Yost, L.J., R.A. Schoof, and R. Aucoin. 1998. Intake of inorganic arsenic in the North American diet. Human and Ecological Risk Assessment. Vol. 4, No 1, pp. 137-152.

Ysart, G., P. Miller, M. Croasdale, H. Crews, P. Robb, M. Baxter, C. de L'Argy and N. Harrison. 2000. 1997 UK total diet study - dietary exposures to aluminium, arsenic, cadmium, chromium, copper, lead, mercury, nickel, selenium, tin and zinc. Food Additives and Contaminants. 17(9): 775-786.

Human Health Risk Assessment

Appendix 5

Bioaccessibility and Bioavailability of Metals Following Ingestion of Rodney Street Community Soils / Dusts and Other Environmental Media (Food, Drinking Water)

Table of Contents
Human Health Risk Assessment - Appendix 5
Bioaccessibility and Bioavailability of Metals Following Ingestion of Rodney Street
Community Soils / Dusts and Other Environmental Media (Food, Drinking Water)

A5-1 Bioavailability and Bioaccessibility

In risk assessment, oral exposures are typically stated in terms of the external dose or intake, instead of in terms of absorbed dose or uptake. Most regulatory exposure limits are expressed as intakes. The distinction between intake and uptake is an important one. The process of a chemical entering the body can be described in two steps - contact with an outer boundary (exposure or intake), followed by actual entry (absorption across the boundary into the bloodstream leading to some amount of the chemical reaching target tissues in the body, this is also called uptake). Intake is typically defined as the process by which an agent crosses the outer exposure surface of a human or animal *without passing an absorption barrier* (e.g., through ingestion or inhalation or soil contact with the skin), while uptake is the process by which an agent crosses an absorption barrier (the lining of the digestive system, the outer layer of the skin, or the lining of the nose, throat and lungs) into human or animal (IPCS, 2000).

In broad terms, the bioavailability ("absolute bioavailability") of a compound can be defined as the fraction of an administered dose that reaches the central (blood) compartment, whether through the gastrointestinal tract, skin, or lungs (NEPI, 2000). Bioavailability values are measured *in vivo* using animals or human volunteers. However, when evaluating potential differences in the bioavailability of a compound that is encountered *via* different routes of exposure, it is also useful to understand the "relative bioavailability" of the compound. Relative bioavailability refers to comparative bioavailabilities of different forms of a compound (e.g., metal species) or for different exposure media containing the chemical (e.g., bioavailability of a chemical bound to soil *versus* its bioavailability in water) (NEPI, 2000). Relative bioavailability is typically expressed as a relative absorption factor (RAF), which provides the absorbed fraction of the compound from a particular exposure medium relative to the fraction absorbed from the dosing vehicle used in the toxicity study for that particular compound (NEPI, 2000). Consideration of relative bioavailability allows variations in bioavailability of a chemical in a different exposure media to be adjusted to make intakes comparable.

Bioaccessibility is the fraction of a chemical in an environmental medium that is available for absorption based on *in vitro* extraction *but not necessarily absorbed*. This depends on the relation between *in vitro* chemical extraction systems and what is measured *in vivo* (in animals or humans). *In vitro* extraction methods were developed as an inexpensive alternative to more expensive *in vivo* experiments (Ruby et al., 1999). The term "bioaccessible" will be used to indicate the *in vitro* fraction of the chemical intake that is directly available for absorption. As the current assessment is primarily driven by the consideration of nickel ingestion, the focus of this discussion will be on the use of bioaccessibility in estimating oral exposures to nickel in different exposure media.

When considering exposure to a metal or other chemical contained in a complex environmental matrix such as soil, there are several aspects of how exposure to the chemical may be estimated.

First, there is the total chemical content of the soil. This is measured by harsh *in vitro* chemical extraction. Second, there is the fraction of the chemical which is extractable using gentler, *in vitro* chemical extraction procedures which simulate chemical conditions in the GI tract (the bioaccessible fraction). Third, there is the fraction of the chemical in the soil that can be absorbed *in vivo* (the bioavailable fraction).

Exposure assessment of the oral route of exposure considers ingestion of food, water and soil. The default relative bioavailability factor for intake estimates from ingestion is 1.0 in different exposure media. Use of this default value assumes that the bioavailability of the chemical being evaluated in any exposure medium will always be the same as its bioavailibilty using a different exposure medium than the one being assessed. More recently, as our understanding of how chemicals behave as they pass through the digestive tract is applied to exposure assessment methodologies, variations in relative bioavailability and the bioaccessible fraction of ingested chemicals is being taken into account. The US EPA has indicated that when the oral exposure limit is based on studies where the bioavailibilty of the chemical administered to the test animals differs from the exposure medium being considered, some adjustment of the oral exposure estimate is permitted (US EPA, 1989, 2001). What this means, in the case of soil ingestion, is that not all the metal that can be extracted from the soil using chemical methods that simulate the stomach and intestines is actually absorbed into the body, and that we can adjust the intake estimate using this information to account for differences in the relative bioavailability between that used in the toxicity study used to derive the R*f*D compared with the chemical in soil.

In the case of many metals, pH is an important variable controlling the bioaccessible fraction of ingested metals. As ingested materials containing these metals pass through the digestive tract, the low (acid) pH of the stomach can increase the bioaccessible fraction considerably while passing through this part of the digestive tract. After the stomach, the digestive tract reverts to higher pH (neutral to alkaline) and the bioaccessible fraction of metals may change. Investigators have tried to simulate conditions in the digestive tract experimentally using *in vitro* tests. These involve various degrees of complexity from simple acid solutions to controlled pH regimes and the addition of digestive enzymes, bile, food and other components of digestion (Ellickson et al., 2001, Rodriguez et al., 1999, Ruby et al., 1999). It should be kept in mind that the pH of the stomach can vary from pH 1 to pH 1.5 under fasting conditions and up to pH 4 during active digestion of food. The transit time of ingested materials is related to gastric emptying and can vary from less than an hour for liquids to over three hours for solid food. Whether the food is carbohydrate, protein or fat can also control transit time. Consequently, *in vitro* stomach acid tests using pH 1 to pH 1.5 represent fasting stomach conditions and are highly conservative. Bioaccessibility measured *in vitro* using simulated GI tract fluids may be a nonequilibrium process given the limited time for passage through the GI tract which may not allow the metal to completely dissolve. The validity of some *in vitro* models has been assessed by comparison with *in vivo* bioavailability assays in test animals.

Bioaccessibility of a metal is also affected by a large range of soil properties including particle size, whether the metal is part of the crystalline structure of a mineral in the soil or is adsorbed onto the surface of soil components, and soil pH. As noted in section A5-6, the preparation of the soil sample for bioaccessibility testing is important and should reflect as closely as possible the state of the soil that is likely to be ingested. Since hand-to-mouth activity accounts for most soil ingestion, particles of a size range that are most likely to adhere to skin on the hands, i.e., that pass through a 250 μm mesh screen, should be tested.

For nickel, our knowledge of oral bioavailability is based on soluble nickel salts administered to test animals and human subjects in the diet or in water (see discussion in sections A5-2 and A5-3). There is little information on oral exposure to insoluble nickel compounds. These studies indicate that soluble nickel salts administered under fasting conditions (stomach pH about 1), results in up to 30% absolute bioavailability (uptake will be 30% of intake). Under non-fasting conditions and in the presence of food (stomach pH about 4), bioavailability (uptake) of soluble nickel salts is much lower (2% to 5%). Limited information indicates that the bioavailability of insoluble nickel compounds is < 1%. Based on the data of Griffin et al., (1990) and Ishimatsu et al., (1995), it is assumed that soil bound insoluble nickel would be less bioavailable than soluble forms.

This appendix describes the available literature on the *in vivo* bioavailability (absolute and relative) of metals, particularly nickel. It should be noted that the focus of the exposure assessment part of the risk assessment is in developing intake estimates, not uptake estimates. The use of *in vitro* bioaccessiblity factors based on soil sieved to 250 μm(which is more likely to adhere to the skin and be transferred to the mouth by children) to adjust soil ingestion intakes will tend to overestimate relative bioavailability factors, and is a conservative and realistic method of estimating metal intakes from soil ingestion.

A5-2 Bioavailability of Nickel Ingested in Water or Food

As discussed in section A2-9, an important issue relating to nickel toxicity is its speciation (metallic, salt, oxide, etc.) and the solubility of different nickel compounds which strongly influences their absorption. There is little information on oral exposure to insoluble nickel compounds and it is assumed that insoluble forms of nickel would be less accessible than soluble forms. Several studies have indicate a lower bioavailability of insoluble forms of nickel are, Ishimatsu et al., (1995) and Griffin et al., (1990). These studies are cited and described in sections A5-3 and A5-4. None of these studies address *in vitro* bioaccessibility of nickel, though Griffin et al., (1990) provide indirect information on the relative bioavailability of soluble nickel in drinking water versus soil mixtures.

There are several studies of nickel administered to human volunteers where urinary excretion of nickel was measured and absorption of the administered dose was calculated. A recent review

summarized most of these studies (Diamond et al., 1998). The following table (Table A5-1) updates that review. It should be noted that all estimates of % bioavailability of nickel which are primarily based on cumulative urinary excretion of nickel were calculated using simplified pharmacokinetic models or the cumulative excretion of nickel as a percentage of the ingested dose over three, four or five days.

The studies not described by Diamond et al., (1998) address orally administered isotopes of nickel (Templeton et al., 1994b; Patriarca et al., 1997; Nielsen et al., 1999). Templeton et al., (1994b) administered ^{61}Ni orally in water to a human volunteer after an overnight fast. Urinary excretion of ^{61}Ni demonstrated absorption of 30% of the administered dose. Pairs of human subjects were administered ^{62}Ni in water (Patriarca et al., 1997); urinary and fecal excretion of ^{62}Ni followed for five days. Gastrointestinal absorption of ^{62}Ni was estimated as the difference between ingested nickel and nickel excreted in urine and the feces. The absorbed dose was 33.1± 4.9%. Nielsen et al., (1999) performed two studies to examine the influence of fasting and food intake on the absorption and retention of nickel added to drinking water. In the first study, volunteers drank nickel dissolved in water either four hours or 1.5 hours before or during a scrambled egg breakfast, or 0.5 to one hour after the breakfast. The results show that bioavailability is lowest when nickel is co-administered with food, intermediate when food is taken within about an hour before or after the food and highest (approaching the high bioavailability shown by the fasting stomach) at four hours after eating.

All of these bioavailability studies involve ingestion of soluble nickel either in drinking water or mixed into food. Inspection of Table A5-1 shows fairly clearly that gastrointestinal absorption of soluble nickel in drinking water is maximal at 23% to 33% under fasting conditions (Cronin et al., 1980; Nielsen et al., 1999; Patriarca et al., 1997; Sunderman et al., 1989; Templeton et al., 1994b). Absorption of nickel is much lower in the presence of food (Christensen and Lagesson, 1981; Gawkrodger et al., 1986; Horak and Sunderman, 1973; McNeely et al., 1972; Menne et al., 1978; Nielsen et al., 1999; Spruit and Bongaarts, 1977; Sunderman et al., 1989). It is not clear whether the lowered absorption of nickel in the presence of food is due to nickel binding to food components or pH changes in the stomach associated with the presence of food. Patterns of peristalsis and gastric emptying into the intestines during ingestion may influence nickel absorption (Nielsen et al., 1999). No differences in nickel absorption related to gender or nickel sensitivity were found (Menne et al., 1978; Nielsen et al., 1999; Patriarca et al., 1997).

A5-3 Oral Bioavailability of Nickel in Animals

Studies in mice, rats and dogs indicate that over10% of nickel, given as soluble nickel, nickel sulphate, or nickel chloride in the diet or by gavage, is rapidly absorbed by the gastrointestinal tract (Ambrose et al., 1976; Ho and Furst 1973; Ishimatsu et al.,1995; Nielsen et al., 1993; Phatak and Patwardhan, 1952; Severa et al., 1995; Tedeschi and Sunderman, 1957). Many of these studies with high oral bioavailability results only tested single doses up to 48 hours,

whereas long term studies suggested that overall oral bioavailability is much lower (Ambrose et al., 1976; Severa et al., 1995).

Absorption by the gastrointestinal tract depends on the solubility of the nickel compound. In a study in which rats were treated with a single gavage dose of a nickel compound (10 mg nickel) in a 5% starch saline solution, the absorption was found to be directly correlated with the solubility of the compound (Ishimatsu et al., 1995). The percentages of the dose absorbed were 0.01% for green nickel oxide, 0.09% for metallic nickel, 0.04% for black nickel oxide, 0.47% for nickel subsulphide, 11.12% for nickel sulphate, 9.8% for nickel chloride, and 33.8% for nickel nitrate. Absorption was higher for the more-soluble nickel compounds. Unabsorbed nickel is excreted in the feces.

$^{57}NiCl_2$ was administered either orally by gastric intubation or by intraperitoneal injection to *ad libitum* fed mice in doses equivalent to the average human daily dietary nickel intake (Nielsen et al., 1993). Whole body retention was measured by whole body counting of the radiation emitted by ^{57}Ni. The intestinal absorption was estimated to be 1.7% to 10% after 20 to 50 hours.

Wistar strain rats were given 100 mg Ni/L (as nickel sulphate) in drinking water for six months (Severa et al., 1995). The urinary excretion of the orally administered nickel was estimated to be 2% assuming a 1% absorption of the administered dose, however, recalculation of the reported nickel concentrations in blood and urine suggest that the actual uptake may have been much lower than 1%.

A5-4 Bioavailability of Nickel Ingested in Soil

The oral bioavailability of nickel in soil is not well characterized. Currently, there are no published reports on the human bioavailability of nickel in soil. In the only study available, Griffin et al., (1990) compared the oral bioavailability of nickel chloride, a soluble salt, administered to rats either as an aqueous slurry with sandy loam, or clay loam soil, or in water. Nickel bioavailability was estimated from levels of nickel in the blood. The soil bound nickel had a reduced bioavailability relative to nickel chloride in water. The relative bioavailabilities of nickel in the sandy and clay loam slurries were 63% and 34%, respectively. The mean absolute bioavailabilities were 3% and 1.5% for the sandy and clay loam slurries, respectively. These absolute bioavailabilities are similar to those reported for nickel administered in animal diets.

A5-5 Bioavailability and Bioaccessibility of Other Metals in Soil

Recent papers discussing the oral bioavailability of metals in soil as assessed by *in vitro* and *in vivo* techniques focus on arsenic and lead because of their large historic database (Ellickson et al., 2001; Hamel et al., 1998; Ruby et al., 1999). For both lead and arsenic, the bioavailability in soil has been shown to be reduced when compared to the bioavailability of soluble forms, i.e., the relative bioavailability is less than the default value of 1.0. *In vivo* and *in vitro* studies have yielded similar results (Ruby et al., 1999).

Table A5-1: Summary of Studies to Determine Oral Bioavailability of Soluble Nickel Salts

Study	n	Exposure medium	Duration	Fasting Status	% Bioavailability
Sunderman et al., 1989	88	water breakfast	acute acute	fasted fasted	29.3 1.8
Cronin et al., 1980	5	gelatin capsule + 100 mL of water	acute	fasted	12 - 32
Christensen and Lagesson, 1981	8	capsule	acute	w/meal	5.7
Gawkrodger et al., 1986	3	capsule	acute	w/meal	2.7, 2.8
Menne et al., 1978	6	capsule	acute	not fasted	2.2 (women)
Menne et al., 1978	7	capsule	acute	not fasted	1.7 (men)
Spruit and Bongaarts, 1977	1	solution	acute	not fasted	0.8
Horak and Sunderman, 1973	10 - 50	food	chronic	not fasted	1
McNeely et al., 1972	1920	food + water food	chronic chronic	not fasted not fasted	1.6 1.2
Templeton et al., 1994a	1	1 L water	acute	fasted	30
Patriarca et al., 1997	4	capsules + water	acute	fasted	33.1 (men and women)
Nielsen et al., 1999	1	eggs 4.5 hours prior to Ni in 250 mL water	acute	fasted	23.2 (men)
Nielsen et al., 1999	1	eggs 1.5 hours prior to Ni in 250 mL water	acute	fasted	7.1 (men)
Nielsen et al., 1999	1	Ni in 250 mL water and eggs 0.5 or 1hour later	acute	fasted	12.8 (men) 16.7 (men)
Nielsen et al., 1999	1	Ni in 250 mL water and eggs together	acute	fasted	3.4 (men) 2.3 (men)
Nielsen et al., 1999	20 20	Ni in 250 mL water and eggs 4 hours later	acute acute	fasted fasted	10.8 (1.8 - 29.5) (women) 11.3 (4 - 25.1) (women)

A5-6 *In vitro* Bioaccessibility Testing

In vitro methods for assessing oral bioavailability of heavy metals found in impacted soils and subsequently in exposure assessments of the human digestive system have been under active development for the past decade. As noted in section A5-1, *in vitro* methods may overestimate *in vivo* measurements. Establishment of validated models for extraction of metals in digestive fluids to provide a tool for determining the metal exposure from any soil has been the focus of a number of groups (US Naval Facilities Engineering Command, 2000, Solubility/Bioavailability Research Consortium (Ruby et al., 1999), NEPI, 2000). Imitation biological fluids of the digestive tract can be prepared chemically, and their extraction capabilities examined to provide a measure of the maximum amount of the metal that can be made accessible in the gastrointestinal tract (bioaccessible). However, the amount that may cross the digestive tract membranes and be absorbed into the tissues requires an *in vivo* method. The artificial fluids evaluated in various studies of sequential extraction are 1) saliva, 2) gastric juice, and 3) duodenal fluid, which represent the major portions of the gastrointestinal tract involved in metal uptake by the body.

Ingested metals are transported by saliva to the stomach where the low pH enhances the dissolution of many metals. The first region of the small intestine, the duodenum, is the primary site of absorption of most metals, however, the conditions in the mouth and stomach may be important preconditions to uptake in this portion of the intestine. Enzymes in the prepared bio-fluids, the soil type chosen, and the soil matrix appear to also play a role in extraction capacity. Accurate simulation of the human digestive system includes consideration of fluid volumes, pH, additional digestive components (enzymes, salts, etc.), peristaltic behavior (fasting vs. presence and type of food) and residence time.

Saliva is 99.5% water and contains sodium, potassium and chloride as the dominant ions. The main digestive enzymes are ptyalin (salivary amylase) secreted by the salivary glands. Lingual glands secrete a lipase. Saliva also contains mucin, a glycoprotein which lubricates the food. The optimal pH for ptyalin is pH 6.7. The normal flow rate of saliva ranges from 0.25 mL/min in the un-stimulated state up to 1 mL/min following stimulation by chewing. Consequently, over a one hour period the maximal production of saliva would be about 60 mL. The role of saliva in absorbing metals in the mouth does not appear to have been studied widely but since the pH of saliva is close to neutral, it is not expected to facilitate metal dissolution from soil.

Gastric secretion is stimulated by the presence of food in the stomach. Gastric acid contains 0.2% to 0.5% HCl (approx. 0.17 N HCl). The lowest pH is 0.87, but the resting human stomach is pH 1 to pH 1.5. Gastric fluid is mainly water with mucin, inorganic salts, pepsin (activated by HCl), rennin and gastric lipases. The stomach can hold two to three L, and produces up to 2.5 L of gastric fluids daily (ICRP, 1984, Ganong, 1989).

The small intestine is the largest part of the gastrointestinal tract and is composed of the duodenum which is about one foot long, jejunum (five - eight feet long), and ileum (16 - 20 feet long). The duodenum is the major portion of the small intestine where enzyme secretion takes

place. The small intestine secretes sucrase, maltase, and lactase. It also secretes peptidase, and lipase. The duodenum receives bile from the liver and gallbladder. Lipase, amylase, trypsin, chymotrypsin and sodium bicarbonate to neutralize the acid chyme from the stomach are received from the pancreas. Epithelial cells in the small intestine secrete several litres/day of a neutral fluid. Brunner's glands in the duodenum secrete mucus. Almost 90% of the daily fluid intake is absorbed in the small intestine.

Altogether food can remain in the small intestine between three - ten hours normally. Depending on what is in the stomach (water, solid food or both), gastric emptying into the duodenum can occur within 75 minutes to 3.5 hours (up to five hours) (ICRP, 1984, Ganong, 1989).

MOE used two methods to assess the *in vitro* bioaccessibility of metals of concern in Rodney Street community soils. Initially MOE used fasting stomach simulation tests based on 24 hour extractions, which are described in section A5-6.1. In addition, to validate the MOE fasting stomach simulation tests, and to account for metal bioaccessibility in other parts of the gastrointestinal tract, Rodney Street community soil samples were also sent out for testing by Exponent using the Standard Operating Protocol developed by the Solubility / Bioavailability Research Consortium (Ruby et al., 1999). This protocol has been validated by comparison to animal studies for arsenic and lead, but not for nickel. The results of the bioaccessiblity testing by the external laboratory are reported in section A5-6.2.

A5-6.1 Simulated Stomach Acid Leachate Tests

MOE adapted the traditional Leachate Extraction Procedure (formerly described in Reg. 347), which is based on the Toxicity Characteristic Leaching Procedure, Method 1311 that appears in the United States Environmental Protection Agency Publication SW-846 entitled "Test Methods for Evaluating Solid Waste, Physical/Chemical Methods", as amended from time to time and available online at http://www.epa.gov/epaoswer/hazwaste/test/main.htm to simulate leaching of metals from soil under the acidic conditions found in the stomach. This method was originally designed to investigate the leaching behavior of various hazardous chemicals in municipal, industrial and hazardous wastes.

Soil samples were analyzed for bioaccessibility utilizing this adapted leaching method to simulate stomach digestion conditions. In this method bioaccessibility is estimated by comparing the amount of total metal extracted by routine ICP analysis with the amount of metal extracted from the soil samples using the simulated stomach acid digestion. The MOE Leachate Extraction Procedure was modified as follows:

- The 0.5 N acetic acid was replaced with 0.17 N HCL;
- 20 g dry weight soil sample used instead of 50 g;
- 400 ml acid used instead of 800 ml acid; and
- Initial pH adjusted to pH 1 instead of pH 5.

This approach has been used to assess the amount of metal that would be released into the

stomach from soil samples in other MOE community exposure studies.

Ten soil samples from the Rodney Street community containing very high levels of nickel were selected for simulated stomach acid leach testing. For each soil sample, 20g of dried, sieved material (355 μm mesh size) was added to 400 ml of 0.17 N HCl (pH 1.0). The samples were agitated for 24 hours on a rotary extractor. The mixture was then filtered through a 4.5 micron filter and the filtrate was analyzed for metals and hydrides. For each sample, the percentage leached was calculated by dividing the mass of the metal in the leachate by the mass in the original soil sample and then multiplying the ratio by 100. The results of the analyses are shown in Tables A5-2 through A5-7.

It is recognized that the use of a 24 hour digestion period, which is longer than the typical residency times for food in the stomach, will overestimate the amount of metal that will be released and available. However, these simulated stomach acid leach tests were run at room temperature not at body temperature. Subsequent extractions using this method at higher temperatures and for shorter time periods (not reported) did not result in substantially reduced results. It was believed that as a precautionary measure, that the potential overestimation of exposures was justified.

The Simulated Stomach Acid Leachate Test results displayed below (Tables A5-2 to A5-7) are the same laboratory data used in the March 2001 report. However, the data have been re-calculated to reflect the mass of metal extracted as a percentage of the mass of metal in the soil sample. Beryllium and cadmium levels in leachate were below detection limits.

Table A5-2: Simulated Stomach Acid Leachate Test: Antimony

Level in Soil (ppm)[1]	Mass in 20 g (µg)	Amount Leached (ppm)[1]	Mass in Leachate (µg)	% Leached by Mass
2.5	49.1	0.004	1.6	3.3
1.8	36.4	0.0035	1.4	3.9
2.1	41.9	0.0033	1.3	3.2
2.3	46.7	0.0033	1.3	2.8
2.8	56.5	0.0036	1.4	2.6
2.5	50.5	0.003	1.2	2.4
2.2	44.2	0.0033	1.3	3.0
2.0	40.2	0.0026	1.0	2.6
2.4	48.3	0.0025	1.0	2.1
2.1	41.0	0.0023	0.9	2.2
Averaged Values				
2.3	45.5	0.00314	1.3	2.8
Minimum % Leached				2.1
Maximum % Leached				3.9

[1] ppm is equivalent to µg/g in soil and µg/ml in leachate.
Note: numbers may not equate due to rounding.

Table A5-3: Simulated Stomach Acid Leachate Test: Arsenic

Level in Soil (ppm)[1]	Mass in 20 g (µg)	Amount Leached (ppm)[1]	Mass in Leachate (µg)	% Leached by Mass
52.0	1040	0.7	281.6	27.1
39.1	782	0.6	222.4	28.4
45.0	900	0.6	230.4	25.6
50.2	1004	0.7	275.6	27.5
63.1	1262	0.4	158.4	12.6
44.8	896	0.4	154.4	17.2
43.1	862	0.4	172.0	20.0
42.2	844	0.5	210.4	24.9
62.3	1246	0.5	217.6	17.5
37.6	752	0.5	205.6	27.3
Averaged Values				
47.9	958.8	0.5	212.8	22.8
Minimum % Leached				12.6
Maximum % Leached				28.4

[1] ppm is equivalent to µg/g in soil and µg/ml in leachate.

Note: numbers may not equate due to rounding.

Table A5-4: Simulated Stomach Acid Leachate Test: Cobalt

Level in Soil (ppm)[1]	Mass in 20g (µg)	Amount Leached (ppm)[1]	Mass in Leachate (µg)	% Leached by Mass
200	4000	2.0	784	19.6
180	3600	1.7	664	18.4
130	2600	1.2	468	18.0
140	2800	1.2	492	17.6
210	4200	2.4	940	22.4
160	3200	1.7	684	21.4
220	4400	1.7	676	15.4
150	3000	1.9	740	24.7
230	4600	1.4	576	12.5
120	2400	1.3	516	21.5
Averaged Values				
174	3480	1.6	654	19.2
Minimum % Leached				12.5
Maximum % Leached				24.7

[1] ppm is equivalent to µg/g in soil and µg/ml in leachate.

Note: numbers may not equate due to rounding.

Table A5-5: Simulated Stomach Acid Leachate Test: Copper

Level in Soil (ppm)[1]	Mass in 20g (µg)	Amount Leached (ppm)[1]	Mass in Leachate (µg)	% Leached by Mass
990	19800	17.2	6880	34.8
770	15400	17.1	6840	44.4
1000	20000	19.1	7640	38.2
780	15600	14.2	5680	36.4
1000	20000	15.9	6360	31.8
840	16800	14.7	5880	35.0
1000	20000	20.5	8200	41.0
980	19600	20.7	8280	42.2
970	19400	16.1	6440	33.2
640	12800	14.0	5600	43.8
Averaged Values				
897	17940	17.0	6780	38.1
Minimum % Leached				31.8
Maximum % Leached				44.4

[1] ppm is equivalent to µg/g in soil and µg/ml in leachate.

Note: numbers may not equate due to rounding.

Table A5-6: Simulated Stomach Acid Leachate Test: Lead

Level in Soil (ppm)[1]	Mass in 20g (µg)	Amount Leached (ppm)[1]	Mass in Leachate (µg)	% Leached by Mass
400	8000	15.6	6240	78.0
480	9600	21.1	8440	87.9
350	7000	12.8	5120	73.1
310	6200	11.1	4440	71.6
400	8000	13.3	5320	66.5
370	7400	14.4	5760	77.8
300	6000	9.17	3668	61.1
350	7000	11.4	4560	65.1
360	7200	15.4	6160	85.6
290	5800	13.1	5240	90.3
Averaged Values				
361	7220	13.7	5495	75.7
Minimum % Leached				61.1
Maximum % Leached				90.3

[1] ppm is equivalent to µg/g in soil and µg/ml in leachate.

Note: numbers may not equate due to rounding.

Table A5-7: Simulated Stomach Acid Leachate Test: Nickel

Level in Soil (ppm)[1]	Mass in 20g (µg)	Amount Leached (ppm)[1]	Mass in Leachate (µg)	% Leached by Mass
8800	176000	86.2	34480	19.6
9200	184000	107	42800	23.3
11000	220000	93	37200	16.9
11000	220000	87.9	35160	16.0
12000	240000	88.5	35400	14.8
13000	260000	96.9	38760	14.9
14000	280000	127	50800	18.1
14000	280000	115	46000	16.4
16000	320000	104	41600	13.0
17000	340000	99.9	39960	11.8
Average				
12600	252000	100.5	40216	16.5
Minimum % Leached				11.8
Maximum % Leached				23.3

[1] ppm is equivalent to µg/g in soil and µg/ml in leachate.

Note: numbers may not equate due to rounding.

A5-6.2 External Bioaccessibility Studies
A5-6.2.1 Methods

The following sections describe the manner in which the soil samples were prepared for analysis, the methods by which the bioaccessibility analyses were performed, and the results of these analyses.

Sample preparation included some preparation by the MOE Phytotoxicology Laboratory (described below). Further sample preparation and bioaccessibility extractions were performed in Exponent's laboratory in Boulder, Colorado (Exponent, 2001). Analyses for total metals concentrations for the eight metals of interest in the sample substrates and extraction fluids were conducted by Columbia Analytical Services, Inc. (CAS) in Kelso, Washington.

A5-6.2.2 Sample Preparation and Analysis

Twenty samples were received at Exponent's Boulder laboratory in two separate shipments in May, 2001. The first shipment consisted of ten samples. Prior to receipt by Exponent, these samples had been dried, ground, and sieved to <350 µm, and each sample had been labeled with a three or four digit sample number. Exponent assigned six digit sample numbers; "SLO" was prefixed to the three digit sample numbers, and "SL" was prefixed to the four digit sample numbers. The dry samples were then sieved to <250 µm. These samples are referred to as "ground soil" in the tables.

The second shipment also consisted of ten samples. The soil samples in this shipment were "bulk" samples which had not been ground and sieved to <350 µm. Each sample had been similarly labeled with a three or four digit sample number. Again, Exponent assigned six digit sample numbers; "CSO" was prefixed to the three digit sample numbers, and "CS" was prefixed to the four digit sample numbers. The samples were then dried in the oven at 100°C for 24 hours, after which they were sieved to <250 µm. These samples are termed "sieved fine soil" in the tables.

The <250 µm soil size fraction was used for bioaccessibility testing, because it is believed to represent the fraction of soil that is most likely to adhere to human hands and become ingested during hand-to-mouth activity (Maddaloni et al., 1998). A one gram aliquot of each substrate was collected and subjected to the *in vitro* extraction procedure (described below). The rest of the sample was used for analysis of total metal concentrations for the eight metals of interest. As a quality control measure, an additional aliquot of one soil sample (sample number CS3740) was submitted as a triplicate for total metal analysis.

A5-6.2.3 Bioaccessibility Testing

The sieved soil samples (<250 µm size fraction) were subjected to bioaccessibility testing according to the Standard Operating Procedure (SOP) developed by the Solubility/ Bioavailability Research Consortium (SBRC) (Ruby et al., 1999, Exponent, 2001). This protocol

is provided below *(in italics)*. The testing included extraction and analysis of one sample in triplicate (sample number SLO 415).

Deviations from the SBRC method with regard to sample preparation and analysis included the following:

- Analyses were conducted for antimony, beryllium, cadmium, cobalt, copper, and nickel, in addition to the analyses for arsenic and lead as specified in the SOP.

- The bioaccessibility test was modified to include a simulation of the small intestinal environment (i.e., a second phase, at neutral pH, was added to the extraction procedure). This was done to evaluate whether an extraction procedure that simulates the environment of the small intestine would influence the bioaccessibility of metals from the sample substrates (e.g., by affecting either the metal solubility or the integrity of the soil matrix that contains the metals). This was accomplished by adding the following steps at the end of the standard SBRC extraction procedure:

1. At the end of the one hour extraction, a 5mL sample of the extraction fluid was collected and preserved with nitric acid for analysis (as in the methods described in the SOP).

2. The extraction fluid in each bottle was then titrated to pH 7.0 ± 0.2 with NaOH (50% w/w) (this required approximately 20 drops of NaOH solution).

3. Once the extraction fluid had been neutralized, 175 mg of bile salts and 50 mg of pancreatin were added to each extraction bottle, and the bottles were returned to the extractor for an additional four hours of extraction time.

4. At the end of the small-intestinal-phase extraction, a 10mL sample of the extraction fluid was collected from each bottle and filtered for analysis.

5. Matrix interference required that the arsenic analyses be conducted by graphite furnace atomic absorption (GFAA) rather than the inductively coupled plasma (ICP) method as specified in the SOP.

All of the extracts produced from the bioaccessibility testing were shipped to CAS for analysis of total concentrations of antimony, arsenic, beryllium, cadmium, cobalt, copper, lead and nickel.

It should be noted that the Exponent extraction method includes both acid and neutral pH extractions with realistic residency times in the stomach (one hour) and intestines (four hours).

Solubility/Bioavailability Research Consortium Standard Operating Procedure

In vitro Method for Determination of Lead and Arsenic Bioaccessibility

Procedure

Sample Preparation - *All soil/material samples should be prepared for testing by oven drying (<40°C) to reduce volatilization of any volatile metals and sieving to <250 μm. The <250-μm size fraction is used because this particle size is representative of that which adheres to children's hands. Sub-samples for testing in this procedure should be obtained using a sample splitter.*

Apparatus and Materials

Equipment - *The main piece of equipment required for this procedure consists of a Toxicity Characteristic Leaching Procedure (TCLP) extractor motor that has been modified to drive a flywheel. This flywheel in turn drives a Plexiglass block situated inside a temperature controlled water bath. The Plexiglass block contains ten 5cm holes with stainless steel screw clamps, each of which is designed to hold a 125mL wide mouth high density polyethylene (HDPE) bottle. The water bath must be filled such that the extraction bottles are immersed. Temperature in the water bath is maintained at 37±2°C using an immersion circulator heater (for example, Fisher Scientific Model 730). Additional equipment for this method includes typical laboratory supplies and reagents, as described in the following sections.*

The 125mL HDPE bottles must have an air tight screw cap seal (for example, Fisher Scientific 125mL wide mouth HDPE Cat. No. 02-893-5C), and care must be taken to ensure that the bottles do not leak during the extraction procedure.

Standards and Reagents - *The leaching procedure for this method uses a buffered extraction fluid at a pH of 1.5. The extraction fluid is prepared as described below.*

The extraction fluid should be prepared using ASTM Type II deionized (DI) water. To 1.9 L of DI water, add 60.06 g glycine (free base, Sigma Ultra or equivalent). Place the mixture in a water bath at 37°C until the extraction fluid reaches 37°C. Standardize the pH meter using temperature compensation at 37°C or buffers maintained at 37°C in the water bath. Add concentrated hydrochloric acid (12.1 N, Trace Metal grade) until the solution pH reaches a value of 1.50 ±0.05 (approximately 120 mL). Bring the solution to a final volume of 2 L (0.4 M glycine).

Cleanliness of all reagents and equipment used to prepare and/or store the extraction fluid is essential. All glassware and equipment used to prepare standards and reagents must be properly cleaned, acid washed, and finally, rinsed with DI water prior to use. All reagents must be free of lead and arsenic, and the final fluid should be tested to confirm that lead and arsenic concentrations are less than 25 and 5 μg/L, respectively.

Leaching Procedure - *Measure 100 ±0.5 mL of the extraction fluid, using a graduated cylinder, and transfer to a 125mL wide mouth HDPE bottle. Add 1.00 ±0.05 g of test substrate (<250 μm) to the bottle, ensuring that static electricity does not cause soil particles to adhere to the lip or outside threads of the bottle. If necessary, use an antistatic brush to eliminate static electricity*

prior to adding the soil. Record the volume of solution and mass of soil added to the bottle on the extraction test checklist. Hand tighten each bottle top, and shake/invert to ensure that no leakage occurs, and that no soil is caked on the bottom of the bottle.

Place the bottle into the modified TCLP extractor, making sure each bottle is secure and the lid(s) are tightly fastened. Fill the extractor with 125mL bottles containing test materials or Quality Control samples.

The temperature of the water bath must be 37±2°C. Record the temperature of the water bath at the beginning and end of each extraction batch on the appropriate extraction test checklist sheet.

Rotate the extractor end over end at 30±2 rpm for 1 hour. Record start time of rotation.

When extraction (rotation) is complete, immediately remove bottles, wipe them dry, and place them upright on the bench top.

Draw extract directly from reaction vessel into a disposable 20ml syringe with a Luer-Lok attachment. Attach a 0.45-µm cellulose acetate disk filter (25 mm diameter) to the syringe, and filter the extract into a clean 15mL polypropylene centrifuge tube or other appropriate sample vial for analysis. Store filtered sample(s) in a refrigerator at 4°C until they are analyzed.

Record the time that the extract is filtered (i.e., extraction is stopped). If the total elapsed time is greater than1 hour 30 minutes, the test must be repeated.

Measure and record the pH of fluid remaining in the extraction bottle. If the fluid pH is not within ±0.5 pH units of the starting pH, the test must be discarded and the sample re-analyzed as follows.

If the pH has dropped by 0.5 or more pH units, the test will be re-run in an identical fashion. If the second test also results in a decrease in pH of greater than 0.5 s.u., the pH will be recorded, and the extract filtered for analysis. If the pH has increased by 0.5 or more units, the test must be repeated, but the extractor must be stopped at specific intervals and the pH manually adjusted down to pH 1.5 with dropwise addition of HCl (adjustments at 5, 10, 15 and 30 minutes into the extraction, and upon final removal from the water bath [60 minutes]). Samples with rising pH values must be run in a separate extraction, and must not be combined with samples being extracted by the standard method (continuous extraction).

Extracts are to be analyzed for lead and arsenic concentration using analytical procedures taken from the US EPA publication (US EPA, 2000), Test Methods for Evaluating Solid Waste, Physical/Chemical Methods, SW-846 (current revisions). Inductively coupled plasma (ICP) analysis, method 6010B (December 1996 revision) will be the method of choice. This method should be adequate for determination of lead concentrations in sample extracts, at a project required detection limit (PRDL) of 100 µg/L. The PRDL of 20 µg/L for arsenic may be too low for ICP analysis for some samples. For extracts that have arsenic concentrations less than five

times the PRDL (e.g., <100 µg/L arsenic), analysis by ICP-hydride generation (method 7061A, July 1992 revision) or ICP-MS (method 6020, September 1994 revision) will be required.

Calculation of the Bioaccessibility Value *- A split of each solid material (<250 µm) that has been subjected to this extraction procedure should be analyzed for total lead and/or arsenic concentration using analytical procedures taken from the US EPA publication (US EPA, 2000), Test Methods for Evaluating Solid Waste, Physical/Chemical Methods, SW-846 (current revisions). The solid material should be acid digested according to method 3050A (July 1992 revision) or method 3051 (microwave-assisted digestion, September 1994 revision), and the digestate analyzed for lead and/or arsenic concentration by ICP analysis (method 6010B). For samples that have arsenic concentrations below ICP detection limits, analysis by ICP-hydride generation (method 7061A, July 1992 revision) or ICP-MS (method 6020, September 1994 revision) will be required.*

The bioaccessibility of lead or arsenic is calculated in the following manner:

Bioaccessibility value = $$\frac{(Concentration\ in\ vitro\ extract,\ mg/L)(0.1L)}{(Concentration\ in\ solid,\ mg/kg)(0.001\ kg)} \times 100$$

Chain-of-Custody/Good Laboratory Practices *- All laboratories that use this SOP should receive test materials with chain-of-custody documentation. When materials are received, each laboratory will maintain and record custody of samples at all times. All laboratories that perform this procedure should follow good laboratory practices as defined in 40 CFR Part 792 (US EPA, 1999) to the extent practical and possible.*

Data Handling and Verification *- All sample and fluid preparation calculations and operations should be recorded in bound and numbered laboratory notebooks, and on extraction test checklist sheets. Each page must be dated and initialed by the person who performs any operations. Extraction and filtration times must be recorded, along with pH measurements, adjustments, and buffer preparation. Copies of the extraction test checklist sheets should accompany the data package.*

Quality Control Procedures

Elements of Quality Assurance and Quality Control (QA/QC) *- A standard method for the in vitro extraction of soils/solid materials, and the calculation of an associated bioaccessibility value, are specified above. Associated QC procedures to ensure production of high quality data are as follows:*

- *Reagent blank—Extraction fluid analyzed once per batch.*
- *Bottle blank—Extraction fluid only run through the complete extraction procedure at a frequency of no less than 1 per 20 samples or one per extraction batch, whichever is more frequent.*
- *Blank spikes—Extraction fluid spiked at 10 mg/L lead and/or 1 mg/L arsenic and run*

> through the extraction procedure at a frequency of no less than every 20 samples or one
> per extraction batch, whichever is more frequent. Blank spikes should be prepared using
> traceable 1,000 mg/L lead and arsenic standards in 2 percent nitric acid.
> • Duplicate—duplicate extractions are required at a frequency of 1 for every 10 samples.
> At least one duplicate must be performed on each day that extractions are conducted.
> • Standard Reference Material (SRM)—National Institute of Standards and Technology
> (NIST) material 2711 (Montana Soil) should be used as a laboratory control sample
> (LCS).

Control limits for these QC samples are delineated in Table A5-8, and in the following
discussion.

Table A5-8: Summary of QC Samples, Frequency of Analysis, and Control Limits

QC Sample	Minimum Frequency of Analysis	Control Limits
Reagent Blank	Once per batch (min. 5%)	<25 µg/L lead <5 µg/L arsenic
Bottle Blank	Once per batch (min. 5%)	<50 µg/L lead <10 µg/L arsenic
Blank Spike	Once per batch (min. 5%)	85–115% recovery
Duplicate	10%	±20% RPD
SRM (NIST 2711)	2%	9.22 ±1.50 mg/L Pb 0.59 ±0.09 mg/L As

QA/QC Procedures - *Specific laboratory procedures and QC steps are described in the
analytical methods and should be followed when using this SOP.*

Laboratory Control Sample (LCS) - *The NIST SRM 2711 should be used as a laboratory control
sample for the in vitro extraction procedure. Analysis of 18 blind splits of NIST SRM 2711 (105
mg/kg arsenic and 1,162 mg/kg lead) in four independent laboratories resulted in arithmetic
standard deviations of 1.50 mg/L lead and 0.09 mg/L arsenic. This SRM is available from the
National Institute of Standards and Technology, Standard Reference Materials Program, Room
204, Building 202, Gaithersburg, Maryland 20899 (301/975-6776).*

Reagent Blanks/Bottle Blanks/Blank Spikes - *Reagent blanks must not contain more than 5
µg/L arsenic or 25 µg/L lead. Bottle blanks must not contain arsenic and/or lead concentrations
greater than 10 and 50 µg/L, respectively. If either the reagent blank or a bottle blank exceeds
these values, contamination of reagents, water, or equipment should be suspected. In this case,
the laboratory must investigate possible sources of contamination and mitigate the problem
before continuing with sample analysis. Blank spikes should be within 15% of their true value. If
recovery of any blank spike is outside this range, possible errors in preparation, contamination,
or instrument problems should be suspected. In the case of a blank spike outside specified limits,*

the problems must be investigated and corrected before continuing sample analysis.

Analytical Methods

Extraction fluids (from both the stomach-phase and intestinal-phase extractions) were analyzed for metals (antimony, arsenic, beryllium, cadmium, cobalt, copper, lead and nickel). Inductively coupled plasma/mass spectrometry (ICP/MS) (EPA Method 200.8), ICP/atomic emission spectrometry (ICP/AES), and graphite furnace atomic absorption (GFAA) (EPA Method 7060A) were used for the analyses. The laboratory reported arsenic results by GFAA for selected samples because of matrix interference encountered for arsenic during the ICP analyses. Most of the data were reported from the ICP/MS analysis, however, the laboratory used the ICP/AES method for samples in which high levels of target analytes were present. All solid and aqueous/fluid samples were shipped to CAS under chain of custody.

A5-6.2.3.1 Results

The results of the external bioaccessibility testing for metals of concern in the Rodney Street community soils are shown in Tables A5-9 to A5-16. All averages are reported as two significant figures.

Table A5-9: *In vitro* Bioaccessibility Testing of Antimony

Ground Soil

Soil Sb content	acid extract		neutral extract	
(μg/g)	actual (μg)	%	actual (μg)	%
1.3	0.2	17	0.3	21
1.3	0.3	21	0.5	36
0.5	0.2	32	0.2	36
1.1	0.1	11	0.2	15
1.4	0.3	19	0.4	24
1.2	0.3	21	0.3	23
0.9	0.2	25	0.2	26
3.0	0.5	18	0.9	29
2.2	0.2	7	0.5	21
4.1	1.8	44	2.6	64
min		7		15
max		44		64
avg		22		30

Sieved Fine Soil

Soil Sb content	acid extract		neutral extract	
(μg/g)	actual (μg)	%	actual (μg)	%
1.3	0.2	13	0.2	18
0.8	0.2	32	0.3	39
3.4	0.2	5.2	0.2	6.1
1.0	0.1	14	0.4	42
1.1	0.3	28	0.4	40
1.4	0.5	35	0.6	44
0.8	0.6	84	0.2	31
2.3	0.5	23	0.8	33
1.5	0.1	6.6	0.3	20
2.4	0.8	34	1.1	45
min		5.2		6.1
max		84		45
avg		28		32

Table A5-10: *In vitro* Bioaccessibility Testing of Arsenic

Ground Soil

Soil As Content (µg/g)	Acid Extract Actual (µg)	%	Neutral Extract Actual (µg)	%
31.8	8.6	27	8.3	26
50.1	21.5	43	23.1	46
30.5	8.9	29	9.2	30
20.4	4.5	22	7.1	35
49.0	13.2	27	10.8	22
29.2	5.6	19	5.0	17
35.3	9.9	28	6.7	19
30.3	9.4	31	5.2	17
37.2	19.0	51	13.4	36
34.4	11.4	33	10.0	29
min		19		17
max		51		46
avg		31		28

Sieved Fine Soil

Soil As content (µg/g)	acid extract actual (µg)	%	neutral extract actual (µg)	%
46.0	12.4	27	9.2	20
28.9	13.9	48	13.3	46
38.9	10.9	28	3.3	8.5
19.3	5.0	26	10.8	56
46.7	17.3	37	16.8	36
42.5	9.8	23	5.1	12
20.9	7.3	35	1.9	9.2
31.8	11.8	37	6.0	19
29.1	13.7	47	8.4	29
33.4	13.0	39	7.4	22
min		23		8.5
max		48		56
avg		35		26

Table A5-11: *In vitro* Bioaccessibility Testing of Beryllium

Ground Soil

Soil Be content (µg/g)	acid extract actual (µg)	%	neutral extract actual (µg)	%
0.7	0.3	42	0.08	11
0.5	0.3	53	0.08	16
0.6	0.3	57	0.08	14
0.5	0.3	48	0.08	15
0.8	0.4	43	0.08	10
0.9	0.4	39	0.08	8.6
0.7	0.3	45	0.07	9.6
1.1	0.7	61	0.08	7.0
4.3	3.4	79	0.08	1.9
0.7	0.4	49	0.07	10
min		39		1.9
max		79		16
avg		52		10

Sieved Fine Soil

Soil Be content (µg/g)	acid extract actual (µg)	%	neutral extract actual (µg)	%
0.8	0.4	48	0.08	11
0.5	0.3	58	0.08	16
0.7	0.4	59	0.07	11
0.5	0.3	55	0.08	16
0.8	0.5	59	0.08	10
1.0	0.5	51	0.08	7.8
0.9	0.5	58	0.08	8.8
1.2	0.8	70	0.08	6.8
4.2	3.4	81	0.08	1.9
0.9	0.4	52	0.08	9.4
min		48		1.9
max		81		16
avg		59		10

Table A5-12: *In vitro* Bioaccessibility Testing of Cadmium

Ground Soil

Soil Cd content (μg/g)	acid extract actual (μg)	%	neutral extract actual (μg)	%
1.5	1.0	63	0.9	59
1.4	1.2	83	0.7	51
1.6	0.9	58	1.0	62
2.3	1.6	70	1.1	48
2.2	1.6	69	1.1	49
1.2	0.9	73	0.7	56
1.8	1.2	68	1.3	75
4.7	3.4	72	2.8	59
3.4	2.4	69	0.3	9
2.5	1.9	77	1.5	62
min		58		9
max		83		75
avg		70		53

Sieved Fine Soil

Soil Cd content (μg/g)	acid extract actual (μg)	%	neutral extract actual (μg)	%
2.2	1.5	68	1.2	54
1.2	1.0	86	0.7	57
1.5	1.0	65	1.2	79
2.2	1.7	78	1.5	69
2.5	2.0	79	1.5	59
1.9	1.4	73	1.2	62
1.5	1.1	74	1.2	75
5.3	4.3	80	3.2	60
2.4	1.7	71	0.1	3.8
1.9	1.6	83	1.2	63
min		65		3.8
max		86		79
avg		76		58

Table A5-13: *In vitro* Bioaccessibility Testing of Cobalt

Ground Soil

Soil Co content (µg/g)	acid extract actual (µg)	%	neutral extract actual (µg)	%
78.6	14.9	19	8.7	11
46.2	11.6	25	7.9	17
63.6	16.5	26	14.6	23
171	39.3	23	30.8	18
113	17.0	15	10.7	9.5
71.7	10.8	15	8.6	12
100	18.0	18	16.0	16
111	37.7	34	27.8	25
56.6	10.8	19	8.5	15
82.3	20.6	25	14.8	18
min		15		9.5
max		34		25
avg		22		16

Sieved Fine Soil

Soil Co content (µg/g)	acid extract actual (µg)	%	neutral extract actual (µg)	%
109	21.8	20	13.1	12
28.7	10.1	35	6.0	21
72.5	23.2	32	18.9	26
163	53.8	33	12.2	7.5
66.6	21.3	32	12.7	19
90	18.9	21	14.4	16
60.1	19.2	32	14.4	24
102	32.6	32	24.5	24
37.1	11.5	31	8.5	23
65.8	15.8	24	11.2	17
min		20		7.5
max		35		26
avg		29		19

Table A5-14: *In vitro* Bioaccessibility Testing of Copper

Ground Soil

Soil Cu content (µg/g)	Acid Extract actual (µg)	%	Neutral Extract actual (µg)	%
438	114	26	149	34
359	111	31	176	49
436	166	38	209	48
631	177	28	227	36
880	273	31	370	42
453	149	33	181	40
798	239	30	303	38
601	162	27	240	40
820	3	0.4	90	11
520	239	46	255	49
min		0.4		11
max		46		49
avg		29		39

Sieved Fine Soil

Soil Cu content (µg/g)	Acid Extract actual (µg)	%	Neutral Extract actual (µg)	%
637	178	28	236	37
247	104	42	124	50
592	207	35	255	43
628	232	37	377	60
819	360	44	401	49
656	249	38	282	43
737	280	38	332	45
647	265	41	304	47
494	3.4	0.7	43	8.8
460	207	45	216	47
min		0.7		8.8
max		45		60
avg		35		43

Table A5-15: *In vitro* Bioaccessibility Testing of Lead

Ground Soil

Soil Pb content (µg/g)	Acid Extract actual (µg)	%	Neutral Extract actual (µg)	%
187	114	61	15.7	8.4
239	165	69	33.5	14
120	94	78	3.7	3.1
231	176	76	13.9	6.0
383	260	68	23.0	6.0
209	134	64	4.2	2.0
343	240	70	17.2	5.0
1030	680	66	36.9	3.6
838	285	34	14.6	1.7
911	738	81	79.3	8.7
min		34		1.7
max		81		14
avg		67		6

Sieved Fine Soil

Soil Pb content (µg/g)	acid extract actual (µg)	%	neutral extract actual (µg)	%
232	167	72	8.8	3.8
211	167	79	12.0	5.7
190	150	79	7.0	3.7
222	178	80	21.1	9.5
430	314	73	20.6	4.8
389	335	86	14.4	3.7
320	253	79	12.8	4
1210	1016	84	48.4	4
973	487	50	12.7	1.3
511	393	77	19.9	3.9
min		50		1.3
max		86		9.5
avg		76		4

Table A5-16: *In vitro* Bioaccessibility Testing of Nickel

Ground Soil

Soil Ni content	Acid Extract		Neutral Extract	
(µg/g)	actual (µg)	%	actual (µg)	%
5170	517	10	517	10
3130	657	21	657	21
5010	852	17	1002	20
7870	944	12	1023	13
10500	945	9	1050	10
4670	514	11	514	11
9550	726	7.6	879	9.2
4610	922	20	1014	22
5450	763	14	763	14
4750	760	16	713	15
min		7.6		9.2
max		21		22
avg		14		15

Sieved Fine Soil

Soil Ni content	Acid Extract		Neutral Extract	
(µg/g)	actual (µg)	%	actual (µg)	%
7310	804	11	804	11
1840	515	28	442	24
5370	1074	20	1074	20
6410	1154	18	1090	17
5620	1293	23	1124	20
5730	917	16	917	16
6200	682	11	744	12
5290	1058	20	1005	19
3040	608	20	638	21
4270	769	18	726	17
min		11		11
max		28		24
avg		19		18

A5-7 Discussion of Bioaccessibility Studies for Metals of Concern in Rodney Street Community Soils

The mean percent bioaccessibilities and the range from minimum to maximum values for the MOE and external laboratory tests are shown in Table A5-17. In the case of the external laboratory test results, the highest combined bioaccessibility value for either the acid or neutral pH extractions for both the ground and sieved and the sieved only soil samples is shown. In this context, perusal of Tables A5-9 to A5-16 shows that for arsenic, beryllium, cobalt, lead, cadmium and nickel, the extraction of the metal was greatest under acid conditions, and that under subsequent neutral pH extraction conditions, the overall bioaccessibility of these metals did not change appreciably (arsenic, cobalt, copper, nickel) or was much less (beryllium, lead). This lowered bioaccessibility at neutral pH following an acid extraction suggests that these metals become less available in the small intestine. The other metals (antimony and copper) showed further extraction of the metal under neutral pH subsequent to the acid extraction indicating that the combined acid and neutral pH extraction gives a more realistic value for overall bioaccessibility. The MOE and the external laboratory's bioaccessibility results for cobalt, copper, lead and nickel are comparable.

Table A5-17: Comparison of Corrected MOE and Exponent Bioaccessibilities (%)

Metal	MOE Mean (range)	Exponent* Mean (range) (Ground & Sieved)	Exponent* Mean (range) (Sieved Only)	Bioaccessibility Value used for Exposure Estimates
Antimony	2.8 (2.1 - 3.9)	30 (7 - 64)**	32 (5-84)	32
Arsenic	22.8 (12.6 - 28.4)	31 (17 - 51)	35 (23-56)	-
Beryllium	N/A	52 (39 - 79)	59 (2-81)	59
Cadmium	N/A	70 (9 - 83)	76 (4-86)	76
Cobalt	19.2 (12.5 - 24.7)	22 (10 - 34)	29 (8-35)	29
Copper	38.1 (31.8 - 44.4)	39 (<1 - 49)	43 (1-60)	43
Lead	75.7 (61- 90.3)	67 (2 - 81)	76 (10-86)	-
Nickel	16.5 (11.8 - 23.3)	15 (9 - 22)	19 (11-28)	19
* = highest bioaccessibility value from either acid or neutral pH extractions				
** = based on only 21% recovery from NIST soil standard N/A = Not Available				

The external laboratory (Exponent, 2001) notes that while neither the measured concentrations of metals in the soils nor the bioaccessibility differed significantly between the ground and unground samples, the data suggest that there is a slight trend toward higher bioaccessibility in the unground soil samples. On this basis, and given the small number of samples analyzed, the bioaccessibility values used to estimate the intake of metals from ingestion of Rodney Street community soils (Section A3-1.5) were selected from the unground (sieved only) soil sample data set (Table A5-17). Even though the external laboratory had difficulty extracting and analyzing antimony, the highest % bioaccessibility for antimony measured by them was selected. The mean bioaccessibility value was selected to account for the wide range of bioaccessibility values obtained which was assumed to reflect the considerable heterogeneity of the soil samples. In general, the percent metal bioaccessibility decreased with increasing soil nickel concentration. This latter trend is, in part, due to the non-equilibrium conditions involved in simulating processes in the gastrointestinal tract, which are themselves non-equilibrium processes, and, the solubility of the form or species of nickel present in the soils containing the higher soil nickel concentrations.

There is some information on the bioaccessibility of metals in other soils, however, the emphasis is on arsenic and lead (Ellickson et al., 2001; Ruby et al., 1999; Hamel et al., 1998). Hamel et al., (1998) do provide some information on the acid extraction of cadmium and nickel from a Standard Reference Material (SRM) 2710 from the US National Institute of Standards and Technology (NIST) and nickel in a chromium contaminated soil from Jersey City, NJ. NIST SRM 2710 is a Montana soil with a certified nickel content of 14.3 mg/kg. The percentage bioaccessibility of nickel in the SRM 2710 ranged from 11% to 14%, and was 23% to 40% in the Jersey City soil sample. The external laboratory that performed the bioaccessibility testing also performed the acidic or stomach phase part of the bioaccessibility protocol on another NIST SRM 2711 as part of the validation of their approach. NIST SRM 2711 is also a Montana soil, however, its certified nickel content is 20.6 mg/kg. The results of the acid bioaccessibility testing of SRM 2711 are shown in Table A5-18. Without further information on the speciation of nickel in these other soils, direct comparison with the bioaccessibility of nickel in Rodney Street community soils is difficult.

It should be noted that while concentrations of some metals are certified in NIST SRMs, not all metals have certified concentrations. For example, concentrations of antimony, beryllium and cobalt are not certified. Other publications (Roelandts and Gladney, 1998 and Church et al., 1999) report antimony concentrations of 12 mg/kg for NIST 2711, beryllium concentrations of 1.6 to 2 mg/kg, and cobalt concentrations of 9.5 and 12 mg/kg. The external laboratory's analytical procedure resulted in only a 21% recovery of antimony based on the certified value of 19.4 mg/kg. However, as noted another laboratory found 12 mg Sb/kg (Roelandts and Gladney, 1998). This would bring the recovery up to 34.6%, which is still low.

Two technical issues have been raised concerning the *in vitro* extraction methods used. It has been claimed that MOE and Exponent used too low a ratio of extraction fluid to soil sample and that the bioaccessibility value would be larger for greater extraction fluid:soil sample ratios, e.g., if a larger extraction fluid volume had been used, more metal would have been extracted and a

larger bioaccessibility value obtained. The paper by Hamel et al., (1998) contains detailed information on the effect of different soil: extraction fluid ratios on the bioaccessibility of nickel and other metals in contaminated soils. The authors examined soil: extraction fluid ratios from 100:1 up to 5000:1 and concluded that the bioaccessibility of metals in the soils extracted *in vitro* with synthetic extraction fluids will only be affected slightly by changes in extraction fluid to solid ratios for the range 100:1 to 5000:1. This study was undertaken by a university laboratory in New Jersey with no connection to Exponent or MOE and provides independent validation of this aspect of the Exponent and MOE extraction methodology. The data of Hamel et al., (1998) indicates that the extraction method of Exponent is unlikely to underestimate the bioaccessibility of nickel in Rodney Street community soils and that the use of a 100% bioaccessibility value is unnecessary.

Table A5-18: Bioaccessibility Testing of Standard Reference Material NIST 2711

Metal	Soil			Stomach-phase Extraction	
	Certified Value (mg/kg)	Measured Value (µg/g)	% Recovery of Lab. Analysis	Measured Value (µg/100 mL)	% Bioaccessiblity[1]
Antimony*	19.4	4.15	21	1.59	38.3
Arsenic	105	91.1	87	54.2	59.5
Beryllium*	1.6	0.92	57.5	0.383	41.6
Cadmium	41.7	37.7	90	35.2	93.4
Cobalt*	10	7.8	78	3.5	44.9
Copper	114	106	93	49.1	46.3
Lead	1162	1080	93	864	80
Nickel	20.6	15.9	77	4.08	25.7

1 = measured value (stomach extract) as a percentage of measured soil value

* = not certified values

The other issue concerned the role of glycine buffer in nickel bioaccessibility tests. Inco has provided information that the glycine buffer used in the Exponent bioaccessibility test method may form aqueous complexes with nickel which enhance the extraction of nickel at pH 7, and thus overestimate the bioaccessibility by a four-fold factor. The new extraction data performed by Inco and the information in Fischer et al., (1992) do indicate that the presence of glycine in extraction fluids results in greater extraction of nickel at pH 7. This effect also occurs for copper, cadmium and zinc but not for lead (Fischer et al., 1992). The effect does not seem to occur at pH 1.5 (Inco's data) and is not so pronounced at pH 4.5 (Fischer et al., 1992). This loss of glycine's ability to form complexes with nickel and other metals at more acidic pH is related to the dissociation of glycine below its isoelectric point of pH 6. This affects glycine's ability to form

complexes with nickel at low pH.

Inspection of Tables A5-7, A5-16 and Inco's data clearly indicate that the percentage of nickel extracted from Rodney Street community soils at low pH either exceeds or is equivalent to the amount of nickel extracted at neutral pH (pH 7). As indicated in section A5-6, MOE selected the highest average bioaccessibility value for either the acid or neutral pH extractions of the sieved not ground soil samples analyzed by Exponent. Extraction of nickel was greatest under acid conditions. The 19% bioaccessibility value was obtained under acid extraction and therefore the presence of glycine in the extraction fluid would have no influence on the percentage of nickel extracted. The MOE stomach acid leachate data which was obtained without glycine (Table A5-7) and the Inco data for acid extraction of nickel *without glycine* from Rodney Street community soils support MOE's selection of the 19% bioaccessibility value obtained under acid pH conditions.

A5-8 References

Ambrose, A.M., P.S. Larson, J.F. Borzelleca, G.R. Hennigar Jr., 1976. Long term toxicologic assessment of nickel in rats and dogs. J. Food Sci. Technol. 13:181-187.

Church, S.E., B.A. Kimball, D.L. Fey, D.A. Ferderer, T.J. Yager, and R.B. Vaughn. 1999. Source, Transport, and Partitioning of Metals between Water, Colloids, and Bed Sediments of the Animas River, Colorado. U.S. Geological Survey Open-File Report 97-0151.

Christensen, O.B. and V. Lagesson, 1981. Nickel concentration of blood and urine after oral administration. Ann. Clin. Lab. Sci. 11:119-125.

Cronin, E., A. Di Michiel, S.S. Brown, S.S. 1980. Oral challenge in nickel-sensitive women with hand eczema. In: Brown, S.S., Sunderman, F.W.Jr., Eds. Nickel Toxicology, London: Academic Press, pp. 149-152.

Diamond, G.L., P.E. Goodrum, S.P. Felter, W.L. Ruoff, 1998. Gastrointestinal absorption of metals. Drug Chem. Toxicol. 21:223-251.

Ellickson, K.M., R.J. Meeker, M.A. Gallo, B.T. Buckley, and P.J. Lioy, 2001. Oral bioavailability of lead and arsenic from a NIST standard reference soil material. Arch. Environ. Contam. Toxicol. 40:128-135.

Exponent. 2001. Bioaccessibility of metals from soils. Prepared for MOE by Exponent, Boulder, CO, USA. June 2001 (and Cadmium Addendum, July, 2001).

Fischer, K., C. Rainer, D. Bieniek and A. Kettrup. 1992. Desorption of heavy metals from typical soil components (clay, peat) with glycine. Intern. J. Environ. Anal. Chem. 46:53-62.

Ganong, W.F. 1989. Review of Medical Physiology. 14th Ed. Published by: Appleton and Lange.

Gawkrodger, D.J. S.W. Cook, G.S. Fell, J.A. Hunter, 1986. Nickel dermatitis: The reaction to oral nickel challenge. Br. J. Dermatol.115:33.

Griffin, S.R., R. Rubenstein, S. Irene, C. DeRosa, and H. Choudhury, 1990. Bioavailability in rats of metals adsorbed to soils. U.S. Environmental Protection Agency, Washington, D.C., Hazelton Laboratories, America, Inc. Poster presented at the Society of Toxicology 29th Annual Meeting, Miami Beach, FL, February 12-16. Poster paper No. 623.

Hamel, S.C., B. Buckley, and P.J. Lioy, 1998. Bioaccessibility of metals in soils for different liquid to solid ratios in synthetic gastric fluid. Environ. Sci. Technol. 32:358-362.

Ho, W. and A. Furst, 1973. Nickel excretion by rats following a single treatment. Proc. West Pharmacol. Soc. 16:245-248.

Horak, E. and F.W. Sunderman Jr., 1973. Fecal nickel excretion by healthy adults. Clin. Chem. 19:429-430.

International Commission on Radiological Protection (ICRP). 1984. Report of the Task Group on Reference Man. International Commission on Radiological Protection No. 23. Pergamon Press.

International Programme for Chemical Safety, (IPCS). 2000. Short list definitions. Harmonization Project (Terminology). Exposure Assessment Planning Workgroup. [http://www.ipcsharmonize.org/]. Accessed October 17, 2001.

Ishimatsu, S., T. Kawamoto, K. Matsuno, and Y. Kodama, 1995. Distribution of various nickel compounds in rat organs after oral administration. Biological Trace Element Research 49:43-52.

Maddaloni, M., N. Lolacono, W. Manton, C. Blum, J. Drexler, and J. Graziano, 1998. Bioavailability of soil borne lead in adults, by stable isotope dilution. Environ. Health Perspect.106 (Suppl. 6):1589-1594.

Mastromatteo., E. 1986. Yant memorial lecture: Nickel. Am. Ind. Hyg. Assoc. J 47:589-601.

McNeely, M.D., M. W. Nechay, F.W. Sunderman Jr., 1972. Measurements of nickel in serum and urine as indices of environmental exposure to nickel. Clin. Chem. 18:992-995.

Menne, T., H.L. Mikkelsen, and P. Solgaard, 1978. Nickel excretion in urine after oral administration. Cont. Dermatol. 4:106-108.

NEPI. 2000. Assessing the Bioavailability of Metals in Soil for Use in Human Health Risk Assessments. Bioavailability Policy Report Phase II: Metals Task Force Report. National Environmental Policy Institute (NEPI). Summer 2000.

Nielsen, G.D., O. Andersen, and M. Jensen, 1993. Toxicokinetics of nickel in mice studied with the γ-emitting isotope ^{57}Ni. Fundam. Appl.Toxicol. 21:236-243.

Nielsen, G.D., U. Soderberg, P.J. Jorgensen, D.M. Templeton, S.N. Rasmussen, K.E. Andersen, P. Grandjean, 1999. Absorption and retention of nickel from drinking water in relation to food intake and nickel sensitivity. Toxicol. Appl. Pharmacol. 154:67-75.

Patriarca, M., T.D.B. Lyon, and G.S. Fell, 1997. Nickel metabolism in humans investigated with an oral stable isotope. Am. J. Clin. Nutr. 66:616-621.

Phatak, SS. and VN. Patwardhan. 1952. Toxicity of nickel accumulation of nickel in rats fed on nickel-containing diets and its elimination. J. Scientific and Industrial Research. 11B:173-176.

Rodriguez, R.R., N.T. Basta, S.W. Casteel, and L.W. Pace. 1999. An *in vitro* gastrointestinal method to estimate bioavailable arsenic in contaminated soils and solid media. Environ. Sci. Technol. 33:642-649.

Roelandts, I and E. S. Gladney. 1998. conference contribution: Consensus values for NIST biological and environmental Standard Reference Materials. Fresenius' Journal of Analytical. 360:327-338.

Ruby, M.V., R. Schoof, W. Brattin, M. Goldale, G. Post, M. Harnios, D. E. Mosby, S.W. Casteel, W. Berti, M. Carpenter, D. Edwards, D. Cragin, and W. Chappell. 1999. Advances in evaluating the oral bioavailability of inorganics in soil for use in human health risk assessment. Environ. Sci. Technol. 33:3697-3705.

Severa, J., A. Vyskocil, Z. Fiala, and M. Cizkova, 1995. Distribution of nickel in body fluids and organs of rats chronically exposed to nickel sulphate. Human and Experiential Toxicology 14:955-958.

Spruit, D. and P.J.M. Bongaarts, 1977. Nickel content of plasma, urine, and hair in contact dermatitis. Dermatologica. 154:291-300.

Sunderman, F.W., Jr., S.M. Hopfer, K.R. Sweeney, A.H. Marcus, B.M. Most, and J. Creason. 1989. Nickel absorption and kinetics in human volunteers. Proc. Soc. Exp. Biol. Med. 191:5-11.

Tedeschi, R.E. and F.W. Sunderman, 1957. Nickel poisoning V. The metabolism of nickel under normal conditions and after exposure to nickel carbonyl. Arch. Ind. Health. 16:486-488.

Templeton DM, F.W. Sunderman Jr., F.M. Herber, 1994a. Tentative reference values for nickel concentrations in human serum, plasma, blood, and urine: Evaluation according to the TRACY protocol. Sci Total Environ. 148:243-251.

Templeton, D.M., S.X. Xu, L. Stuhne-Sekalec, 1994b. Isotope-specific analysis of Ni by ICP-MS: Applications of stable isotope tracers to biokinetic studies. Sci. Total Environ.148:253-262.

US EPA. 1989. U.S. Environmental Protection Agency. Risk Assessment Guidance for Superfund. Volume 1: Human health evaluation manual (Part A). EPA/540/1-89/002.

US EPA. 1999. U.S. Environmental Protection Agency. 40CFR Part 792. National Archives and Records Administration. Code of Federal Regulations, Title 40: Protection of the Environment, Part 792: Good Laboratory Practice Standards. Revised as of July, 1999. [http://www.access.gpo.gov/nara/cfr/waisidx_99/40cfr792_99.html] (Accessed October 18, 2001).

US EPA. 2000. U.S. Environmental Protection Agency, Office of Solid Waste. Test Methods for Evaluating Solid Wastes. Physical/Chemical Methods. SW-846. Available on-line at: [http://www.epa.gov/epaoswer/hazwaste/test/main.htm] Last updated August 1, 2000. Accessed March 5, 2002.

US EPA. 2001. U.S. Environmental Protection Agency. Risk Assessment Guidance for Superfund. Volume 1: Human health evaluation manual (Part E, Supplemental guidance for dermal risk assessment). EPA/540/R/005.

US Naval Facilities Engineering Command. 2000. Guide for incorporating bioavailability adjustments into human health and ecological risk assessments at U.S. Navy and Marine Corps Facilities - Part 1: Overview of metals bioavailability (NFESC User's Guide UG-2041-ENV). US Naval Facilities Engineering Command, Washington, D.C. 20374-5065.

Human Health Risk Assessment

Appendix 6

Intake Factors and Receptor Assumptions for the Assessment of the Rodney Street Community

Table of Contents
Human Health Risk Assessment – Appendix 6
Intake Factors and Receptor Assumptions for the Assessment of the Rodney Street Community

A6-1 Intake Factors and Receptor Assumptions

Several sources were considered in selecting the physiological and behaviourial characteristics used to assess the Rodney Street community. These included:

- Compendium of Canadian Human Exposure Factors for Risk Assessment (O'Connor, 1997);
- EPA Exposure Factors Hand Book (US EPA, 1997);
- EPA Dermal Exposure Assessment and Supplemental Guidance (US EPA, 1992; 2000a);
- EPA Child-Specific Exposure Factors Handbook (US EPA, 2000b);
- Environmental Health Directorate Working Group on Reference Values (EHD, 1993);
- Canadian Environmental Protection Act (1994);
- Canadian Council of the Ministers of the Environment (CCME, 1996; 2000); and
- Health Canada (1995).

The following sections provide detailed information, in the form of tables and text where appropriate, for each of the receptor parameters utilized in the assessment. Selected values are highlighted and discussed. For the most part, the Compendium of Canadian Human Exposure Factors for Risk Assessment (O'Connor, 1997) was used as a primary source of receptor data. This source was selected since it characterizes Canadian populations and as a result would best represent the Rodney Street community. It relies on published and reliable reference sources, such as Health Canada, Statistics Canada and the Canadian Fitness and Lifestyles Research Institute and has been used in the past on several assessments conducted by the ministry and the CCME. In cases where this data set was unable to adequately describe certain time activity patterns and/or behaviourial/physiological characteristics, other data sources, such as the US EPA Exposure Factors Handbook (US EPA, 1997) were used.

A6-2 Time Spent in Age Group

As defined by Health Canada (1995) and CEPA (1994), the following age classifications were used for the HHRA:

- Infant (0 to 6 months);
- Toddler (7 months to <5 years);
- Child (5 years to <12 years);
- Teen (12 to <20 years); and
- Adult (20 years and over).

A6-3 Body Weight

Available body weight data are summarized in Table A6-7a. Mean values provided by O'Connor (1997) and CCME (2000) were selected for the HHRA, as this was the most recent data

characterizing the Canadian population, and is similar to other data sources available for the Canadian and U.S. populations.

A6-4 Inhalation Rate

Inhalation of airborne chemicals is a potential source of chemical exposure for the residents of the Rodney Street community. Available inhalation rate data are summarized in Table A6-7b. Mean values provided by O'Connor (1997) were selected for the HHRA, as this was the most recent data characterizing the Canadian population. It should be noted that this data need not be used in the direct assessment of inhalation toxicity since the dose response factors used in the HHRA to characterize inhalation toxicity are expressed on a concentration basis ($\mu g/m^3$), rather than a dose basis ($\mu g/kg/day$), allowing direct comparison of these factors with measured and/or predicted air concentrations. However, inhalation rate data is necessary in the calculation of incremental exposures through this pathway.

A6-5 Water Intake Rate

Drinking water is municipally supplied in the Rodney Street community with water taken from Lake Erie that is not likely to be directly impacted by contamination in the Port Colborne area. None the less, drinking water is a potential source of exposure to the chemicals of concern in the Rodney Street community. Available water intake data are summarized in Table A6-7c. Mean values provided by O'Connor (1997) were selected for the HHRA, as this was the most recent data characterizing the Canadian population, and is similar to other data sources available for the Canadian and U.S. populations.

A6-6 Soil Ingestion Rate

The ingestion of soil is a potential source of exposure to chemicals in the Rodney Street community. The potential for exposure to chemicals through inadvertent soil ingestion is greater for children as a result of behavioral patterns present during childhood. Inadvertent soil ingestion among children may occur through the mouthing of objects or hands. This mouthing behavior is considered to be a normal phase of childhood development. Adults may also ingest soil or dust particles that adhere to food, cigarettes, or their hands. Deliberate soil ingestion is defined as pica and is discussed below. Available soil ingestion data are summarized in Table A6-7d. The average soil ingestion rates recommended by the EPA (US EPA, 2000b; 1997) for assessing risks associated with chemicals in soil were selected for the assessment of toddlers and children. Recent publications (Stanek et al., 2001) and past assessment conducted by the MOE are in agreement with the assumptions made in this assessment. Ingestion rates recommended by CCME (1996) and used by MOE in the past were selected for other receptors.

A6-6.1 Pica

Deliberate soil ingestion is defined as pica and is relatively uncommon in the general population. The scientific literature define pica as "the repeated eating of non-nutritive substances" (Feldman, 1986), however, for this study, the concern is related to deliberately high soil ingestion. Information on the incidence of soil pica is limited, but it appears that soil pica is not common. Five key tracer studies (Binder et al., 1986; Clausing et al., 1987; Van Wijnen et al., 1990; Davis et al., 1990; and Calabrese et al., 1989) reviewed by the US EPA (1997) revealed only one child out of the more than 600 children involved in these studies ingested an amount of soil significantly greater than the range for other children. These studies did not include data for all populations and were representative of short term ingestion only. However, it can be assumed that the incidence rate of deliberate soil ingestion behavior in the general population is low. US EPA (1997, 2000b) recommend a soil ingestion rate of 10 g/day for children who deliberately ingest soil. However, this value is only intended for use in acute exposure assessments and these exposures cannot be compared with chronic health criteria.

A6-6.2 Soil/Dust Ratio Methodology

In the outdoor environment, soil ingestion, hence the intake of various contaminants, represents a significant exposure pathway for children. Studies have shown that soil enters the house as dust by atmospheric transport, as well as being brought into the house by animals and humans on their bodies, clothes or shoes (ATSDR, 1988). While there is adequate evidence of a relationship between the levels of contaminants found outside the homes with those inside the homes, the extent of the relationship is not clear.

Study undertaken by Rutz et al., (1997) suggested that between 20 - 30% of indoor contamination was from outdoor soil sources. The authors found that the tracking of soil was the primary mechanism for uranium to enter the residence with the re-entrained dust being a factor as well.

The Integrated Exposure Uptake BioKinetic (IEUBK) model used by the EPA for modeling the metal lead has suggested that the mass fraction of soil in indoor dust is related to the level of lead in the outdoor soil concentration by a ratio of about 0.7. This means that the indoor lead dust levels on a weight-by-weight basis were designated to be about 0.7 of the outdoor soil level. According to the EPA, this value is only a recommended value for input into the IEUBK model in the absence of site-specific data on lead as current information regarding practical techniques to estimate the indoor/outdoor mass fraction ratio are limited. Additionally, it has been noted that lead is not an appropriate model for nickel due to the significant indoor sources of lead in indoor dust.

In the course of this risk assessment, the MOE was provided with a study conducted by PTI Environmental Services (PTI, 1994) at a zinc contaminated site in Bartlesville, Oklahoma. One

component of this study was the evaluation of the relationship between the soil concentration and the indoor dust concentration of the metal. This study measured the indoor and outdoor levels of arsenic, cadmium, lead, and zinc and found the ratio of indoor dust level to the soil concentration levels to be 0.20, 0.35, 0.50 and 0.36, respectively.

Recently, a study conducted by Rasmussen et al., (2001) in the city of Ottawa, found that dust generated from sources in the house contribute significantly to exposures to elements such as lead, cadmium, antimony and mercury. While the study did consider the indoor and outdoor levels of the aforementioned metals, the study concluded that it would be "difficult-to-impossible" to predict indoor dust concentration based on the low metal levels found in the local soils. Additionally, this study only looked at particles in the 100 - 250 um size range which is of questionable relevance.

Hwang et al., (1997) and Calabrese (unpublished, as reported in Walker and Griffin, 1997) have undertaken comprehensive studies which in part examine the relationship of interior surface dust levels of arsenic to outdoor soil concentration of arsenic. Both Hwang et al., (1997) and Calabrese (unpublished) sampled interior dust and outdoor soil concentrations using different methodologies from the same subset of 25 households in Anaconda, Montana. The reported average outdoor soil and interior dust concentrations were significantly different, 192 mg/kg and 75.14 mg/kg, respectively from the Hwang et al., (1997) study, versus 75 mg/kg and 29 mg/kg from the Calabrese study, respectively. However, the relationship between the average interior surface dust concentration and outdoor soil concentrations were very similar. The ratio of the average interior dust to outdoor soil concentrations were calculated to be 0.391 and 0.387 for the Hwang and Calabrese studies, respectively. These ratios have been validated in a recent study conducted in Deloro, Ontario where indoor dust samples were compared with outdoor soil samples (MOE, 1999).

Calabrese and Stanek (1992) have estimated that 30% of household dust is derived from outdoor soil, with the remaining 70% originating from other sources. Other studies have found similar relationships between outdoor soil and indoor dust concentrations for several metals (Murgueytio et al., 1998).

It was concluded that using the most comprehensive environmental media sampled within the Rodney Street community (outdoor soil) and the relationship described by the Hwang and Calabrese studies above would not only result in the appropriate expression of interior dust levels for use in an exposure assessment, but would also inherently correlate soil and interior dust on a spatial basis. The ratio derived from the Hwang and Calabrese studies (0.39) was selected as it considered metals similar to nickel with respect to the expected relationship of indoor dust and outdoor soil concentrations, it has been validated in a recent Ontario study (MOE, 1999) and it is substantiated by the other studies cited above (Murgueytio et al., 1998; PTI, 1994; Calabrese and Stanek, 1992; Rutz et al., 1997).

Based on Walker and Griffin (1997), it has been assumed that 55% of a receptors soil ingestion rate is allocated to indoor dust exposure, while the remaining 45% is allocated to outdoor soil exposures. The ratio of soil intake to dust intake is not proportional to the ratio of the number of waking hours that a child spends outdoors versus indoors. Children spend only 15 to 30% of their waking hours playing outside but are more likely to be in contact with bare soil areas during this time. The default outdoor:indoor ingestion ratio of 45:55 has been adopted by the US EPA (1994) as an IEUBK model default.

A6-7 Dermal Routes of Exposure

Dermal exposure can occur during a variety of activities in different environmental media, such as:

- Water (e.g., bathing, washing, swimming);
- Soil (e.g., outdoor recreation, gardening, construction);
- Sediment (e.g., wading, fishing);
- Liquids (e.g., use of commercial products);
- Vapors/fumes (e.g., use of commercial products); and
- Indoors (e.g., carpets, floors, counter tops).

For the Rodney Street assessment, only dermal contact with soils and dusts (indoors) are of concern.

A6-7.1 Soil Adhesion to Skin

Soil adherence to the surface of the skin is a required parameter to calculate dermal dose when the exposures involving dermal contact with chemicals in soil or dust are of concern. Available soil adhesion to skin data are summarized in Table A6-7e. Adhesion factors reported in the literature vary greatly depending on activity and soil conditions. US EPA (2000b) recommends a default adhesion factor of 0.2 mg/cm^2 for children and 0.07 mg/cm^2 for adults. These values were selected for the Rodney Street assessment.

A6-7.2 Area of Exposed Skin Derivation

In order to calculate the area of exposed skin for various receptors, several methods are available. US EPA (1997) recommends an approach where in a moderate climate, 25, 10, 10 and 5% of the whole body surface area is exposed for summer, spring, fall and winter, respectively. Others have considered the surface area of specific body parts considered exposed for each season. Both approaches yield similar results. The latter approach was selected for the Rodney Street assessment, with mean surface areas reported by O'Connor (1997) (Table A6-1). Available surface area data are summarized in Table A6-7f. For the Rodney Street community, the following assumptions were made:

- • Hands, arms, legs and feet were considered exposed in July and August;
- • Hands, arms and legs were considered exposed in June and September; and
- • Hands and arms were considered exposed in all other months.

Table A6-1: Mean Percentage of Total Body Surface Area Exposed

| Body Part | Skin Surface Areas in m^2 | | | | |
| | Receptor | | | | |
	Infant	Toddler	Child	Teen	Adult
Hands	0.032	0.043	0.059	0.08	0.089
Arms	0.055	0.089	0.148	0.223	0.25
Legs	0.091	0.169	0.307	0.497	0.572
Feet	0.025	0.043	0.072	0.108	0.119
Totals	0.203	0.344	0.586	0.908	1.03

Source: O'Connor, 1997.

A6-8 Backyard Fruit and Vegetable Consumption

The assessment of potential health risks for people living in the homes on Rodney Street, Port Colborne, considers exposures to the metals of concern from all relevant pathways. Eating vegetables grown in backyards where metal levels are above typical levels, represents a potential exposure pathway if the metals present in the soil are taken up into the vegetables. The exposures received by people eating such produce depends upon the concentration of the metals in the vegetables and the amount of vegetables consumed from backyard gardens on an annual basis. Specific data on backyard garden vegetable consumption patterns for the homes on Rodney Street are not available. Therefore it was necessary to estimate likely consumption rates based on studies conducted in other communities in Ontario (MOEE, 1995).

The amounts and types of produce that people might consume from a backyard garden are affected by the size of the garden, the preferences of individuals for the types of crops grown and the yields achieved. In previous risk assessments in other communities, the MOE developed an estimate of backyard garden crop yield of 1.4 kg/m^2 for mixed produce (MOEE, 1995). An assumed garden size of 30 m^2 was used to provide an estimated total annual yield of 42 kg of produce. These assumptions have been used to estimate backyard garden produce consumption for people living on Rodney Street.

In order to estimate the proportion of home grown produce which is fruit and that which is vegetables, it was first necessary to determine the proportion of fruit and fruit juice consumption which could be derived from a home garden in Ontario. The apparent per capita food consumption rates (Statistics Canada, 1991) indicated that the proportion of fruits consumed by typical Canadians (1988 to 1989) which could be grown in this region of Canada (e.g., apples, blueberries, cherries, plums, raspberries and strawberries) was 37 percent of the total fruit consumption. Thus, a typical adult and child would consume fruit which could be grown in Canada at a rate of 90.65 and 99.16 g/d, respectively (see Table A6-2). All of the vegetables consumed by Rodney Street residents were assumed to be grown in Canada, and therefore, potentially in a home garden. Therefore, the total consumption of fruits and vegetables

potentially grown in this region of Canada, and potentially in a home garden, would be 416 and 358 g/d, for adults and children, respectively (i.e., 90.65 g/d + 325 g/d; 99.16 + 259 g/d, respectively) (Table A6-2). Thus, the total consumption of potentially home grown fruits and vegetables for a typical family (2 adults, 2 children) would be 565 kg/year (i.e., 416 g/d x 365 d/yr x 2 + 358 g/d x 365 d/yr x 2 = 565,000 g/year) (Table A6-3). In addition, Table A6-7g lists fruit and vegetable consumption rates from various sources.

Assuming the proportions of fruits versus vegetables in a typical home garden would equal the proportions of potentially home grown fruits and vegetables consumed, as calculated above, this would mean that 26% and 74% of home grown produce would be fruits and vegetables, respectively. Apportioning the backyard garden yield of 42 kg/year between fruits and vegetables would therefore indicate that 11 kg/year (or 26% of 42 kg/year) would be fruits, while 31 kg/year would be vegetables (Table A6-3).

Using the estimated yields of a home garden of 11 and 31 kg/year for fruits and vegetables, respectively, the overall contribution of the home garden to fruit and vegetable consumption can be calculated. For a family of four, given a total fruit and fruit juices consumption of 375.5 kg/year and a total vegetable intake of 426.3 kg/year, the proportion potentially obtained from a home garden would be 2.91% and 7.29% for fruits and vegetables, respectively (Table A6-4). This incorporates the assumption that the total yield from the garden is consumed, i.e., that there is no loss due to wildlife browsing or spoilage. It should be noted that this calculation, due to its basis on the yield of a home garden, would not take into account the harvesting and consumption of wild fruits such as berries. However, berry consumption is highly seasonal and would comprise a small fraction of the overall fruit consumption, and thus a very small proportion of the total diet. Therefore, it was concluded that consumption of wild berries would contribute negligibly to total intake via home garden produce.

Table A6-2: Daily Produce Consumption Rates for the Canadian Population[1]

	Produce Consumption Rates (g/day)				
	Infant (0 - 6 mo)	Toddler (7 mo - <5 yr)	Child (5 - <12 yr)	Teen (12 - <20 yr)	Adult (20+ yr)
Root Vegetables	83	105	161	227	188
Other Vegetables	72	67	98	120	137
Fruits and Fruit Juices	136	234	268	258	245
% Fruit Locally grown	37%	37%	37%	37%	37%
Local Fruit	50.32	86.58	99.16	95.46	90.65
Total Daily Consumption of Locally Grown Produce	205	259	358	442	416
Root as a % of Total Daily Local Consumption	40%	41%	45%	51%	45%
Other as a % of Total Daily Local Consumption	35%	26%	27%	27%	33%
Local Fruit as a % of Total Daily Local Consumption	25%	33%	28%	22%	22%

1. From O'Connor, 1997.

Table A6-3: Estimation of Backyard Produce Contribution to Total Produce Consumption

Receptor	Daily Consumption (g/day)	Number	Total Daily Consumption (g/day)	Days/Year	Total Annual Consumption	
					g/year	kg/year
Adult	416	2	832	365	303,680	304
Child	358	2	716	365	261,340	261
Annual Family Consumption of Local Produce					565,020	565
Annual Produce Yield from Backyard Garden[1]					42,000	42
% of backyard garden - vegetables					74%	74%
Annual Vegetable Yield from Backyard Garden					31,080	31
Total Family Vegetable Consumption (all sources)					426,300	426
% of backyard garden - fruits					26%	26%
Annual Fruit Yield from Backyard Garden					10,920	11
Total Family Fruit Consumption (all sources)					375,500	376
% of Annual Vegetable Consumption that comes from Backyard Gardens					7.29%	7.29%
% of Annual Fruit Consumption that comes from Backyard					2.91%	2.91%

1. From MOEE, 1995.

Table A6-4: Estimated Daily Consumption of Backyard Garden Produce for all Age Groups

	Produce Consumption Rates (g/day)				
	Infant (0 - 6 mo)	Toddler (7 mo - <5 yr)	Child (5 - <12 yr)	Teen (12 - <20 yr)	Adult (20+ yr)
Total Daily Consumption of Root Vegetables	83	105	161	227	188
% Consumed as Backyard Garden Vegetables	7.29%	7.29%	7.29%	7.29%	7.29%
Daily Consumption of Backyard Root Vegetables	6.05	7.7	11.7	16.5	13.7
Total Daily Consumption of Other Vegetables	72	67	98	120	137
% Consumed as Backyard Garden Vegetables	7.29%	7.29%	7.29%	7.29%	7.29%
Daily Consumption of Other Backyard Vegetables	5.25	4.88	7.14	8.7	10.0
Total Daily Consumption of Fruits	136	234	268	258	245
% Consumed as Backyard Garden Fruits	2.91%	2.91%	2.91%	2.91%	2.91%
Daily Consumption of Backyard Fruits	3.96	6.81	7.80	7.5	14.1

A6-9 Time Activity Patterns

The US EPA Exposure Factors Handbook ,Volume III (US EPA, 1997) contains a wide variety of summarized activity pattern statistics for a broad range of categories (i.e., gender specific, seasonal, regional, age class, etc.). Data describing the number of minutes spent outdoors (outside the residence) per 24 hour period were used to help define activity values for the residents of the Rodney Street community.

During the two months of summer where toddlers, children and adolescents are not in school, it has been estimated that a receptor may spend up to eight hours/day, seven days/week outside. For the remaining six months of the summer season (for the purpose of characterizing receptor activities, it was assumed that "summer" in Port Colborne included mid March to mid November), receptors were assumed (based on literature estimates) to spend approximately three hours/day, seven days/week outside. The following illustrates the calculations used to estimate the average amount of time spent outside during the summer season.

$$\text{Time}_{avg} = [(2/8 \text{ months}) * (8\text{hrs}/\text{day} * 7\text{days}/\text{wk})] + [(6/8) * (3.0\text{hrs}/\text{day} * 7\text{days}/\text{wk})]$$

The daily average value has therefore been estimated to be approximately 4.3 hours/day outside in the summer. For winter months, original mean estimates based on literature data were used for these receptors. For infants and adults, literature data was used for both summer and winter. The

following table contains the typical mean and plausible maximum daily average values for each receptor. Other available data are summarized in Table A6-7h.

Table A6-5: Average Time Spent Outdoors During Summer and Winter

Receptor Description	Time Spent Outdoors During the Summer[*] (hours/day)	Time Spent Outdoors During the Winter (hours/day)
Infant	3	2
Toddler	4.3	2
Child	4.3	2
Teen	4.3	2
Adult	3	2

[*] For the purpose of characterizing receptor activities, it was assumed that "summer" in Port Colborne included mid March to mid November.

Although the amount of time spent outside versus inside should be estimated as accurately as possible, it is not expected to have a great influence on overall exposure estimates. Since exposure from soil ingestion is not proportional to the amount of time a receptor spends outside (Walker and Griffin, 1997), it is expected this variable will have a negligible impact on exposure estimates relative to other parameters.

A6-9.1 Amount of Time Spent Within the Rodney Street Community

Typical values have been assigned to the amount of time a receptor may spend in the Rodney Street community, either at home or at parks in the immediate area. The following is a breakdown of the average time assumed to be spent away from the Rodney Street community per day for all receptors (i.e., at school and/or work where appropriate). These activity patterns were assumed not to vary significantly throughout the year, except for the two summer months per year which children and adolescents are not in school (July and August). During this time it was assumed these receptors would spend more time within the Rodney Street community (Table A6-6).

Table A6-6: Average Amount of Time Spent Within
the Rodney Street Community Per Day

Receptor	Typical	July and August
Infant	23 hours/day (1 hour/day)	23 hours/day (1 hour/day)
Toddler	23 hours/day (1 hour/day)	23 hours/day (1 hour/day)
Child	16 hours/day (8 hours/day)	23 hours/day (1 hour/day)
Teen	14 hours/day (10 hours/day)	23 hours/day (1 hour/day)
Adult[*]	23 hours/day (1 hour/day)	23 hours/day (1 hour/day)

( ) represents the amount of time spent away from the Rodney Street community but still within Port Colborne.
[*]Adults were assumed to be stay at home parents.

A6-10 References

ATSDR. 1988. The nature and extent of lead poisoning in children in the United States: A report to Congress. Agency for Toxic Substance and Disease Registry, Atlanta, GA. U.S. Department of Health and Human Services.

Binder, S., D. Sokal, D. Maughan, 1986. Estimating soil ingestion: The use of tracer elements in estimating the amount of soil ingested by young children. Arch. Environ. Health. 41(6):341-345.

Calabrese, E.J., H. Pastides, R. Barnes, C. Edwards, P.T. Kostecki, P.T., et al., 1989. How much soil do young children ingest: An epidemiologic study. In: Petroleum Contaminated Soils, Lewis Publishers, Chelsea, MI. 20 pp. 363-397.

Calabrese, E.J. Unpublished. Unpublished data available in the administrative record for the Anaconda superfund site. Cited In: Walker and Griffin, 1997.

Calabrese, E and E. Stanek, 1992. What proportion of household dust is derived from outdoor soil? J Soil Contam. 1:253-263.

CEPA. 1994. Human Health Risk Assessment for Priority Substances. Canadian Environmental Protection Act. Health Canada. ISBN 0-662-22126-5.

CCME. 1996. A Protocol for the Derivation of Environmental and Human Health Soil Quality Guidelines. Canadian Council of Ministers of Environment. Winnipeg, MB, CCME-EPC-101E. En 108-4/8-1996E. ISBN 0-662-24344-7.

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

Clausing, P., B. Brunekreef, J.H. Van Wijnen, 1987. A method for estimating soil ingestion by children. Int. Arch. Occup. Environ. Health (W. Germany). 59(1):73-82. 30.

Davis, S., P. Waller, R. Buschbon, J. Ballou, P. White, 1990. Quantitative estimates of soil ingestion in normal children between the ages of 2 and 7 years: Population based estimates using aluminum, silicon, and titanium as soil tracer elements. Arch. Environ. Health. 45:112-122.

EHD. 1993. Reference Values for Canadian Populations. Updated May 1997. Environmental Health Directorate Working Group on Reference Values, Health Canada.

Feldman, M.D. 1986. Pica: Current Perspectives. Psychosomatics USA. 27(7):519-523.

Gradient Corporation. 1995. Exposure Assessment Placer Dome Balmertown, Ontario, Canada.

Health Canada. 1995. Investigating Human Exposure to Contaminants in the Environment: A Handbook for Exposure Calculations. ISBN-0-662-23543-6.

Hwang, Y.H., R.L. Bornschein, J. Grote, W. Menrath, and S. Roda, 1997. Environmental arsenic exposure of children around a former copper smelter site. Environ Res. 72(1):72-81.

MOEE. 1994. Scientific Criteria Document for Multimedia Environmental Standards Development - Lead. Ministry of the Environment and Energy, March 1994.

MOEE. 1995. Health Risk Assessment of Mercury Contamination in the Vicinity of ICI Forest Products Cornwall, Ontario. Ontario Ministry of Environment and Energy. May 1995. PIBS 3352.

MOE. 1999. Deloro Village Exposure Assessment and Health Risk Characterization for Arsenic and Other Metals. Ontario Ministry of the Environment.

Murgueytio, A.M., R.G. Evans, D.A. Sterling, S.A. Clardy, B.N. Shadel and B.W. Clements. 1998. Relationship between lead mining and blood levels in children. Arch. Environ. Health.53 (6): 414-423.

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

PTI. 1994. Remedial Investigation Report. National Zinc Site. PTI Environmental Services. September 1994.

Rasmussen, P.E., K.S. Subramanian, B.J. Jessiman, 2001. A multi-element profile of house dust in relation to exterior dust and soils in the city of Ottawa, Canada. The Science of the Total Environment. 267:125-140.

Rutz, E., J. Valentine, R. Eckart, and A.Yu, 1997. Pilot study to determine levels of contamination in indoor dust resulting from contamination of soils. J. Soil Contamination. 6(5): 525-536.

Stanek, E.J., E.J. Calabrese, and M. Zorn, 2001. Soil Ingestion Distributions for Monte Carlo Risk Assessment in Children. HERA. 7(2):357-368.

Statistics Canada. 1991. Apparent Per Capita Food Consumption In Canada, 1989. Statistics Canada, Ottawa, ON, Part 2. 32-230 (Part II).

US EPA. 1989. Risk Assessment Guidance for Superfund: Volume 1-Human Health Evaluation Manual (Part A), EPA/540/1-89/002, Office of Emergency and Remedial Response, Washington, D.C.

US EPA. 1991. Risk Assessment Guidance for Superfund Volume I: Human Health Evaluation Manual Supplemental Guidance - Standard Default Exposure Factors [interim final]. OSWER Directive 9285.6-03. Office of Solid Waste and Emergency Response, Washington, DC.

US EPA. 1992. Dermal Exposure Assessment: Principles and Applications. Interim Report. (U.S.) Environmental Protection Agency. EPA/600/8-91/011B.

US EPA. 1994. Guidance Manual for the Integrated Exposure Uptake Biokinetic Model for Lead in Children. OSWER Directive 9285.7-15-1. Washington, DC: Environmental Protection Agency, Office of Emergency and Remedial Response, 1994. Cited in Walker and Griffin,1997.

US EPA. 1997. Exposure Factors Handbook. U.S. Environmental Protection Agency.

US EPA. 2000a. Risk Assessment Guidance for Superfund Volume I: Human Health Evaluation Manual (Part E, Supplemental Guidance for Dermal Risk Assessment), Interim Guidance.EPA/540/R-99/005. Office of Solid Waste and Emergency Response, Washington, DC. PB99-963312. As cited by US EPA Region IX.

US EPA. 2000b. Child-Specific Exposure Factors Handbook. U.S. Environmental Protection Agency. NCEA-W-0853.

Van Wijnen, J.H., P. Clausing, B. Brunekreff, 1990. Estimated soil ingestion by children. Environ. Res. 51:147-162.

Walker and Griffin. 1997. Site-specific Data Confirm Arsenic Exposure Predicted by the U.S. Environmental Protection Agency. Susan Walker, Susan Griffin. AGEISS Environmental, Inc., Denver, CO 80202 USA. Environmental Health Perspectives. Vol. 106, pp. 133-139.

Table A6-7a: Body Weight Receptor Parameters

	Unit	Infant	Toddler						Child	Teen	Adult	Source
		0-0.5y	0.5-1 y	1-1.5 y	1.5-2 y	2-2.5y	3y	4y	5-11y	12-19y	20+ y	
Time Spent in Age Group	Yrs	0.5	4.5						7	8	50	
Body Weight	Kg	7	13						27	57	70	CEPA, 1994; Health Canada, 1995; CCME, 1996
		6.28 F 7.20 M	9.23 F 9.83 M	10.39 F 10.7 M	11.66 F 12.5 M	12.17 F 12.69 M	13.94 F 14.85 M	16.42 F 17.14 M	26.4 F 26.4 M	54.3 F 60.1 M	70	EHD[1], 1993
		-	16.5						-	-	70.7	CCME, 2000
		8.2	16.5						32.9	59.7	70.7	O'Connor, 1997

Parameter	Unit	Infant	Toddler					Child	Teen	Adult	Source
		0 - 0.5 y	6-11 mo	1 y	2 y	3 y	4 y	5 - 11 y	12 -19 y	18-74 y	
Time Spent in Age Group	Yrs	0.5	0.5	1	1	1	1	7	8	57	
Body Weight	Kg	n/av	9.1	11.3	13.3	15.3	17.4	29.2	58.3	71.8	US EPA, 1997[2]

1. EHD (Environmental Health Directorate Working Group on Reference Values). 1993. Reference Values for Canadian Populations. Updated May 1997. Infant, toddler, and child data are from the Nutrition Canada Survey, 1970-72; teen data are from the Canada Fitness Survey, 1981.

2. US EPA recommended values; from NCHS, 1997; weights include clothing weight estimate 0.09-0.28kg; no body weight value available for infants 0-5 months of age; adult range here is 18 to 74 years of age, thus 18 and 19 year olds are accounted for in both the teen and adult age categories.

EHD and US EPA data presented in this table are summarized from data from individual years.

n/av = value not available.

Table A6-7b: Inhalation Rate

Parameter	Unit	Infant 0-0.5y	Toddler 0.5-4y	Child 5-11y	Child 9-11y	Teen 12-19y	Adult 20+y	Source
Number of Years Spent in Age Group	Yrs	0.5	4.5	7	-	8	50	
Inhalation Rate	m^3/day	2	5	12	-	21	23	CEPA, 1994; Health Canada, 1995; EHD, 1993; CCME, 1996
		-	9.3	-	-	-	16.2	CCME, 2000
		2.1	9.3	-	14.5	15.8	15.8	O'Connor, 1997

Parameter	Unit	<1 y	1-2 y	3-5 y	6-8 y	9-11 y	12-14 y	15-18 y	19-65+ y	Source
Number of Years Spent in Age Group	Yrs	1	2	3	3	3	3	4	51	
Inhalation Rate	m^3/day	4.5	6.8	8.3	10	14M 13F	15 M 12 F	17 M 12F	11.3 F 15.2 M	US EPA, 1997
		-	-	-	-	-	-	-	20	US EPA, 1991[1]

[1]. Adult age range not specified.

M = male; F = female.

Table A6-7c: Water Intake Rate

Parameter	Unit	Infant		Toddler		Child				Teen		Adult	Source
		0-0.5y	<1 y	<3 y	0.5-4y	1-10 y	3-5y	5-11y	6-17y	11-19y	12-19y	20+ y	
Number of Years Spent in Age Group	Yrs	0.5			4.5			7			8	50	
Water Intake	L /day	0.75	-	-	0.8	-	-	0.9	-	-	1.3	1.5	CEPA, 1994[1]; Health Canada, 1995; CCME, 1996
		-	-	-	0.6	-	-	-	-	-	-	1.5	CCME, 2000
		0.3	-	-	0.6	-	-	0.8	-	-	1	1.5	O'Connor, 1997
		0.4/0.8	-	0.61	-	-	0.87	-	1.14	-	-	1.49	EHD[2], 1993
		-	0.3	-	-	0.74	-	-	-	0.97	-	1.4	US EPA, 1997
		-	-	-	-	-	-	-	-	-	-	2	US EPA, 1991[3]

1. Infants here are non-breast fed; it is assumed that exclusively breast fed infants do not require supplementary liquids.
2. Regarding values quoted for infants, values refer to bottle fed infants consuming concentrated and powdered milk, respectively; adult estimate refers to adults 18 years and older.
3. Adult age range not specified; estimate based on consumption 350 days/year for 30 years.

Soil Investigation and Human Health Risk Assessment for the Rodney Street Community, Port Colborne: March 2002

Table A6-7d: Soil Ingestion Rate

Parameter	Units	Infant		Toddler			Child	Teen	Adult	Source
		0-0.5y	0.5-4y	<6 y	1-6 y	1-7y	5-11y	12-19y	20+ y	
Number of Years Spent in Age Group	Years	0.5	4.5	6	6	7	7	8	50	
Soil Ingestion	mg /day	35	50	-	-	-	35	20	20	CEPA, 1994; Health Canada, 1995
		-	-	-	-	70	-		20	EHD, 1993
		20	80	-	-	-	20	20	20	CCME, 1996, 2000
		100 (best estimate of mean) 200 (conservative estimate of mean)-							50	US EPA, 1997[1]
		100 (best estimate of mean) 400 (upper %percentile, short duration) 10,000 (pica, acute only)							-	US EPA, 2000b
		-	-	-	200	-	-	-	100 (7+ y)	US EPA, 1991[2]
		-	-	median 24 mg/day (sd = 4 mg/day) 95th percentile 91 mg/day (sd = 16.6 mg/day)				-	-	Stanek et al., 2001
		35	80	-	-	-	80	20	20	MOEE, 1995[3]

1. 100 mg/day is the best estimate of the mean for children under 6 years of age; 200 mg/day may be used as a conservative estimate of the mean; for pica children, 10 g/day is a reasonable value to be used in acute exposure assessments. Adult age range not specified.

2. Used for calculating a 30 year residential exposure; there is a 6 year exposure duration which accounts for the period of highest soil ingestion and lowest body weight (15 kg), followed by a 24 year exposure for older children and adults, using and adult body weight (70 kg).

3. Soil ingestion rates typically used by MOE in assessing risks associated with chemicals in soil.

Part B - Human Health Risk Assessment: Appendix 6

Soil Investigation and Human Health Risk Assessment for the Rodney Street Community, Port Colborne: March 2002

Table A6-7e: Soil Adhesion to Skin

Parameter	Unit	Infant	Toddler	Child		Teen	Adult	Source
		0-0.5y	0.5-4y	5-11y	6-17y	12-19y	20+ y	
Number of Years Spent in Age Group	Yrs	0.5	4.5	7	12	8	50	
Soil Adhesion to Skin	g /day	2.2	3.5	5.8	-	9.1	8.7	Health Canada, 1995
	mg /cm²	-		0.1 (hands only) 0.01 (whole body)				CCME, 2000
		-	-	0.5 - 1.5		-	0.58 - 1.40	US EPA, 1997[1]
				0.2				US EPA, 1992[2]
						0.07	0.2	US EPA, 2000a

1. Studies cited specify adherence values within the given ranges. Age ranges were not specified.
2. United States Environmental Protection Agency (US EPA), Exposure Assessment Group, Office of Health and Environmental Assessment. Dermal Exposure Assessment: Principles and Applications. EPA/600/8-91/011B. January 1992, Interim Report. Range of values in studies were 0.2 - 1.5 mg/cm²; since this range is derived from hand measurements only, it may overestimate average adherence for the entire exposed skin area; thus, the lower end of this range (0.2) may be the best value to represent an average overall exposed skin and 1 mg may be a reasonable upper limit. Age ranges not specified.

Soil Investigation and Human Health Risk Assessment for the Rodney Street Community, Port Colborne: March 2002

Table A6-7f: Body Surface Area

Parameter	Unit	Infant	Toddler	Child	Teen	Adult	Source
		0-6 months	7 months- 4 years	5-11 years	12-19 years	20+ years	
Number of Years Spent in Age Group	Years	0.5	4.5	7	8	50	CCME, 1996
Total Body	m²	0.3	-	-	-	-	
Head, Arms, Hands & Lower Legs		-	0.26	0.41	-	-	
Head, Arms & Hands		-	-	-	0.43	0.43	
Arms		0.052	0.075	0.121	0.227	0.255	Health Canada, 1995
Hands	m²	0.0185	0.035	0.048	0.081	0.091	
Legs		0.077	0.139	0.2705	0.518	0.582	
Feet		0.022	0.04	0.0725	0.113	0.1275	
Total (Arms, Hands, Legs, & Feet)		0.17	0.289	0.512	0.939	1.06	
Arms			-			0.412	EHD, 1993
Hands			-			0.082	
Legs	m²		-			0.62	
Feet			-			0.13	
Total (Arms, Hands, Legs, & Feet)			-			1.24	

Soil Investigation and Human Health Risk Assessment for the Rodney Street Community, Port Colborne: March 2002

Parameter	Unit	Infant 0-6 months	Toddler 7 months- 4 years	Child 5-11 years	Teen 12-19 years	Adult 20+ years	Source
Number of Years Spent in Age Group	Years	0.5	4.5	7	8	50	O'Connor, 1997
Hands	m²	0.032	0.043	0.059	0.08	0.089	
Arms		0.055	0.089	0.148	0.223	0.25	
Legs		0.091	0.169	0.307	0.497	0.572	
Feet		0.025	0.043	0.072	0.108	0.119	
Total (Hands, Arms, Legs, & Feet)		0.203	0.344	0.586	0.908	1.03	

Parameter	Unit	Infant <1 year	Toddler 1–4 years	Child 6 years	Child 9 years	Teen 12-17 years	Adult 18+ years	Source
Arms (Means)	%	13.7	13.0 - 14.0	13.1	12.3	12.1 - 17.5	-	US EPA, 1997
Hands (Means)		5.3	5.3 - 6.07	4.71	5.3	5.11 - 5.68	-	
Legs (Means)		20.6	23.1 - 27.8	27.1	28.7	30.5 - 33.6	-	
Feet (Means)		6.54	6.27 - 7.29	6.9	7.58	6.93 - 8.02	-	
Total (Arms, Hands, Legs, & Feet)	%	46.1	47.7 - 55.2	51.8	53.9	54.6 - 64.8	-	
Exposed Parts of Body	%	In moderate climates, it may be reasonable to assume that 5% of the skin is exposed during the winter, 10% during the spring and fall, and 25% during the summer.						

Soil Investigation and Human Health Risk Assessment for the Rodney Street Community, Port Colborne: March 2002

Parameter	Unit	Toddler 2-4 years	Child 5-11 years	Teen 12-17 years	Adult 18+ years	Source
Arms (Means)		-	-	-	0.219	US EPA, 1997; US EPA, 1992
Hands (Means)		-	-	-	0.0793	
Legs (Means)		-	-	-	0.497	
Feet (Means)		-	-	-	0.105	
Total (Arms, Hands, Legs, & Feet) (Means)	m^2	-	-	-	0.9	
Total Body (Medians)		0.579 - 0.731	0.779 - 1.30	1.34 - 1.80	1.815	

Parameter	Unit	Toddler 3 y	Toddler 3-5 y	Child 6 y	Child 6-8 y	Child 9 y	Child 9-11 y	Teen 12-14 y	Teen 15-17 y	Adult 18+ y	Source
Arms (Males Only)		0.096	-	0.11	-	0.13	-	-	-	0.23	US EPA, 1989 (medians)
Hands (Males Only)		0.04	-	0.041	-	0.057	-	-	-	0.082	
Legs (Males Only)	m^2	0.18	-	0.24	-	0.31	-	-	-	0.55	
Total Body (Males & Females)		-	0.72	-	0.925	-	1.16	1.49	1.68	1.82	

Table A6-7g: Fruit and Vegetable Consumption

Parameter	Unit	Infant 1-2 y	Toddler 3-5 y	Child 6-11 y	Teen 12-19 y	Adult 20-39 y	Adult 40-69 y	Adult 70+ y	Source
Number of Years Spent in Age Group	Years	n/av[1]	n/av[1]	n/av[1]	n/av[1]	n/av[1]	n/av[1]	n/av[1]	
Backyard - Fruits	g/kg/day	8.74	4.07	3.59	1.94	1.95	2.66	2.25	US EPA, 1997
		All Ages: 2.31 (central city), 2.41 (non-metro), 3.07 (suburb), 2.68 (all groups) 1.05 (suburb in northeast), 0.929 (all groups in northeast)							
Backyard - Fruits	g/day	5.2	42 (reasonable worst case) (ages not specified)						US EPA, 1991
Backyard - All Vegetables.	g/kg/day		2.46	2.02	1.48	1.47	2.07	2.51	US EPA, 1997
		All Ages: 1.40 (central city), 2.68 (non-metro), 1.82 (suburb), 2.08 (all groups) 3.05 (non-metro northeast), 1.59 (suburb northeast), 1.78 (all groups northeast)							
Backyard - All Vegetables.	g/day	3.48	80 (reasonable worst case) (ages not specified)						US EPA, 1991
Backyard - Exposed Vegetables	g/kg/day		1.74	1.39	1.07	1.05	1.6	1.68	US EPA, 1997
		All Ages: 1.11 (central city), 1.87 (non-metro), 1.35 (suburb), 1.52 (all groups) 1.65 (all groups in northeast)							
Backyard - Protected Vegetables	g/kg/day	2.46	1.3	1.1	0.776	0.762	0.93	1.05	US EPA, 1997
		All Ages: 0.996 (central city), 1.07 (non-metro), 0.926 (suburb), 1.01 (all groups) 0.701 (all groups in northeast)							
Backyard - Root Vegetables.	g/kg/day	2.52	1.28	1.32	0.937	0.874	1.13	1.22	US EPA, 1997
		All Ages: 0.749 (central city), 1.43 (non-metro), 1.06 (suburb), 1.16 (all groups) 0.838 (all groups in northeast)							

Soil Investigation and Human Health Risk Assessment for the Rodney Street Community, Port Colborne: March 2002

Parameter	Unit	Infant 0-0.5 y	Toddler 0.5-4 y	Child 5-11 y	Teen 12-19 y	Adult 20+ y	Source
Number of Years Spent in Age Group	Years	0.5	4.5	7	8	50	
Backyard - Root Vegetables.	g/day	8.18	10.3	15.9	22.4	19.3	MOEE, 1995
Backyard - Other Vegetables.	g/day	7.09	6.6	9.65	11.8	14.1	MOEE, 1995
Backyard - All Vegetables.	kg/harvest			22.8			Gradient, 1995

Parameter	Unit	Child 1-4 y	Adult 20+ y	Source
Number of Years Spent in Age Group	Years	n/av[1]	n/av[1]	
Backyard - All Fruits & Vegetables.[2]	g/day	26	34	MOEE, 1994[2]

Parameter	Unit	Infant 0-0.5 y	Toddler 0.5-4 y	Child 5-11 y	Teen 12-19 y	Adult 20+ y	Source
Number of Years Spent in Age Group	Years	0.5	4.5	7	8	50	

Part B - Human Health Risk Assessment: Appendix 6

Soil Investigation and Human Health Risk Assessment for the Rodney Street Community, Port Colborne: March 2002

Parameter	Unit	Infant	Toddler	Child	Teen	Adult	Source
		0-0.5 y	0.5-4 y	5-11 y	12-19 y	20+ y	
Root Vegetables.	g/day	83	105	161	227	188	O'Connor, 1997
Other Vegetables.	g/day	72	67	98	120	137	O'Connor, 1997
All Vegetables.	g/day	42	125	198	250	250	Health Canada, 1995 CEPA, 1994 EHD, 1993
Vegetables.	g/day			200 (ages not specified)			US EPA, 1991
Fruits & Juices	g/day	136	234	268	258	245	O'Connor, 1997
Fruit & Fruit Products	g/day	112	189	202	160	186	EHD, 1993 Health Canada, 1995 CEPA, 1994
Fruit	g/day			140 (ages not specified)			US EPA, 1991

[1] Number of years in age categories not specified.
[2] Calculated as totals of all food group types consumed.

Soil Investigation and Human Health Risk Assessment for the Rodney Street Community, Port Colborne: March 2002

Table A6-7h: Activity Patterns

Parameter	Unit	Infant	Toddler	Child	Teen	Adult	Source
		0-0.5 year	0.5-4 years	5-14 years	15-19 years	20+ years	
Number of Years Spent in Age Group	Years	0.5	4.5	10	5	50	
Time Spent Outdoors	Min/Day	-	-	-	85	85	O'Connor, 1997

Parameter	Unit	Infant	Toddler	Child	Adult		Source
		0-0.5 year	0.5-2 years	3-11 years	12+ years		
Number of Years Spent in Age Group	Years	0.5	2.5	9	58		
Time Spent Outdoors	Hour/Day	-	-	5 (weekdays) 7 (weekends)	1.5		US EPA, 1997

Human Health Risk Assessment

Appendix 7

Dermal Uptake Coefficients for Metals

Table of Contents
Human Health Risk Assessment - Appendix 7
Dermal Uptake Coefficients for Metals

A7-1 Dermal Uptake Coefficients for Metals

Daily contact with metals through soil present on the skin represents a potential route of exposure. However, the insoluble nature of most metals in soil limits their bioaccessability for uptake into and through the skin. Where data is available, it shows that dermal uptake of metals is low (Paustenbach, 2000). The rate at which a metal is taken up into the outer layers of the skin is referred to as the *dermal uptake coefficient* (DUC). Studies of the dermal absorption of nickel have suggested that the outer layer of the skin, the stratum corneum, can act as a collector for dermally applied nickel before it enters the underlying tissue (Fullerton et al., 1992). While there is little information available in the scientific literature on dermal uptake of the other metals of concern in this assessment, it is reasonable to assume that similar mechanisms will govern their absorption into the body. This process can be considered to be equivalent to ingestion or inhalation intakes where the material is delivered into the gut or lungs, but cannot be considered to have entered the body proper until it is absorbed through the gut or lung lining and into the underlying tissue or blood.

Therefore, for the purposes of this assessment, dermal uptake coefficients will be used to estimate the amount of each metal that could be delivered to the skin through contact with soil (referred to as *Dermal Intake*). The calculation of dermal intakes for each metal is provided in Appendix 3.

Dermal absorption studies typically use exposure periods extending well past 24 hours. However, the amount of time a human receptor is in contact with a chemical/soil mixture on the skin should correspond with soil contact times normally encountered (US EPA, 1992). In real life situations, soil is likely to remain in contact with the skin for only a few hours. Consequently, dermal intake estimates from studies with longer than 24 hours are likely to overestimate normal human exposures.

Metal specific dermal uptake coefficients have been identified for two of the six metals (cobalt and nickel) considered in the detailed exposure assessment. The selection of the dermal uptake coefficient for each metal is discussed below.

A7-2 Dermal Uptake Coefficient for Nickel

Studies of how nickel can penetrate the skin in humans and animals are limited. Only studies of intact organisms where nickel is measured in blood or urine can show whether nickel has penetrated through the skin layers into the bloodstream. This is important since permeation of nickel into the upper layers of the skin does not automatically mean that nickel has been absorbed into the body. Similarly, contact dermatitis reactions following dermal application of nickel solutions may not imply complete penetration but only irritation of the deep layers of the skin. There are few studies that address the uptake of nickel through human skin available in the literature. Human studies may be separated into application of nickel to intact skin in individuals

(Norgaard; 1955, 1957), and, *in vitro* studies with excised human skin in diffusion cells (Fullerton et al., 1986; 1992, Frankild et al., 1995, Samitz and Katz, 1976). In addition, reviews are available (ATSDR, 1997, Hostynek et al., 1993). The available studies on nickel uptake in human skin have focused primarily on the uptake of nickel and its relationship with nickel contact dermatitis (Norgaard, 1955; Fullerton et al., 1986; 1992; Frankild et al., 1995; Samitz and Katz, 1976).

A7-3 Intact Skin Studies

These studies have examined the uptake of soluble forms of nickel into the outer layers of the skin. Two types of study protocols are used to measure dermal uptake; studies where nickel compounds were applied to skin and secured with some form of patch occluding the skin and studies where the applied material were not secured with a patch. Norgaard (1955) applied aliquots of radioactive ^{57}Ni to the forearm. This area was occluded and the radioactivity measured by placing the counter directly over the treated area. Loss of radioactivity with time was interpreted as resorption through the skin, however, no measures of radioactivity in blood or urine were taken. On this basis, dermal uptake rates that ranged between 55% and 77% over a 24 hour period when nickel sulphate was applied to occluded skin reported (Norgaard, 1955). However, it could not be determined if the nickel in this study was actually bound in the outer layers of the skin (ATSDR, 1997). This limits the utility of the study for assessing dermal absorption of nickel compounds.

A7-4 *In vitro* Studies of Excised Skin

Nickel diffusion through excised cadaver skin was studied using ^{63}Ni (as nickel sulphate) (Samitz and Katz, 1976). The diffusion of nickel (0.001M to 0.1M) from physiological saline or human sweat through the epidermis was slight. However, considerable amounts of nickel were bound to the epidermis. No diffusion of nickel through the epidermis took place within five hours, even after 48 hours, less than 0.1% of the nickel diffused through the skin. The excised epidermis does not seem to have been occluded.

In a study that applied nickel chloride to excised human skin, Fullerton et al., (1986) reported that 0.23% of the applied dose was absorbed over a 144 hour period in unoccluded skin while 3.5% was absorbed by occluded skin. In a follow-up study designed to determine the efficacy of different vehicle carriers for dermal patch testing, Fullerton et al., (1992) reported that dermal uptake of nickel sulphate in excised human skin, ranged between 3% and 5% of the applied dose in occluded skin over a 93 hour testing period. The study further showed that the level of absorption was dependent on the carrier vehicle used, and that the dermal absorption of dissolved nickel was greater than that of undissolved or crystalline nickel (Fullerton et al., 1992). Analysis of nickel levels in the stratum corneum, epidermal, and dermal layers of skin also showed that the outer stratum corneum layer held the highest levels of nickel. The study also found that little nickel was able to penetrate through all layers of the skin to the underlying tissue (Fullerton et al.,

1992). The authors suggest that this layer of the skin may act as a reservoir for nickel that could allow nickel to move into other tissue and that as the level of nickel increases in this layer, subsequent exposures would allow greater amounts of nickel to move through the skin (Fullerton et al., 1992).

Frankild et al., (1995) examined the time- and dose-related effect of the detergent, sodium lauryl sulphate (SLS), on *in vitro* percutaneous penetration of radio labeled ^{63}Ni chloride in excised human cadaver skin. Simultaneous application of SLS (0.25%, 2% and 10%) and nickel chloride resulted in a significant dose response relationship between SLS concentration and penetration of nickel. Inspection of the data indicates that as in the studies reported by Fullerton et al., (1986, 1992), no penetration occurred over the first 48 hours of the experiments, and all the SLS-mediated penetration occurred after 48 hours. No penetration of nickel chloride occurred in the SLS free controls (Frankild et al., 1995).

This information suggests that while soluble nickel may accumulate in the upper layers of the skin in the initial 48 hours, penetration, if any, does not occur until later. In the context of nickel in soil in contact with the skin for periods of less than 24 hours, assuming normal hygiene, it is difficult to make a case for any dermal penetration of nickel. Consequently, prorating dermal penetration rates based on cumulative penetration after 69 hours or 96 hours *in vitro* exposures will overestimate dermal nickel intake estimates. As pointed out by Frankild et al., (1995), *in vitro* skin models are limited in that the metabolic pathways of the skin and normal repair mechanisms are not functioning.

It should be stressed that the work of Fullerton et al., in 1992 was conducted with occluded skin. This is not representative of dermal contact with soil where exposures would not be expected to last for more than 24 hours. Further, Hostynek et al., (1993) note that occlusion increases skin penetration ten fold over unoccluded conditions. The authors further note that sweat contains significantly higher levels of nickel than normal blood serum and that it is a significant excretory pathway for the metal (Hostynek et al., 1993). Thus, it would appear that dermal absorption of nickel from soil is likely to be very limited and that much of what is absorbed into the outer layers of the skin is likely to be lost from the skin due either to removal in sweat or through the normal loss of outer skin cells from the stratum corneum.

The study using unoccluded skin most closely resembles the dermal exposures to nickel in soil that could be expected in the Rodney Street community. Therefore, the absorption factor of 0.23%, reported by Fullerton, et al., (1986) was used to develop a dermal uptake coefficient for nickel in Port Colborne.

As noted above, Fullerton et al., 1986 reported that 0.23% (0.0023) of an applied dose of nickel chloride was absorbed over a period of 144 hours. However, bathing activities can be expected to limit skin contact with nickel bearing soil to a maximum of 24 hours. Therefore, it is necessary to correct the uptake coefficient reported by Fullerton et al., (1986) to account for the difference in

the expected exposure duration of 24 hours and the 144 hours used in the Fullerton study. In developing a corrected dermal uptake coefficient for nickel oxide, it has been assumed that soil would remain in contact with the skin for a period of 24 hours before being removed by bathing activities. The derivation of dermal uptake coefficient for nickel is shown in equation A7-1.

Eq A7-1:

$$DUC_{Ni} = 0.0023 * \left(\frac{24 hours}{144 hours} \right) = 0.00038 = 3.8 X 10^{-4}$$

Where:	DUC_{Ni}	=	Dermal Uptake Coefficient for nickel
	24 hrs	=	Expected exposure duration
	144 hrs	=	Duration of experimental exposure
	0.0023	=	Reported dermal absorption of Nickel Chloride

It should be noted that this approach assumes a linear relationship between the length of exposure and the amount of nickel available to the skin for absorption. It should also be noted that there is a marked difference in water solubilities between the nickel chloride used by Fullerton et al., (1986) and nickel oxide which is the predominant form of nickel found in the soil on Rodney Street and elsewhere in Port Colborne. Reported solubilities are 642 g/L and 0.0011 g/L for nickel chloride and nickel oxide respectively (ATSDR, 1997). Further, the nickel chloride used by Fullerton et al., (1986) was applied in solution and was freely available for absorption by the skin. In Port Colborne, the nickel oxide is associated with soil particles and must dissociate (dissolve) from the soil particles before it is available for absorption by the skin. Therefore, using a dermal dose factor derived for dissolved nickel chloride to estimate the dermal dose of undissolved nickel oxide, will significantly overestimate the amount of nickel oxide available for absorption by the skin. Thus, the DUC_{Ni} factor selected for use at Rodney Street in Port Colborne will provide conservative estimates of dermal exposure for all age groups considered in the assessment.

A7-4.1 Dermal Bioaccessibility

For the NiO in the soil to become available for penetration through the layers of the skin, the nickel has to be in a soluble form. The nickel oxide in the soil may dissolve to some degree in the layer of sweat on the skin during perspiration. The soluble nickel speciation data from the Lakefield testing (Part A, Table 10) may not be equivalent to extraction in synthetic sweat but may indicate some estimate of the soluble nickel available to penetrate the skin. The soluble nickel leaching approach used by Lakefield extracted the soil samples in hot water for 30 minutes. Soil pH (Part A, Table 9) ranged from pH 6.85 to pH 7.75, however, while the pH of the hot water extracts was not reported, it is not expected to be much below pH 7. Hot water was used instead of the 0.1 M ammonium citrate (pH 4.4) used by Zatka et al., (1992), a fluid that is similar to artificial sweat solutions used in other studies because the hot water extraction gave a more accurate extraction of soluble nickel salts. Freshly perspired sweat has a neutral pH, however, it becomes slightly acidic (pH 4 to pH 6) depending on the presence of resident skin

bacteria. The mean soluble nickel value in hot water is 0.37% (range = 0.26% to 0.68%). Tests to measure plant-extractable nickel in Port Colborne soils averaged 0.22% for pH neutral mineral soils (Part A, section 6.1.3).

Inco has provided information to suggest that at pH 7 *without the glycine buffer* used by Exponent in their bioaccessibility studies (Appendix 5), nickel extractability is 1.5 %. This information (also discussed in section A2-9.2.4) suggests that despite the generally insoluble nature of the nickel compounds present in Rodney Street community soils, some soluble nickel may leach from the soil into human sweat. The data of Fullerton et al., (1986), Frankild et al., (1995) and Samitz and Katz, (1976) indicating that nickel ions do not penetrate the upper layers of human skin for at least 48 hours support the DUC factor selected for Rodney Street community soils.

A7-5 Dermal Uptake Coefficient for Cobalt

Paustenbach (2000) cites a dermal uptake coefficient of 0.0004 for cobalt chloride. Information on the cobalt species present in Rodney Street soil is not available. Therefore, it has been assumed that the dermal uptake coefficient for cobalt chloride is representative of the dermal uptake coefficient for cobalt in soil in the Rodney Street community.

A7-6 Dermal Uptake Coefficients for Antimony, Beryllium, Cadmium and Copper

Dermal uptake coefficients for the remaining metals are not available. In the absence of such values, a default value of 0.01 is recommended by the US EPA for assessing dermal exposure to inorganic compounds such as metal salts (US EPA, 1992). However, this recommendation is based on the conservative assumption that all metal delivered to the skin is available for uptake into the skin. Sweat contains many electrolytes (mainly sodium chloride, potassium and calcium salts), urea and lactic acid. The primary secretion is similar to plasma and initially would have a neutral to slightly acidic pH. Artificial sweat solutions contain high levels of NaCl (around 10%) and lactic acid (around 5%), with a pH of about 5. An adult man secretes 650 mL/day (50 to 1600 mL/day). As noted elsewhere in this report, the amount of each metal that could be released for the soils from Rodney Street, under acidic and neutral pH conditions has been assessed (Appendix 5). For all metals, maximal % bioaccessibility occurred at acid pH. Assuming a slightly acidic (pH 5) sweat layer on the skin, the bioaccessibility of these metals from soil in contact with the skin is likely to be much less than the maximal % bioaccessibility under more acidic (pH 1.5) conditions. These values represent an over estimate of the amount of each metal that could be expected to be released from the soil while in contact with skin. Therefore, the most recent default values for dermal absorption recommended by US EPA Region III (US EPA, 1995) have been used as the dermal uptake coefficients for estimating dermal exposure to these metals.

The dermal uptake coefficients used in this report are summarized in Table A7-1.

Table A7-1: Dermal Uptake Coefficients

	Antimony	Beryllium	Cadmium	Cobalt	Copper	Nickel
Coefficient	0.01	0.01	0.01	0.0004	0.01	0.00038

A7-7 References

ATSDR (Agency for Toxic Substances and Disease Registry), 1997. U.S. Department of Health and Human Services. Toxicological Profile for Nickel. Atlanta, Georgia, USA.

Frankild, S., K.E. Andersen and G.D. Nielsen. 1995. Effect of sodium lauryl sulfate (SLS) on *in vitro* percutaneous penetration of water, hydrocortisone and nickel. Contact Dermatitis. 32:338-345.

Fullerton, A., J.R. Andersen, A. Hoelgaard, T. Menne. 1986. Permeation of nickel salts through human skin *in vitro*. Contact Dermatitis. 15:173-177.

Fullerton, A. et al., 1992. Topical nickel salts: The influence of counterion and vehicle on skin permeation and patch test response. In: *Nickel and Human Health: Current Perspectives*. 1992. Eds. E. Nieboer and J.A. Nriagu. J. Wiley and Sons, Inc. pp. 211-222.

Hostynek, J.J., R.S. Hinz, C.R. Lorence, M. Price, and R.H. Guy. 1993. Metals and the skin. CRC Critical Reviews in Toxicology. 23: 171-235.

Norgaard, O. 1955. Investigation with radioactive Ni-57 into the resorption of nickel through the skin in normal and in nickel-hypersensitive persons. Acta Derm. Venereol. 35:111-117.

Norgaard, O. 1957. Investigations with radioactive nickel, cobalt and sodium on the resorption through the skin in rabbits, guinea pigs and man. Acta Derm. Venereol. 37:440-445.

Paustenbach, D.J. 2000. The practice of exposure assessment: A state-of-the-art review. J. Toxicol. Environ. Health. Part B. 3:179-291.

Samitz, M.H. and S. Katz. 1976. Nickel - epidermical interactions: diffusion and binding. Environ. Res. 11:34-39.

US EPA. 1992. Dermal Exposure Assessment: Principles and Applications. Washington, D.C. US Environmental Protection Agency. EPA/600/8-91/011B.

US EPA. 1995. U.S. Environmental Protection Agency, Region III Technical Guidance Manual Risk Assessment: Assessing Dermal Exposure from Soil. EPA/903-K-95-003.

Zatka, V.J., J.S. Warner, D. Maskery. 1992. Chemical speciation of nickel in airborne dusts: Analytical method and results of an interlaboratory test program. Environ. Sci. Tech. 26:138-144.

Human Health Risk Assessment

Appendix 8

Statement of the Expert Peer Review Panel

Expert Peer Review Panel Members

The science of risk assessment, as it is used to evaluate the potential human health risks of exposure to environmental contaminants, is rapidly advancing. To ensure that the most recent and most appropriate science was used for the Rodney Street community risk assessment the ministry assembled an international expert peer review panel which included toxicologists and risk assessors from Canada, the United States and Norway. The panel members were selected on the basis of their extensive knowledge and international reputation in the areas of toxicology, risk assessment, and the health effects of nickel. Panel members included:

Dr. Ambika Bathija, United States Environmental Protection Agency, Washington, D.C., USA

Dr. Lynne Haber, Toxicology Excellence for Risk Assessment, Cincinnati, Ohio, USA

Dr. Robert Jin, Ontario Ministry of Health and Long-Term Care, Toronto, Ontario, Canada

Dr. Tor Norseth, Norwegian National Institute of Occupational Health, Oslo, Norway

Dr. Rosalind Schoof, Gradient Corporation, Seattle, Washington, USA

Dr. John Wheeler, Agency for Toxic Substances and Disease Registry (ATSDR), Atlanta, Georgia, USA

Statements of the Peer Review Panel

In August 2001 the panel members critically reviewed a working draft of the ministry's report. A professional facilitator was hired and a meeting of the panel members and ministry scientists was held in Toronto on September 20 and 21, 2001 to resolve issues raised by the panel's review. Follow-up teleconferences were held on October 16 and 19, 2001. The report was revised based on consensus decisions reached with panel members and the October 2001 draft report was placed on the EBR for a 30 day public consultation period. Following the September meeting and October teleconferences the panel members agreed to the following statement:

> The draft document entitled *Soil Investigation and Human Health Risk Assessment for the Rodney Street Community, Port Colborne: October 2001*, and the report entitled *Recommendations and Conclusions from the International Expert Panel Meeting*, as received October 12, 2001, together with revisions addressing verbal comments tabled in the teleconferences held on October 16 and October 19, 2001, appropriately address the issues raised at the expert panel meeting held in Toronto, Ontario on September 20 - 21, 2001. Panel members also reviewed revisions to the October 2001 draft made as a result of the November 2001 public consultation period and concurred that the revisions and the ministry's response to the public comments were appropriate.

The ministry received twelve submissions on the *Soil Investigation and Human Health Risk Assessment for the Rodney Street Community, Port Colborne: October 2001* draft report as a

result of the 30 day November 2001 EBR public consultation period. The Environmental Bill of Rights Act stipulates that the ministry must take every reasonable step to ensure that all comments relevant to the proposal received as part of the public participation process are considered in it's decision (S. 35 EBR Act). Based on the EBR comments, the October 2001 draft report was revised and a summary of the public comments and how the ministry responded to them, was provided to the panel members in February 2002. The ministry asked the panel to review the report's revisions, the public comments, and the ministry's response to the comments. All panel members concurred with the following statement:

> As an expert panel member for the Ontario Ministry of the Environment's Port Colborne human health risk assessment project, I have seen the final draft of the report, as provided to me under cover letter dated January 30, 2002, and I have reviewed the comments received by the ministry through their public consultation process. I concur that, where the changes to the report and the comments pertain to the science and expert judgement that guided the development and application of the risk assessment model, the ministry has responded appropriately. I have not been asked to, nor would I comment on or be expected to concur with, issues relating to how the ministry addressed public opinion or administered government policy.

Recommendations and Conclusions
From the International Peer Review Panel Meeting

**Meeting to Review the Draft Revised Human Health Risk Assessment of the Rodney Street
Community completed by the Ontario Ministry of the Environment (MOE)**

**Radisson Plaza Hotel Admiral
Toronto, Ontario, Canada
September 20 and 21, 2001**

The Ontario Ministry of the Environment assembled a panel of internationally recognized
scientists to conduct a peer review of the ministry's draft report entitled *Soil Investigation and
Human Health Risk Assessment for the Rodney Street Community: Port Colborne (Revised 2001)*
(hereinafter referred to as the Revised 2001 HHRA Study). The Panel's recommendations and
conclusions from the review of the study primarily focus on nickel, and are summarized in the
following consensus statements.

Statement as to Conflict of Interest:

The members of the assembled International Peer Review Panel have declared they have no
existing conflict of interest with respect to review of this assessment. They are not employed by,
under contract to, or received monies from Inco with respect to the matter under review.

Overall Conclusion:

The International Peer Review Panel concluded that:

- In general, the methods followed in the study are consistent with the currently generally
  accepted risk assessment paradigm.

- The Panel supports the process and methodology as applied for the purpose of the Rodney
  Street assessment.

- A number of specific revisions were recommended by the Panel and are outlined below in
  this recommendations and conclusions document.

- The Panel came to consensus on the set of data and parameters which should be used for
  establishing a soil intervention level for nickel for the Rodney Street community.

- The available scientific data on dermal sensitization are inadequate to estimate a soil
  intervention level that would protect sensitized individuals from nickel dermatitis, or that
  would protect people from being sensitized to nickel.

- The additional analyses on nickel speciation and bioaccessibility conducted for the

Revised 2001 HHRA study improved the confidence and reduced uncertainties in interpreting the results of the assessment. This is a significant contribution to the site-specific risk assessment methodology for nickel.

Major Strengths Identified by the International Peer Review Panel

The following major strengths of the Revised 2001 HHRA Study were noted by the International Peer Review Panel:

- The methods described in the report of Revised 2001 HHRA Study provided a clear, easily followed outline of how the risk assessment process is currently used by regulatory agencies to develop intervention levels of chemicals and metals in various environmental media that are considered protective of human health.

- The multi-media, multi-pathway approach followed in the Revised 2001 HHRA Study was appropriate and clearly described.

- The speciation analysis of nickel appropriately evaluated the forms of nickel present in the community, and the implications of that speciation on nickel exposure in the risk assessment.

- The assessment of bioaccessibility/bioavailability of nickel from Rodney Street soils was properly conducted. The estimate of the relative bioavailability of soil nickel *versus* soluble nickel was accurately incorporated into the risk assessment.

- The Reference Dose (R$_f$D) of 20 micrograms per kilogram body weight per day (US EPA) was considered to be appropriate and protective of human health.

- The Revised 2001 HHRA Study accurately demonstrated that oral exposure was the major exposure pathway of concern for nickel in soils and that inhalation and dermal exposure pathways had a minor contribution to the intake of total nickel from soils.

Action Items Recommended by the International Peer Review Panel

The International Peer Review Panel recommends that the following action items be addressed in the Revised 2001 HHRA Study:

1.   Oral Toxicity and Exposure Criteria

1.1   There is uncertainty in the extrapolation to humans of effects arising from nickel exposure in laboratory studies, both in the concordance of endpoints and in the determination of appropriate intake criteria. These uncertainties, and the relevance to humans of the endpoints in animal studies should be discussed in greater depth.

Response: Addressed in chapter 6 (Part B, Main document) and Appendix 2 (section A2-1).

1.2   The Review Panel considers the US EPA RfD of 20 µg Ni/kg/day appropriate for use in this study. The confidence in this RfD was increased by the findings of a recent two-generation reproduction study where rats were exposed to nickel sulphate hexahydrate (Springborn, 2001). This study addresses a key data gap in the data used for the development of the current US EPA RfD value. A preliminary RfD of 20 µg Ni/kg/day based upon the Springborn (2001) study is under review by the US EPA. These issues should be discussed in the overall strengths and uncertainty analysis of the assessment.

Response: Addressed in Appendix 2 (section A2-9.2.2 and A2-9.3.2).

1.3   Some of the toxicological endpoints attributable to nickel in laboratory studies using animals (e.g., systemic effects on b and A2-body weight) have not been evaluated in epidemiological studies of workers exposed to high levels of nickel. However, some similarities have been observed in sensitive endpoints in both laboratory animals and humans (e.g., possible adverse reproductive outcomes in female workers in the nickel refining industry and in reproduction toxicity observed in laboratory studies in animals exposed to nickel). A discussion of these issues should be included in the uncertainties section in the revised document.

Response: Addressed in Appendix 2 (section A2-1).

1.4   The rationale for application of uncertainty factors typically used in risk assessment to establish regulatory exposure criteria would be most useful in the methodology section (Appendix 2). This documentation could then be referred to in the main text of the report, and in the discussion of strengths and uncertainties of the overall assessment.

Response: Addressed in Appendix 2 (section A2-1).

1.5   All the RfDs and equivalents are expressed as µg Ni/kg/day, so a conversion to nickel sulphate is not required for the TERA value. The text in the draft report should be modified appropriately to correct this issue.

Response: This issue has been addressed in the oral exposure limit sections of Appendix 2. However, due to the way inhalation exposure limits are described, it has not been addressed for those limits. This issue is being addressed.

1.6   The existing discussion of the rationale for different regulatory exposure values by various agencies (e.g., US EPA, Health Canada, WHO) should be expanded by adding information from the Springborn (2001) study, and develop an appropriate discussion of the rationale for the selection of criteria used in the MOE's assessment.

Response: Addressed in Appendix 2 (section A2-9.2.2 and A2-9.3.2).

1.7   It should be clearly stated in the uncertainty discussion (in addition to the discussion in Appendix 2) that the EPA definition of an RfD provides a recommended exposure criteria

estimate within an order of magnitude. This uncertainty impacts the degree of confidence in the selected soil intervention level.

EPA's IRIS database provides the following definition of an RfD: "In general, the RfD is an estimate (with uncertainty spanning perhaps an order of magnitude) of a daily exposure to human population (including sensitive subgroups) that is likely to be without an appreciable risk of deleterious effects during a lifetime."

Response: Addressed in Appendix 2 (section A2-1).

2. Inhalation Toxicity and Exposure Criteria

2.1 It is recommended that inhalation risk estimates should be calculated and discussed using the EPA, WHO and Health Canada cancer potency factors for inhalation of nickel. This information will provide an estimate of the range in predicted risks based on current regulatory exposure criteria. It is important to indicate that the regulatory potency criteria are primarily developed to ensure the safety and the health of the public. The estimates of occupational exposures associated with cancers, as documented in various epidemiological studies, should be contrasted with the estimated inhalation exposures from the Rodney Street assessment. A discussion of this comparison is needed to provide a rationale for the conclusion that health risks from inhalation exposures based on the Rodney Street data are minimal.

Response: Addressed in section 5.6.2 (Part B, Main document) and Appendix 2 (sections A2-9.2.3.2 and A2-9.3.1).

2.2 The discussion in the uncertainty analysis of the cancer assessment, particularly in the estimates of exposure in the epidemiological studies used to derive the cancer slope factor, requires further discussion.

Response: Addressed in Appendix 2 (section A2-9.3.1).

2.3 Impacts of particle size on deep deposition of nickel into the lungs, resulting in higher tissue dose, requires expanded discussion. The cancer slope factor for nickel refinery dust is based on lung cancer, not nasal or total cancer. Therefore, a discussion of the issue of particle size and degree of penetration into the lungs is important.

Response: Addressed in Appendix 2 (section A2-9.2.1.1).

2.4 Using nickel concentrations in air expressed as TSP (total suspended particulate) rather than PM_{10} (particulate matter less than 10 microns in diameter) is more appropriate because the cancer unit risk values are based on nickel refinery dusts of variable sized particles, including a significant fraction of large particles. The recent, preliminary air monitoring from the Rodney Street area should be presented and discussed to demonstrate MOE's due diligence on this issue.

Response: Addressed in section 5.6.2 (Part B, Main document) and Appendix 2 (section A2-9.2.1.1).

2.5 The statement in the document that the inhalation cancer risks are 10-fold (an order of magnitude) lower than those calculated based upon the Health Canada value should be removed.

Response: Done.

2.6 Information on nickel concentrations in ambient air from other locations in Canada/North America will help put the air nickel concentration data from the Rodney Street community into context.

Response: Due to delays in verifying what sort of air sampling equipment was used and the importance of comparing air monitoring data appropriately (discussed in Appendix 2 (section A2-9.2.1.1)), this information was not ready for inclusion in the current draft. This issue is being addressed.

2.7 The Panel agreed with the following items, but recommended a more detailed discussion be added to the report for clarification.

2.7.1 The air pathway is insignificant in the systemic dose calculation. Nonetheless, this pathway was included in the current assessment.

Response: Addressed in section 4.3.3 (Main document).

2.7.2 Because the link between soil levels and ambient air concentrations cannot be reliably estimated, the cancer risk from inhalation exposure was appropriately not used to derive the soil intervention level.

Response: Addressed in section 7-1 (Main document).

3. Market Food Basket and Home Garden Data

Market Food Basket

3.1 The Review Panel recommended the use of mean intake values from the Dabeka and McKenzie (1995) data for estimating exposures to nickel from the normal diet.

3.1.1 In the Panel's view, upper bound intake values from short-term food consumption surveys may not provide an appropriate estimate of typical long-term food consumption patterns, and the MOE use of a mean value was appropriate.

Response: No action required.

3.1.2   The Dabeka data have been peer-reviewed and published, and represents the author's analysis and conclusions. However, the Panel recognized that the Dabeka data are from one study from the city of Montreal, and may not be fully representative of dietary habits of the Port Colborne population. These uncertainties should be communicated within the uncertainty section of the revised document.

Response: Addressed in section 6.2 (Part B, Main document).

3.1.3   Studies from other jurisdictions (e.g., the U.S. and U.K. market intake surveys) show lower intake of nickel from foods. This provides confidence that the nickel intake from diet would not be underestimated in this assessment.

Response: Addressed in section 6.2 (Part B, Main document).

3.2   The potential reasons for the reported differences between available studies on the Food Basket data on nickel should be discussed in greater detail, particularly the impacts of the use of different cooking utensils and vessels on metal content in foods during preparation/cooking, as well as variation in analytical laboratory methodologies and detection limits. The procedures used to address non-detect nickel concentrations reported in produce need to be investigated and discussed as appropriate in the report.

Response: Addressed in Appendix 4 (section A4-2.3). The treatment of non-detect values has not been addressed in this draft. This issue is being addressed.

3.3   Infant consumption rates for market basket foods should not be based on a proportionate adjustment of adult intake information as conducted by CEPA (1994). The Panel recommended using a more age-appropriate infant diet, as presented by the MOE in the discussion of alternatives within the draft document.

Response: Addressed in Appendix 4 (section A4-2.3). It should be noted that the CEPA infant diet is not derived from adult intake data, but is based on 1970s infant intake survey data.

Home Garden

3.4   The discussion of home grown produce consumption values needs to be expanded and the uncertainty of the various estimates presented. For example, the home-grown produce consumption rate of 9.9% of total produce that was used is higher than those observed in other jurisdictions. These issues should be addressed in the sensitivity analysis, and discussed in the section on strengths and uncertainty of the assessment.

Response: Addressed in section 6.3 (Part B, Main document) and Appendix 6 (section A6-7).

3.5   There is insufficient data to support the use of a linear regression to calculate a soil-to-plant uptake factor for nickel. Rather, the Panel recommended that the ministry re-evaluate the available data and select an appropriate upper bound value for plant uptake of nickel.

Response: Addressed in Appendix 3 (section A3-1.4).

3.6   There are a number of different fruits listed in the "vegetable" nickel analyses table (page 12 of Appendix 1). The data should be re-evaluated to determine whether fruits, in addition to vegetables, were present in the home gardens found in the Rodney Street community.

Response: Addressed in Appendix 3 (section A3-1.4).

3.7   It is likely overly conservative to have potential consumption of home garden produce, in addition to full daily consumption from market basket (i.e., background). The Panel recommends adjusting the market produce consumption rate by the home garden produce consumption, to avoid double counting and exceeding the overall typical daily fruit/vegetable consumption.

Response: Market basket intakes were not adjusted for home grown produce because the use of an upper bound plant tissue nickel concentration from all the aggregated produce types precludes a detailed accounting of the intake of each produce type from either source.

3.8   It should be noted in the report that very few of the residences in the Rodney Street area have home gardens. However, the Panel agrees with the inclusion of this pathway so that the assessment may be sufficiently conservative to enable residents to consume home grown garden produce.

Response: Addressed in Appendix 3 (section A3-1.4).

4.   Dermal Contact (Contact Dermatitis and Impacts of Oral Intake)

4.1   The discussion of nickel-induced contact dermatitis within the assessment report needs to be expanded. The discussion should emphasize that higher levels of exposure are necessary to cause sensitization to nickel than to elicit a response in a sensitized individual. This is a common feature of chemical sensitization in general. However, the available information is inadequate to estimate the magnitude of nickel exposures that could cause sensitization.

Response: Addressed in Appendix 2 (section A2-9.2.4).

4.2   The discussion of contact dermatitis should emphasize that contact of sensitized individuals with nickel-contaminated soils has the potential to cause contact dermatitis. The assessment should indicate that the available information does not enable the

identification of a soil nickel concentration that would result in contact dermatitis. Evidence of the low levels of exposure that are needed to cause contact dermatitis from other agents should be summarized to support the conclusion that protection of sensitized individuals by limiting soil nickel concentrations may not be practically achievable.

Response: Addressed in section 7.1 (Part B, Main document).

4.3   The Eastside Community Health Study, to be conducted over the next eight to ten months, will be testing people living in the Rodney Street community for nickel contact dermatitis. The results of this study may provide information on the extent and degree of concern on this issue.

Response: Not addressed in this draft. This information is not available yet.

5.   Indoor/Outdoor Dust Exposures

5.1   The MOE risk assessment treated indoor dust as comparable to outdoor soil. It would be preferable to treat indoor dust as a separate exposure media with distinct characteristics.

Response: In the absence of reliable information on metal concentrations in indoor dust, the contribution of outdoor soil to indoor dust was modeled using literature values (section A6-5.2).

5.2   The use of the 10% winter soil cover adjustment to soil ingestion rates and dermal exposures is not considered appropriate for estimating nickel exposure from indoor dust. The Panel recommends removing this adjustment, and include a discussion of the conservatism involved in using the same indoor dust exposure year-round.

Response: Done, discussed in section 6.2 (Part B, Main document).

5.3   The relative bioavailability of nickel from outdoor soil and indoor dust required additional discussion. Very little information on this topic is available. One study indicated that bioavailability of dust was slightly higher than that of outdoor soil. This issue requires further discussion in the uncertainty section.

Response: Addressed in section 6.2 (Part B, Main document).

5.4   The occurrence of indoor dust reservoirs in inaccessible areas of homes requires additional discussion. Remediation of outdoor soil may not immediately affect indoor dust reservoirs, arising from historical air emissions.

Response: Addressed in section 6.2 (Part B, Main document).

5.5   If these indoor dust reservoirs are not disturbed (i.e., extensive renovations), one would not expect indoor dust concentrations to be significantly elevated. Although events such as renovations may result in an elevation of ambient concentrations of nickel of indoor air

and dust, one would expect this to only result in the potential for short-term exposure. As these exposures would only be short-term and are expected to occur infrequently, it would be inappropriate to include in the overall risk assessment.

Response: Addressed in section 6.2 (Part B, Main document).

5.6 In the current risk assessment, indoor dust levels were considered to be 39.5% of the outdoor soil level. Available data do indicate that at sites without a significant air source of metals, indoor dust concentrations are generally less than outdoor soil concentrations. However, additional justification should be provided for the value used in the current risk assessment.

Response: Addressed in Appendix 6 (section A6-5.2).

6. Other Metals

6.1 The toxicology discussion of cadmium should reference the association between cadmium exposure and osteoporosis. Further information can be found in the following articles:

Järup, L. et al., 1999. Cadmium may be a risk factor for osteoporosis. Occup Environ Med 55:435-439.
Staessen, J.A. et al., 1999. Environmental exposure to cadmium, forearm bone density, and risk of fractures: prospective population study. Lancet 353.

Response: Addressed in Appendix 6 (section A2-5.2).

6.2 The toxicology discussion of antimony should be expanded based upon the new NRC study. The new study is considered more appropriate for exposure from environmental sources. However, antimony was not an issue using the lower exposure criteria. Therefore, the current assessment has an extra margin of safety.

Response: Addressed in Appendix 2 (section A2-2.3.1).

6.3 The section on the toxicology of lead should reference the polymorphism of delta-aminolevulinic acid dehydrogenase. Further information can be found in the following articles:

Gerhardsson, L. et al., 1999. Chelated lead in Relation to Lead in Bone and ALAD Genotype. Environ Res Section A 80:389-398.
Landrigan, P.J. et al., 2000. The Reproductive Toxicity and Carcinogenicity of Lead: A Critical Review. Am J Ind Med 38:231-243.

Response: Addressed in Appendix 2 (section A2-8.1).

7.   Bioavailability

7.1   While the Panel concluded that the bioavailability adjustments in the assessment were appropriately conducted, the documentation in Appendix 5 requires further clarification of the concept of relative bioavailability in the context of risk assessment.

Response: Done (see Appendix 2 (section A2-1).

7.2   It is recommended that future work on bioavailability should be based on unground soil samples sieved to <250 microns. This approach would ensure that particles so large that they would not adhere to skin, and thus would be unlikely to be ingested, would be removed from the assessment.

Response: Standard MOE SOP is sieving to <350 microns. Changes in our SOP will be considered in future investigations.

8.   Uncertainty Discussion

The discussion of the strengths and uncertainties of the Revised 2001 HHRA Study communicates important information to the reader on the degree of confidence in the final soil intervention level for nickel. Therefore, the International Review Panel identified the following issues that could be addressed to improve the overall discussion of uncertainty within the report.

8.1   The Panel recommends that the discussion of uncertainty and variability, and the distinction between the two, be clarified. This can be achieved by creating a table that identifies the critical parameters that have uncertainty associated with them, the magnitude/impact of the uncertainty associated with these parameters, and the rationale for the magnitude of the impacts on assessment results.

Response: Addressed in section 6.1 (Main document).

8.2   The report should present a table that identifies the critical parameters that have uncertainty associated with them, the magnitude/impact of the uncertainty associated with these parameters, and the rationale for the magnitude of the impacts on assessment results.

Response: A table indicating the impact of changing model parameters as compared with our March 2001 model is included in today's package. This could be the basis for addressing this issue.

8.3   A quantitative sensitivity analysis should be conducted to evaluate the relative impact of selected critical parameter on the overall assessment results and conclusions.

Response: See response to 8.2 above.