TECHNICAL REPORT

Assessment of Potential Health Risks of Reported Soil Levels of Nickel, Copper and Cobalt in Port Colborne and Vicinity

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EXECUTIVE SUMMARY

In 1991 the Phytotoxicology section of the Ontario Ministry of the Environment conducted soil and vegetation surveys in the vicinity of the International Nickel Company (INCO) refinery facility in Port Colborne Ontario. These surveys were part of an on-going assessment carried out in this area in response to local concerns over emissions of nickel and other metals from the INCO facility. Analyses of surface soil samples (0-5cm depth) showed that the levels of nickel, copper and cobalt exceeded the Phytotoxicology *Upper Limit of Normal* guidelines (ULN) at many of the 37 sites examined.

As a result of these findings an evaluation of health effects was undertaken to determine whether there is any evidence of risk to human health associated with exposures to nickel, copper or cobalt, in the soils of the Port Colborne area. This was done in two ways. Firstly, a site-specific risk assessment was undertaken to determine the potential health effects which may result from current exposures in the Port Colborne area. Secondly, population health was evaluated to determine if there is any evidence of increased disease incidence in the Port Colborne area compared with the rest of Ontario.

The site-specific risk assessment provided estimates of intakes for all routes of exposure (inhalation, oral and dermal) and integrated these into a total multi-media exposure estimate. Since the primary concern is the levels of metals in the soils, this assessment focused on exposures from soils and dusts and from the consumption of backyard garden produce. Inhalation and drinking water exposures were found to be minor components (less than 1%) of the total exposure for all three metals. Summaries of total exposure and potential health risks for nickel, copper and cobalt are provided below.

There was no evidence of an elevation in the rate of reproductive failure or birth defects in the Port Colborne population, compared with the Ontario population. The most recent cancer incidence rates for all sites and both sexes were within expected limits. However, a greater than expected number of lung cancer cases were observed amongst Port Colborne males in the time period 1979-1983. This excess may be related to earlier occupational exposures to lung carcinogens or other factors associated with lung cancer.

Nickel:

Estimates of chronic nickel exposure ranged between 352 and 1240 µg/day for children and 348 and 690 µg/day for adults for people using a municipal drinking water supply. For these people, the combined lifetime averaged chronic daily intake (CDI) calculated for the maximum estimated intake was 17 µg/kg/day. For people who rely on well water as a drinking water supply, estimates of chronic nickel intake based on the highest reported well water nickel levels, range between 379 µg/day and 1270 µg/day for children and 438 µg/day and 780 µg/day for adults. The CDI for this latter group was 18.5 µg/kg/day. Both of these CDI values are lower than the United States Environmental Protection Agency (USEPA) lifetime averaged exposure reference dose (RfD) of 20 µg/kg/day, which is defined as the dose below which, exposure averaged over a lifetime, is unlikely to result in adverse health effects.

Comparison of intakes between children and adults, on a body weight basis, showed that at maximum soil nickel levels, children receive a dose which is approximately ten-fold greater than that of adults. At lower soil nickel levels, the difference is about four-fold. These differences in dose are due to differences in body weight (15 kg for a child and 70 kg for an adult) and soil consumption (80 mg/day for a child and 20 mg/day for an adult). No indication that children exhibit age-specific health effects relating to nickel exposure could be located in the literature. Also, given that nickel is an essential dietary element, the tolerance range is likely to be broad and the levels required to produce toxic effects are likely to be high. In addition, the RfD value suggested by the US EPA incorporates a large margin of safety into the 20 µg/kg/day value. The maximum CDI values calculated for the Port Colborne area are below this US EPA value. Based on this information, it is unlikely that exposure to nickel in the soils from the Port Colborne area will result in adverse health effects in children or adults.

Copper:

Estimates of maximum total exposure to copper for children and adults in the Port Colborne area are 878 and 2084 µg/day, respectively. Both of these values fall below the National Academy of Sciences *Estimated Safe and Adequate Daily Dietary Intake* (ESADDI) ranges of 1000 to 1500 µg/day for children and 2000-3000 µg/day for adults, the FAO/WHO acceptable maximum daily intake of 500 µg/kg/day for the general population (this would be an intake of 7500 µg for a 15 kg child) and the allowable daily intake (ADI) value of 2650 µg/day suggested in the U.S. EPA Drinking Water Criteria Document. Based on this information, exposure to copper in the soils in the Port Colborne area is not expected to pose any appreciable risks to area populations as total multi-media intakes are below current exposure limits.

Cobalt:

Estimates of maximum total exposure to cobalt for children and adults in the Port Colborne area are 333 and 375 µg/day, respectively. Data on cobalt exposure from food is highly variable and ranges between 300 µg/day and 1800 µg/day. Exposures to cobalt in the Port Colborne area do not appreciably alter the total exposures. Thus, excess exposures are not expected at the environmental concentrations which exist in the Port Colborne area. On the basis of this assessment, no adverse health effects are anticipated as a result of cobalt exposure in Port Colborne soils.

In conclusion, 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. Furthermore, the review of population health data did not indicate any adverse health effects which may have resulted from environmental exposures.

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1.0 INTRODUCTION

In 1991 the Phytotoxicology Section of the Ontario Ministry of the Environment conducted soil and vegetation surveys in the vicinity of the INCO facility in Port Colborne Ontario. These surveys were part of an on-going assessment carried out in this area in response to concerns over emissions of nickel and other metals from the INCO facility. The results from these surveys are contained in a separate report (MOEE 1994). Analyses of surface soil samples (0-5 cm depth) showed that the levels of nickel, copper and cobalt exceeded the Phytotoxicology *Upper Limit of Normal* guidelines (ULN) at many of the 37 sites examined. The levels of nickel exceeded the ULN value by the greatest margin and at the largest number of sites (36 of 37). The difference between the maximum reported concentrations of copper and cobalt and their respective ULN values were smaller than that for nickel, and exceedances occurred at fewer sites (18 and 16 sites of 37 for copper and cobalt respectively).

As a result of these findings an evaluation of health effects was undertaken to determine whether there is any evidence of risk to human health associated with exposures to nickel, copper or cobalt, in the soils of the Port Colborne area. This was done in two ways. Firstly, a site-specific risk assessment was done to estimate the potential health effects which may result from current exposures in the Port Colborne area. Secondly, population health was evaluated to determine if there is any evidence of increased disease incidence in the Port Colborne area compared with the rest of Ontario.

The site-specific risk assessment makes use of environmental monitoring data and recent toxicological information to estimate exposures and potential health effects. It examines current toxicological information 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 modelling to estimate the total exposures 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, dose-response assessment, exposure assessment and risk characterization have been described in detail in a previous health risk assessment report which evaluated potential health risks associated with exposures to various metals in the soils in the Port Hope area (MOE 1991). The risk assessment and the conclusions for nickel, copper and cobalt are contained in sections 2, 3 and 4 of this document.

Population health effects which may have occurred were evaluated by examining population data for evidence of elevated incidence of cancer and adverse reproductive outcomes within the Port Colborne population. The evaluation and conclusions are contained in Section 5.

1.1 Sources of Information

The hazard identification and the dose-response information were taken from the work prepared for the "*Assessment of Human Health Risk of Reported Soil Levels of Metals and Radionuclides in Port Hope*" (MOE 1991). Additional toxicological information for nickel came from a recent scientific review of nickel toxicity (MOEE, 1994b).

The exposure assessment models for estimating intakes from air, drinking water and soils and dusts were the same as those used in the Port Hope Assessment (MOE, 1991). The equations used to estimate these intakes can be found in the appropriate sections of the individual assessments. This report differs from that prepared for Port Hope in that the contributions to intakes made by the consumption of backyard garden produce grown in the Port Colborne area are considered in the estimates of total intakes. The equations used to model these exposure components, the exposure scenarios considered and the assumptions upon which they rest are described in detail in a *Technical Report* prepared by the MOEE (MOEE, 1994c). A brief summary is provided below.

Information on the uptake of nickel, copper and cobalt by various vegetables was provided by the Phytotoxicology Section of the Standards Development Branch of the MOEE. This contained plant uptake data specific to the Port Colborne area.

The levels of the three metals in Port Colborne drinking water were taken from 1990-1994 averaged levels detected in the Port Colborne system under the MOEE *Drinking Water Surveillance Program*. Residents living outside the area supplied with municipal water obtain drinking water either from cisterns or wells. Metals levels data were available for 6 residential wells in the Port Colborne vicinity. The levels of copper and cobalt in these wells were comparable to those reported in the municipal drinking water supply. Therefore, for these metals, drinking water intake estimates have been based solely on municipal drinking water data. The levels of nickel in four of the six tested wells exceed those found in the municipal drinking water supply. The highest reported level was used to estimate drinking water intakes from well water supplies. Air monitoring data for the Port Colborne area was available only for nickel.

1.2 Consumption of Backyard Garden Produce: Exposure Models

Estimating the intakes of contaminants from home-grown fruits and vegetables requires two pieces of information;

1) the concentration of the contaminant in the fruit/vegetable on a fresh weight basis.
2) an estimate of the consumption rate for backyard garden produce.

From this information, intakes from backyard garden produce can be estimated using the following equation;

Eq 1 $$Intake = C_{food} \times IR$$

where C is the concentration of the contaminant in the food on a fresh weight basis (mg/kg) and IR is the produce consumption rate in kg/day. Contaminant concentrations in vegetables are frequently reported on a dry weight basis. These values must be converted to fresh weight values by using conversion factors. The conversion factors used in this report are provided in the intake estimate tables for each metal.

Estimating the consumption rate of backyard garden produce requires the use of a number of assumptions about garden size, crop yield and the percentage of the total diet that the consumption of backyard produce represents. A detailed derivation of the values used for each of these

assumptions is provided in a technical report prepared by the MOEE (MOEE, 1994c), and will not be provided herein. The assumed values are listed in table 1.1. These values were used to estimate daily intakes of metals from backyard produce and total daily intakes.

Table 1.1 Assumptions and Values Used in Estimating Intake Rates of Backyard Garden Produce

Assumption	Value	
Garden Size	30.0 m^2	
Crop yield	1.40 kg/m^2	
	Child	Adult
Yearly Averaged Consumption of Backyard Garden Produce	26.0 g/day	34.0 g/day
Percent of Total Daily Food Intake	1.8%	2.2%

Intake estimates from backyard garden produce were calculated using two different exposure scenarios (models). These models and the assumptions and equations used by each are described in detail in the MOEE technical report (MOEE,1994c). A brief summary of each model is provided below.

Model 1: Chronic Exposure Model

The chronic exposure model uses metal concentrations in a single type of vegetable to represent the concentration in all vegetables. Usually, the vegetable with the highest metal concentration is selected as the surrogate. This model also uses a yearly averaged consumption rate for backyard produce as an estimate of daily intakes. In deriving the value it is assumed that backyard garden produce contributes only a portion of the total daily vegetable consumption and that this consumption occurs at a fixed rate every day of the year. The value is derived by dividing the total yearly consumption of backyard garden produce by 365 days/year to produce the yearly averaged daily consumption rate. The values calculated for children and adults are 26.0 g/day and 34.0 g/day respectively.

Model 2: Acute Exposure Model

The acute exposure model is used to account for days when all of the vegetables consumed come from backyard gardens. This is most likely to occur during the latter part of the growing season when backyard produce will be most available. The model uses metal concentrations in a single type of vegetable to represent the concentration in all vegetables. Usually, the vegetable with the highest metal concentration is selected as the surrogate. The vegetable consumption values used in this model are 98 g/day for children and 326 g/day for adults.

Estimates of exposure derived from Model 1 are based on an assumed garden size and yield (see table 1.1). The consumption of this yield is averaged over 365 days/year. Model 2 differs from Model 1 in that it is independent of garden size or yield.

Both of these models have been used to estimate intakes for backyard garden produce for each of the three metals under consideration. These values have also been used in the estimation of total exposures. The exposure estimates derived for each of the metals are contained in sections 2, 3 and 4.

2.0 NICKEL

2.1 HAZARD IDENTIFICATION

2.1.1 Pharmacokinetics

The absorption of nickel by an organism is largely dependent on the solubility of the nickel compounds. Soluble salts such as nickel chloride or nickel sulphate are more readily absorbed by all routes of exposure than insoluble nickel compounds such as nickel sulphide or nickel oxide. Elemental nickel (Ni) may exist in oxidation states which range between -1 and +4, but the most prevalent in the environment is Ni^{+2} (NiII).

The contributions to nickel intakes from the gastrointestinal tract from utensils and equipment used in food processing and preparation can be quite high. However, the absorption of soluble nickel compounds from the gastrointestinal tract is only 2 to 5 percent, although it may be higher on an empty stomach. Bioavailability also appears to be dependent upon dietary composition. For example, in humans, nickel (as nickel sulphate) absorption from drinking water has been reported to be forty-fold greater than that from food.

Nickel is inhaled in the form of particulate-bound salts or as aerosols. The pulmonary deposition pattern of inhaled nickel compounds varies with the particle size. Particles larger than 2 μm in diameter usually remain in the upper respiratory tract. Smaller particles are distributed throughout the lower respiratory tract. The amount of particulate nickel absorbed from the pulmonary tract is a function of both the solubility and the physical form of the particles. Insoluble particulate nickel is absorbed only very slowly, while soluble nickel salts are cleared rapidly (rates vary between several hours to a few days).

From the site of absorption, nickel is carried throughout the body by the blood. Nickel has been found to bind to serum albumin and macroglobulins. Albumin is the primary macro-molecular carrier of nickel in man and other species. Regardless of the route of exposure, most nickel is rapidly transferred to the kidney where it is excreted in the urine. Biliary excretion of nickel is negligible in animals and it is not known if it occurs in humans. As a result of the low absorption, faecal elimination of nickel is roughly equivalent to the dietary intake. Animal studies suggest that the biological half-life of absorbed nickel is on the order of days. There is little evidence for the tissue accumulation of nickel once exposure has stopped.

Age dependent accumulation appears to occur only in the lung. This can be explained by an active phagocytic mechanism for insoluble or only slightly soluble nickel compounds. Once inside the cells, the particles form an intracellular source of bioavailable nickel ions. The active phagocytic uptake appears to be related to particle size and to the surface charge (negatively charged particles are phagocytized while positively charged particles do not appear to be).

Placental transfer has been demonstrated experimentally in rodents, and there is evidence that NiIII is able to cross the placental barrier in humans.

2.1.2 Toxicology

The toxicology of nickel and nickel compounds has been previously reviewed in detail (Stokinger, 1981a; MOL, 1986; ECETOC, 1989; IARC, 1990a; ASTDR, 1987). The information presented in this section is drawn from these reviews. Nickel is an essential nutrient in several species and may, in minute amounts, have essential biochemical functions in humans, although this has not been established conclusively. Nickel deprivation in mammals has an adverse effect on body weight, on reproductive capability and on the viability of offspring and it induces anemia through reduced absorption of iron. Nickel also appears to be required in several proteins and enzymes (MOL, 1986).

Absorbed nickel affects the immune system. Sensitization for contact dermatitis occurs as a result absorption through the skin of soluble nickel compounds which are released through the corrosion by sweat of objects made of nickel metal and alloys. A large fraction (10-20%) of the female population is sensitized during the teen years as a result of daily skin contact with objects made from nickel alloys. Sensitization may also occur from occupational exposures. Provocation of dermatitis (hand eczema) occurs through additional dermal contact and is also possible after ingestion.

Respiratory allergy in the form of asthma can result from occupational exposures to aerosols from nickel plating or polishing of nickel alloys or to welding fumes.

Nickel has been shown in experimental studies to cause immunotoxic effects, such as increased infectiousness, decreased natural killer-cell activity, inhibition of interferon production and interference with functions of the complement system. Although some of these effects have been demonstrated at concentrations likely to occur under occupational exposure conditions, the health significance of these findings is difficult to evaluate. It has been speculated that, since the immune system has important functions in the defence against foreign cells, its weakening could possibly be related to the increased occurrence of respiratory cancers in occupational settings.

There is no information on reproductive effects in humans, even at occupational doses. However, experimental studies with animals have demonstrated effects at doses that are higher than the likely human exposures. Reduced fertilization rates has been found in male mice, and decreased birth weight, reduced number of live pups per dam and increased number of neonatal deaths have been observed with female mice and rats. Significant embryotoxicity and malformations were found when mice were injected intra-peritoneally.

Nickel does not appear to be a mutagen in bacteria or phages but is possibly a mutagen in mammalian cells, however, the experimental data are of poor quality. It may also cause DNA lesions. Experimental results from animals provide doubtful evidence for chromosomal damage *in vivo*. There is weak positive evidence for chromosomal damage in exposed workers. Increased rates of sister chromatid exchange (SCE) have been found *in vitro* but not in exposed workers.

Studies of cellular transformations *in vitro* are almost all positive, and epithelial changes have been found in exposed workers. Manganese appears to prevent such transformations *in vitro*. The transformations are enhanced if organic carcinogens are added together with the nickel. Cigarette smoke appears to have a synergistic effect.

There is no evidence that ingestion of nickel causes cancer in humans. In the single study (Schroeder et al., 1964) when nickel was administered orally in drinking water, the tumour frequency in exposed animals was less than in the controls. No tumours developed when the mucosa of the cheek pouches of hamsters were painted with nickel subsulfide (Ni_3S_2) (Sunderman et al., 1978). However, there are numerous rodent studies which show that nickel compounds are carcinogenic via ingestion and that the potential varies for different compounds. The experiments suggest that the NiII ion is the ultimate carcinogen (MOL, 1986).

Inhalation of finely powdered nickel metal by rats and hamsters produced carcinogenic effects in the upper pulmonary tract, but the results were negative with nickel oxide. Epidemiological studies indicate that various forms of nickel cause cancer of the nasal cavities and lungs of exposed workers. The evidence is not clear-cut, but exposure to a mixture of oxidic and sulfidic nickel at very high concentrations causes cancer. Exposure to oxidic nickel at large concentrations in the absence of sulfidic nickel is also associated with an increased risk of lung and nasal cancer. There is evidence that soluble nickel increases the cancer risks and may enhance risks associated with exposure to less soluble forms. There is no evidence that metallic nickel causes lung or nasal cancers, and there is no substantial evidence that occupational exposures to nickel in any form are likely to cause cancer in other parts of the body. The available evidence suggests that respiratory cancer risks are primarily related to exposures to soluble nickel at concentrations >1 mg nickel/m^3 and to exposures to less soluble forms at concentrations >10 mg nickel/m^3 (ICNC, 1990).

IARC (1990) concluded that nickel compounds such as nickel sulphate (and the combinations of nickel sulphides and oxides encountered in the nickel refining industry) are carcinogenic to humans as there is sufficient evidence that they cause lung and nasal cancer. Metallic nickel is possibly carcinogenic to humans although there is only inadequate evidence in humans. The evidence of carcinogenicity in animals is sufficient for metallic nickel and nickel sulphides and limited for nickel alloys and some nickel salts.

2.2 DOSE-RESPONSE INFORMATION/CURRENT EXPOSURE LIMITS

The U.S. EPA has developed an oral reference dose for non-carcinogenic effects of 20 µg/kg/day (lifetime dose). It is believed that a daily exposure at this level is without an appreciable risk of deleterious effects to the human population, including sensitive subgroups, during a lifetime. The RfD is based on a No Observable Adverse Effects Level (NOAEL) found in a study where rats were fed a diet containing 100 µg nickel salts/g (equivalent to 5 mg/kg/day). The results were decreased body and organ weights. An uncertainty factor (UF) of 100 was multiplied by a modifying factor of 3

because of certain inadequacies in the study and divided into the NOAEL to derive the RfD (U.S. EPA, 1991).

Ontario has not set an objective for nickel in drinking water. In 1985 the U.S. EPA set a lifetime Adjusted Acceptable Daily Intake (AADI) of 350 µg/L for nickel in drinking water. This is equivalent to a daily intake of 700 µg/day, assuming a consumption of 2 L/day of water. The AADI is based on the same study as the 1988 RfD value of 20 µg/kg/day, which translates into an intake of 1400 µg/day for a 70 kg person. In the 1985 calculations, the ADI was divided by an uncertainty factor of 100 and multiplied by a further factor of 0.2, to account for an assumed difference in absorption of nickel in water versus milk to allow for the fact that the nickel was not given to the rats in drinking water.

Ontario has set an interim air quality standard (24 hour averaging period) of 2 µg nickel/m^3, with a limiting effect based on vegetation effects and soiling.

The U.S. EPA has estimated that the incremental unit risk for lung cancer due to lifetime occupational exposure to nickel refinery dust containing 1 µg nickel/m^3 is 2.4×10^{-4}, based on epidemiological data. This value is the median of the range of risk estimates from 1.1×10^{-5} to 4.6×10^{-4}, based on studies from different refineries. The quantitative unit risk estimate for Ni_3S_2, which is the most carcinogenic nickel compound in animal studies, is twice that for nickel refinery dust because it makes up about 50% of the refinery dust.

2.3 HUMAN EXPOSURE ASSESSMENT

Although speciation data for nickel in soil were not found in the literature, nickel may exist as minerals (chiefly ferromagnesium minerals or precipitates) and as free ion chelated metal complexes in soil solution or adsorbed on surfaces. A lowering of the pH will release nickel from the soil. As pH increases, nickel adsorption by iron and manganese oxides increases, and at pH values greater than 9, nickel carbonate or hydroxide may precipitate (Adriano, 1986).

NiII, which is the predominant form in fresh waters at pH 5 to 9, forms stable complexes in water with inorganic and organic ligands. It is also associated with iron and manganese oxides. Therefore, generally >95% of nickel in rivers and about 40% in lakes is in particulate form. About 40% of the total nickel in water is bioavailable (Moore and Ramamoorthy, 1984).

Nickel in air is in particulate form. The predominant forms appear to be nickel sulphate (from combustion of fossil fuels, the predominant source), complex oxides of nickel and other metals, chiefly iron, nickel oxide, and, in small quantities, metallic nickel and Ni_3S_2 (IARC, 1990a; U.S. EPA, 1986).

2.3.1 Estimated Intake from Individual Sources

2.3.1.1 Air

Ambient air monitoring data for 1993 from the Port Colborne area shows that the geometric mean concentration for nickel in total suspended particulate was 0.013 µg/m^3. This concentration was used to estimate the exposure to inhaled nickel.

Table 2.3-1: Estimated Nickel Intakes From Air

	Estimated Air Intakes (µg/day)	
	Child (1-6 years)	Adult
Average Level (ng/m^3)	0.013	0.013
Volume Inhaled (m^3/day)	5.00	22.0
Estimated Intake (µg/day)	0.065	0.29

2.3.1.2 Drinking Water

The drinking water concentrations of nickel, based on treated water from the Port Colborne treatment plant for 1990-1994 averaged 1.2 µg/L. Residential tap water data for nickel are unavailable. Data from standing samples in the distribution system averaged 1.6 µg/L over the same period. This latter value, which likely represents the highest nickel levels, was used to estimate nickel drinking water intakes. Residents who live outside the area supplied with municipal water obtain their drinking water from cisterns or wells. Nickel levels data are available for six residential wells in the Port Colborne vicinity. Reported levels ranged between < 2 µg/l (2 of 6 wells) to a maximum of 46.2 µg/l. The highest value was used to estimate nickel intakes from drinking water from a well water supply.

Metals in well water have the potential to contribute to soil levels and vegetable uptakes if the water supply is used to irrigate backyard gardens. However, for the purposes of this assessment it has been assumed that watering backyard gardens from a well water supply is an historic and on-going practice. Thus, recent soil levels data which provide total soil levels will include the contributions made to soils from watering activities. Estimates of intake from the well water to soil to human pathway and the well water to soil to plant to human pathway will already have been accounted for in the estimates of intake from soils and dusts (sections 2.3.1.3) and from backyard produce

(2.3.1.4.2).

Table 2.3-2: Estimated Nickel Intakes from Drinking Water

	Drinking Water Intake µg/day			
	Child (1-6 years old)		Adult	
Source of Supply	Municipal	Well	Municipal	Well
Assumed Tap Water Concentration (µg/L)	1.60	46.0	1.60	46.0
Daily Consumption L/day	0.60	0.60	2.00	2.00
Estimated Intake µg/day	0.96	28.0	3.20	92.4

2.3.1.3 Soil and Dust

Sampling of surface soils (0-5cm) from the Port Colborne area was done in July of 1991. Nickel levels in the 37 samples taken ranged from 36 µg/g to 9800 µg/g. This large range in concentrations makes it difficult to determine an overall average which will adequately represent the entire group. To provide soil level intake estimates which would be of greater utility predicting exposures at each site, the available data were grouped into three concentration ranges (see table 2.3-3). Average nickel concentrations were determined for each of these ranges and these values were used to estimate nickel intakes from soils and dusts and from backyard produce (see Section 2.3.1.4.2). The estimated nickel intakes from soils and dusts for each of the concentration ranges examined as well as for the minimum and maximum reported nickel levels are shown in table 2.3-3.

Soil nickel levels exceeding 20,000 µg/g have been documented on private property immediately east of INCO in undisturbed surface soil in rural woodlots. These soil concentrations are much higher than adjacent residential and agricultural properties because of the specific soil and site characteristics. Primarily, the sites (rural) have not experienced significant disturbance, therefore, the surface soil has remained relatively intact. Nickel from atmospheric deposition from INCO settled on the surface and accumulated in the upper soil layers, resulting in the highest concentration in the top 5 cm of soil. With no surface soil disturbance, the nickel continued to accumulate with time. Agricultural soil the same distance away from INCO, and therefore exposed to the same amount of nickel deposition, usually has surface soil nickel concentrations that are 1/4 to 1/2 of those found on the woodlots. Agricultural soil is continuously cultivated to about 15 cm, which mixes the more contaminated upper soil layer with the less contaminated lower layer, diluting the overall nickel concentration. Another

reason for the higher soil nickel concentrations in the woodlots is the abundance of organic matter in the surface layer of the forest soil. The top few centimetres of the forest soil is very rich in leaf litter and related organic matter, which can bind with metal ions and slow down the normal downward leaching, thereby enhancing the rate of accumulation of nickel in the upper layer. In addition, tree foliage, will absorb nickel from the air. When these leaves fall to the forest floor and decompose, the foliar nickel is released, adding to the nickel in the surface soils which was deposited directly from the air.

Table 2.3-3: Estimated Nickel Intakes From Soils and Dusts

Soil Ni Concentration Ranges (µg/g)	[Ni] Range Averages	Estimated Intakes (µg/day)	
		Child (1-6 years)[1]	Adult[2]
[Ni] < 100	67.0	5.4	1.30
100 ≤ [Ni] <1000	398	31.8	7.96
[Ni] ≥ 1000	4290	343	86.0
Minimum	36	2.88	0.72
Maximum	9750	780	195

1 Based on a total soil intake of 80 mg/day
2 Based on a total soil intake of 20 mg/day

These highly contaminated forest soils are restricted to a few locations immediately adjacent to and downwind (east) of INCO. They are privately owned and are therefore not publicly accessible. In addition, they cannot be used for private or commercial crop production unless the trees are removed and the soil cultivated. This would result in a substantial dilution of the soil nickel contamination. For these reasons, these very high soil nickel concentrations were not used in the risk assessment.

2.3.1.4 Food

2.3.1.4.1 Food: Non-Home Sources

There is little information on average daily intakes of nickel available in the literature. A 1974 study showed that the Canadian intake of nickel ranged from 374 to 546 µg Ni/day with a mean value of 460 µg/day (Adriano 1986), This value is above the upper limits which have been cited for other countries (ICRP, 1984; MOL, 1986; IARC, 1987) which include:

- USA: 160 µg/day
- Italy: generally <300 µg/day
- Denmark: 155 µg/day
- USSR: 290-500 µg/day

Nickel can also be leached from stainless steel utensils, especially at low pH. Food processing, such as the milling of flour and catalytic hydrogenation of fats and oils with nickel catalysts also can add nickel. Intakes may reach as high as 1,000 µg/day. The best dietary data for the U.S. indicates that a value of 350 µg/day is a reasonable estimate of dietary intake. Therefore this value will be used to estimate nickel intakes for food from other sources. Backyard produce can account for up to 1.8 percent of the total daily food intake for children and 2.2 percent for adults (MOEE, 1994c). Thus, food from other sources accounts for 98.2 percent of the daily intake for children and 97.8 percent for adults. Therefore, the value of 350 µg/day is adjusted by a factor of 0.982 for children and 0.978 for adults to allow for the incorporation of backyard produce intake estimates into the total daily intake estimates. The values used for food from other sources are (350 µg/day x 0.982) = **344 µg/day** for children and (350 µg/day x 0.978) = **342 µg/day** for adults.

2.3.1.4.2 Food: Backyard Produce

Nickel levels in backyard produce will depend on the soil nickel concentration, the soil characteristics and on gardening practices. Data on the uptake of nickel is available for celery leaf and stalk and for lettuce (MOEE, 1994). The data come from greenhouse and field experiments conducted in 1983/84 at the MOE Phytoxicology facility in Brampton, Ontario. The plants used in these studies were grown in soils from the Port Colborne area. Thus, the uptake data should be representative of the uptake rates which could be expected in the Port Colborne area. Unfortunately, the nickel concentrations in the experimental soils did not match the nickel levels in any of the 37 samples collected during the 1991 survey nor the average concentrations for each of the ranges considered. Therefore intakes from backyard produce were estimated by using uptake data from experimental soil nickel concentrations closest to the value being considered. The nickel concentration in the soils for each of the plant uptake data are shown in table 2.3-4.

Intake estimates have been made for both children and adults at each of the identified soil nickel levels, for each of the vegetables identified and for both of the exposure scenarios considered (See Tables 2.3-5 and 2.3-6).

In order for nickel (and other contaminants) to be taken up from the soil and accumulate in plant tissue the metal must be free to move in the soil water. Certain soil characteristics effectively "bind" metals to soil particles and therefore inhibit bioavailability. Generally, this "binding capacity" increases as soil organic matter, cation exchange capacity and particularly pH increase. Therefore, increases in soil nickel concentrations are not necessarily followed by proportionate increases in plant nickel uptake. This can be seen with the uptake data for copper (see Section 3.3.1.4.2), where increases in

soil levels from 30 µg/g to 600 µg/g do not appreciably alter the levels of copper in the plants.

The soil characteristics in the Port Colborne area vary considerably. The area of commercial vegetable production has an *organic-type* soil whereas the residential community has a *mineral-type* soil. Mainly because of the low pH and moderate ion-exchange capacity, the organic soil would likely have a poorer binding capacity and therefore, more nickel would be available for plant uptake, compared with the mineral soils of the residential gardens, which would tend to bind more nickel. Since most of the uptake data for the risk assessment were derived from the Phytotoxicology experiments which used (mostly) organic soils, the estimates of nickel uptake by plants grown in residential gardens may be slight overestimates. Further, soil nickel concentrations greater than 1,000 µg/g can cause injury to sensitive species of garden crops, such as celery. Celery is grown commercially in Port Colborne and some of the farms have nickel contaminated soils. Nickel toxicity symptoms include generally stunted growth, dark brown-to-black water soaked lesions on the foliage that is noticeably cupped or curled upwards around the margins and chlorotic in colour. In addition, yields are lower because of poorer plant growth and because stress due to metal toxicity predisposes the plants to insect and disease organisms. These symptoms are obvious, and therefore the plants are less likely to be consumed because they are unappealing to consumers. This has two implications. First, the home gardener may actually eat less of the contaminated produce because there is less of it, or because it looks less appetizing. Second, commercial production would be less profitable because of lower yields, greater losses, and difficulty in obtaining markets for obviously injured produce.

Table 2.3-4: Averaged Soil Nickel Levels and Corresponding Soil Nickel Levels from Plant Uptake Data

Soil Ni Concentration Range (µg/g)	[Ni] Range Average (µg/g)	Nickel Levels Used in Plant Uptake Studies (µg/g)
[Ni] < 100 (*LOW*)	67.0	35.0
100 ≤ [Ni] < 1000 (*MIDDLE*)	398	920
[Ni] > 1000 (*HIGH*)	4290	5000
Minimum	36.0	35.0
Maximum	9750	5000

Based on this information, it is unlikely that using plant uptake data from lower nickel concentrations to estimate intakes at the low and high ranges will appreciably underestimate intakes from backyard produce. The estimates derived from this data should adequately represent expected intakes from these areas.

Table 2.3-5: Estimated Nickel Intakes From Backyard Produce; Children

Soil Level (µg/g)		Vegetable	Ni Level in Vegetable (µg/g)			Estimated Daily Intake (µg/day)	
Pt. C[1]	Exp[2]		Dry	C.F.[4]	Fresh	Model 1 25 g/day	Model 2 98 g/day
Low	35.0	Celery L[3]	1.00	0.059	0.059	1.53	5.78
		Celery S	1.00	0.059	0.059	1.53	5.78
		Lettuce	1.00	0.045	0.045	1.17	4.41
Middle	920	Celery L	18.0	0.059	1.06	27.6	104
		Celery S	4.00	0.059	0.236	6.14	23.1
		Lettuce	4.00	0.045	0.180	4.68	17.6
High	5000	Celery L	75.0	0.059	4.43	115	434
		Celery S	23.0	0.059	1.36	35.3	133
		Lettuce	41.0	0.045	1.85	48.0	181
Minimum	35.0	Celery L	1.00	0.059	0.059	1.53	5.78
		Celery S	1.00	0.059	0.059	1.53	5.78
		Lettuce	1.00	0.045	0.045	1.17	4.41
Maximum	5000	Celery L	75.0	0.059	4.43	115	434
		Celery S	23.0	0.059	1.36	35.3	133
		Lettuce	41.0	0.045	1.85	48.0	181

1) Nickel concentrations in Port Colborne soils
2) Nickel concentration in soils from experimental plant uptake studies
3) L = Leaf; S = Stalk
4) Dry to Fresh Weight Conversion Factors

2.4 RISK CHARACTERIZATION

The estimated total daily intakes of nickel from all sources for children and adults using a municipal

drinking water supply are shown in tables 2.4-1 and 2.4-2 respectively. Total exposures using the maximum reported well water nickel concentration are provided in table 2.4-3 and 2.4-4. All exposure estimates are provided for both backyard garden produce exposure scenarios, at each of the soil concentration ranges considered as well as for the minimum and maximum reported nickel soil concentrations. The data show that for both children and adults, the consumption of celery leaf produces the highest estimated exposures at each of the nickel concentrations examined. Therefore, the estimations of risk have used these exposures only.

Table 2.3-6: Estimated Intakes From Backyard Produce; Adult

Soil Level (µg/g)		Vegetable	Ni Level in Vegetable (µg/g)			Estimated Daily Intake (µg/day)	
Pt. C[1]	Exp[2]		Dry	C.F.[4]	Fresh	Model 1	Model 2
Low	35	Celery L[3]	1.00	0.059	0.059	2.01	19.2
		Celery S	1.00	0.059	0.059	2.01	19.2
		Lettuce	1.00	0.045	0.045	1.53	147
Middle	920	Celery L	18.0	0.059	1.06	36.1	346
		Celery S	4.00	0.059	0.236	8.02	76.9
		Lettuce	4.00	0.045	0.180	6.12	58.7
High	5000	Celery L	75.0	0.059	4.43	150	1442
		Celery S	23.0	0.059	1.36	46.1	442
		Lettuce	41.0	0.045	1.85	62.7	601
Minimum	35	Celery L	1.00	0.059	0.059	2.01	19.2
		Celery S	1.00	0.059	0.059	2.01	19.2
		Lettuce	1.00	0.045	0.045	1.53	14.7
Maximum	3800	Celery L	75.0	0.059	4.43	150	1442
		Celery S	23.0	0.059	1.36	46.1	442
		Lettuce	41.0	0.045	1.85	62.7	601

1) Nickel concentrations in Port Colborne soils
2) Nickel concentration in soils from experimental plant uptake studies
3) L = Leaf; S = Stalk
4) Dry to Fresh Weight Conversion Factors

The highest estimates of exposure come from model 2 which simulates exposures where the daily vegetable consumption consists primarily of backyard garden produce (see model 2 in tables 2.4-1 through 2.4-4). Exposures of this nature are likely to occur for a limited number of days a year, and thus, they may be considered to represent acute exposures. The maximum calculated exposures, for children and adults, are 1559 µg/day and 1982 µg/day respectively when a municipal drinking water supply is used (see table 2.4-1 and 2.4-2). When the drinking water is taken from a well, these estimates rise to 1590 µg/day and 2070 µg/day for children and adults respectively (see table 2.4-3 and 2.4-4). There is little information available on the effects of acute nickel exposures on human health available in the literature. The Agency for Toxic Substances and Disease Registry (ATSDR), which provides a summary of available data lists several acute toxic effects on the gastrointestinal tract (vomiting and cramps), renal system (increased albumin in the urine) and on the muscles (increased muscle pain). The lowest observable adverse effect levels (LOAEL) reported for these effects was approximately 7100 µg/day. One death was also reported following the ingestion of a single large dose (570,000 µg) of nickel sulphate (ATSDR, 1993). The total exposure levels in the Port Colborne area for both children and adults are significantly below these LOAEL levels when municipal or well water supplies of drinking water are considered. Therefore, it is unlikely that the occasionally high exposures which may occur through the consumption of larger amounts of backyard garden produce, will result in any adverse health effects.

To estimate potential health risks arising from chronic exposures, the chronic exposure values (model 1, celery leaf) in tables 2.4.1 through 2.4-4 have been compared with the oral RfD of 20 µg/kg/day developed by the U.S. EPA (U.S. EPA, 1991). The U.S. EPA RfD is defined as the dose below which, exposure, averaged over a lifetime, is unlikely to result in adverse health effects. This value incorporates differences in exposure levels and duration between children and adults to produce a single average lifetime daily dose. For Port Colborne, estimates of chronic nickel exposure ranged between 352 and 1240 µg/day for children and 348 and 690 µg/day for adults for people who use a municipal drinking water supply and between 379 µg/day and 1270 µg/day for children and 438 µg/day and 780 µg/day for adults who use well water as a drinking water supply. These estimates represent day-to-day intakes, and are not lifetime averaged values. In order to adequately compare these estimated exposures with the U.S. EPA RfD, and to better determine the levels of risk, it is necessary to convert these daily exposures to lifetime averaged values. Lifetime average intakes (Chronic Daily Intakes (CDI)) are calculated from combined child and adult exposures, corrected for differences in body weight and exposure duration and are based on an assumed lifetime of 70 years (see equation 1) (MOEE, 1994).

The CDI values calculated from the Model 1 intake estimated for celery leaf, for municipal and well drinking water supplies, for each of the soil concentration ranges examined are shown in tables 2.4-5 and 2.4-6 respectively. The maximum CDI for municipal and well water supplies were 17.2 µg/kg/day, and 18.5 µg/kg/day respectively (see tables 2.4-5 and 2.4-6). Both of these values are below the U.S EPA RfD of 20 µg/kg/day. This U.S. EPA RfD incorporates a large margin of safety. As the maximum CDI values calculated for the Port Colborne area are below the RfD, it is unlikely that chronic exposure to nickel in the soils of the Port Colborne area will result in any chronic health effects.

Table 2.4-1: Total Nickel Exposure Estimates for Children (µg/day) Municipal Water Supply

Air	Drinking Water	Food Other	Soil Estimated Intakes (µg/day)		Vegetable	Backyard Produce Estimated Intake (µg/day)		Estimated Total Intakes (µg/day)	
µg/day	µg/day	µg/day	Level	Intake		Model 1	Model 2	Model 1	Model 2
0.065	0.96	344	Low 67.0g/g	5.40	Celery L	1.53	5.80	352	356
				5.40	Celery S	1.53	5.80	352	356
				5.40	Lettuce	1.17	4.40	351	355
			Middle 398 µg/g	31.8	Celery L	27.6	104	404	481
				31.8	Celery S	6.14	23.1	383	400
				31.8	Lettuce	4.68	17.6	381	394
			High 4290 µg/g	343	Celery L	115	434	803	1122
				343	Celery S	35.3	133	723	821
				343	Lettuce	48.0	181	736	869
			Minimum 36.0g/g	2.88	Celery L	1.53	5.80	349	353
				2.88	Celery S	1.53	5.80	349	353
				2.88	Lettuce	1.17	4.40	349	352
			Maximum 9750 µg/g	780	Celery L	115	434	1240	1559
				780	Celery S	35.3	133	1160	1258
				780	Lettuce	48.0	181	1173	1306

1) Models 1 and 2

Table 2.4-2: Total Nickel Exposure Estimates for Adult (µg/day) Municipal Water Supply

Air	Drinking Water	Food Other	Soil Estimated Intakes (µg/day)		Vegetable	Backyard Produce Estimated Intake (µg/day)		Estimated Total Intakes (µg/day)	
µg/day	µg/day	µg/day	Level	Intake		Model 1	Model 2	Model 1	Model 2
0.286	2.4	342	Low 67.0g/g	1.30	Celery L	2.00	19.2	348	365
				1.30	Celery S	2.00	19.2	348	365
				1.30	Lettuce	1.50	14.5	348	361
			Middle 398 µg/g	8.00	Celery L	36.1	346	389	699
				8.00	Celery S	8.00	76.9	361	430
				8.00	Lettuce	6.10	58.7	359	412
			High 4290 µg/g	86.0	Celery L	150	1442	581	1873
				86.0	Celery S	46.0	442	477	873
				86.0	Lettuce	62.7	601	494	1032
			Minimum 36.0g/g	0.70	Celery L	2.00	19.2	348	365
				0.70	Celery S	2.00	19.2	348	365
				0.70	Lettuce	1.50	14.7	347	360
			Maximum 9750 µg/g	195	Celery L	150	1442	690	1982
				195	Celery S	46.0	442	586	982
				195	Lettuce	62.7	601	603	1141

l) Models 1, 2

Table 2.4-3: Total Nickel Exposure Estimates for Children (µg/day) Well Water Supply

Air	Drinking Water	Food Other	Soil Estimated Intakes (µg/day)		Vegetable	Backyard Produce Estimated Intake (µg/day)		Estimated Total Intakes (µg/day)	
µg/day	µg/day	µg/day	Level	Intake		Model 1	Model 2	Model 1	Model 2
0.065	27.7	344	Low 67.0g/g	5.40	Celery L	1.53	5.8	379	383
				5.40	Celery S	1.53	5.8	379	383
				5.40	Lettuce	1.17	4.4	378	382
			Middle 398 µg/g	31.8	Celery L	27.6	104	431	508
				31.8	Celery S	6.14	23.1	410	427
				31.8	Lettuce	4.68	17.6	408	421
			High 4290 µg/g	343	Celery L	115	434	830	1150
				343	Celery S	35.3	133	750	848
				343	Lettuce	48.0	181	763	896
			Minimum 36.0g/g	2.88	Celery L	1.53	5.8	376	380
				2.88	Celery S	1.53	5.8	376	380
				2.88	Lettuce	1.17	4.4	376	379
			Maximum 9750 µg/g	780	Celery L	115	434	1270	1590
				780	Celery S	35.3	133	1190	1280
				780	Lettuce	48.0	181	1200	1330

1) Models 1 and 2

Table 2.4-4: Total Nickel Exposure Estimates for Adult (µg/day) Well Water Supply

Air	Drinking Water	Food Other	Soil Estimated Intakes (µg/day)		Vegetable	Backyard Produce Estimated Intake (µg/day)		Estimated Total Intakes (µg/day)	
µg/day	µg/day	µg/day	Level	Intake		Model 1	Model 2	Model 1	Model 2
0.286	92.4	342	Low 67.0g/g	1.30	Celery L	2.00	19.2	438	455
				1.30	Celery S	2.00	19.2	438	455
				1.30	Lettuce	1.50	14.5	438	451
			Middle 398 µg/g	8.00	Celery L	36.1	346	478	789
				8.00	Celery S	8.00	76.9	451	520
				8.00	Lettuce	6.10	58.7	449	501
			High 4290 µg/g	86.0	Celery L	150	1442	671	1960
				86.0	Celery S	46.0	442	568	963
				86.0	Lettuce	62.7	601	583	1120
			Minimum 36.0g/g	0.70	Celery L	2.00	19.2	437	455
				0.70	Celery S	2.00	19.2	437	455
				0.70	Lettuce	1.50	14.7	437	450
			Maximum 9750 µg/g	195	Celery L	150	1442	780	2070
				195	Celery S	46.0	442	676	1070
				195	Lettuce	62.7	601	692	1230

1) Models 1, and 2

$$\text{Eq 1: CDI} = \frac{\text{intake(child)} \times 7 \text{ years}}{15\text{kg} \times 70 \text{ years}} + \frac{\text{intake(adult)} \times 63 \text{ years}}{70\text{kg} \times 70 \text{ years}}$$

The high levels of nickel in the soils at some for these sites means that intakes from nickel from soils and dusts and from the consumption of backyard produce can make a significant contribution to the total daily nickel intakes for both children and adults. The elevated nickel levels in well water supplies, compared to those found in municipal supplies means that at these sites, drinking water can also make a significant contribution to total daily nickel intakes. The contribution that each exposure pathway makes to total nickel intakes has been calculated based on Model 1 and celery leaf consumption for both municipal and well water supplies for each of the soil nickel concentrations considered (see tables 2.6-7 through 2.4-10).

At lower soil nickel levels (< 100 µg/g) at locations where municipal water provides the drinking water supply, drinking water accounts for 0.27 percent and 0.69 percent of total nickel intake respectively for children and adults (see table 2.4-7 and 2.4-8). Soils and dusts and backyard garden produce account for approximately 1.3 percent and 0.78 percent of total nickel exposures in children and adults. At soil nickel levels greater than 1000 µg/g. the contribution of soils and dusts and backyard garden produce rises significantly to 72 percent in children and 50 percent in adults. The contributions from drinking water drop to 0.08 percent for children and 0.35 percent for adults. This indicates that in areas where drinking water is derived from the municipal supply, nickel levels in the soils of the Port Colborne area can add substantially to the total nickel intakes of the people in the area, and that drinking water does not make a substantial contribution to the total intake.

In locations where well water is used to supply drinking water, drinking water can make a significant contribution to total daily intakes of nickel in both children and adults (see table 2.4-9 and 2.4-10). In areas where soil nickel levels are low (< 100 µg/g) drinking water contributes 7.31 percent of the total daily nickel intake in children. Soils and dusts and backyard produce account for only 1.69 percent of the total intake. At higher soil nickel levels, the contribution from drinking water drops to approximately 2 percent while the total contribution from soils and dusts and the consumption of backyard produce rises to 70 percent of the total daily intakes for children. For adults, at low soil nickel levels, drinking water can contribute almost 21 percent of the total daily intake while soils and dusts and backyard garden produce consumption account for a total of 0.75 percent of the total nickel intake. At the highest soil nickel concentrations considered, the drinking water contribution falls to approximately 11.8 percent while the contributions from soils and dusts and backyard garden produce consumption rise to a total of 44 percent of the total daily intake. This data shows that at sites where well water is used as a drinking water supply, nickel contained in the water can make a significant contribution to the total daily intake. This differs from those sites which use municipally supplied water. At these latter sites, drinking water accounts for less than 1 percent of the total daily intake and that total intakes are driven by soil levels (see above). At sites which rely on well water, drinking water soil and dusts levels, backyard produce consumption and drinking water all drive the total daily nickel intakes.

The combination of high nickel levels in well water and soil results in an estimated CDI value of 18.5 µg/kg/day (see table 2.4-6) which approaches the oral RfD of 20 µg/kg/day. This is a worst case estimate and represents exposures which are unlikely to occur. In the Port Colborne area, the highest soil nickel levels were found in relatively close proximity to the Inco facility, in areas which receive municipal drinking water supplies. Consequently, high exposures resulting from elevated levels in the soil and drinking water are unlikely to occur. Areas which do not have access to the municipal water supply are generally located some distance from the Inco facility in areas where soil concentrations are lower (< 500 µg/g) (based on the soil nickel profiles data contained in the Phytotoxicology Survey Report (MOEE, 1994)). At these lower soil concentrations, CDI values for well water supply are less than 10 µg/kg/day (see table 2.4-6), which is well below the 20 µg/kg/day RfD value. Thus, it is unlikely that the nickel exposures which result from the use of well water as a drinking water supply will be sufficiently large to result in adverse health effects.

A comparison of chronic intakes between children and adults, on a body weight basis, shows that at maximum soil nickel levels, children receive a dose which is approximately ten-fold greater than that of adults. At lower soil nickel levels, the difference is about four-fold. These differences in dose are due to differences in body weight and soil consumption. No indication that children exhibit age-specific health effects relating to nickel exposure could be found in the literature. Also, nickel is an essential dietary element. Consequently, the tolerance range is likely to be broad and the levels required to produce toxic effects are likely to be high. Therefore, it is unlikely that the increased doses experienced by children will result in any adverse health effects.

Table 2.4-5: Estimates of Life-time Averaged Chronic Daily Intakes of Nickel (Municipal Drinking Water Supply)

	Estimated Daily Intakes in µg/day (Model 1)		
Concentration Range (µg/g)	Child	Adult	CDI g/kg/day
[Ni] < 100 (*LOW*)	352	348	6.82
100 ≤ [Ni] < 1000(*MIDDLE*)	404	389	7.69
[Ni] > 1000 (*HIGH*)	803	581	12.8
Minimum	349	348	6.80
Maximum	1240	690	17.2

Table 2.4-6: Estimates of Life-time Averaged Chronic Daily Intakes of Nickel (Well Water Supply)

	Estimated Daily Intakes in µg/day (Model 1)		
Concentration Range (µg/g)	Child	Adult	CDI µg/kg/day
[Ni] < 100 *(LOW)*	379	438	8.17
100 ≤ [Ni] < 1000 (*MIDDLE*)	431	478	9.04
[Ni] > 1000 (*HIGH*)	830	671	14.2
Minimum	376	437	8.14
Maximum	1270	780	18.5

Table 2.4-7: Relative Contributions of Individual Pathways to Total Nickel Exposure (Model 1, Celery Leaf); Children; Municipal Water Supply

	Percentage of Total Nickel Exposure					
Range	Air	D.W.	Food	Soil	Backyard	Total
Low	0.02	0.27	97.7	1.54	0.44	100
Middle	0.02	0.24	85.1	7.87	6.83	100
High	0.01	0.12	42.8	42.7	14.33	100
Minimum	0.02	0.27	98.4	0.83	0.44	100
Maximum	0.01	0.08	27.7	62.9	9.28	100

Table 2.4-8: Relative Contributions of Individual Pathways to Total Nickel Exposure (Model 1, Celery Leaf); Adult; Municipal Water Supply

	Percentage of Total Nickel Exposure					
Range	Air	D.W.	Food	Soil	Backyard	Total
Low	0.08	0.69	98.3	0.37	0.57	100
Middle	0.07	0.62	88.0	2.06	9.28	100
High	0.05	0.41	58.9	14.8	25.8	100
Minimum	0.08	0.69	98.5	0.20	0.58	100
Maximum	0.04	0.35	49.6	28.3	21.7	100

Table 2.4-9: Relative Contributions of Individual Pathways to Total Nickel Exposure (Model 1, Celery Leaf); Children; Well Water Supply

	Percentage of Total Nickel Exposure					
Range	Air	D.W.	Food	Soil	Backyard	Total
Low	0.17	7.31	90.8	1.29	0.401	100
Middle	0.015	6.43	79.8	7.38	6.40	100
High	0.008	3.34	41.4	41.3	13.9	100
Minimum	0.017	7.37	91.5	0.767	0.407	100
Maximum	0.003	2.18	27.1	61.4	9.06	100

Table 2.4-10: Relative Contributions of Individual Pathways to Total Nickel Exposure (Model 1, Celery Leaf); Adult; Well Water Supply

	Percentage of Total Nickel Exposure					
Range	Air	D.W.	Food	Soil	Backyard	Total
Low	0.0653	21.1	78.1	0.297	0.457	100
Middle	0.0598	19.3	71.5	1.66	7.55	100
High	0.0426	13.8	51.0	12.8	22.4	100
Minimum	0.0654	21.1	78.3	0.165	0.458	100
Maximum	0.0367	11.8	43.8	25.0	19.2	100

Inhalation Pathway:

All available evidence suggests that nickel is carcinogenic only when inhaled in particulate form and at concentrations normally associated with occupational exposures. Based on excess lung and nasal cancers among nickel refinery workers, the US EPA has established a cancer unit risk slope for nickel refinery dust of 2.4×10^{-4} $(\mu g/m^3)^{-1}$. Using this value to estimate the cancer risk in Port Colborne shows that the estimated risk level is relatively low. In Port Colborne, the yearly average nickel level is 0.013 $\mu g/m^3$, which translates to a risk level of 3.1×10^{-6}. The risk value cited above has not been reviewed during the preparation of this report. It is provided solely as an illustration of the type of risk which may be expected. A more precise estimate of the cancer risk would require a more in depth evaluation of the assumptions used in the US EPA model. However, the average concentrations of nickel in the air in Port Colborne are similar to those found throughout Ontario, and are therefore unlikely to pose an increased risk to those living in the area. As inhalation is a minimal exposure here, threshold effects are the principle consideration in assessing the possible health effects from nickel exposure in the Port Colborne area.

Summary:

The maximum estimates of acute exposure (model 2) for residents in the Port Colborne area, are 1559 µg/day for children and 1983 µg/day for adults for people using municipally supplied drinking water. For people who rely on well water, the maximum levels of exposure using the highest reported well water levels and exposure model 2 rise to 1590 µg/day for children and 2070 µg/day for adults. All of These values are well below reported LOAEL levels of 7100 µg/day for gastrointestinal, muscular and renal effects.

The maximum estimated CDI (chronic exposure) for Port Colborne area residents who rely on well water (18.5 µg/kg/day) is below the U.S. EPA oral RfD value of 20 µg/kg/day. Chronic Daily intake values for all other exposure scenarios examined are below this value. Thus, exposures to nickel in the Port Colborne area are unlikely to pose either acute or chronic health risks to persons in the area.

3.0 COPPER

3.1 HAZARD IDENTIFICATION

3.1.1 Pharmacokinetics

Copper is an essential element in the diet of humans, and it is widely distributed in the body. Biologically, it is involved including erythrocyte formation, the release of tissue iron and the development of bone, connective tissue and the central nervous system. In addition, copper is necessary for the proper functioning of many enzyme systems and is associated with certain elements of blood.

3.1.2 Toxicology

The biology of copper has previously been the subject of a number of more comprehensive reviews (NAS, 1977, 1980; Stokinger, 1980; WHO, 1984; U.S. EPA, 1985c) and the more salient characteristics are summarized here.

Animal experiments indicate that copper deficiencies are associated with anemia, depigmentation, depressed growth, bone dystrophies and gastro-intestinal disorders.

Copper is absorbed from the lungs or gastrointestinal tract following exposure. The highest tissue concentrations of copper are found in the brain, kidney, heart liver and pancreas. Copper appears to be excreted in the faeces and at a constant rate by the kidneys. Excess copper can be eliminated in faeces with little change in the rate of elimination from the kidneys.

Although not thoroughly understood, homeostatic mechanisms in normal humans provide a balance between copper intake and elimination. It is known that serum copper levels may vary widely with age, sex, and hormonal and nutritional status. The homeostatic mechanisms prevent toxicity from the range of normal variations in copper ingestion.

Copper is rarely systemically toxic when ingested unless large amounts are absorbed. Acute poisoning from oral ingestion of copper is rare due to its emetic effect. However, in instances of large oral exposures, effects such as mucosal irritation, capillary damage, hepatic and renal injury and central nervous system irritation have been reported. Severe gastro-intestinal irritation is also associated with large single doses of copper. Application of concentrated copper salts on the skin has led to papulovesicular eczema and other symptoms, reflecting the corrosiveness of the salts.

Airborne exposures to copper have occurred in occupational settings. Pulmonary exposures have been observed to result in irritation of the respiratory tract, nausea and metal-fume fever. In some cases, discoloration of the skin and hair has been observed.

The scientific literature is replete with studies of the nutritional essentiality of copper, but copper

toxicity from chronic exposure has not been well investigated. There are no data indicating that human exposure to copper results in chronic toxic effects. Diets containing up to 5.8 mg of copper/day produced no noticeable effects in humans. Long-term inhalation of copper fumes and fine aerosols may result in metal-fume fever. Wilson's disease (hereditary hepatolenticular degeneration), a rare inborn error of metabolism, appears to be the only manifested form of chronic copper toxicity by ingestion in humans. This is a condition characterized by high levels of copper, accumulating in the brain, liver and kidney. Another group at increased risk from chronic high level ingestion are individuals with glucose-6-phosphate deficiencies.

There is no evidence that any copper compounds are carcinogenic. The International Agency for research on Cancer has not evaluated copper or copper compounds for carcinogenicity. Copper is classified by the U.S. EPA in the Group D, inadequate data in humans and animals.

3.2 DOSE-RESPONSE INFORMATION/CURRENT EXPOSURE LIMITS

In the case of copper there are two types of limits to consider; 1.) Recommended daily intakes which are based on a minimum nutritional requirement and 2.) Maximum permissible intakes.

For children, a World Health Organization recommended daily intake (WHO-RDI) of 0.08 mg/kg body weight (1.2 mg/day for a 15 kg child) is thought to be necessary for proper development. The National Academy of Sciences (1980) has a recommended daily allowance (RDA) for copper of 2.0-3.0 mg/day for adults and 1.5-2.5 mg/day for children. The 1980 estimated safe and adequate daily dietary intake (ESADDI) ranges for the adult diet is 2.0-3.0 mg/day; the ESADDI ranges for the infant and toddler diets were 0.7-1.0 mg/day and 1.0-1.5 mg/day respectively. The joint FAO/WHO Expert Committee on Food Additives has set a limit of 0.5 mg/kg body weight (7.5 mg/day for a child; 35 mg/day for an adult) as an acceptable maximum daily intake for copper.

In the development of a drinking water criterion for copper, the U.S. EPA (1985), focused on human studies which suggest that ingestion of between 5.3 and 32 mg of copper/person resulted in gastro-intestinal disorders, vomiting, nausea and diarrhoea. Because no lasting adverse effects were reported, and the symptoms have been the result of local gastro-intestinal irritation, the single oral dose of 5.3 mg was considered as a lowest observed adverse effect level (LOAEL). In determining an ADI for copper, the WHO applied a safety factor of 2 to this LOAEL, yielding a value of 2.65 mg/day. These data were also used to calculate a lifetime adjusted ADI, as the acute effects appear to be the effects of concern from exposure to copper.

3.3 HUMAN EXPOSURE ASSESSMENT

3.3.1 Estimated Intakes from Individual Sources

3.3.1.1 Air

Results of the air monitoring program for Ontario suggest annual mean concentrations of copper in particulate of 0.017 $\mu g/m^3$ for various Ontario locations. No air monitoring data for copper specific to the site was located, so this average concentration is was used.

Table 3.3-1: Estimated Copper Intakes From Air

	Child (1-6 years)	Adult
Average Annual Air Level ($\mu g/m^3$)	0.017	0.017
Volume Inhaled (m^3/day)	5	22
Estimated Intake (μg/day)	0.085	0.374

3.3.1.2 Drinking Water

The drinking water concentration of copper, based on treated samples at the Port Colborne water treatment plant for 1990-94, averaged 1.3 µg/L. Data from standing samples in the distribution system averaged 11.4 µg/L over the same time period. Data from consumer taps in the area where soil surveys were conducted were not available. Consumer tap water may contain more copper than the original water supply because of the dissolution of copper from copper piping, which is common in residential housing. The most relevant data of this type comes from a integrated monitoring survey of consumed tap water in Ontario (MOE, 1989b). The average concentration of copper in water over a one week sampling period was 176 µg/L. Therefore conservative value was used for the drinking water intake estimate. Data on the levels of copper found in well water was available for six wells in the Port Colborne area. The copper levels contained in these samples were comparable to the levels found in the municipal supply. Therefore, the copper level in the municipal supply was used to represent well water exposure scenarios, and a separate exposure evaluation was not undertaken.

Table 3.3-2: Estimated Copper Intakes From Drinking Water

	Child (1-6 years)	Adult
Tap Water Level (µg/L)	176	176
Daily Consumption (L/day)	0.6	2.0
Estimated Intake (µg/day)	106	352

3.3.1.3 Soil and Dust

Soil samples from the Port Colborne area, collected during July of 1991, show that copper concentrations in surface soils (0-5cm) range between 22 µg/g and 865 µg/g. The copper concentration in soils from four of the Port Colborne sample site (sites 5,20,31 and 39) match copper soil concentrations used in studies which have examined copper uptake in plants (30, 40, 140 and 380 µg/g). Therefore, estimates of intake from soil and dust were made for each of these concentrations. Providing intake estimates for these sites will allow more accurate estimates of total exposure than using plant uptake studies where the experimental soil concentrations do not match those at the various sites tested. Estimates of soil contributions are also provided for the minimum and maximum reported values and the average levels determined from all 37 sites (see table 3.3-3).

Table 3.3-3: Estimated Copper Intakes from Soils and Dusts

		Estimated Intake µg/day	
Site	Soil [Cu] (µg/g)	Child (1-6 years)[1]	Adult[2]
Site 39	31	2.50	0.62
Site 20	40	3.20	0.80
Site 5	140	11.2	2.80
Site 31	380	30.4	7.60
Average	242	19.4	4.80
Minimum	22	1.80	0.44
Maximum	865	69.2	17.3

1 Based on a total soil intake of 80 mg/day
2 Based on a total soil intake of 20 mg/day

3.3.1.4 Food

3.3.1.4.1 Food: Non-home Sources

Dietary copper is generally the primary source for copper exposure. No figures specific to Ontario were found that would provide an estimated intake for this medium. Therefore, the 1980 average dietary intakes of 680 µg/ day children and 1600 µg/day for adults, as determined by the U.S. Food and Drug Administration (USFDA) were used. The consumption of backyard produce accounts for approximately 1.8 percent of the total daily food intake for children and 2.2 percent for adults (see Section 1.2). The contribution to the total dietary intake made by backyard produce is considered

separately from that of other food sources. In estimating the dietary intake from other sources, it has been assumed that these make up 98.2% and 97.8% of the total daily intake for children and adults, respectively. Therefore, the estimated intakes from the USFDA were adjusted by factors of 0.982 and 0.978 for children and adults to allow for the incorporation of backyard produce. Thus, the values used for food from non-home sources for children and adults were 668 µg/day and 1565 µg/day, respectively.

3.3.1.4.2 Food: Backyard Produce

The levels of copper found in backyard produce is dependent upon the copper concentration in the surrounding soils, and on the type of soil the plants grow in. Data on the uptake of copper are available for celery and lettuce (MOEE, 1994). The data come from both greenhouse and field experiments conducted at the MOEE Phytotoxicology facility in Brampton Ontario. The plants used in these studies were grown on soils taken from the Port Colborne area. Thus, the uptake data should be representative of the uptake rates which could be expected in the Port Colborne area.

Four of the soil copper concentrations used in the plant uptake studies match soil copper levels of four of the Port Colborne sites. This plant uptake data has been used to estimate the copper uptakes in backyard produce. Plant uptake data could not be found to match the minimum, maximum or average soil copper concentrations in the Port Colborne area. Intake estimates were made for these concentrations by using plant uptake values from the next highest concentration. For example; the minimum soil copper level is 22 µg/g. Intakes were estimated for this level by using the plant uptake for a soil level of 30 µg/g copper. The plant uptake data used to estimate intakes from the average soil concentration are shown in Tables 3.3-4 and 3.3-5. The maximum soil concentration reported in Port Colborne (865 µg/g) is higher than the highest concentration of copper used in the plant uptake experiments. Intake estimates for the maximum soil level were made using the highest available soil concentrations from the plant uptake data (600 µg/g). Intakes have been estimated for both children and adults at each of the copper concentrations and for both of the exposure scenarios considered (see Table 3.3-4 and 3.3-5). These data are based on an assumed garden size of approximately 30 m^2.

The intake estimates for the minimum and average copper soil concentrations may over estimate exposure from backyard produce, because the plant uptake values used are for higher soil copper concentrations. The calculated intakes for the maximum concentration are likely to underestimate actual exposures as these values were determined from plant uptake data which used lower copper soil concentrations. In both cases, it is unlikely that the errors in estimation will be appreciable. The vegetation uptake data for copper shown in Tables 3.3-4 and 3.3-5 indicate that copper levels in plants do not change significantly between 30 µg/g and 600 µg/g copper in soil. Thus, it is unlikely that copper levels in vegetables at the minimum and maximum copper levels in soils would differ from those reported. Therefore, it is likely that the intakes at the minimum and maximum levels of copper will be reasonable estimates of actual exposures.

3.4 RISK CHARACTERIZATION

The estimated total daily intakes of copper from all sources for children and adults are shown in Tables 3.4-1 and 3.4-2 respectively. Estimates are provided for both of the backyard garden exposure scenarios, for the average, minimum and maximum reported soil concentrations and for each of the vegetables identified. Estimates are also provided for each of the four sites where soil copper levels matched those used in the cited plant uptake data. The intake values used for food from other sources are based on USFDA values which are determined for U.S. diets and may not hold for Ontario. Therefore, a degree of caution must be used when interpreting these estimates.

Intakes from soil and dust, based on the minimum, maximum and average reported soil copper concentrations range between 1.8 and 69.2 µg/day for children and 0.4 and 17.3 µg/day for adults. At maximum soil concentrations, these values represent approximately 8 percent of the total estimated intake for children and less than 1 percent for adults. The highest estimated intakes from backyard produce for both children and adults were found for lettuce grown in a soil where copper concentrations were 270 µg/g (used as the average soil level for plant uptake in Tables 3.3-4 and 3.3-5), and not as would be expected for plants grown on the most heavily contaminated soils. This is due to the higher plant uptakes reported for lettuce at the 270 µg/g soil copper level. The available plant uptake data show that for a given soil copper level, lettuce appears to show higher plant levels than either celery leaf or stalk, and as a result the estimated intakes are higher for lettuce. Therefore, in considering total exposures, attention has been focused on intakes estimates using lettuce.

At the highest estimated exposure levels, intakes from backyard produce account for 1.1 percent of the total daily intake for children (Model 1) and approximately 0.62 percent for adults (Model 1). The relative contributions of each pathway to the total exposure at the minimum, maximum and average soil copper concentrations and for the consumption of lettuce, for both children and adults are shown in Table 3.3-6.

To characterize the potential risks associated with copper contamination of the soil, the age-specific exposures from Tables 3.4-1 and 3.4-2 are compared to current exposure limits for copper. Estimated maximum total exposures (878 µg/day for children and 2084 µg/day for adults) fall below the NAS ESADDI ranges of 1.0-1.5 mg/day for children and 2.0-3.0 mg/day for adults, the FAO/WHO acceptable maximum daily intake of 0.5 mg/kg/day (7.5 mg/day for a 15 kg child) and the ADI value of 2.65 mg/day suggested by the 1985 U.S EPA Drinking Water Criteria document. Maximum intake based on the maximum soil concentration and maximum consumption of backyard produce would not appear to add appreciably to daily intake.

The copper levels in soil on these sites were estimated to result in exposures which are below currently recommended maximum acceptable health limits. These are not predicted to pose any appreciable risk to area populations, as total multimedia intakes are below current exposure limits.

Table 3.3-4: Estimated Copper Intakes from Backyard Produce; Children

Soil Level (µg/g)		Vegetable	Cu Level In Vegetable (µg/g)			Estimated Daily Intake (µg/day)	
Pt. C[1]	Exp[2]		Dry	C.F.[4]	Fresh	Model 1	Model 2
31	30	Celery L[3]	6	0.059	0.354	9.2	34.69
	30	Celery S	3	0.059	0.177	4.6	17.35
	30	Lettuce	6	0.045	0.27	7.02	26.46
40	40	Celery L	5	0.059	0.295	7.67	28.91
	40	Celery S	3	0.059	0.177	4.60	17.35
140	140	Lettuce	7	0.045	0.315	8.19	30.87
380	380	Celery L	5	0.059	0.295	7.67	28.91
	380	Celery S	3	0.059	0.177	4.60	17.35
	380	Lettuce	9	0.045	0.405	10.53	39.69
Avg (242)	270	Celery L	6	0.059	0.354	9.20	34.69
	270	Celery S	3	0.059	0.177	4.60	17.35
	270	Lettuce	11	0.045	0.495	12.87	48.51
Min (22)	30	Celery L	6	0.059	0.354	9.20	34.69
	30	Celery S	3	0.059	0.177	4.60	17.35
	30	Lettuce	6	0.045	0.270	7.02	26.46
Max (865)	600	Celery L	6	0.059	0.354	9.20	34.69
	600	Celery S	2	0.059	0.118	3.07	11.56
	600	Lettuce	8	0.045	0.360	9.36	35.28

1) Copper concentration in Port Colborne Soils
L = Leaf; S = Stalk

2) Copper concentrations in soils from experimental Plant uptake studies
4) Dry to Fresh weight conversion factors

Table 3.3-5: Estimated Copper Intakes from Backyard Produce; Adults

Soil Level (µg/g)		Vegetable	Cu Level In Vegetable (µg/g)			Estimated Daily Intake (µg/day)	
Pt. C[1]	Exp[2]		Dry	C.F.[4]	Fresh	Model 1	Model 2
31	30	Celery L[3]	6	0.059	0.354	12.04	115.40
	30	Celery S	3	0.059	0.177	6.02	57.70
	30	Lettuce	6	0.045	0.27	9.18	88.00
40	40	Celery L	5	0.059	0.295	10.03	96.17
	40	Celery S	3	0.059	0.177	6.02	57.70
140	140	Lettuce	7	0.045	0.315	10.71	102.69
380	380	Celery L	5	0.059	0.295	10.03	96.17
	380	Celery S	3	0.059	0.177	6.02	57.70
	380	Lettuce	9	0.045	0.405	13.77	132.03
Avg (242)	270	Celery L	6	0.059	0.354	12.04	115.40
	270	Celery S	3	0.059	0.177	6.02	57.70
	270	Lettuce	11	0.045	0.495	16.83	161.37
Min (22)	30	Celery L	6	0.059	0.354	12.04	115.40
	30	Celery S	3	0.059	0.177	6.02	57.70
	30	Lettuce	6	0.045	0.270	9.18	88.00
Max (865)	600	Celery L	6	0.059	0.354	12.04	115.40
	600	Celery S	2	0.059	0.118	4.01	38.47
	600	Lettuce	8	0.045	0.360	12.24	117.30

1) Copper concentration in Port Colborne Soils
2) Copper concentrations in soils from experimental Plant uptake studies
3) L = Leaf; S = Stalk
4) Dry to Fresh weight conversion factors

Table 3.3-6: Relative Contribution of Individual Pathways to Total Copper Exposure (Model 1, Celery Leaf)

Substrate or Medium	Percentage of Total Contribution					
	Child (1-6 years)			Adult		
Soil Concentration	Min	Max	Avg	Min	Max	Avg
Food (other Sources)	85.4	78.4	82.9	81.2	80.4	80.7
Backyard Produce	0.90	1.1	1.6	0.48	0.62	0.87
Soil & Dust	0.23	8.12	2.4	0.02	0.89	0.25
Drinking Water	13.5	12.4	13.1	18.3	18.1	18.2
Air	< 0.01	< 0.01	< 0.01	< 0.02	< 0.02	< 0.02
Total	100	100	100	100	100	100

Table 3.4-1: Total Copper Exposure Estimates for Children (µg/day)

Air	Drinking Water	Food Other	Soil Estimated Intakes (µg/day)		Vegetable	Backyard Produce Estimated Intake (µg/day)		Estimated Total Intake (µg/day)	
µg/day	µg/day	µg/day	Level	Intake		Model 1	Model 2	Model 1	Model 2
0.085	105.6	668	31	2.5	Celery Leaf	9.2	34.7	785	811
					Celery Stalk	4.6	17.4	781	794
					Lettuce	7.02	26.5	783	803
			40	3.2	Celery Leaf	7.67	28.9	785	806
					Celery Stalk	4.60	17.4	781	794
			140	11.2	Lettuce	8.19	30.9	793	816
			380	30.4	Celery Leaf	7.67	28.9	812	833
					Celery Stalk	4.60	17.4	809	821
					Lettuce	10.53	39.7	815	844
			Avg (242)	19.4	Celery Leaf	9.20	34.7	802	828
					Celery Stalk	4.60	17.4	798	810
					Lettuce	12.87	48.5	806	842
			Min (22)	1.8	Celery Leaf	9.20	34.7	785	810
					Celery Stalk	4.60	17.4	780	793
					Lettuce	7.02	26.5	783	824
			Max (865)	69.2	Celery Leaf	9.20	34.7	852	878
					Celery Stalk	3.07	11.6	846	854
					Lettuce	9.36	35.3	852	878

Table 3.4-2: Total Copper Exposure Estimates for Adults (µg/day)

Air	Drinking Water	Food Other	Soil Estimated Intakes (µg/day)		Vegetable	Backyard Produce Estimated Intake (µg/day)		Estimated Total Intake (µg/day)	
µg/day	µg/day	µg/day	Level	Intake		Model 1	Model 2	Model 1	Model 2
0.374	264	1565	31	0.6	Celery Leaf	12.0	115	1930	2033
					Celery Stalk	6.0	57.7	1924	1976
					Lettuce	9.2	88.0	1927	2006
			40	0.8	Celery Leaf	10.0	96.2	1928	2014
					Celery Stalk	6.0	57.7	1924	1976
			140	2.8	Lettuce	10.7	103	1931	2023
			380	7.6	Celery Leaf	10.0	96.2	1935	2021
					Celery Stalk	6.0	57.7	1931	1983
					Lettuce	13.8	132	1939	2057
			Avg (242)	4.8	Celery Leaf	12.0	115	1934	2038
					Celery Stalk	6.0	57.7	1928	1980
					Lettuce	16.8	161	1939	2084
			Min (22)	0.4	Celery Leaf	12.0	115	1930	2033
					Celery Stalk	6.0	57.7	1924	1975
					Lettuce	9.2	88.0	1927	2003
			Max (865)	17.3	Celery Leaf	12.0	115	1947	2050
					Celery Stalk	4.0	38.5	1939	1973
					Lettuce	12.2	117	1947	2052

4.0 COBALT

4.1 HAZARD IDENTIFICATION

4.1.1 Pharmacokinetics

Cobalt exists in nature as a metal and in two valence states - CoII and CoIII, which form numerous organic and inorganic salts. Cobalt is an essential nutrient functioning as a cofactor for several enzymes, and it is required for the synthesis of vitamin B_{12} (MOL, 1988).

Cobalt and its salts are readily absorbed in the gastro-intestinal tract, where the amount absorbed decreases with increasing dose. Although some individual variations exist, humans absorb about 25% if ingested cobalt salts. However, more than 75% of the cobalt in food is absorbed (ICRP, 1984; MOL, 1988). Absorption also seems to be dependent on diet (ICRP, 1984; MOL, 1988; Stokinger, 1981b). In animals, where large doses of cobalt have been given orally, approximately 80% is excreted in the faeces and the remainder in the urine (Taylor and Marks, 1978 as cited in Domingo, 1989; MOL, 1988). In humans, the absorbed cobalt is excreted predominately through the urine, with about 10% in the faeces and some in sweat (ICRP, 1984; MOL, 1988). The initial excretion is rapid, but some may be retained for several months.

4.1.2 Toxicology

In sufficiently large doses, cobalt salts can cause gastrointestinal tract irritation. Acute exposures in patients receiving cobalt for treatment of anemia have shown symptoms of hypothyroidism, nausea, tinnitus and neurogenic deafness. Cobalt may also elicit other neurotoxic and cardiotoxic effects. Polycythemia (elevated red blood cell levels) is the characteristic response of most mammals, including humans, to ingestion of excessive amounts of cobalt.

Small epidemics of severe cardiomyopathy have been observed to result from the heavy consumption of beer to which cobalt compounds had been added. Doses may have been as high as 10 mg per day (Friberg, 1986).

Inhalation of cobalt and cobalt compounds in occupational settings has been associated with various effects. Occupational exposures to dusts containing cobalt mixed with other materials can cause a severe type of pneumoconiosis as well as obstructive lung disease at concentrations of >60 μg Co/m^3. Allergic dermatitis has also been reported in workers exposed to cobalt-containing materials (MOL, 1988; Stokinger, 1981b).

Cobalt has not been shown to cause significant teratogenic or reproductive effects in humans, although some *in vitro* studies have been positive. Oral administration of cobalt did not produce teratogenicity or significant fetotoxicity in the rat at daily doses as high as 100 mg $CoCl_2$/kg (Domingo, 1989).

There is very little information available on the mutagenicity of cobalt, but the available data do not suggest that it has strong mutagenic properties.

Single or repeated injections of cobalt powder or cobalt salts have induced malignant tumours at the site of injection in rats but not in mice. There was no increase in the incidence of lung tumours in hamsters exposed to cobalt oxide dust as compared to controls. To date induction of cancer in experimental animals has not been possible except by injection. Epidemiological evidence for the carcinogenicity of inhaled cobalt compounds among industrial workers is conflicting because exposures have been to mixtures of dusts. Therefore, it cannot be concluded that there is a correlation between occupational exposure to cobalt and cancer.

According to a review by Domingo (1989), most authors conclude that cobalt poses no recognized health hazard at environmental concentrations to non-occupationally exposed individuals.

4.2 DOSE-RESPONSE INFORMATION/CURRENT EXPOSURE LIMITS

Ontario drinking water objectives have not been set for cobalt. It has been reported that young rats exposed to 500 ppm of $CoCl_2$ in drinking water for three months showed various adverse effects including hypertrophy of the spleen, lungs and heart, and decrease in the values of the nutritional parameters. Studies in rats involving chronic drinking water exposure at a level of 2 mg Co/L have been shown to affect the erythropoietic system, cause immunosuppression and inhibit reflex learning (Carson et al., as cited in Mol, 1988). These effects were not seen at 200 μg/litre doses.

An ambient air guideline of 100 ng/m^3 has been proposed (MOL, 1988) based on a study where a LOEL of 100 µg/m^3 was reported in miniature swine. This guideline incorporates an uncertainty factor of 1000. This value was chosen because a LOEL rather than a NOEL was used to account for interspecies variation. It also accounts for variation within the human population.

4.3 HUMAN EXPOSURE ASSESSMENT

Cobalt exists probably only as CoII in both soil and water, as CoIII is unstable in aqueous media (Stokinger, 1981b).

4.3.1 Estimated Intake from Individual Sources

4.3.1.1 Air

The concentration at Port Colborne is not known. It has been estimated that the intake is <0.1 µg/day (ICRP, 1984).

4.3.1.2 Drinking Water

The drinking water concentrations of cobalt, based on treated samples from the Port Colborne treatment plant for 1990-1994 averaged 0.156 µg/L. Data from standing samples in the distribution system averaged 0.161 µg/L over the same period. This latter value, which likely represents the highest cobalt level, was used to estimate cobalt intakes from drinking water (see Table 4.3-1). Testing data from six well water supplies in the Port Colborne area showed that the cobalt levels in the well water were comparable to those found in the municipal supply. Therefore the municipal supply was used to represent all drinking water exposures to cobalt.

Table 4.3-1: Estimated Cobalt Intakes From Drinking Water

	Child (1-6 years)	Adult
Tap Water Level (µg/L)	0.161	0.161
Daily Consumption (L/day)	0.6	2.0
Estimated Intake (µg/day)	0.097	0.32

4.3.1.3 Soil and Dust

The sampling of soil in the Port Colborne area during July of 1991 showed that cobalt concentrations in surface soil (0-5cm) are fairly evenly distributed over most of the sampling area, with levels ranging between 8 and 195 µg/g (MOEE, 1994). Of the 37 sample examined, only 5 showed cobalt levels greater than 100 µg/g. These five are all located in the immediate vicinity of the INCO facility. From these 1991 data, the following concentrations have been determined:

- average 46 µg/g (n=37)
- minimum 8 µg/g
- maximum 195 µg/g

Based on these concentrations, the estimated cobalt intakes from soil/dust were calculated and are shown in Table 4.3-2.

Table 4.3-2: Estimated Cobalt Intakes from Soils and Dusts

		Estimated Intake µg/day	
Concentrations	Soil [Co] (µg/g)	Child (1-6years)[1]	Adult[2]
Average	46	3.7	0.9
Minimum	8	0.6	0.2
Maximum	195	15.6	3.9

1 Based on a total soil intake of 80 mg/day
2 Based on a total soil intake of 20 mg/day

4.3.1.4 Food

4.3.1.4.1 Food: Non-home Sources

There is only limited information available on the intake of cobalt from food. Average intakes of approximately 300 µg/day with a maximum of about 600 µg/day have been reported in the literature, although levels as high as 1800 µg/day have been suggested (ICRP, 1984; MOL, 1988; Stokinger, 1981b). The consumption of backyard garden produce can account for approximately 1.8 percent of the total daily food intake for children and 2.2 percent for adults (see Section 1.2). The contribution to the total dietary intake made by the consumption of backyard garden produce is considered separately from the contributions made by other foodstuffs. In estimating the dietary intake from other food sources, it has been assumed that these make up 98.2 percent of the total dietary intake for children and 97.8 percent for adults. Therefore, the estimated total dietary intakes reported in the literature have been adjusted by factors of 0.982 and 0.978 to allow for the incorporation of backyard produce data. Thus, the values used for food from non-home sources for children and adults were 295 µg/day and 293 µg/day, respectively.

4.3.1.4.2 Food: Backyard Produce

Cobalt levels in backyard produce will depend on the cobalt concentration in the soils. Uptake from the soils is also dependent upon the soil characteristics. Data on the vegetation uptake of cobalt is available for celery, lettuce and onions (MOEE, 1994). The data come from plants grown in the Bradford Marsh area of Ontario. The soil type found at this site (muck soil) is similar to that found in the vicinity of Port Colborne. Thus, the data should be reasonably representative of potential plant uptake of cobalt in Port Colborne soils. Unfortunately, the maximum soil Cobalt concentration available from the Bradford Marsh data is 5 µg/g, which is lower than the lowest soil concentration reported in the Port Colborne area. Therefore, the estimation of potential cobalt exposure from the consumption of backyard garden produce in Port Colborne is limited. Intakes have been estimated for the lowest cobalt concentration in the Port Colborne area (8 µg/g) by using the Bradford Marsh plant uptake data where soil concentrations were 5 µg/g. The difference between the two soil values is unlikely to have a significant effect on either plant uptake or estimated exposures. Intakes have been estimated for both children and adults for each of the vegetables listed and for both exposure

scenarios. The data shown in Table 4.3-2 are based on a garden size of 30 m^2.

The highest intake estimates come from celery leaf data. Therefore, these values have been used to estimate total exposures (see Table 4.4-1). In using these values it must be recognized that the soil cobalt concentration used in the plant uptake study is lower than the lowest cobalt soil level reported in Port Colborne. Therefore these exposure estimates are likely to underestimate the maximum possible exposures. While it is difficult to state quantitatively, what exposures are likely from these higher soil cobalt concentrations, it is possible to qualitatively estimate the potential for exposure.

Table 4.3-3: Estimated Cobalt Intakes from Backyard Garden Produce

				Estimated Daily Intakes (µg/day)			
Vegetable	[Co] in Vegetable (µg/g)			Model 1		Model 2	
	Dry[1]	CF[2]	Fresh[3]	Child	Adult	Child	Adult
Celery Leaf	4	0.059	0.236	6.14	8.02	23.13	76.94
Celery Stalk	3	0.059	0.177	4.6	6.02	17.35	57.70
Lettuce	2	0.045	0.090	2.34	3.06	8.82	29.34
Onion	1	0.11	0.110	2.86	3.74	10.78	35.86

1) Dry Weight Co Concentration in vegetable
2) Dry weight to Fresh Weight Conversion Factor
3) Fresh Weight Co Concentration in vegetable

Plant uptake of cobalt is a function of the soil cobalt concentration and the capacity of the soil to bind cobalt, thereby making it unavailable to the plant. Soils high in organic matter content, neutral to moderately alkaline pH and high cation exchange capacity have a greater binding capacity. This could mean that soil cobalt would have to be relatively high before cobalt is available to plants. The plant uptake data for copper shows that increases in soil copper concentration from 30 µg/g to 600 µg/g results in little appreciable difference in the plant concentration of copper (see section 3). It is likely that Co would behave in a similar manner. Therefore, it is unlikely that the cobalt concentration found in backyard produce would vary greatly between the various sites and that exposure to cobalt from backyard produce grown at the most heavily contaminated site (195 µg/g) would be significantly different from that estimated above.

4.4 RISK CHARACTERIZATION

The estimated total daily intakes of cobalt from all sources are shown in Table 4.4-1. Estimates are provided for both of the backyard garden exposure scenarios at the average, minimum and maximum reported cobalt soil concentrations. The estimates at the average and maximum soil concentrations are underestimates because the plant uptake data relied on a single cobalt soil concentration (5 µg/g)

(see above). However, it is unlikely that the figures presented in Table 4.4-1 significantly under-estimate the potential exposure. The data in Table 4.4-2 shows that food from other sources is the largest contributor to total cobalt exposure. The consumption of backyard garden vegetables contributes up to 7.6 percent of the total food intake in children (Model 2) and 22 percent in adults (Model 2). When the total food intake of cobalt is compared to cobalt from other sources, it can be seen that soil and dust is the largest non-food contributor to total cobalt intake. In children exposed to soils and dusts which carry the highest reported cobalt levels (195 µg/g), this contribution reaches about 5 percent of the total daily intake. For other locations and exposure scenarios, the contribution from soils and dusts are significantly less and frequently less than 1 percent of the daily total (see Table 4.4-3).

The toxicological information regarding the chronic effects of cobalt exposure via ingestion is limited. No specific reference doses or tolerable intake values for chronic human exposure were located.

The exposure data from food, taken to be 300 µg/day (adjusted for children and adults) is highly variable and the reported data range between 300 and 1800 µg/day (Calabrese et al., 1985 as cited by Domingo). Thus, the maximum contribution from backyard garden produce (76.94 µg/day for adults (model 1)) is not a significant increase in the total cobalt intake and represents a total estimated intake which lies well within the normal range indicated in the literature.

Estimated exposures from air are negligible and are estimated to be less than 0.03% of total exposure. Estimated exposures from drinking water are also negligible and do not exceed 0.07% of the total daily exposure.

Excess exposures are not expected at these environmental concentrations. On the basis of this exposure assessment, no adverse health effects as a consequence of cobalt exposure from soils in Port Colborne is predicted.

Table 4.4-3: Relative Contributions of Individual Pathways to Total Cobalt Exposure (Model 1, Celery Leaf)

Substrate or Medium	Percentage of Total					
	Child (1-6 years old)			Adult		
Soil Concentration	Min	Max	Avg	Min	Max	Avg
Food (Other Sources)	97.70	93.08	96.71	97.19	95.96	96.91
Backyard Produce	2.03	1.94	2.01	2.66	2.63	2.65
Soil & Dust	0.20	4.92	1.21	0.01	1.28	0.30
Drinking Water	0.03	0.03	0.03	0.11	0.10	0.11
Air	0.03	0.03	0.03	0.03	0.03	0.03
TOTAL	100%	100%	100%	100%	100%	100%

Table 4.4-1: Total Cobalt Exposure Estimates for Children (µg/day)

Air	Drinking Water	Food Other	Soil Estimated Intakes (µg/day)		Vegetable	Backyard Produce Estimated Intake (µg/day)[1]		Estimated Total Intake (µg/day)	
µg/day	µg/day	µg/day	Level	Intake		Model 1	Model 2	Model 1	Model 2
0.1	0.097	295	Avg	3.7	Celery Leaf	6.14	23.13	305	322
					Celery Stalk	4.60	17.35	303	316
					Lettuce	2.34	8.82	301	308
					Onion	2.86	10.78	302	310
			Min	0.6	Celery Leaf	6.14	23.13	302	319
					Celery Stalk	4.60	17.35	300	313
					Lettuce	2.34	8.82	298	305
					Onion	2.86	10.78	299	307
			Max	15.6	Celery Leaf	6.14	23.13	317	334
					Celery Stalk	4.60	17.35	315	328
					Lettuce	2.34	8.82	313	320
					Onion	2.86	10.78	314	322

1) Only one soil cobalt concentration was used in estimating intakes from backyard produce. Therefore, these intake values were used in estimating total exposures for all three soil concentration considered.

Table 4.4-2: Total Cobalt Exposure Estimates for Adults (µg/day)

Air	Drinking Water	Food Other	Soil Estimated Intakes (µg/day)		Vegetable	Backyard Produce Estimated Intake (µg/day)[1]		Estimated Total Intake (µg/day)	
µg/day	µg/day	µg/day	Level	Intake		Model 1	Model 2	Model 1	Model 2
0.1	0.32	293	Avg	0.9	Celery Leaf	8.02	76.94	302	371
					Celery Stalk	6.02	57.70	300	352
					Lettuce	3.06	29.34	297	324
					Onion	3.74	35.86	298	330
			Min	0.02	Celery Leaf	8.02	76.94	302	371
					Celery Stalk	6.02	57.70	300	351
					Lettuce	3.06	29.34	297	323
					Onion	3.74	35.86	297	329
			Max	3.9	Celery Leaf	8.02	76.94	305	374
					Celery Stalk	6.02	57.70	303	355
					Lettuce	3.06	29.34	300	327
					Onion	3.74	35.86	301	333

1) Only one soil cobalt concentration was used in estimating intakes from backyard produce. Therefore, these intake values were used in estimating total exposures for all three soil concentration considered.

5.0 Population Health Effects

Available population health outcome data for Port Colborne residents were reviewed for evidence of deviation from expected rates which might suggest an environmental effect. Specifically, information regarding adverse reproductive outcomes (i.e., stillbirths, infant deaths and congenital anomalies) provided by the Public Health Branch of the Ministry of Health, and cancer incidence data provided by the Ontario Cancer Treatment and Research Foundation (OCTRF) were examined.

Both adverse reproductive outcomes and site specific cancers are sufficiently uncommon that rates calculated for small populations can provide unstable estimates. As well, data release guidelines preclude the release of low case counts, in order to protect patient confidentiality. The OCTRF guideline requires a case count of greater than or equal to 5. For these reasons, Port Colborne population data were aggregated over a number of years and across age groups.

Reproductive outcome data were examined for the 20 year period, 1970-89. Still births were aggregated for two 10 year intervals, 1970-79; 1980-89. Reported rates of spontaneous abortions were available for the time period 1975-83. Specific congenital anomalies (Down's syndrome, cleft palate and lip, spina bifida) are sufficiently rare that rates were calculated per 10,000 births over the 9 year period 1975-83.

For Port Colborne males, cancer incidence data for all sites combined and the 5 common sites (prostate, lung, colorectal, non-Hodgkin's lymphoma and leukaemia) were available for the grouped years 1979-83, 1984-88 and 1989-91. For females in the Port Colborne area, cancer incidence data were available for the 5 common sites (breast, colorectal, lung, uterus and ovary) and all sites combined, but only for time period 1982-91, Direct age adjustment was used to standardize rates for comparison with the Ontario population.

5.1 Adverse Reproductive Outcomes

There is no direct evidence linking environmental exposures to nickel, copper of cobalt with adverse reproductive outcomes in humans. However, population surveillance of reproductive failure (still births, spontaneous abortions and early infant deaths) and congenital anomalies are considered an "early warning system" for environmental health effects.

Based on data from a report produced by the Public Health Branch of the Ontario Ministry of Health (MOH, 1986), and data routinely analyzed by the Regional Niagara Health Services Department, there is no evidence of reproductive failure in the Port Colborne population over the 20-year period 1970-89. Over this period, the average annual stillbirth rate in Port Colborne was approximately 8.5 per 1000 total births. For the period 1976-83, the spontaneous abortion rate was approximately 7.6 per 1000 live births. These rates do not differ from provincial rates over the same time periods. The still birth rate in Ontario was approximately 8 per 1000 total births and the reported spontaneous abortion rate was

8.3 per 1000 live births. Of the 10 birth anomalies monitored routinely, there was statistical elevation in the reported rate of congenital dislocation of the hip in Port Colborne over the 1975-83 period. However, this was due to the occurrence of 2 cases in 9 years versus an expected 0.5 cases.

5.2 Cancer Incidence

Neoplastic changes are the principal adverse human health effect associated with occupational nickel exposures. Occupational groups exposed to nickel sulphide and nickel oxide compounds via inhalation, have demonstrated elevated incidence of lung cancer [Ca_{lung}] and nasal cancers [Ca_{nasal}].

Cancer incidence rates for all sites combined and specific sites, for Port Colborne females 1982-91 and for males in the time period 1984-88 and 1989-91 were not significantly different from expected rates. Based on data reviewed by the OCTRF, there were fewer than five newly diagnosed nasal-pharyngeal cancer cases in the Port Colborne population between 1979-1991. In the five year period 1979-83, however, the incidence of lung cancer in Port Colborne males was elevated: 60 cases observed compared with an expected rate of 44.4 (Standard Incidence Ratio 1.35 at the 95% Cl 1.03-1.72)

The observed elevation in the incidence of lung cancer for Port Colborne males for the 1979-83 time period may be associated with occupational exposures in the Port Colborne area. Population data cannot directly address this. However, the indirect evidence is suggestive. Lung cancer mortality risks have been shown to be elevated in workers exposed to leaching, calcining, and sintering process in the Port Colborne INCO nickel refinery (ICNC, 1990). The incidence rate of lung cancer in males in Port Colborne declined over the observed time period 1979-91. IN the period 1979-83, the age adjusted lung cancer incidence was 87.9/100,000 falling to 68.6/100,000 for the period 1984-88, and further to 58.2/100,000 in the 1989-91 period. In contrast, the incidence of lung cancer in Ontario males was 61.5/100,000 in 1979. This rate peaked at 66.2/100,000 in 1986 and declined to 61.1/100,000 in 1991. Given the limitations of examining outcome data only, any explanation of the observed effect is speculative. However, the pattern of cancer incidence rates in Port Colborne does not indicate an excess population risk due to persistent and pervasive environmental exposures.

6.0 SUMMARY

The conclusions drawn from the assessment of each of the metals are summarized below.

Nickel:

Estimates of chronic nickel exposure ranged between 352 and 1240 µg/day for children and 348 and 690 µg/day for adults for people using a municipal drinking water supply. For these people, the combined lifetime averaged chronic daily intake (CDI) calculated for the maximum estimated intake was 17.2 µg/kg/day. For people who rely on well water as a drinking water supply, estimates of chronic nickel intake based on the highest reported well water nickel levels, range between 376 µg/day and 1260 µg/day for children and 431 µg/day and 773 µg/day for adults. The CDI for this latter group was 18.4 µg/kg/day. Both of these CDI values are lower than the US EPA lifetime averaged exposure reference dose (RfD) of 20 µg/kg/day, which is defined as the dose below which, exposure averaged over a lifetime, is unlikely to result in adverse health effects.

Comparison of intakes between children and adults, on a body weight basis, showed that at maximum soil nickel levels, children receive a dose which is approximately ten-fold greater than that of adults. At lower soil nickel levels, the difference is about four-fold. These differences in dose are due to differences in body weight (15 kg for a child and 70 kg for an adult) and soil consumption (80 mg/day for a child and 20 mg/day for an adult). No indication that children exhibit age-specific health effects relating to nickel exposure could be located in the literature. Also, given that nickel is an essential dietary element, the tolerance range is likely to be broad and the levels required to produce toxic effect are likely to be high. In addition, the RfD value suggested by the US EPA incorporates a large margin of safety into the 20 µg/kg/day value. The maximum CDI values calculated for the Port Colborne area are below this US EPA value. Based on this information, it is unlikely that exposure to nickel in the soils from the Port Colborne area will result in adverse health effects in children or adults.

Copper:

Maximum estimates of total exposure to copper for children and adults in the Port Colborne area are 878 µg/day and 2084 µg/day respectively. Both of these values fall below the National Academy of Sciences *Estimated Safe and Adequate Daily Dietary Intake* (ESADDI) ranges of 1000 to 1500 µg/day for children and 2000-3000 µg/day for adults, the FAO/WHO acceptable maximum daily intake of 500 µg/kg/day for the general population (this would translate to an intake of 7500 µg for a 15 kg child) and the allowable daily intake (ADI) value of 2650 µg/day suggested in the U.S. EPA Drinking Water Criteria Document. Based on this information, exposure to copper in the soils in the Port Colborne area are not expected to pose any appreciable risks to area populations as total multi-media intakes are below current

exposure limits.

Cobalt:

Maximum estimates of total exposure to cobalt for children and adults in the Port Colborne area are 333 and 375 µg/day respectively. Data on cobalt exposure from food is highly variable and ranges between 300 µg/day and 1800 µg/day. Exposures to cobalt in the Port Colborne area do not appreciably alter the total exposures. Thus, excess exposures are not expected at the environmental concentrations which exist in the Port Colborne area. On the basis of this assessment, no adverse health effects as a consequence of cobalt exposure in Port Colborne soils are anticipated.

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