Assessment of Potential Health Effects of Ingestion of Garden Produce containing Arsenic and Cobalt grown in Cobalt, Ontario.

S.W. Fleming
Senior Regulatory Toxicologist
Risk Assessment Unit
Hazardous Contaminants Branch

The following is a review of the potential health implications of consumption of garden produce grown in Cobalt, Ontario. The request stems from analytical results of produce collected by the Sudbury regional office in the summer of 1990 from several residential gardens. These are from areas located within the Town of Cobalt, which has a long mining history. By way of clarification, these are not in the vicinity of the Old Cobalt refinery from which other produce samples have been previously assessed by the Ministry of Labour. The analytical data provided indicate analysis for a number of metals in the produce. As per the request for advice, the analysis will focus on the levels of arsenic and cobalt found.

The current assessment provides the following information: a.) a summary of the known human health effects of ingested arsenic and cobalt; b.) estimation of possible exposures through the vegetable consumption pathway utilizing the analytical data provided and; c.) characterization of possible risks through comparison of these modelled intakes to the toxicological information, as well as recommended limits for human exposure.

Toxicology

1. Arsenic

People are chronically exposed to low levels of inorganic and organic arsenic in the environment. The main pathways of exposure for humans are through ingestion and to a lesser extent, inhalation. Available evidence shows that organic arsenicals in the environment, such as those found in fish and other foods, are absorbed after ingestion but have very low toxicity to man and are excreted unchanged. These organic compounds are, on the whole, poorly characterized. There is considerable evidence, both from man and from other organisms, of the toxic effects of inorganic arsenic, which is present as compounds of either the AsIII or AsV form. There is, however, little information describing the inorganic compounds to which humans are exposed to environmentally. This is especially true of inorganic arsenic in food.

The fraction of inorganic arsenic absorbed from the gastro-intestinal tract will vary, depending on the chemical species, solubility, dose administered and the matrices of the compounds administered. In general, it is expected that arsenic in aqueous solutions would be better absorbed than arsenic bound to

particulate matter. About 80% of the inorganic arsenic in food is absorbed in the gastro-intestinal tract, although this may vary with type of food.

Once absorbed, arsenic is transported to other tissues of the body by the red blood cells and the blood plasma. The liver detoxifies the inorganic arsenic by methylating it to dimethyl arsenic acid. The excretion of the absorbed arsenic is largely through the urine. Fecal arsenic represents ingested insoluble particulates. Total intake and excretion, when averaged over a week, are in balance.

Health Effects

Some question exists as to whether arsenic is a nutritionally essential dietary element, and this has been the subject of previous scientific review (U.S EPA, 1988). Studies with chickens and small mammals fed a diet almost free of inorganic arsenic suggest that a certain daily dose of arsenic is needed for normal development and health of the animals. There is no direct evidence that arsenic is an essential element for humans. Marcus and Rispin (1988) have suggested that the nutritional requirement for animals for arsenic lies between 12 and 50 µg/day. This is based on research work using chickens, goats and rats fed on arsenic-deficient diets. It is considered plausible that there is a nutritional requirement for arsenic in humans, although no arsenic deficiency disease in humans has been reported.

The toxicological aspects of inorganic arsenic has been reviewed in detail in a number of reports (WHO, 1981; U.S. EPA, 1984; U.S. EPA, 1988; MOE, 1991). Arsenic has a number of known toxic effects in humans. Occupational exposure to arsenic dust and ingestion of water with high concentrations leads to irritation of mucous membranes, hyperpigmentation and hyperkeratosis, and neurotoxic and cardiovascular effects.

Arsenic is a potent teratogen in animals, producing a wide spectrum of malformations. The effective teratogenic dose is near the maternal toxic dose. The few studies in humans have produced inconclusive results.

Arsenic induces sister chromatid exchange (SCE) and chromosome aberrations in vitro but does not cause gene mutations. It does not directly affect DNA but rather appears to inhibit DNA repair processes. Both AsIII and AsV are co-mutagenic and inhibit the growth of cells in vitro. AsIII is about ten times more potent than AsV.

Although arsenic does not appear to induce chromosome aberrations in vivo in experimental animals, several studies suggest that humans exposed to high levels have higher frequencies of SCE and chromosomal aberrations in peripheral lymphocytes.

The toxic effects of arsenic on various organ systems, including vascular, neurological, dermal, haematological, hepatic and renal systems, all occur at doses higher than the dose correlated with the induction of carcinogenicity. Therefore, the most significant toxicological endpoint in evaluating the health risk of ingested arsenic is the long-term effect of skin cancer.

The International Agency for Research on Cancer (IARC) states that there is "sufficient evidence that inorganic arsenic compounds are skin and lung carcinogens in humans. The data suggesting an increased risk for cancer at other sites are inadequate for evaluation" (IARC, 1980). Animal studies generally have not shown increased rates of tumour formation. The only positive studies involve particulate arsenic that has been instilled intratracheally in hamsters. The mechanism(s) by which arsenic induces cancer is not known.

There is clear evidence that workers exposed to occupational levels of arsenic dust through inhalation in non-ferrous smelters have increased rates of lung cancer. Studies from Taiwan and Mexico show a strong link between arsenic in drinking water and skin cancer. Studies in the USA have not found a similar relationship. This is ascribed to lower exposures and possibly increased sensitivity to arsenic in the Taiwanese and Mexicans because of a poorer diet. There is a suggestion in the Taiwanese studies of a linkage between ingested arsenic and internal cancers.

2. Cobalt

Cobalt exists in nature as the metal and in two valence states - CoII and CoIII, which forms 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).

Man absorbs about 25% of ingested soluble cobalt salts, with great individual variation. In general, more than 75% of the amount of cobalt in food will be ingested. Cobalt and its salts are readily absorbed in the gastro-intestinal tract but the degree is dependent on dose where the amount absorbed decreases with increasing dose. Absorption also seems to be dependent on diet. In animals where large doses of cobalt are given orally approximately 80% is excreted in the feces and the remainder in the urine. In humans, the absorbed cobalt is excreted predominately through the urine, with about 10% in the feces and some in sweat. The initial excretion is rapid, but some may be retained for several months.

Health effects

Toxicological properties of cobalt have been previously reviewed (Stokinger, 1981; MOL, 1988; Domingo, 1989). Cobalt salts, like copper salts, in sufficiently large doses 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 effects and cardiotoxic effects. Polycythemia (elevated red blood cell level) is the characteristic response of most mammals, including humans, to ingestion of excessive amounts of cobalt.

Small epidemics of severe cardiomyopathy have been observed resulting from heavy consumption of beer to which cobalt compounds had been added. Doses may have been as high as 10 mg per day.

Inhalation of cobalt and compounds in the 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 .

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.

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 the exposure has been to a mixture 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 nonoccupational exposed individuals.

Exposure Assessment

1. Vegetable Consumption Patterns

The measured levels of arsenic and cobalt in garden produce in the Cobalt survey taken in the summer of 1990 and are summarized in Table 1. Sampling was performed at 4 garden locations and results provided represent triplicate samples taken for the specified vegetables. The vegetables samples are of the root variety, namely potato, beet and carrot. Such varieties will generally have greater accumulation of metal from soil than leafy varieties. Three of the four sampling locations show levels for Co and As at or below the limit of detection for each of the vegetables sampled. For these, the limit indicated is assumed as a worst case. The Box 251 garden location had detectable quantities. This site is located adjacent to a horsitorical miming site. For the Box 251 location, the average of the triplicate samples (on a fresh weight basis) are utilized for the calculation of intakes. Where the maximum level reported is somewhat higher than the mean value, a separate intake estimate is also presented.

Modelling of the fruit and vegetable exposure route requires two sets of information

1) fresh weight concentration of the vegetables in question

2) estimate of the consumption rates for homegrown produce.

The estimated human intake is given by the algorithm

$$\text{Intake} = C_{food} \times IR$$

where C is the contaminant concentration in food (fresh weight, mg/kg) and IR is ingestion rate in kg/day.

The data provided are measured on a dry weight basis. In order to model intake through consumption of these vegetable types, it is first necessary to estimate the metal concentrations on a fresh weight basis. To obtain this, multiplication by an appropriate conversion factor is applied. The calculated fresh weight concentrations of arsenic and cobalt are shown in Tables 2 and 3 respectively. The vegetable specific factors utilized are derived in the revised MOE Field Investigation Manual (D. McLaughlin, 1991) based upon "normal" per cent moisture content as cited in the Agriculture Handbook No. 8, U.S. Dept. of Agriculture.

The assumptions regarding consumption of vegetables are as follows. The amounts and types of produce that people might consume from a backyard garden are influenced by the size of the garden, the yields of the crop grown, and the preferences of the individuals. The actual dose received will likely be lowered by the reductions

Table 1. Analytical Results: Levels of Arsenic and Cobalt in Garden Produce in Cobalt, Ontario.

Sampling Location (residence)	Field Sample	Vegetable Species	Cobalt (µg/g DW)	Arsenic (µg/g DW)
Trailer 9	12617	Potato	<.2<W	<0.20<W
	12618	Potato	<0.2<W	<0.20<W
	12619	Potato	<0.2<W	<0.20<W
Box 251	12620	Beet	9.4	4.80
	12621	Beet	10.0	5.00
	12622	Beet	11.0	2.00
	12623	Carrot	2.4	2.20
	12624	Carrot	2.1	1.60
	12625	Carrot	1.3	2.10
Box 686	12626	Beet	.2<T	<0.20<W
	12627	Beet	.2<T	<0.20<W
	12628	Beet	.2<T	<0.20<W
	12629	Carrot	<.2<W	<.20<W
	12630	Carrot	<.2<W	<.20<W
Ouevillon	12631	Carrot	.2<T	<.20<W
	12632	Beet	.7<T	<.20<W
	12633	Beet	.7<T	<.20<W
	12634	Carrot	<.2<W	-
	12635	Carrot	<.2<W	-

W= minimum measurable value
T= minimum reliable value

Table 2. Estimated Fresh Weight Concentrations of Arsenic in Vegetables

Sampling Location	Vegetable Type	Conversion Factor	Arsenic Concentration	
			Dry Weight (µg/g)	Fresh Weight (µg/g)
Trailer 9	Potato	0.20	<0.20<W	0.040
			<0.20<W	0.040
			<0.20<W	0.040
Box 251	Beet	0.13	4.80	0.62
			5.00	0.65
			2.00	0.26
	Carrot	0.12	2.20	0.26
			1.60	0.19
			2.10	0.25
Box 686	Beet	0.13	<0.20<W	0.026
			<0.20<W	0.026
			<0.20<W	0.026
	Carrot	0.12	<.20<W	0.024
			<.20<W	0.024
Ouevillon	Carrot	0.12	<.20<W	0.024
	Beet	0.13	<.20<W	0.026
			<.20<W	0.026
	Carrot	0.12	-	ND
			-	ND

ND = not determined

Table 3. Estimated Fresh Weight Concentrations of Cobalt in Vegetables

Sampling Location	Vegetable Type	Conversion Factor	Cobalt Concentration	
			Dry Weight (µg/g)	Fresh Weight (µg/g)
Trailer 9	Potato	0.20	<.2<W	0.04
			<0.2<W	0.04
			<0.2<W	0.04
Box 251	Beet	0.13	9.4	1.2
			10.0	1.3
			16611.0	1.4
	Carrot	0.12	2.4	0.28
			2.1	0.25
			1.3	0.16
Box 686	Beet	0.13	.2<T	0.03
			.2<T	0.03
			.2<T	0.03
	Carrot	0.12	<.2<W	0.03
			<.2<W	0.03
Ouevillon	Carrot	0.12	.2<T	0.03
	Beet	0.13	.7<T	0.09
			.7<T	0.09
	Carrot	0.12	<.2<W	0.03
			<.2<W	0.03

ND = not determined

of concentrations in produce that result when produce is washed, peeled and boiled.

According to the produce module of the AERIS (Aid for Evaluating the Redevelopment of Industrial Sites) Model for soil-related exposures, the yields of various types of crops generally range from 0.3 to 2.6 kg/m^2. For a mixture of crops that might be found in a backyard garden, a yield of 1.4 kg/m^2 has been recommended, although it is possible to achieve higher yields with special techniques or extraordinary efforts. The model assumes that a garden area is 30 m^2 with a yield of 1.4 kg/m^2; this gives a total of 42 kg, which represents 13% of the vegetables and fruits that one adult and one child would consume in one year. If one assumes a family size of four, then approximately 7% of fruits and vegetables would be garden-grown. This also assumes that the family consumes all of the vegetables grown.

Nutrition Canada Survey (1972) data for Ontario indicate average daily consumption of fruits, fruit products and vegetables combined as 372 g/day (26% of total diet) for young children (one to four years) and 489 g/day (32% of total diet) for adult males, which convert to 136 kg/year and 178.5 kg/year respectively. Leaf and root vegetable consumption (excluding potatoes) in Ontario on average is 152 (144-180) grams/day for male adults; 83 (63-131) grams/day for female adults and 49 grams/day for children (1-4 years) (NCS,1972). Average Ontario intakes for potatoes + other vegetables (this excludes fruits) are approximately 98 grams/day for children (1-4 yrs.) and 326 g/day for adult males (20-39 years).

If 7% of fruit and vegetable consumed are homegrown, then average consumption of homegrown produce would be 26 g/day for children (1.8% of diet) and 34 g/day for adults (2.2% of diet) averaged over a year. This would hold assuming that the Cobalt gardens involved are of a size of roughly $30m^2$.

One can also look at the amounts of the specific types of vegetables involved which people might consume. Suggested mean daily intake figures for beets, carrots and potato are shown in Table 4 below.

The exposures to arsenic and cobalt are calculated using three vegetable consumption scenarios. For evaluation of chronic toxicity, time-averaged exposures are required; whereas incidental exposure estimates are required for consideration with acute effects level and acceptable daily intakes. The differences also provide an indication of some of the uncertainty inherent in making such estimates. These models are:

1.) low-level chronic exposure model. The vegetables sampled are representative of a varied garden produce which is consumed at an rate which is averaged over a year. Considers that only a

certain fraction of fruits and vegetables will be provided by the garden over the course of the year. Assumes freezing of some vegetables for later consumption.

2.) limited low level chronic exposure model (only beets or carrots are eaten). The root vegetables sampled are consumed at the typical consumption rates, averaged over a year (table 4). All beets and carrots cor the year are provided by the garden

3.) acute exposure model. Beets/carrots account for all of the daily potatoes + other vegetables intake on particular days. Fruits are not included in this consumption value. This scenario is provided to account for those days, particularly during growing season, where all of the daily intake of vegetables is taken from the gardens. For example, on a particular day, an adult consumes a meal or two consisting of roughly 300 grams of carrots/beets in total.

The major difference of model 3 from models 1 and 2 is that the exposures are not averaged over the year but rather try to look at the plausible scenario of all vegetables being consumed from the garden for short periods. Models 1 and 2 estimate chronic exposure which is appropriate to compare to dose-response information based on chronic exposure.

Table 4. Average Vegetable-specific Food Consumption Rates

Vegetable type	Average Food Intake (grams/person/day)	Reference
Potatoes	Child (1-4 years): 61 Males (20-39 yrs): 174 (87-235) Females (20-39 yrs): 83 (63-131)	NCS, 1972
Beets	Child (0.5-4 yrs): 0.43 Adults (20 yrs +): 1.8	HWC, unpublished data
Carrots	Child (0.5-4 yrs): 8.14 Adults (20 yr+): 14.19	HWC, unpublished data

2. Estimated Arsenic Intakes

Estimated arsenic intakes under the three different consumption scenarios are provided in Table 5. For the three plots not exhibiting levels above detection, estimated intakes are relatively low using all three models, ranging from as little as 0.01 µg/day/child if considering beets alone (Box 686) to a maximum of 13 ug/adult/day if the levels in potatoes are encountered for the total daily consumption of all vegetables.

At the Box 251 location levels above detection were encountered for bot beets and carrots types, with the maximum fresh weight concentrations in beets. Estimated intakes based on model 2, where the consumption of beets alone averaged over a year is considered, provide the lowest intake estimate of 0.22 µg/child/day and 0.9 µg/adult/day. If concentrations in beet are considered to represent all vegetables consumed, intakes for children range from 16.9 µg/day (model 1) to 63.7 µg/day if beets comprise the total daily consumption of vegetables for a particular meal (model 3; assumes 100 % homegrown). Similarly, respective adult intakes based on beets are 22.1 and 212 µg/adult/day. Intakes estimates are approximately one-third of these values based on the levels in carrots.

3. Estimated Cobalt Intakes

Estimated cobalt intakes under the three different consumption scenarios are given in Table 6. For the three plots not exhibiting levels above detection, estimated intakes are quite small using all three models. As with arsenic, detectable quantities of cobalt are seen in both root vegetable types at Box 251. Calculated average intakes from this garden based on model 1 range from 5.5 -34 µg/day for a child and 7.8-44.2 µg/day for adults. Based on model 3, an occasional daily consumption of beets alone would provide upwards of 400 µg/day for an adult; carrrots would provide up to 75 µg cobalt to adults.

Risk Characterization

1. Arsenic

There are relatively few regulations or tolerances limits relating to allowable levels of arsenicv in foodstuffs. In Canada, there are no specific tolerance limits for arsenic in fruits or vegetables. The tolerance limit for arsenic in fruit juices and nectors is 0.1 ppm under the Food and Drug Act.

In order to assess the exposure to arsenic predicted above, it is useful to examine what is generally known about human exposure to this substance. Arsenic is a known multimedia contaminant. In other words, it is expected that individuals will normally be exposed to

Table 5. Estimated intakes of arsenic from background vegetable consumption.

Sampling Location	Vegetable Type	Average Arsenic Concentration (μg/g, FW)	Estimated intake (μg/day), A=adult, C=child		
			Model 1[1]	Model 2[2]	Model 3[3]
Trailer 9	Potato	0.040	1.0 (C) 1.4 (A)	2.4(C) 6.9(A)	3.9(C) 13.0(A)
Box 251	Beet	0.51	13.3 (C) 17.3 (A)	0.22 (C) 0.91 (A)	50.0 (C) 166 (A)
		(high, 0.65)	16.9 (C) 22.1 (A)	0.3 (C) 1.2 (A)	63.7 (C) 212.0 (A)
	Carrot	0.23 (high, 0.26)	6.0 (C) 7.8 (A)	1.9 (C) 3.3 (A)	22.5 (C) 75.0 (A)
Box 686	Beet	0.026	0.68 (C) 0.88 (A)	0.01 (C) 0.05 (A)	2.6 (C) 8.5 (A)
	Carrot	0.024	0.62 (C) 0.82 (A)	0.21 (C) 0.34 (A)	2.4 (C) 7.8 (A)
Ouevillon	Carrot	0.024	0.62 (C) 0.82 (A)	0.21 (C) 0.34 (A)	2.4 (C) 7.8 (A)
	Beet	0.026	0.68 (C) 0.88 (A)	0.01 (C) 0.05 (A)	2.6 (C) 8.5 (A)
	Carrot	-	-	-	-

1. Average consumption of homegrown produce throughout year: child= 26g; adult= 34g.

2. See Table 4 for ingestion rates, time-averaged.

3. Average daily consumption of potatoes and vegetables in Ontario based on the Nutrition Canada Survey: child (1-4yrs)= 100 g; adult= 326g.

Table 6. Estimated intake of cobalt from backyard vegetable consumption.

Sampling Location	Vegetable Type	Average Cobalt Concentration (μg/g, FW)	Estimated intake (μg/day), A=adult, C=child		
			Model 1	Model 2	Model 3
Trailer 9	Potato	0.04	1.0 (C) 1.4 (A)	2.4(C) 6.9(A)	3.9(C) 13.0(A)
Box 251	Beet	1.3 (high, 1.4)	33.8 (C) 44.2 (A)	0.55 (C) 2.3 (A)	127.4 (C) 423.8 (A)
	Carrot	0.23	5.5 (C) 7.8 (A)	1.9 (C) 3.3 (A)	22.5(C) 75.0(A)
		(high, 0.28)	7.3 (C) 9.5 (A)	2.3 (C) 3.9 (A)	27.4 (C) 91.3 (A)
Box 686	Beet	0.03	0.8 (C) 1.0 (A)	0.01 (C) 0.05 (A)	2.9 (C) 9.7 (A)
	Carrot	0.03	0.8 (C) 1.0 (A)	0.24 (C) 0.42 (A)	2.9 (C) 9.7 (A)
Ouevillon	Carrot	0.03	0.8 (C) 1.0 (A)	0.24 (C) 0.42 (A)	2.9 (C) 9.7 (A)
	Beet	0.09	2.2 (C) 3.0 (A)	0.03 (C) 0.16 (A)	8.7 (C) 29.1 (A)
	Carrot	0.03	0.8 (C) 1.0 (A)	0.24 (C) 0.42 (A)	2.9 (C) 9.7 (A)

arsenic through a variety of exposure pathways including air, dusts, food and drinking water. Exposure assessment for Ontario (MOE, 1991) suggests that ingestion of inorganic arsenic is predominantly in food accounting for upwards of 97 % of typical urban exposures (see appendix 1). Total exposures are approximately 14 µg/day for children and 18 µg/day for adults on average.

Background intakes of arsenic in food in Ontario have been estimated to range from 13 ug/day for children to 23.1 µg/day for adolescent males. Dabeka and co-workers in a duplicate diet study of Canadian adults report mean dietary intakes of 16.7 µg/day or 0.26 µg/kg/day Dabeka et al., 1987). The range of intakes reported was 2.6 to 101 µg/day. Typical intake values of 50 µg/day (U.S. EPA , 1983) and 62 µg/day (Gartrell et al., 1985) in adults have been reported in the United States.

The estimated arsenic exposures for the Trialer 9, Box 686, and Ouevillon garden vegetables falls within and\or below the range of normal dietary intakes for adults and children.

With respect to the Box 251 site, the estimated intakes based on average daily intakes of beets alone or carrots alone are quite small (Model 2). If one assumes that beets or carrots are representative of vegetables consumed and it is assumed that only a fraction of yearly consumption is yielded by the garden (model 1), then arsenic intakes are estimated to range from 6.0 -17.3 µg/day for a child; and from 7.8-22.1 µg/day for adults. If considered as additional intake above background this would represent additional exposure of at least 50 % to slightly greater than double total exposure. Calculations based on all vegetable intake during the day being composed of beet or carrot yield higher predicted intakes. These values are about 4-10 X higher than background exposure for adults.

The exposure values determined using model 3 represent potential short term exposures which may occur on intermittent days. These may be compared against the World health Organization provisional maximally acceptable daily intake of 2µg inorganic arsenic per kg body weight based on drinking water. For an adult of 70 kg, this dose means an ingested amount of 140 µg inorganic arsenic per day and for a child of 15 kg, an equivalent of 30 µg per day. The estimated intakes at Box 251 site exceed this value for adults based on both beets and carrot, and for children based on beets. Exposure estimates based on the other sites fall well below the permissable intake.

Arsenic in certain food types is known to be in a chemically complex form that is relatively toxicologically inert and is rapidly excreted intact. It is thought that the amount of absorbed food arsenic considered to be toxicologically significant is relatively small compared to total arsenic intake (U.S. EPA, 1983). In fruits and vegetables only 10-15% of the total arsenic is

suggested to be of the more toxic inorganic form. However, in light of the lack of precise breakdown of the arsenic species, the intakes modelled here assumes that the arsenic measured is in inorganic form. This is a conservative measure which may tend to overestimate actual inorganic arsenic exposure.

Chronic low level exposure to arsenic can be assessed using mathematical dose-response models. Such approaches assume that there is no level below which there is not some incremental amount of carcinogenic risk. For non-threshold carcinogens, risks may be estimated as the incremental probability of an individual developing a particular cancer over a lifetime as a result of exposure to a chemical agent. A skin cancer risk calculation based on the Box 251 data is provided (Table 7). The highest exposure estimate for beets based on longer averaging periods (Model 1) is used. The calculated incremental risk is 5×10^{-4}. This risk level is moderately greater than risk levels which are generally considered in the negligible range (10^{-5} to 10^{-6}). This value may overestimate risk as levels in beets may be higher than other vegetables. It may underestimate risk if the consumer eats a greater proportion of their fruits and vegetables from the garden than the model assumes.

In summary, the consumption of the root vegetables from the garden at location Box 251 may result in intakes that exceed the WHO permissable daily intake for inorganic arsenic. The individual cancer risk estimates based on long term exposure provide estimates which are moderately above the negigible range. Taken together, potential exposures exceeding the tolerable intake, and which may be several fold above typical total intake rates, are of concern.

Consumption at the other sites in Cobalt would not appear to pose a significant hazard with respect to arsenic.

2. Cobalt

Like arsenic, exposures to cobalt come primarily from food (i.e greater than 95%). There is only meagre information available on the background daily intake from food. The average intake is around 300 μg/day, and the maximum close to 600 μg/day (ICRP, 1984; MOL, 1988; Stokinger, 1981b). Dietary intakes as high as 1800 μg/g have been suggested.

Cobalt is an essential dietary element and is often provided in vitamin tablets. It is present in trace quantities in all foods. Vegetables are common sources of cobalt, with typical concentrations ranging from 8 to 100 μg/g dry weight.

No tolerance limits for cobalt in foodstuffs in Canada were located.

TABLE 7 CANCER RISK ESTIMATE OF INORGANIC ARSENIC IN VEGETABLES

Sample	Chronic Daily Intake[1] (μg/kg/day)	CDI[2] Adjust for Absorption (μg/kg/day)	Type of Cancer	Weight of Evidence[3]	Slope Factor[4] (μg/kg/day)	Pathway[5] Risk Level
Box 251 (beets)	0.40	0.32	skin	A	1.5×10^{-3}	5×10^{-4}

1. The chronic daily intake is the estimated intake from that pathway daily as averaged over a lifetime and adjusted for body weight.
In this case, $CDI = \frac{\text{intake(child) x 7 years}}{\text{15 kg x 70 years}} + \frac{\text{intake(adult) x 63 years}}{\text{70 kg x 70 years}}$

2. Where absorption from the media ingestion is different from that which the cancer slope factor is derived, the CDI is adjusted for absorption. Absorption from vegetables is assumed at 80%.

3. A = U.S. EPA weight of Evidence Cancer classification (U.S. EPA, 1991). Human carcinogen.

4. Slope factor is the maximum likelihood estimate of skin cancer risk due to 1 μg/kg/day of arsenic intake, based on U.S.EPA cancer potency for drinking water of 5×10^{-5} $(\mu g/L)^{-1}$. The slope factor is based on dose administered in drinking water and an assumed 100% absorption. These risk estimates are based on a dose-response model that assumes linearity at low doses and would overestimate risk if risk decreases faster than linear or if a threshold exists for arsenic-induced skin cancer (U.S.EPA, 1988).

5. Cancer risk estimates are expressed as one significant figure only.

The calculated intakes of cobalt from all sites, regardless of the model used, is within the range of background dietary intake. The highest intake based on beets at Box 251 (423 µg/day, adult) would be sightly greater than background food intake, and thus total daily intake may be approximately doubled to perhaps 700 µg (0.7 mg). This assumes consumption of 326g of beets/day.

There is relatively little dose-repones information against which to assess this intake level. No specific reference doses or tolerable intake values for chronic human exposure were found in the scientific literature. Limits for cobalt in food have not been established. Anecdotal information regarding cobalt poisoning in beer-drinkers suggested amounts ingested of up to 10 mg/day. Animal studies in general indicate lethal levels by the oral route of a least 100 mg/kg/day and systemic oral toxicity at 1 mg/kg/day.

Based upon the above considerations, it is not considered that these exposures to cobalt would lead to adverse health impacts provided that consumption of the root vegetables is not excessive.

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