Chapter 12

Pollution and Human Health

Contents

12.1. Introduction

The chapter on human health will describe the levels of contaminants in humans and their effects on human health. It has not been written as an overview of the general health of the peoples of the Arctic. Rather, this chapter is an evaluation of the current knowledge of the consequences to Arctic peoples of environmental exposure to priority contaminants as defined in the AMAP mandate. Indirect health implications of climate change, oil pollution, and waste sites are treated in other chapters of this report.

Many factors contribute to health and illness of human populations living in the Arctic, these include socioeconomic conditions, availability of health services, societal and cultural factors, individual lifestyles and behaviors, and genetics. Environmental contaminants, although just one of these factors, can have a significant influence on human health. There are numerous population groups in the Arctic of which many are indigenous. These groups may be more threatened by contaminants than immigrants from the south since they generally live their whole lives in the Arctic and subsist to a large extent on local food. This chapter, however, deals with the contaminant-related health implications for all Arctic inhabitants.

Observations from most parts of the Arctic ecosystem indicate that it is far from pristine. Pollution, both local and due to long-range transport of contaminants, is prevalent at levels which pose a concern for human health. For some indigenous populations of the Arctic, blood mercury levels and concentrations of persistent organic pollutants in blood and fat are 10-20 times higher than those found in most temperate regions. Although there are currently no confirmed diagnoses of illness in these populations which can be causally linked to contaminants, the high concentrations of toxic substances are a cause for concern.

It is important to be able to estimate the potential risk associated with pollution and develop appropriate preventive measures with due respect for local cultural traditions. One prerequisite for this is the continued monitoring of concentrations of pollutants in humans in order to determine spatial and temporal trends. This monitoring has been initiated through the 1994-96 AMAP data collection initiative and should be continued in the future.

Environmental medicine is a discipline which studies the impact on human health of pollutants in air, water, and food, together with physical factors such as radiation exposure or noise. Some of these factors are more relevant for populations in the Arctic than elsewhere, and this is reflected in the AMAP Human Health Monitoring Program. The core program consists of monitoring for heavy metals (mercury, lead, cadmium), essential trace elements (zinc, selenium, copper), and chlorinated organic contaminants (including pesticides, dioxins and PCBs) in pregnant women and newborn babies. In addition to this core program which should be followed by all countries, the monitoring of other contaminants of concern, such as nickel, arsenic and radionuclides, in specific parts of the circumpolar Arctic should be considered on a subregional basis.

Other contaminants of growing health concern, such as heterocyclic amines and some less persistent contaminants, are not addressed in this report as they are not included in the AMAP mandate, but should be considered for relevance to the Arctic in future programs.

Human exposure to pollutants through the diet is of central concern in the Arctic. Many of the relevant pollutants, including mercury and persistent organic pollutants, are biomagnified through Arctic food chains. Because humans in the Arctic traditionally feed on other carnivores such as seals and whales, they are at the top of food chains which include other top level predators. Thus, indigenous peoples consuming traditional diets are more likely to be exposed to higher concentrations of these pollutants than populations elsewhere in the world. However, the traditional diet is often important for the social, spiritual, and cultural identity of indigenous peoples. The negative attitude which can develop as a result of, e.g., bans on consumption of local foods, can disrupt this identity and can have other effects on health. Furthermore, the traditional diet including marine mammals, birds and fish is rich in vitamins, minerals, protein and fatty acids. The n-3 fatty acids which are widely marketed in the south as 'fish oils' are known to prevent arteriosclerosis. This component of the diet is probably one of the reasons for the very low mortality from ischemic heart disease among indigenous Arctic populations.

As described in chapter 5, indigenous peoples of the circumpolar region represent different ethnic, historical and cultural backgrounds. In addition, their living conditions vary according to climatic conditions, socio-economic conditions, and their access to traditional and market foods. These differences will influence the susceptibility of the various circumpolar population groups to contaminant exposures and their

Table 12·1. Toxicological characteristics of persistent organic pollutants.

Contaminant	Acute oral lethality (LD_{50} rats, mg/kg bw)	Human carcinogenicity (IARC 1987)[p]	Acceptable / tolerable daily intakes, μg/kg bw/d	Main sources of exposure
DDT	113[a]	2B	20[b]	Fish, marine mammals
DDE	880[a]	n.a.[c]	20[b]	
Toxaphene	80-90[d]	2B	0.2[e]	
Dioxins (2,3,7,8-TCDD)	0.022-0.340[f]	2B	0.00001[g]	
Furans (2,3,7,8-TCDF)	n.a.	n.a.	0.00001[g]	
Mirex	365-3000[h]	2B	0.07[e]	
Chlordane	127-430[i]	3	0.05 (total)[j]	
Heptachlor	71[k]	3	0.1 (total)[l]	
HCH	88 (γ-HCH)[m]	2B (mixture)	0.3 (total)[j] 8 (γ-HCH)[n]	
PCBs	1010-4250 (various Aroclors)[o]	2A	1.0 (total PCBs)[e]	
HCB	1000-10000[q]	2B	0.27[e]	
PAH (benzo[a]pyrene)	n.a.	2A	n.a.	Tobacco smoke, smoked foods, home fuel combustion (wood)

a. ATSDR 1994a. b. WHO 1984b. c. n.a.: not available. d. ATSDR 1994b. e. Provisional TDI (PTDI) Health Canada 1996. f. WHO 1989b. g. WHO 1992b. h. WHO 1984c. i. ATSDR 1993a. j. TDI Health Canada 1996. k. ATSDR 1993b. l. WHO 1991c. m. ATSDR 1992. n. WHO 1989c. o. ATSDR 1995. p. Group 1: The agent is carcinogenic to humans. Group 2A: The agent is probably carcinogenic to humans. Group 2B: The agent is possibly carcinogenic to humans. Group 3: The agent is not classified as to its carcinogenicity to humans. Group 4: The agent is probably not carcinogenic to humans. q. Government of Canada 1993.

related effects. Consequently, no definitive description of the interrelationship between environmental contaminants and human health for any particular group of northerners can be provided. This chapter, therefore, presents a description of the available information on specific exposure conditions and possible human health consequences for seven of the eight Arctic countries individually.

Previous chapters of this report have presented data on contaminant concentrations in wildlife. Ideally, this information could be combined with dietary intake data to estimate human exposure and make quantitative risk assessments. Such an attempt, however, would be premature. Comprehensive and quantitative dietary data are lacking for nearly all Arctic communities. In addition to measurements of food intake, such data would need to include information on the age of animals consumed, storage and cooking methods, seasonal variations in amounts and types of food consumed, and other factors influencing contaminant burden in foods as they are consumed. The absence of this information prevents anything more than a general analysis of dietary intakes of contaminants. In addition, scientific understanding of the effects of long-term low-dose exposure to contaminants, either individually or in combination, is poor, such that even if quantitative estimates of exposure were available, risk characterization would remain imprecise. Since both accurate dietary data and a sound understanding of the risk of chronic exposure to mixtures of contaminants are unavailable, any attempt to quantify human exposures in the Arctic and then assess their associated risks would be of low predictive value. Inclusion of such speculative risk estimates in this report would either imply a greater validity than is justified, or cast doubt on other data that are presented. For this reason, no attempt to make such calculations has been undertaken. The need for such assessments, however, is identified and recommendations are made for future work to overcome the obstacles outlined above.

The effects of environmental pollutants on health are most often subtle, since they usually occur at concentrations which are not expected to result in acute toxic symptoms. What might be expected are long-term, possibly transgenerational, effects on the neurologic and reproductive systems. These effects are confounded by many other factors and can be difficult to identify even in long-term epidemiological studies in large populations. In the small Arctic populations, it may prove impossible to conduct epidemiological studies with enough power to detect the effects of concern. It is therefore important to recognize that the paucity of health data or the absence of overt illness or malfunction does not imply that the exposure of peoples in the Arctic to contaminants is without effects. Even biochemical changes in blood and tissue must be regarded as undesirable effects of pollution.

Existing guideline values for a range of contaminants are presented in Annex Tables 12·A1 to 12·A11.

12.2. Toxicology and health effects of contaminants

The toxicological considerations, as described in the following sections, have been restricted to include information that is directly relevant for human health. Additional descriptions of pathways and ecotoxicological aspects of the contaminants can be found elsewhere in the report, see chapters 6, 7, 8 and 10.

12.2.1. Toxicology of persistent organic pollutants (POPs)

Availability of information on contaminant levels in the tissues of northern residents is very recent, and the quantity is increasing rapidly. While trends are difficult to determine, there is a clear indication in the National Reports (section 12.4) and in the comparison of data from an international study (section 12.5) that a number of persistent substances are significantly elevated in the tissues of several Arctic ethnic groups.

This section provides a brief overview of the toxicological concerns related to a variety of POPs. A summary of the toxicological findings are provided in Table 12·1. Detailed descriptions of the past and present uses, sources, transport, fate, and presence of these substances in biota other than humans has been covered in earlier chapters of this report, and are not repeated here.

Assessing the human health impacts of exposure to POPs is a very difficult task. Human populations are always exposed to mixtures of POPs in the ecosystem, never to single compounds. Hence, toxicological risk assessments that make use of animal test data on individual chemicals rather than mixtures of chemicals, and their applicability to humans, is frequently in question. The actual levels of individual contaminants in the mixture of POPs to which populations are exposed varies by region (because of differences in environmental occurrence and food consumption patterns), making

comparisons of possible effects between populations very difficult. Human exposures to POPs are usually to lower levels than those chosen for animal studies. Humans are also typically exposed to these contaminants over their entire lifetime, commencing with conception, and not merely for the limited life stages chosen for most animal studies. Finally, confounding factors of lifestyle, diet, age, reproductive status, gender, and general health also affect how individual POPs will influence the onset of disease or adverse effects.

12.2.1.1. **DDT/DDE/DDD**

The use of DDT has been declining in the temperate regions of the Northern Hemisphere since the 1960s, and especially since the mid-1970s when it was banned by many western nations. Its persistence in the environment, and its continued entry into the Arctic region via long-range atmospheric transport and in some river systems, has meant that it is detectable in almost all compartments of the ecosystem and in human tissues. Levels of total DDT in human tissue in the Arctic are considerably higher than those in southern populations, reflecting the greater consumption of high trophic level species for food.

DDT and its metabolites are stored in fatty tissue and are excreted very slowly, primarily via urine and feces. Because DDT and its metabolites are found in breast milk, 'excretion' also occurs during breast feeding. DDT can readily cross the placenta.

Acute lethal (LD_{50}) oral doses in test animals range between approximately 115-800 mg/kg bw (body weight). No-observed-adverse-effect levels (NOAELs) for chronic exposure to DDT, DDE and DDD for most mammalian test species range between 10-100 mg/kg bw/d for respiratory, cardiovascular, gastrointestinal, hematological, hepatic and renal outcomes. However, the more serious effects (neurological, developmental, reproductive and carcinogenic) have lowest-observed-adverse-effect level (LOAEL) values more in the 8-50 mg/kg bw/d range for chronic exposure (ATSDR 1994a). Some forms of DDT are considered to have weak estrogen-like responses with potencies relative to estradiol of between approximately 0.01 and 0.0001 (Soto *et al.* 1992). This estrogenic effect is probably responsible for DDT impacts on reproduction in animals (ATSDR 1994a). DDE has recently been shown to be a potent androgen receptor antagonist (Kelce *et al.* 1995).

In humans, acute lethal exposures to DDT are probably greater than 250 mg/kg bw. Very little data on the effect of chronic exposure is available. Long-term exposure of volunteers to amounts up to approximately 0.6 mg/kg bw/d did not lead to any observable neurological signs (Hayes *et al.* 1956). The World Health Organization (WHO) have proposed a Tolerable Daily Intake (TDI) of 20 μg/kg bw/d for DDT (including metabolites).

Epidemiology studies have not established an association between DDT exposure and cancer, even though cancer is an outcome of long-term animal-feeding studies. The International Agency for Research on Cancer (IARC) has classified DDT and DDE as 'possibly' carcinogenic to humans based on evidence from animal studies.

Suppression of reflexes in neonates appears to be associated with levels of DDE in breast milk exceeding 4 μg/g lipid, however, it has not been substantiated that DDE is the causative factor (Rogan *et al.* 1986). Elevated levels of DDE in human breast milk (≥3 μg/g lipid) have been correlated with a shortening of breast-feeding duration, and inhibition of lactation was hypothesized as the cause (Rogan *et al.* 1987).

12.2.1.2. **Toxaphene**

Toxaphene, also known as camphechlor, is an insecticidal mixture of over 670 chemicals. It enters the Arctic region via long-range atmospheric transport. Significant amounts have been reported in the Yukon Territory and coastal regions of Arctic Canada. It is commonly found in human tissue, however, its historical quantification has been compromised by difficulties in analysis and in the estimation of amounts of the various chemicals that make up toxaphene. As a result, comparisons using published data on toxaphene are difficult.

The lethal oral dose (LD_{50}) of technical toxaphene in rats is between 80 and 90 mg/kg bw (Gaines 1969), however a range of other effects, including death, can follow chronic exposure to levels of 20 to 30 μg/kg bw/d (ATSDR 1994b). Large doses of toxaphene are likely to affect the nervous system (seizures, tremors, convulsions, paralysis and both behavioral and biochemical effects), the liver (enzyme induction) and the kidney (enzyme release, fatty degenerative changes and focal necrosis). Intermediate exposure duration for toxaphene may affect the adrenals, the immune system and fetal development. This research base is mostly limited to short-term and intermediate-term (i.e., less than a lifetime) animal studies conducted with technical grade toxaphene. Although there is one chronic (eighty week) study, there are no suitable studies to confirm the effects of technical or environmentally available toxaphene on human populations. Because so little is known about the effects of long-term exposure to both technical and environmental toxaphene in animals and humans, there is considerable uncertainty over the applicability of a TDI. Canada uses 0.2 μg/kg bw/d. In the USA, the Agency for Toxic Substances and Disease Registry (ATSDR 1994b) has proposed an 'intermediate-duration oral exposure minimal risk level' of 1 μg/kg bw/d. The WHO has not proposed a TDI.

Animal studies suggest that toxaphene is an animal carcinogen. It has been classified by IARC (1987) as 'possibly' carcinogenic to humans. Toxaphene does not appear to be a very active estrogenic mimic (Soto *et al.* 1992). Recent data, however, indicate that toxaphene is a potent estrogen receptor antagonist (Jørgensen pers. comm.). Furthermore, it has been shown by Arnold *et al.* (1996), that the potency of the antagonistic effect of toxaphene was greater by an order of magnitude in synergistic interaction with other chlorinated contaminants such as dieldrin and endosulfan.

Toxaphene is readily absorbed. Intakes in Arctic populations are entirely dependent on the type and amount of food consumed. It is likely that the highest levels in food occur in narwhal and beluga blubber in the eastern Canadian Arctic (Kuhnlein *et al.* 1995a) and in some fish in a small area of the western Canadian Arctic. Toxaphene is known to be transported northward from past high-use areas in the southern USA. Measurements from other regions of the Arctic are few. Absorbed toxaphene is readily metabolized and excreted (90% in 24-36 hours), however, some constituents remain in fatty tissues for prolonged periods (ATSDR 1994b). Stern *et al.* (1992) report that the two most common compounds retained are an octachlorocamphene (T_2) and a nonochlorocamphene (T_{12}). There is no toxicological information available for T_2 and T_{12}.

12.2.1.3. **Hexachlorocyclohexanes (HCHs)**

HCHs are a group of organochlorine pesticides: alpha- (α-), beta- (β-), gamma- (γ-, more commonly known as lindane), delta- (δ-) and epsilon- (ϵ-) are the most stable isomers. Many countries in the world use large amounts of lindane. There are some minor registered uses for lindane in some circumpolar

jurisdictions (e.g., control of head lice). Like other POPs, most human exposure to HCHs results from food consumption.

γ-HCH (lindane) is the most toxic of the HCH isomers. Excessive exposures can affect the liver, the nervous system, the kidney, the reproductive system, and perhaps the immune system. IARC (1987) classifies it as 'possibly' carcinogenic to humans. No effects have been reported in populations exposed to low-level environmental concentrations. Exposure levels for HCH via consumption of store-bought food in several countries were approximately 0.005 μg/kg bw/d (α-HCH), 0.0003 μg/kg bw/d (β-HCH) and 0.03 μg/kg bw/d (γ-HCH) (Gorchev and Jelinek 1985). The TDI for total HCHs is 0.3 μg/kg bw/d (WHO 1991a).

HCHs, especially β-HCH, accumulate readily in fatty tissues and are excreted slowly via feces, breast milk and urine (WHO 1991a). Levels of α-, β- and γ-HCH in breast milk in the general populations of Europe, Canada and the United States are in the ranges 10-40 ng/g lipid, 10-500 ng/g lipid, and <1-10 ng/g lipid, respectively. The most recently measured 'background levels' of HCHs in blood, serum, plasma, milk and adipose tissues are relatively low compared to values reported during the 1960s and 1970s and much lower than levels reported from countries with extensive current usage. Because of its persistence, β-HCH is found at the highest level of the four isomers reported.

The exposure of babies, resulting from β-HCH concentrations found in breast milk, has been identified as a matter of concern by the WHO (1991a), but not as a reason to stop promoting breast feeding. The high β-HCH levels that have been found in some breast milk samples in countries using HCH, indicate that some infants may exceed the TDI of 0.3 μg/kg bw/d, temporarily and locally. The β-HCH concentrations in the blood of babies lie within the same range as those in the mothers.

12.2.1.4. **Mirex**

Mirex has never been used in any of the circumpolar jurisdictions. It has been manufactured and applied extensively in the continental USA and has become widely distributed via long-range atmospheric transport. It is now found at low levels in human tissues throughout North America (Government of Canada 1991) and Greenland (see section 12.4.2). It is highly persistent.

The acute lethal (LD_{50}) oral toxicity of mirex ranges from 365 to 3000 mg/kg bw in laboratory mammals. The effects of long-term, low-level exposure to mirex have not been extensively studied; the primary organs affected by mirex in laboratory species are the liver (at 50 μg/kg bw/d), kidneys, eyes and thyroid (IPCS 1984). Mirex is considered a 'possible' human carcinogen (IARC 1987) and also has fetotoxic and teratogenic effects on laboratory species in the 1-6 mg/kg bw/d range. There is no WHO TDI, however, the Canadian provisional TDI is 0.07 μg/kg bw/d (Health Canada 1996). Mirex is readily absorbed and stored in fatty tissues. Metabolism to photomirex is slow and elimination is mainly via feces and breast milk.

Because of its persistence and accumulation in the food chain, mirex levels in breast milk are above average for communities consuming high amounts of fish and marine-bird eggs (Dewailly *et al.* 1991).

12.2.1.5. **Chlordane, oxychlordane and *cis*- and *trans*-nonachlor**

Technical chlordane is a mixture of chlordane, nonachlor, heptachlor and other chlordane isomers. It is not registered for use in circumpolar jurisdictions and enters the Arctic ecosystem primarily via long-range transport through the atmosphere.

The acute lethal (LD_{50}) oral dose of technical chlordane is between 127 and 430 mg/kg bw in rats (ATSDR 1993a). High dose exposures affect the neurological and immune systems (no-effect levels are 4-6 mg/kg bw/d). Long-term exposure is likely to cause cellular changes in the liver at levels of approximately 0.5 mg/kg bw/d (WHO 1984a). Chlordane has been classified as a probable human carcinogen by the US Environmental Protection Agency (EPA) based on tumor identification in mice (LOAEL of 3.9 mg/kg bw/d for mice). The TDI for chlordane is 0.5 μg/kg bw/d (WHO 1984a).

There are few estimates of intakes for chlordane and its metabolites. US estimates indicate that only very small amounts are consumed, 0.002-0.005 μg/kg bw/d (Gunderson 1988).

Chlordane and its related compounds accumulate in fat and are found in human tissues. In general, only small amounts of chlordane are found in tissues. However, they often contain relatively larger (one or two orders of magnitude higher) amounts of *trans*-nonachlor and the metabolite oxychlordane. Excretion of chlordane is primarily through feces and breast milk.

12.2.1.6. **Dioxins (PCDDs) and furans (PCDFs)**

Polychlorinated dibenzodioxins and polychlorinated dibenzofurans are two structurally similar families of compounds that include 75 congeners (different compounds) and 135 congeners, respectively. These compounds enter the Arctic ecosystem almost exclusively via long-range atmospheric transport. Seventeen members of these two families of chemicals are highly toxic and contribute most to the toxicity of complex mixtures of dioxins and furans. The seventeen more toxic congeners in both families have chlorine substitutions in the 2, 3, 7 and 8 positions. These more toxic congeners cause a wide range of deleterious effects in laboratory animals, these effects varying significantly between species (Environment Canada and Health Canada 1990).

The acute lethal (LD_{50}) oral dose for 2,3,7,8-TCDD in mammals varies almost 10 000-fold (0.6 μg/kg bw for guinea pigs and 5051 μg/kg bw for hamsters). Common signs of acute and chronic toxicity in animals include loss of body weight and thymic atrophy. Longer-term exposure to doses below the LD_{50} can lead to discoloration and thickening of skin, skin eruptions, hair loss, liver damage, hematological changes and immune system dysfunction (IPCS 1989). Exposure of laboratory animals during gestation can lead to developmental deficits and altered sexual differentiation (Lindström *et al.* 1995).

Laboratory rats have developed cancer when exposed to 2,3,7,8-substituted tetrachloro- and hexachlorodioxins (NOEL for 2,3,7,8-TCDD is 1 ng/kg bw/d) (Kociba *et al.* 1978). IARC (1987) lists 2,3,7,8-TCDD as 'possibly' carcinogenic to humans. Dioxins and furans can directly affect reproduction, with a NOEL for intake of 2,3,7,8-TCDD for this outcome of 1 ng/kg bw/d (Murray *et al.* 1979). The majority of TDI values are within an order of magnitude (1-10 pg/kg bw/d). The WHO TDI is 10 pg 2,3,7,8-TCDD/kg bw/d (IPCS 1989). Recent studies, however, suggest that single exposures at close to these 'no-effect' doses leads to impairment of development of the reproductive system in male rats (Mably *et al.* 1992). The antiestrogenic capabilities of TCDD appear to be related to Ah-receptor mediated events (Zacharewski *et al.* 1991).

Due to the different relative toxicity of the seventeen more toxic dioxin and furan congeners, a set of international toxi-

city equivalency factors (I-TEFs) have been adopted, ranging from 1 for 2,3,7,8-TCDD, the most toxic congener, to 0.001 for octachlorodibenzodioxin, the least toxic congener (NATO 1988). By weighting (multiplying) the concentrations of different congeners in a mixture by their respective I-TEFs, and summing the resulting values, the International Toxic Equivalent (2,3,7,8-TCDD TEQ) value can be calculated. This allows the total dioxins and furans in a mixture to be expressed as a single 2,3,7,8-TCDD TEQ value. I-TEF values have also been established for other dioxin-like substances such as certain PCB congeners (see 12.2.1.7, below).

The general population is exposed to small amounts of complex mixtures of PCDDs and PCDFs and other organochlorines. An extensive analysis of adipose tissue samples, from a number of countries, has concluded that almost all humans contain TCDD at concentrations up to, and occasionally greater than, 3 pg/g in lipids (Travis and Hattemer-Frey 1991). These levels in the general population have not been associated with disease. In a few incidents, in the USA, Italy and Japan, where workers and others who have been exposed to very large amounts of a limited number of these compounds, individuals have developed chloracne, a skin disorder. There is also evidence that high level exposure to dioxins and furans can cause variations in serum lipid levels, other dermatological effects related to chloracne, microsomal enzyme induction and gastrointestinal alterations (Schulz *et al.* 1990). Other studies of high level occupational exposures have found associations with some types of cancer (Zober *et al.* 1990, Manz *et al.* 1991, Fingerhut *et al.* 1993, Bertazzi *et al.* 1996). Pluim *et al.* (1993) concluded that *in utero* and lactational exposures to PCDDs/PCDFs are capable of affecting the hypothalamic, pituitary, and thyroid regulatory system in human infants.

The best documented poisonings by PCDFs in humans are the Yusho and Yu-cheng incidents when rice oil was accidentally contaminated with polychlorinated biphenyls (PCBs) from electrical transformer fluid. The PCBs were heavily contaminated with PCDFs. Investigators have reported low birth weight, early tooth eruption, sensory losses, skin discoloration, swollen eyelids (Kuratsune *et al.* 1972) and retarded development (Rogan *et al.* 1986) in infants exposed transplacentally. It is likely that the PCDFs were the cause of many of the reported effects (Rappe *et al.* 1983, Rappe and Nygren 1984). The mean total intake of PCDFs by the Yusho and Yu-cheng patients has been estimated to be 0.9 μg/kg bw/d (Hayabuchi *et al.* 1979) or 3.3 ng 2,3,7,8-TCDD TEQ/kg bw/d (Ryan *et al.* 1990). The smallest amount of total PCDFs causing chloracne has been estimated to be 0.16 μg/kg bw/d (Hayabuchi *et al.* 1979).

Average daily intake of PCDDs and PCDFs over a lifetime is similar in most industrialized regions, between 2 and 10 pg 2,3,7,8-TCDD TEQ/kg bw/d for a 60 kg person (Birmingham *et al.* 1989). Back calculations from human tissue levels in Canada have confirmed this estimated intake, i.e., deriving likely intakes of 1.9 pg/kg bw/d (Environment Canada and Health Canada 1990). The mean PCDD and PCDF concentrations in the breast milk of Arctic and non-Arctic populations are similar: 10-20 pg 2,3,7,8-TCDD TEQ/g lipid (see Ryan *et al.* 1993 for Canada, Nygren *et al.* 1986 for Sweden, Schecter *et al.* 1987 for the USA). Breast milk levels in the Netherlands can be slightly higher: 30 pg 2,3,7,8-TCDD TEQ/g lipid (Koopman-Esseboom *et al.* 1994a).

12.2.1.7. **PCBs**

Polychlorinated biphenyls (PCBs) are a group of 209 structurally similar compounds (congeners) commercially produced as mixtures. Although PCBs have been released due to improper use, storage and disposal, or accidents at military sites in the Arctic, they have primarily contaminated the Arctic ecosystem through atmospheric transport from regions farther south. Their manufacture and new usage is banned in most circumpolar jurisdictions, but they are still present in older electrical transformers and at a number of contaminated industrial sites and waste sites throughout the Arctic.

The toxicity of PCBs as mixtures is complicated by the varying amounts of the 209 congeners in the mixture and the traces of other contaminants also present (e.g., PCDFs). Individual congeners and mixtures can affect liver function, reproduction, infant birth weights, neurobehavioral development and the immune system and may cause cancer in laboratory animals (ATSDR 1995). The TDI for PCBs is 1 μg/kg bw/d (Health Canada 1996).

While levels of total PCBs have declined since the 1980s in breast milk of women in many industrialized countries (Newsome *et al.* 1995), there is no similar information yet available for Arctic residents. Nor are historical values easy to compare because of changing analytical techniques and quantitation methods (reported PCB concentrations based on an Aroclor 1260 standard are typically approximately double those based on a sum of specific PCB congeners).

Assessing the human health effects of PCBs is very difficult because PCB mixtures typically used in animal studies or identified in accidental poisonings frequently contain traces of contaminants such as PCDFs and undergo extensive 'environmental filtering' prior to human exposures. Many of the effects of exposure to PCBs observed in humans (e.g., Yusho and Yu-cheng incidents) reflect exposure to high levels of both PCBs and PCDFs. PCDFs are believed to be responsible for many of the observed human health effects. From studies following the Yusho and Yu-cheng incidents (see section 12.2.1.6, above), the earliest toxicological signs included chloracne. Additional generalized adverse health effects included hepatomegaly (enlarged liver), bronchitis and peripheral neuropathy (nervous system damage) (Safe 1987). In Yu-cheng, increased upper respiratory tract infection rates were associated with decreased serum IgA and IgM plus increased IgG levels (WHO 1988). Occupational exposures to PCBs generally do not include the PCDF contaminants found in the rice oils, so the effects seen are often different.

A small group of PCB congeners have dioxin-like activity and have been assigned dioxin toxic equivalency factors (Ahlborg *et al.* 1994), cf. section 12.2.1.6. In a number of human tissue samples, such as breast milk or adipose tissue, it has been found that the dioxin-like PCBs contributed a large proportion of the total 2,3,7,8 TCDD TEQ (Dewailly *et al.* 1992). Because PCB congeners co-exist with dioxins and furans in the environment, ascribing an effect to one or the other contaminant is almost impossible.

Some studies from Japan have found levels of dioxin-like PCBs, such as the congeners CB 77, 126, and 169, up to several orders of magnitude higher than the levels of 2,3,7,8-TCDD in human adipose tissue samples (Tanabe *et al.* 1987, Kannan *et al.* 1988, Kashimoto *et al.* 1989). Results from analysis of human adipose tissue and serum collected in the USA show that concentrations of coplanar PCBs (cf. chapter 6, section 6.1.1.1.1) can be more than an order of magnitude higher than the concentrations of 2,3,7,8,-TCDD (Patterson *et al.* 1994).

Data obtained from epidemiological studies on cohorts of US infants from Michigan (Jacobson *et al.* 1990, 1992) and North Carolina (Rogan *et al.* 1986, 1987, Gladen *et al.* 1988, 1991) suggest adverse neurobehavioral effects from *in utero* exposure to PCBs (calculated as Aroclor 1260 equivalents). High cord blood concentrations were associated with

low birth weight and small head circumference (Jacobson *et al.* 1990). Birth size among male infants (Inuit) was inversely related to PCB concentration in breast milk of the mother (Dewailly *et al.* 1993a). Perinatal exposures to PCBs/dioxins/furans may impair immune responses to infection, as suggested by a 20-fold higher incidence of infectious diseases (e.g., meningitis, measles) and ear infections (*otitis media*) among 1-year old Inuit with high PCB exposures than among lesser exposed controls (Dewailly *et al.* 1993b). The infectious disease data may be confounded by a lower seroconversion rate (successful immunization) among Inuit compared to controls (Dewailly *et al.* 1993b).

It is not clear whether PCB exposure is the sole factor leading to neurodevelopmental deficits in the Wisconsin or Michigan cohorts, or if other contaminants, such as mercury, or socio-demographic characteristics might also be associated with these results (Ayotte *et al.* 1996). Caution must be used when examining data from the Lake Michigan cohort in relation to assessing health risks for Inuit newborns in the Arctic. The mixture of contaminants to which Lake Michigan infants were exposed may be very different from that found in Arctic ecosystems (regional industrial sources as opposed to long-range atmospheric transport). In addition, the Lake Michigan population exposure was through fish consumption, while the diet of northern Inuit also includes species at higher trophic levels (e.g., marine mammals). These dietary differences may lead to quite different contaminant exposure profiles. Studies underway in northern Quebec and the Faeroe Islands should help to answer these questions.

12.2.1.8. **Hexachlorobenzene**

Hexachlorobenzene (HCB) was widely used as an anti-fungal agent for various seed crops and is also an important industrial feedstock for production of chlorinated solvents and pesticides (Government of Canada 1993). HCB is still widely used in the world and atmospheric transport is a major pathway to the Arctic.

HCB causes a wide range of effects in laboratory animals, including liver pathology, skin lesions (porphyrial cutanea tarda in humans), behavioral changes, reproductive changes in primates, and effects on the immune system. The provisional TDI for HCB is 0.27 μg/kg bw/d (Government of Canada 1993).

HCB is found at higher levels in serum of newborn Inuit from Arctic Canada compared to southern Canadian populations (CACAR 1996).

12.2.2. **Toxicology of PAHs**

Polynuclear aromatic hydrocarbons (PAHs) are a family of ring-structured compounds that do not include chlorine in their molecular structure. Several PAHs are carcinogenic to animals. While PAHs can enter the Arctic from remote locations (with sources including industrial activity and forest fires), the primary sources of human exposure in the Arctic are local. The greatest pathways of exposure to PAHs for the general population are inhalation of tobacco smoke (active or passive), wood smoke and smoke from other fuel sources, indoors and outside, and the ingestion of PAHs through consumption of smoked, fried or broiled food. For non-smokers, food is the main source of exposure (99% of total benzo[a]pyrene (B[a]P) intake), and air a much less important source (0.9% of total B[a]P intake).

Benzo[a]pyrene, one well studied PAH, is a carcinogen in animals and is 'probably' carcinogenic to humans. Several other PAHs are also known to be animal carcinogens (IARC 1987). PAHs are readily absorbed and can be metabolized with relative ease in the liver and kidney. They are excreted in bile, feces and urine. The total daily potential exposure of adult males to carcinogenic PAHs is estimated to be 3 μg (median), and may be as high as 15 μg. For smokers, exposure levels may be twice as high. Exposure to PAHs through cigarette smoke has been associated with reduced fecundity and low fetal birth weight (Weinberg *et al.* 1989) and is thought to be a major factor in lung cancer.

The daily intake of all PAHs from foods has been estimated to be 2-20 μg (Pucknat 1981). PAH concentration in food depends on both the method of preparation and the origin of the food. Barbecuing foods increases their PAH concentration. Smoked and cooked fish and meats are higher in PAHs than uncooked products. Despite considerable potential dietary exposure to PAH from smoked food, there is little risk to health from this route of intake. Inhalation of PAHs from tobacco smoke (active or passive) is associated with a significant risk to health (IARC 1987).

There are no data available on PAH levels in breast milk, fat or blood of Arctic residents.

12.2.3. **Toxicology of heavy metals**

Metals can occur in ecosystems in organic and inorganic form as well as in different oxidation states. These factors will affect the absorption, metabolism and toxicity of metals, making information on their form and speciation crucial for realistic risk estimates.

The toxicology of metals involves approximately eighty elements and their compounds ranging from simple ionic salts to complex molecules such as organometallic compounds.

The term 'heavy metals' is not well defined chemically, but in a biological context has been applied to metals having a specific gravity of 5 or higher. Within this arbitrary group are many of the trace elements essential to plants and animals, as well as metals not known to have any essential property. Some may give rise to toxic reactions, even at low levels of exposure and concentrations only moderately in excess of the background levels. In this latter group, mercury (Hg), lead (Pb), and cadmium (Cd) are regarded as priority contaminants.

The toxicity of metals is often due to their interference with important sites in cellular biochemical systems, such as the sulfhydryl groups in enzyme systems. This interference often results in cell death. Metals may also compete with essential elements as enzyme co-factors, creating a toxic response that is manifested as a deficiency of an essential metal. For this reason, it is also important to consider the status of essential elements when evaluating an exposure to the toxic metals. There is an abundance of data on such interactions (for a review see Nordberg *et al.* 1986). Mercury, lead, and to a lesser degree cadmium, can all cross the placenta and can affect the developing fetus.

Important natural sources of metals in the atmosphere include volcanic activity and forest fires. Rock-weathering, land runoff, and atmospheric deposition are the most important sources in the hydrosphere. Major anthropogenic sources are combustion of fossil fuels, including vehicular emissions, mining and smelting operations, processing and manufacturing industries, and waste disposal. Sources and environmental occurrence of metals, in particular mercury, cadmium, lead and selenium are considered in chapter 7. The following sections include a brief summary of sources of the metals discussed, with more extensive summaries for nickel and arsenic which are important in relation to human health, but have a lower priority as environmental contaminants in the Arctic.

Table 12·2. Some limit values for cadmium, lead and mercury in the environment.

	Limit for air[a]	Limit for drinking water[b]	Provisional tolerable weekly intake[c]	Main sources of exposure
Cadmium	10-20 ng/m^3 (urban air)	3 μg/L	Approx. 7 μg/kg body weight	Occupational; cigarette smoke; food
Lead	0.5-1 μg/m^3	10 μg/L	50 μg/kg body weight	Occupational; pica[d]; deposition from leaded particles
Mercury	1 μg/m^3	1 μg/L	5 μg/kg body weight as total mercury 3.3 μg/kg body weight as methyl mercury (MeHg)	Occupational; marine food

a. Guideline value for upper limit of concentration as time-weighted average over 1 year (WHO). b. Guideline value for upper limit of concentration in drinking water (WHO). c. Maximum acceptable weekly intake for adults (WHO/FAO). The value quoted should be multiplied by the body weight in kilograms to obtain the total maximum acceptable weekly intake for an individual. d. Pica is the habit of eating clay, soil, dirt, and other non-food items. It is an important source of lead intake for children who live in contaminated environments, particularly houses with old lead-based paints.

From the point of view of human exposure, marine foods are of major importance since the traditional diets of many Arctic populations include species from the top of the marine food chain. Historically, technological development and industrialization seem generally to have increased human exposure, especially for mercury and lead (Hansen *et al.* 1989b).

Exposure limit values have been proposed by WHO and FAO (cf. Table 12·2) for most of the metals of concern.

12.2.3.1. Mercury (Hg)

Mercury is ubiquitous in the environment. The primary source is from degassing of the earth's crust especially in connection with geothermal activities (Lindquist *et al.* 1991). Although a minor portion is of anthropogenic origin, it may contribute to the global level and give rise to local pollution, in particular when it enters into lakes and rivers.

Inorganic mercury compounds from natural degassing or industrial waste can be methylated in the aquatic environment, and methylmercury (MeHg) can then be introduced into the aquatic food chains where it is biomagnified. Consequently, exposure to methylmercury is of significance for inhabitants of the Arctic, whereas exposure to the metallic vapors from dental amalgam fillings, a problem discussed in other parts of the world (Vimy and Lorscheider 1985a, 1985b), is of minor importance.

Methylmercury is readily absorbed through the intestinal wall, and blood concentrations at steady state reflect the daily intake. Sherlock *et al.* (1984) have suggested the following relationship between blood and exposure: Hg concentration in blood (μg/L) = 0.8 × daily intake (μg/adult).

This equation can be used to estimate human exposure levels from data on blood Hg concentration.

The provisional tolerable weekly intake (PTWI) for total mercury has been set by the WHO (1990b) at 5 μg/kg bw/week, and for methylmercury at 3.3 μg/kg bw/week or 231 μg/kg/week for a 70 kg person. According to the above-mentioned equation, this intake corresponds to a blood Hg concentration of about 26 μg/L. The lowest blood Hg concentration at which neurological signs have been observed in exposed adults is often accepted as being 200 μg/L. For protection of the fetus, maternal blood Hg should not exceed 50 μg/L.

The biological half-life of methylmercury in humans has been estimated by Åberg *et al.* (1969). Using a one-compartment model, a half-life of 73 days was determined. This corresponds to the elimination of about 1% of the body burden per day. Recently, Smith *et al.* (1994) have estimated a half-life of 44 days and an excretion of 1.6% of the body burden per day using a five-compartment model.

Metabolism

Demethylation of methylmercury occurs in the liver, kidney, intestine and stomach (Norseth and Clarkson 1970, Norseth 1971, Syversen 1974, Berlin *et al.* 1975, Yamamoto *et al.* 1986, Tsubaki and Takahashi 1986). Until recently, it was thought that demethylation does not generally take place in the brain, however, this has now been shown to occur in humans (Tsubaki and Takahashi 1986), in dogs (Hansen *et al.* 1989a, Hansen and Danscher 1995), and in monkeys (Lind *et al.* 1988). The demethylation process in the brain is slow compared to other organs and for this reason it may, therefore, not have been noticed previously in short-term animal experiments. No demethylation seems to take place in skeletal muscles. Hansen *et al.* (1989a), and Hansen and Danscher (1995) found all mercury in muscle tissue from dogs chronically exposed to methylmercury to be in the methylated form (i.e., no transformation to inorganic mercury was observed). Demethylation is thought to take place via reactions with reactive oxygen intermediates. Suda *et al.* (1991) reported that $OH^{\cdot}$ is the responsible free radical. They later suggested that the myeloperoxidase-halide system is also involved in the process (Suda and Takahashi 1992).

The biochemical mechanism for the toxicity of methylmercury seems to be based on: 1) binding to gluthation (GSH), leading to instability of microtubules, i.e., the cytoskeletal system, (Brown *et al.* 1988, Kromidas *et al.* 1990); 2) introduction of lipid peroxidation (Fujimoto *et al.* 1985, Sarafian and Verity 1991); and 3) inhibition of glutathione peroxidase (GSH_{Px}) (Bem *et al.* 1985, Hirota 1986). All three mechanisms involve the influence of oxidative reactions which appear to be of central importance to mercury toxicity.

Toxic effects

Methylmercury is neurotoxic and the incidence of signs and symptoms of Hg poisoning are related to its concentrations in the brain. Few data exist on Hg concentrations in human brain; levels between 1 and 2 mg/kg fresh tissue in brain correspond to the lowest blood Hg concentration at which neurological signs have been observed (Berlin 1986).

Methylmercury (MeHg) readily crosses the placental barrier such that exposure to methylmercury *in utero* can give rise to severe neurological damage in children (Amin-Zaki *et al.* 1974, Harada 1977). The fetal central nervous system (CNS) has been found to have higher concentrations than the maternal CNS in both humans and in experimental animals (Marsh *et al.* 1980, Reynolds and Pitkin 1975). Furthermore, it is likely that the fetal CNS reacts differently and is more sensitive than the maternal CNS. Even if there are similarities in neuropathological findings between adults and infants (Choi *et al.* 1978), there are specific findings in children, such as ectopic cells and cortical disintegration, which are not seen in adults. This suggests an effect on astroglial cells when the fetal CNS is exposed to methylmercury. Peckham and Choi (1988) have shown in experiments on mice that methylmercury disturbs development of astroglia resulting in abnormal distribution of cortical neurons. This may explain the behavioral abnormalities observed after methyl-

mercury exposure. In humans, the development of astroglia starts at gestational week seven and continues throughout the fetal life (Reske-Nielsen *et al.* 1987). Thus, the effects of methyl mercury could be exerted during most of fetal development. Neonatal CNS development can also be affected by exposure to mercury through breast milk.

Methyl mercury may also affect the immune system, however, existing data are not conclusive. Ohi *et al.* (1976), Koller *et al.* (1979), and Blakely *et al.* (1980) did not find significant immunosuppressive effects in mice and rabbits. Petruccioli and Turillazzi (1990) have reported that monkeys (*Macca fasciculans*), exposed orally to 0.4-50 μg/kg bw/d, showed a progressive dose-related reduction of IgG and, in the highest exposed groups, reduction in IgM and IgA. The lowest dose used in the monkey study (0.4 μg/kg bw/d) corresponds to a daily intake by a 70 kg adult human of 28 μg (0.4×70), which is close to the PTWI. Based on these data, immunosuppressive effects of methyl mercury at exposure levels actually reported in several Arctic communities cannot be excluded.

Using the autometallographic technique, it has been demonstrated that methyl mercury exposure leads to heavy accumulation of mercury in the thyroid gland (Hansen *et al.* 1989a, Hansen and Danscher 1995). As this technique only reveals inorganic mercury, this observation is consistent with a high myeloperoxidase-iodine activity with a supposed high demethylation rate. At present, no studies have been carried out to investigate possible adverse effects.

12.2.3.2. **Cadmium (Cd)**

Cadmium was discovered in 1817, but its use in various technologies started late in the 19th century. Since then, the use of cadmium has steadily increased, resulting in pollution of the environment and in increased human exposure. In modern technology, cadmium has a wide spectrum of applications, such as in alloys, pigments, metal coating, and the electronic industry. Cadmium is a by-product of zinc and lead mining and smelting, which are important sources of environmental pollution. The toxicology of cadmium is extensively reviewed by Friberg *et al.* (1986).

For the general population, the two main sources of exposure are diet and tobacco smoking. Concentrations in air are generally low. Cadmium is highly cumulative in animals and humans, with a half-life of more than ten years in humans.

In Western countries, meat, fish and fruit typically contain 1-50 μg/kg Cd, while grains typically contain 10-150 μg/kg. The highest Cd concentrations are found in the livers and kidneys of animals. Of special relevance to the Arctic is cadmium accumulation in the marine food webs and in grazing mammals in cadmium rich areas. Shellfish (mussels, scallops and oysters) are reported to contain 100-1000 μg/kg. In cetaceans in Greenland, levels of 3250 μg/kg and 13 200 μg/kg have been found in liver and kidney, respectively (Paludan Müller *et al.* 1993). Caribou kidneys have been reported to contain high concentrations of cadmium in some regions of Canada.

Population groups with high consumption of traditional food may have cadmium intakes in excess of 700 μg per adult per day. This should be seen in relation to the PTWI set by WHO at 490 μg per person per week, i.e., 70 μg per day for a 70 kg adult.

Absorption and organ distribution

Absorption of inhaled cadmium is about 15-30%. One cigarette contains 1 to 2 μg of cadmium, of which 10% is inhaled (Elinder *et al.* 1983), thus, each cigarette smoked can contribute 0.015-0.06 μg cadmium to the body burden.

Intestinal absorption of cadmium from food is low, generally reported to be ≤ 5%, and influenced by other dietary factors such as iron and protein. Iron status may partially explain why dietary intake of cadmium is only reflected to a minor degree in blood Cd concentrations. Flanagan *et al.* (1978) demonstrated in human studies that cadmium absorption correlated inversely with serum ferritin. Because of these different factors, exposure estimates based on food data alone are not very useful for risk estimates.

Individual blood cadmium concentrations reflect: 1) pulmonary absorption, 2) intestinal absorption, and 3) body burden. In smokers, the pulmonary fraction dominates, therefore, any risk assessments for cadmium should include information on smoking habits. Where there is a constant exposure level, the concentration in blood should reflect body burden.

In animal tissues, cadmium is bound to the protein metallothionein. Metallothionein has been shown to be absorbed intact by the human intestine. Cadmium absorbed through the lungs becomes bound to metallothionein in the liver and transported via the blood stream to the kidney where it is absorbed by the proximal tubular cells in the kidney cortex. Cadmium accumulates in the kidneys over time, in humans until the age of about 50 after which time the renal concentration declines, probably due to the age-related loss of active nephrons. Cadmium also accumulate in the liver, and together the two organs contain approximately 50% of the total body burden of cadmium (Friberg *et al.* 1974, 1984). Based on kinetic studies, Friberg *et al.* (1984) estimated about 33% of the body burden to be present in the kidneys and about 16% in the liver. Based on an autopsy study, Samela *et al.* (1983) estimated that approximately 55% is stored in the kidneys and approximately 20% in the liver. However, these studies concern a very low exposure level. At higher exposures, a larger proportion is expected in the liver and a smaller proportion is expected in the kidney (Friberg *et al.* 1984).

Toxic effects

The kidney cortex is the 'critical target' in chronic cadmium exposure. Exposure may result in disturbances in the renal tubular function with increased excretion of small molecular weight proteins. β_2 microglobulin release in urine is the first warning sign of incipient cadmium damage. Increased excretion of retinol-binding protein, N-acetyl-β-glucosaminidase, amino acids and calcium are also indicators of tubular dysfunction. It has been estimated that one or more of these indicators are increased when the urinary cadmium excretion exceeds 2-4 μg/24 h (Buchet *et al.* 1990).

Urinary excretion of cadmium is related to kidney concentration and therefore to the overall body burden. Ellis *et al.* (1981) have indicated 217 mg/kg kidney cortex as the concentration where 10% of a population will show signs of renal dysfunction, while Roels *et al.* (1983) have calculated a figure of 185 mg/kg. These estimates are based on occupationally exposed workers, and the results could be influenced by the fact that workers (i.e., persons of working age) tend to be healthier than society in general. Recent studies indicate that this is an underestimation of the risk. Elderly people appear to develop renal tubular dysfunction already at cadmium concentrations in the kidney cortex around 50 mg/kg (Elinder and Järup 1996), corresponding to urinary excretion of 2-3 μg/24 h (Buchet *et al.* 1990). If the minimal toxic concentration is indeed about 50 mg/kg kidney cortex, some people, especially elderly and diabetics, living in areas with elevated cadmium levels will be affected. This also implies that the current provisional tolerable weekly intake is too high (Elinder and Järup 1996). This may be a significant problem because cadmium-induced tubular dysfunction is

irreversible (Kido *et al.* 1988) and no effective chelating therapy has yet been found (Jones and Cherian 1990).

Buchet *et al.* (1990) found an association between urinary excretion of cadmium and calcium. Kido *et al.* (1991a, 1991b) have demonstrated that in cadmium-exposed Japanese with renal tubular dysfunction, the serum concentration of the Vitamin K-dependent calcium-binding protein, osteocalcin, was higher in exposed than in individuals in a non-exposed control group, and that the osteocalcin level was associated with indicators of osteopenia. These findings indicate that even a modest cadmium exposure may influence calcium homeostasis. Whether or not this can exacerbate the age-related osteoporotic changes in women with low dietary calcium intake needs further investigation.

It has been hypothesized that environmental exposure to cadmium may lead to hypertension, and as such may also contribute to the pathogenesis of cardiovascular diseases. However, a population study comprising more than 2000 persons carried out in Belgium did not confirm this hypothesis. Staessen *et al.* (1991) found no association between cadmium exposure and elevation of blood pressure or higher prevalence of cardiovascular diseases.

Neurotoxic effects of cadmium have been reported in rats exposed during the perinatal period to doses that were not expected to affect the dams (Wong and Klaassen 1982, Smith *et al.* 1985, Anderson 1996). Clinical studies have indicated a correlation between body burden of cadmium and certain alterations in the behavior of children (Thatcher *et al.* 1982, Marlowe *et al.* 1983).

A number of epidemiological studies have been done to determine the relationship between occupational exposure to cadmium and lung cancer and prostatic cancer. These studies have been reviewed by IARC (1987), with the conclusion that exposure to cadmium may contribute to lung cancer, but confounding factors including cigarette smoking prevent a definitive conclusion. For prostate cancer, the risk appears debatable (Waalkes and Rehm 1994). However, in a later evaluation, IARC (1993) declared cadmium a human carcinogen (Group 1).

The fetus is protected against cadmium because the placenta accumulates cadmium and thereby acts as a partial biological barrier (Wier *et al.* 1990). However, cadmium may have an indirect adverse effect on fetal development as its interaction with zinc can induce a relative zinc deficiency in the fetus.

12.2.3.3. **Lead (Pb)**

Industrial and vehicle-exhaust emissions are regarded as being the most important sources of environmental lead. As such, most of the Arctic is expected to be an area with low human lead exposure. However, in Greenland, blood lead levels have been reported that are comparable to those found in West European cities (Hansen 1981, Hansen *et al.* 1983, 1984). The relatively high blood levels in Greenland have still not been explained, but could possibly be a result of long-range transport of lead by atmospheric particles. The studies of Murozomi *et al.* (1969) and Rahn and McCaffrey (1980) both indicate that a combination of mid-latitude pollution and meteorological conditions could account for the lead exposure in the Arctic.

The pathway from the environment to humans is not clear, and seems to involve several sources of exposure, not just those related to contamination of food. As a consequence, human exposure estimates based only on results of environmental monitoring of consumed foodstuffs are not generally adequate.

Blood samples are most commonly used for estimating lead exposure. Mean blood Pb concentrations for non-occupationally exposed persons in industrialized areas in Europe and North America are often reported to be between 100 and 200 μg/L blood. Studies in remote societies have shown lower concentrations. Poole *et al.* (1980) found a mean blood concentration of 50 μg/L in 100 children from an unpolluted area in Papua New Guinea, and Piomelli *et al.* (1981) found 30 μg/L in 103 Nepalese children and adults. Concentrations below 10 μg/L have been reported among Venezuela Indians (Hecker *et al.* 1974).

Existing data indicate that the northern hemisphere is more polluted with lead than the southern hemisphere, which is in accordance with the fact that most lead-emitting industries and historically the highest concentrations of automobiles burning leaded gasoline are found in the north.

Some studies have indicated a decrease in environmental exposure. Rabinowitz and Needleman (1982) reported a mean annual decline in lead levels of 11% in umbilical cord blood samples taken in Boston between 1979 and 1981. Among children in Chicago, aged 6 months to 5 years, Hayes *et al.* (1994) found a decline from a median blood Pb concentration of 300 μg/L in 1968 to 120 μg/L in 1988. In maternal blood samples collected in Greenland between 1984 and 1989, Hansen *et al.* (1990b) found a mean annual decrease of 7%.

Absorption and organ distribution

The most important sources of lead are generally air, food and drinking water. Respiratory uptake depends upon a number of factors, especially particle size. Particles greater than 0.5 μm are mainly cleared from the lungs and swallowed. The percentage of particles less than 0.5 μm that are retained increases with decreasing particle size. About 90% of ambient air particles containing lead are small enough to be retained. The retained lead is relatively efficiently absorbed through the alveoli. Intestinal absorption in adults is between 5 and 15%. Children are known to have a greater absorption than adults.

More than 90% of the lead in the blood is found in the red blood cells. The mean Pb concentration level in whole blood is significantly lower in females than in males, possibly due to menstrual blood loss or hormone effects.

The total body burden of lead may be divided into at least two kinetic pools with different rates of turnover. The skeletal pool is larger and has the longest biological half-life, of up to 20 years (Rabinowitz 1991); lead in the soft tissue pool is more labile.

Lead crosses the placental barrier. Cord blood levels generally correlate with maternal blood levels, but are slightly lower. Lead is excreted in the urine.

Toxic effects

Toxic effects from lead constitute a continuum from clinically overt effects to subtle biochemical effects involving the hematologic, neurologic and renal systems. In general, children are more sensitive to lead exposure than adults, due to a higher intestinal absorption and high sensitivity of immature tissues. Prenatal life may be a period of particular vulnerability. The earliest and most sensitive effect is inhibition of δ-aminolevulinic dehydratase (ALA-D), which can be observed at blood concentrations <100 μg/L. Lead also inhibits ferro chelatase, resulting in anemia.

In 1979, Needleman *et al.* reported that children exposed to environmental lead who did not exhibit clinical symptoms of lead toxicity, had deficits in psychometric intelligence, speech and language-processing, attention, and classroom performance. In an eleven year follow-up, the neurobehavioral deficits were found to persist (Needlemann *et al.* 1990).

Numerous other studies of the effects of lead on children have been published, and the results have been combined by means of quantitative meta analysis to provide a more valid estimate of the true effect level (Needleman and Gatsonis 1990). A WHO/CEC study on 1800 children in eight European countries confirmed that there are small but detectable exposure-related neurobehavioral effects in school-age children. The study also found that it was not possible to identify an effect threshold (Winneke *et al.* 1990). Epidemiological studies have reported that low-level exposure to lead (≥100 μg/L in blood) during early childhood is inversely associated with neuropsychological development at school-age (Baghurst *et al.* 1992). Today there is an increasing concern about childhood exposure to lead at levels as low as 100 μg/L (Davis *et al.* 1993).

The risk to the fetus from exposure *in utero* is uncertain (Rice 1990, Bellinger *et al.* 1992). The general sensitivity of the prenatal nervous system to chemical insult, the presence of lead in the fetus after maternal exposure, and subtle effects seen in rodents exposed during gestation, all raise concerns.

Recently, Newland *et al.* (1994) have demonstrated prolonged behavioral effects and learning deficits in squirrel monkeys born to mothers exposed to lead during pregnancy and with blood levels equivalent to those tolerated in humans in occupational settings. These data raise the possibility of human fetal hazards at exposure levels actually present in certain occupational environments and in heavily polluted areas.

Studies by Blanzka *et al.* (1994) suggest that low concentrations of lead are capable of inhibiting nitrite produced by the calcium-dependent constitutive form of nitric oxide synthase (cNOS), while the calcium-independent inducible form of nitric oxide synthase (iNOS) is not affected. These data provide a new hypothesis for the mechanism of lead neurotoxicity, as nitric oxide acts as a neurotransmitter in the brain (Snyder 1992).

12.2.3.4. **Nickel (Ni)**

Nickel is mined either as sulfide or lateritic ores. Major deposits occur in Canada, Russia, South Africa, New Caledonia, Cuba, The Dominican Republic and Indonesia. Most of the nickel production is used in the making of alloys, most notably stainless steel. Nickel metal and nickel compounds are used in the electroplating of consumer goods and industrial materials, in the manufacture of nickel-cadmium batteries, and as catalysts in chemical processes (Nieboer 1992). Nickel is extracted from its ores by pyrometallurgical or hydrometallurgical procedures, or some combination of these, and is produced in the pure state through the Mond (nickel carbonyl) process, electrolysis or electrowinning.

Nickel enters surface water and the terrestrial (soil) environments as a result of natural weathering and erosion of geological materials. It is also released as a result of human activities, including mining, smelting, refining, alloy production, plating, fuel combustion and waste incineration.

Ambient levels of nickel are low, typically 1-20 ng/m^3 in air at urban and rural areas with no obvious nickel source. Near nickel refinery sites, however, levels may be as much as a 1000 times higher (Norseth 1994, Chau and Kulikovsky-Cordeiro 1995). The most important anthropogenic sources in ambient air are base metal production (ca. 65%) and fossil fuel combustion. Nickel (in dissolved and particulate form) enters the aquatic environment in effluents and leachates, as well as through atmospheric deposition after anthropogenic release. Surface freshwater typically has nickel levels less than 2 μg/L, while that of marine water is less than 1 μg/L. Concentrations of nickel in drinking water are of similar magnitude, although enhancement from leaching from stainless-steel piping does occur. Background soil and sediment nickel concentrations are less than 10 μg/g (dw). A thousand-fold increase in surface water, sediments and soil levels have been noted in contaminated areas (Chau and Kulikovsky-Cordeiro 1995). Comparable elevations are seen in vegetation (McIlveen and Negusanti 1994). Near nickel-smelting/refining operations, or in naturally nickel-enriched areas, surface water, sediment and soil levels of nickel are within the range to cause harmful effects to sensitive water-column organisms and terrestrial plants, as well as to soil microbial populations (PSL 1994). In contaminated areas, bioconcentration of nickel occurs in aquatic and terrestrial biota; biomagnification through food chains is not known to occur.

Absorption and organ distribution

Inhalation and ingestion are the major routes of nickel intake by humans. Respiratory absorption is the most significant route in occupational settings. Dietary nickel intake has been estimated at 100-300 μg per day, averaging around 150 μg per day (Nieboer and Fletcher 1995). Foods that are relatively rich in nickel include cocoa, soya beans, other dried legumes, nuts and certain grains (Nieboer *et al.* 1992). About 1% of dietary nickel is absorbed, while the amount absorbed from drinking water may be as high as 25%. Background serum nickel levels accepted as normal for non-occupationally exposed individuals are about 0.30 μg/L, with a normal range upper limit of less than 1.1 μg/L, while levels in urine are less than or equal to 2 μg/L (normal range upper limit of 6 μg/L) (Nieboer *et al.* 1992, Templeton *et al.* 1994). Both urinary and serum levels constitute good indices of exposure. On average, urinary nickel concentrations are about a factor of 10 greater than serum levels. Toxicokinetic modeling suggests that an average adult excretes approximately 2-6 μg Ni per day with comparable levels expected for the urinary and biliary routes (Nieboer *et al.* 1992). The major storage compartments in humans are the serum and the tissues. The elimination half-time into urine for nickel absorbed both from drinking water and food has been estimated as 28 ± 9 hours (range 17-48 hours). This also applies to industrial exposure to aerosols of water-soluble nickel compounds; half-times of more than three years have been reported for retired nickel workers exposed to pyrometallurgical intermediates (primarily as particulates).

Toxic effects

Due to its volatility (boiling point of 43°C), nickel tetracarbonyl (generally referred to as nickel carbonyl) is used in commercial refining of nickel to obtain a high purity product (Antonsen 1981). The toxicity of nickel carbonyl is different from the other nickel compounds because of its unique chemical properties (e.g., zero oxidation state and high lipid solubility). Nickel carbonyl poisoning is systemic, although the lung and brain are affected most significantly. Fatalities have been documented.

Compared to inhalation of nickel carbonyl, ingestion of nickel (II) sulfate or chloride is considerably less toxic. In an incident in which 32 electroplating workers drank water accidentally contaminated with nickel sulfate and nickel chloride (estimated as 1.63 g Ni/L; doses were estimated to be 0.5-2.5 g) (Sunderman *et al.* 1988), most of the workers developed some symptoms such as nausea, abdominal cramps or discomfort, giddiness, lassitude, and diarrhea which lasted from a few hours to 1-2 days in the most severe cases. Urinary and serum nickel concentrations observed for these subjects are by far the highest levels recorded by any laboratory recognized for performing reliable nickel analyses: 13-1340

μg/L in serum, compared to baseline values of 0.3±0.2 μg/L; 230-37 100 μg/L in urine compared to normal values of 2±1 μg/L. In a human experimental study, transient neurological disturbance (hemianopsia) occurred at a dose of 50 μg Ni/kg bw administered as nickel sulfate in drinking water (Sunderman *et al.* 1989). No adverse effects were evident at two lower doses (12 or 18 μg Ni/kg bw), where mean peak serum levels for four volunteers in each of the lower dose groups were 20 and 30 μg Ni/L, respectively.

Hypersensitivity reactions

Asthma. Although nickel-related asthma is rare, periodic case reports and case series studies in an occupational context have been documented since 1973 (e.g., McConnell *et al.* 1973, Dolovich *et al.* 1984, Malo *et al.* 1985, Nieboer *et al.* 1988, Shirakawa *et al.* 1992).

Allergic dermatitis. Worldwide studies of populations suggest a rather uniform prevalence of nickel dermatitis, 7-10% in females and 1-2% in males (Maibach and Menne 1989). In dermatological clinics, 10-20% of females react to nickel, compared to 2-10% of males. Primary contact dermatitis has a good medical prognosis, although continued exposure may lead to chronic forms involving other sites, especially the hands (Christensen 1990). The majority of patients are exposed to non-occupational sources, with the leaching of nickel from inexpensive earrings, other jewelry, or wrist watches being the main causes. Ear-piercing is a recognized determinant. Nevertheless, work-related nickel dermatitis is a common diagnosis in reports of permanent disability involving skin diseases (Maibach and Menne 1989). In its chronic form (i.e., presence of hand eczema), flaring of recurrent nickel dermatitis can occur when patients are challenged orally with a nickel (II) salt. Systemic induction, therefore, appears possible. There is a good indication that nickel-restricted diets and avoidance of nickel-rich foods are helpful (Maibach and Menne 1989, Nielsen 1992).

Other immunotoxic responses. Animal studies suggest that nickel compounds can produce immune suppression. The thymus, T-lymphocytes, macrophages, and natural killer-cells appear to be sensitive targets (Nieboer *et al.* 1988, Knight *et al.* 1991, Nicklin and Nielsen 1992). There are no corresponding human data, although inflammatory alterations characterized by a loss of cilia and gradual modification of epithelial cell shape and arrangement have been observed in the nasal mucosa of active and retired nickel workers (Boysen *et al.* 1982, 1994).

Cancer

Association between occupational nickel exposure and enhanced risk of lung and nasal sinus cancers was suspected over 50 years ago (Doll 1990). The first published reports appeared in 1958 (Doll 1958, Morgan 1958). Since that time many studies with positive findings have been published. Initially, exposure to nickel carbonyl was suspected, and in subsequent years it was believed that the greatest cancer risk occurred among nickel smelter workers. A comprehensive reassessment (Doll 1990) concluded that, in addition to pyrometallurgical intermediates in the nickel-refining process (mostly nickel oxides and sulfides), water-soluble salts such as nickel chloride and nickel sulfate also increase lung and nasal cancer risks. The respiratory cancer incidences were primarily related to soluble nickel exposures at concentrations more than 1 mg Ni/m^3 and to less soluble forms at more than 10 mg Ni/m^3. The conclusion that nickel metal (as opposed to Ni compounds) was not linked to lung and nasal cancer risks has been accepted by IARC, although based on animal data it is designated as possibly carcinogenic to humans (IARC 1990). It is also clear that respiratory cancer risks have not been found to extend to the user industries or nickel alloy manufacturing. Exposure to nickel carbonyl, for which there is limited evidence for carcinogenicity in animals, is not implicated.

Miscellaneous toxicological effects

Reproductive and developmental toxicity. Until very recently, reproductive and developmental effects in humans had not been reported in connection with exposure to nickel compounds (e.g., Clarkson *et al.* 1985, WHO 1991b). In a preliminary and qualitative report, Chashschin *et al.* (1994) have expressed concern about apparent increases in spontaneous abortions and structural malformations (especially cardiovascular and musculoskeletal defects) in newborn babies whose mothers were employed in a Russian nickel refinery. This concern requires a more comprehensive and quantitative epidemiological investigation.

Renal toxicity. It may be concluded that nickel compounds have low nephrotoxicity in humans. A number of reports of mild proteinuria are known for individuals exposed occupationally or through consumption of contaminated well water (Gitlitz *et al.* 1975, Sunderman and Horak 1981). Mild transient nephrotoxicity (e.g., proteinuria) was observed with the accidental ingestion incident mentioned earlier (Sunderman *et al.* 1988).

Cardiotoxicity. Nickel (II) chloride induces coronary vasoconstriction in the dog heart *in situ* and in isolated perfused rat heart (Rubanyi *et al.* 1984, Edoute *et al.* 1992). A critical review of the available literature suggests that cardiotoxicity or cardiovascular disease in humans is not a recognized response or outcome (Nieboer *et al.* 1988). Interestingly, in the above mentioned incident whereby men were acutely exposed to nickel due to consumption of contaminated drinking water at work, palpitations lasting up to half a day constituted the extent of heart related symptoms (Sunderman *et al.* 1988).

Concluding remarks

Most of the health effects of Ni described above have been experienced under conditions of occupational exposure or acute toxicity. However, nickel contact dermatitis is primarily a public health disease, that is both debilitating and preventable. It is exacerbated by dietary (including drinking water) intake of nickel and, thus, the pathology of the dermatological response may be influenced by environmental contamination. Urinary and serum nickel levels are sensitive indices of environmental or occupational exposure and, thus, are useful parameters in biological monitoring assessments. In communities where a significant proportion of the population is employed in nickel refining or a comparable industry, such as Nikel and Norilsk, in Russia, environmental health assessments must take cognizance of disease carryover such as reproductive or developmental impairments or cancer risk due to occupational exposure to nickel. On the other hand, the industrial experience must not close the door to other and perhaps unsuspected health effects.

12.2.3.5. **Arsenic (As)**

Arsenic is a constituent of more than 200 different minerals, though in small amounts, and is found most frequently in association with sulfur. The mean concentration of inorganic As in the continental crust is about 1.5-2.0 mg/kg. Soil levels

are reported in the range of 0.3-10 mg/kg in cropland areas and 0.6-15.7 mg/kg in rural areas (values from Wolson 1983). The concentration of total As in ground and surface water is usually below 10 μg/L (Pershagen and Vahter 1979).

As is present in higher concentrations in certain minerals. The average As level in sedimentary iron ores is 400 mg/kg. Certain coals are also rich in arsenic. Inorganic As, in the form of arsenious oxide (As_2O_3), is released to the environment through human activities, e.g., coal burning, mining, smelting, and the use of As-containing pesticides (Fishbein 1981).

The environmental levels of arsenic around As-emitting plants are generally increased. It has been suggested that air levels of As are about 10 to 100 times higher in smelter areas than in urban and rural environments (Wolson 1983).

The background concentration of As in food and drinking water is generally low and the daily intake of As will normally not exceed 50 μg (WHO 1981, Wolson 1983). In certain areas of the world, As concentrations in drinking water have been high with serious adverse health effects after prolonged exposure, e.g., vascular and neurological diseases (WHO 1981).

Toxic effects

Toxicity of inorganic arsenic

The main mechanism behind the toxicity of As(III) compounds is their interaction with sulfhydryl-(SH)-groups in tissues (for a review see Squibb and Fowler 1983). Other mechanisms of enzyme interactions, e.g., competitive inhibition of substrate binding due to structural similarities, may also play a role.

In vivo toxicity of inorganic arsenic is dependent on many different factors, such as the animal species and the chemical form and the solubility of the As compound. As(III) (arsenite) compounds are more toxic than the As(V) (arsenate) salts. Pentavalent arsenicals, due to their structural similarity with phosphate, uncouple oxidative phosphorylation. Part of the arsenate administered in *in vivo* experiments is reduced to arsenite (Hindmarsh and McCurdy 1986).

In humans, as in many animal species, arsenic is methylated and excreted in the urine as monomethylarsonic acid (MMA) and dimethylarsinic acid (DMA) (Smith *et al.* 1977). The metabolism of As is fairly rapid and the biological half-life in humans is about 2-3 days (Crecelius 1977, Pomroy *et al.* 1980, review: Vahter 1983). In a study of Swedish smelter workers, urinary As levels had decreased to normal values after four weeks vacation (Lagerkvist *et al.* 1988).

Different studies on the distribution of inorganic arsenic *in vivo* show the highest concentrations in skin, gastro-intestinal mucosa, liver and hair (Vahter 1983).

Health effects of inorganic arsenic

Adverse health effects after exposure to inorganic arsenic compounds, both in the general and occupational environment, are described in several reports and reviews. Both acute, subacute and chronic effects are reported. Almost every organ system may be affected (reviews: Jenkins 1966, Chhuttani and Chopra 1979, Pershagen and Vahter 1979, WHO 1981, Pershagen 1983, Hindmarsh and McCurdy 1986, Bates *et al.* 1992). Chronic bronchitis, liver cirrhosis, peripheral vascular disease, neuropathy, CNS disturbances, cardiovascular diseases, and cancer in the liver, lung, stomach and other internal organs are considered to be associated with exposure to inorganic arsenic. Skin disorders ranging from dermatitis, melanosis, and hyperceratosis to skin cancer have been described in workers and populations exposed to arsenic (Tseng *et al.* 1968, WHO 1981). Peripheral vascular disease and neuropathy caused by contaminated drinking water are the most common adverse health effects reported in population studies with moderate exposure levels.

Vascular effects. Adverse effects caused by inorganic arsenic on peripheral circulation and even gangrene have been reported in populations with a high intake of arsenic in drinking water, and in vintagers and vintners exposed to As in pesticides and wine (Butzengeiger 1940, Astrup 1968, Grobe 1976, Tseng 1977, Borgoño *et al.* 1977, Lüchtrath 1983).

A high prevalence of gangrene (black foot disease) was reported from Taiwan, where the arsenic in well water ranged from 0.01-1.82 mg/L (Tseng *et al.* 1968, Tseng 1977). Vascular diseases were also reported from northern Chile in a population exposed to arsenic in drinking water at levels of about 0.6 mg/L (Borgoño *et al.* 1977).

An increased prevalence of Raynaud's phenomenon and vasospastic tendency was found in smelter workers in northern Sweden with long-term airborne exposure to inorganic arsenic at relatively low dose (Lagerkvist *et al.* 1986). The mean urinary concentration of inorganic arsenic and its methylated compounds was 71 μg/L (range 10-340), and the mean daily intake of inorganic As was estimated to be 105 μg during the 8-40 years (mean 23 years) period of working at the smelter, with a higher intake in the past.

Neurological effects. Peripheral nerve effects caused by inorganic arsenic, ranging from simple neuralgia to severe paralysis, have been known and discussed for centuries. Geyer cited approximately 200 references in his review of different clinical (including neurological) manifestations of chronic As exposure through As-polluted drinking water (Geyer 1898).

Arsenic neuropathy is considered to be a sensory-motor distal axonopathy with accompanying demyelination (reviews in Chhuttani and Chopra 1979, and Manzo 1985). The biochemical effects of arsenic at the cellular level, e.g., binding to SH-groups and inhibition of oxidative phosphorylation, are consistent with a process leading to distal axonal degeneration (Manzo 1985).

Most of the peripheral nerve lesions described in the literature are caused by comparatively high doses of arsenic (e.g., Jenkins 1966, WHO 1981). There are few studies on nerve effects resulting from occupational low-dose exposure. Hine *et al.* (1977), discussing the medical problems associated with As exposure in the Tacoma smelter, Washington, found no clinical neuropathy in the employees, though they 'might have been heavily exposed to airborne As_2O_3 in the past'.

Subclinical neuropathy in high As exposure groups with a 5-year average periodic urinary arsenic above 200 μg/L has been reported in a study on 70 copper smelter workers and 41 controls from the USA. Subclinical neuropathy was defined as 'one or more nerves with reduced conduction velocities, or two or more nerves with reduced amplitudes' (Feldman *et al.* 1979).

In a study on smelter workers from northern Sweden, Bloom *et al.* (l985) reported subclinical effects on the peripheral nervous system, i.e., reduced nerve conduction velocity (NCV). A statistically significant correlation was found between reduced NCV, decrease of finger blood pressure during cooling (vasospastic tendency) and total estimated absorption of arsenic.

In a study from Alaska on 147 Ester Dome residents exposed to arsenic in well water, the median As concentration in the water was 41 μg/L (range, 1-4781) and in the urine it was 51 μg/L (range 6-4964). The calculated index of the daily ingestion of As was 14.5 μg and correlated closely with the urine As concentration. Median exposure time was five years (Kreiss *et al.* 1983). Symptoms or clinical signs of sensory neuropathy were found in six persons, and one or more abnormal NCVs in 13 persons. However, no dose-response relationship was found between As ingestion and NCVs. The

authors concluded that the As exposure had not resulted in clinical or sub-clinical neuropathy.

The urinary levels of As, assuming that comparable analytical methods were used, were lower in the Alaskan study on environmental exposure of As than in the studies on smelter workers. When monitoring arsenic in urine, inorganic As and its methylated metabolites should be determined since analysis of total As includes also the organic arsenic from fish.

Toxicity of organic arsenic

Arsenate in seawater is transformed into organic compounds by marine algae and accumulates in the food web (Tamaki and Frankenberger Jr. 1992). In fish and other seafood, organic As compounds, e.g., arsenobetaine, $(CH_3)3AsCH_2COOH$, may be present in relatively large amounts. At present, these are not considered to be toxic to humans. Different authors have found no indication of bio-transformation in humans after ingestion of lobster, crab meat, or flounder containing as much as about 2 mg of organic As in one meal (Crecelius 1977, Cannon *et al.* 1979, Charbonneau *et al.* 1980, WHO 1981, review: Vahter 1983).

In recent years, some *in vitro* studies have shown that DMA (one of the urinary metabolites of inorganic As) can act as a clastogen. In one recent experimental study, tumor induction in urinary bladder, kidney, liver, and thyroid gland was enhanced in rats sequentially treated with three different nitrosamines, methylnitrosurea and dimethylhydrazine followed by administration of DMA (Yamamoto *et al.* 1995). The authors concluded that DMA was acting as a promotor. No tumors or preneoplastic lesions were observed in the two control groups (n = 12 in each group) given 100 and 400 mg/kg of DMA for 24 weeks. Thus, to date, neither epidemiological nor experimental studies have demonstrated carcinogenic effects from exposure to DMA alone or to more complex organic As compounds.

Concluding remarks

In conclusion, the major health risk of environmental exposure to arsenic seems to be through drinking water which is locally contaminated with inorganic arsenic. In recent years, it has been suggested that inorganic arsenic might be essential to humans and play a role in the synthesis of taurine (Nielsen 1990, 1991). A possible adequate daily intake of 12 to 25 μg has been suggested (Uthus and Nielsen 1993). In fact, inorganic arsenic is excreted in the urine of people with no known exposure (WHO 1981). In a study on pregnant women in northern Sweden, urinary levels of inorganic arsenic were of the same magnitude as the daily required dose suggested by Uthus and Nielsen (Jakobsson Lagerkvist *et al.* 1993). However, further studies are needed before definite conclusions can be made about whether inorganic arsenic is essential to humans or not.

12.2.4. Health effects of ionizing and non-ionizing radiation

12.2.4.1. Radionuclides

This section provides a brief summary of information presented in more detail in chapter 8, focusing here on human health aspects. As an introduction to the effects of ionizing radiation on human health, Table 12·3 gives a description of radiation effects at various levels of exposure.

Absorbed dose of radiation is measured in grays (Gy). Equivalent dose and effective dose are both measured in sieverts (Sv); reported dose estimates are more commonly associated with the unit millisieverts (mSv). One sievert (i.e., 1000 mSv) corresponds to one joule of gamma radiation energy absorbed per kilogram of tissue, or an amount of any other type of radiation that would do as much biological damage as one joule per kilogram of gamma radiation. The threshold for the onset of acute radiation sickness is about 1000 mSv delivered over a short time period (a few hours or days). As the dose increases, the symptoms become more severe. Survival of humans and other mammals is unlikely for doses above 10 000 mSv. Levels such as these are almost never encountered in the environment. Normal background exposure is about 2 mSv per year, resulting predominantly from naturally-occurring radionuclides in the earth's crust and in the human body, as well as from cosmic radiation.

Table 12·3. Effects of various doses of radiation (UNSCEAR 1988, ICRP 1991).

Dose in millisieverts (mSv)	Description	Cancer risk
10000 in a short time (hours)	survival unlikely	–
1000 in a short time (hours)	onset of acute radiation illness	1 in 20
20 in a year	occupational dose limit	1 in 1000
2 in a year	background radiation dose	1 in 10000
1 in a year	public dose limit	1 in 20000
0.1 per exposure	chest x-ray	1 in 200000

The health effects of chronic exposure to low levels of radiation are not as clearly understood. Ionizing radiation is a known carcinogen and its effects in humans have been well documented in studies of high levels of radiation exposure, e.g., survivors of atomic bombs, occupationally exposed persons, and persons treated medically with radiotherapy (UNSCEAR 1977, BEIR 1990, ICRP 1991). It is generally found that the risk of developing cancer is directly proportional to the amount of radiation received. The risk has been found to be about 0.05 per sievert of exposure (or 5×10^{-5} cancers per mSv of exposure). For exposures below about 100-200 mSv, the number of radiation-induced cancers becomes statistically indistinguishable from cancers produced by other causes. Radiation protection authorities generally assume that there is no threshold for radiation-induced cancer, and that even a very low-level exposure carries some small but finite risk of developing cancer. It should be noted that this is an assumption that is unproven and may be unprovable.

Genetic or hereditary effects of radiation are known to occur in animals and are presumed to occur in humans. However, there is no direct evidence of the latter, even in the very high exposures sustained by A-bomb survivors.

Radionuclides in the Arctic environment may be of natural or artificial origin. Natural radionuclides include potassium (^{40}K) and members of the uranium and thorium decay series. They form a significant part of the natural radiation background to which humankind has always been exposed. Environmental levels of these radionuclides may become enhanced as a result of human activities such as uranium mining.

Artificial radionuclides in the Arctic and elsewhere are mainly products of nuclear fission. During the 1950s and 1960s, a great deal of this material entered the environment as a result of fallout from nuclear weapons testing. The signing of the Limited Test Ban Treaty in 1963 did much to reduce this source of contamination, however, France and China were not parties to the treaty and continued atmospheric testing until 1980. Major nuclear accidents such as that at Chernobyl in 1986 have injected fresh radioactive material into the Arctic region. Nuclear dump sites have also contributed. Discharges from reprocessing plants in western Europe and Russia, and from nuclear dump sites have also contributed significantly to the radioactive contamination of the Arctic seas.

The following is a brief description of those radionuclides, both artificial and natural, that are of greatest concern for human health in the Arctic.

Radiocaesium

Environmental radiocaesium consists primarily of two isotopes, ^{137}Cs with a half-life of 30 years and ^{134}Cs with a half-life of about two years. Radiocaesium from atomic bomb fallout is predominantly ^{137}Cs. Both ^{137}Cs and ^{134}Cs are present in nuclear reactor wastes, in the ratio of about two to one. In biological systems, caesium behaves in a manner similar to potassium, an essential nutrient. Ingested radiocaesium is almost completely absorbed by the intestine and becomes distributed throughout the body, mainly in muscle tissue. It is cleared from the body with a half-life of two to three months. The radiocaesium isotopes are considered to be the artificial radionuclides of greatest concern for human health in the Arctic. They are concentrated particularly by two food chain pathways:

Lichens → reindeer/caribou → humans
Freshwater → fish → humans.

Chapter 8 (Table 8·13) indicates average cumulative doses to Arctic populations of 0.74 to 11.6 mSv from the ingestion of radiocaesium. For selected populations, these doses vary from 10.5 to 152 mSv (Table 8·14). It should be noted that these dose estimates are highly variable. They depend on the local concentrations of radiocaesium in reindeer and caribou herds and in fish, and also on the amount consumed. The latter quantity can be particularly difficult to estimate and 152 mSv should be regarded as an upper limit for individual lifetime exposure. Most of the cumulative exposure is due to global fallout from nuclear weapons testing and the levels are quite similar across all of the circumpolar countries. Fallout from the Chernobyl accident was more variable, with the Nordic countries receiving the largest share, followed by northern Russia and North America.

It is important to note that virtually all of the cumulative radiocaesium dose has already been received. Barring another major input, such as a serious nuclear reactor accident or the resumption of atmospheric nuclear testing, the impact of current levels of radiocaesium on future generations will be minimal.

Radiostrontium

^{90}Sr is a fission product with a half-life of about 30 years. About 30% of ingested strontium is absorbed by the intestine and, being chemically similar to calcium, it becomes deposited in bone where it may remain for many years. It was largely due to concern over this radionuclide, and especially its effects on the bone marrow and on the developing bones of children, that led to the banning of atmospheric nuclear weapons tests in 1963.

^{90}Sr is concentrated particularly by the grass → cow → milk → human pathway. Its impact on Arctic residents is no greater that the impact on other populations groups. Chapter 8 (Table 8·15) gives average ^{90}Sr doses of 0.21 to 0.36 mSv, with 0.06 to 4.4 mSv to selected groups (Table 8·16). These doses are much smaller than those from radiocaesium.

Radionuclides of lead and polonium

Lead-210 and polonium-210, two natural radionuclides, make a greater contribution to the present day exposure of Arctic populations than all artificial radionuclides combined. Both radionuclides are members of the decay series of naturally-occurring ^{238}U. They become airborne as a result of the decay of radon gas seeping from soil. Uranium mining and processing operations may lead to enhanced concentrations of ^{210}Pb and ^{210}Po in the environment. ^{210}Pb has a half-life of 22.3 years. It decays to ^{210}Po (half-life of 138 days). ^{210}Pb and ^{210}Po are transported by atmospheric particulates and tend to settle out on vegetation. They are concentrated particularly in the lichen → reindeer/caribou → human pathway. Some Canadian studies have indicated doses of up to 10 mSv per year to Arctic residents from exposure to these radionuclides. Since they are naturally-occurring and ubiquitous, it is likely that these doses would be about the same in all circumpolar countries where caribou and reindeer are consumed. Furthermore, these doses have remained about the same during the 10 000 to 20 000 years that human populations have inhabited the Arctic.

Other radionuclides

Two other long-lived radionuclides are relevant to the Arctic, radiocarbon (^{14}C) and radioiodine (^{129}I). Both are naturally present in minute traces, but levels are enhanced by anthropogenic sources. ^{14}C is produced as an activation product in the nuclear fuel cycle. The individual doses from this radionuclide in the Arctic are quite small, as indicated in chapter 8, however, its long half-life (over 5000 years) means that it will be available as an exposure source for generations far into the future. ^{129}I is released in minute quantities from fuel reprocessing activities. Its very long half-life (16 million years) means that its potential impact will extend even further into the future.

Neither of these two radionuclides are of major health concern at present. However, their long-term impacts need to be clearly understood, and their levels should be monitored to ensure that they are not increasing.

12.2.4.2. UV radiation

Satellite measurements in the northern polar regions indicate a 10% reduction in levels of stratospheric ozone during the last 15 years (IASC 1995). Upper atmospheric ozone depletion has raised concerns about the effects of increased ultraviolet (UV) radiation exposure on human populations (see also chapter 11). Since stratospheric ozone functions as a filter, reducing the amount of solar radiation at wavelengths below 330 nm that reaches the Earth's surface, reductions in ozone will allow more of these wavelengths, UV-B (290-315 nm) and UV-A (315-400 nm), to reach the biosphere (WHO 1994b). Biological activity increases as wavelength decreases. For every 1 nm shift of the solar spectrum towards shorter wavelengths, the biological activity of sunlight may increase by as much as 15% (Wulf 1994). This is sometimes referred to as the 'Biological Amplification Factor' for solar UV spectral shifts. Such a shift toward shorter wavelengths during the Polar summer will mainly increase biologically active wavelengths below 310 nm. Therefore, even small changes in ozone may have substantial biological consequences and alter the health status of the Arctic population.

In the Arctic, the total annual UV-B exposure at ground level is about two thirds of that in southern Scandinavia. In addition to this surface exposure, the terrain reflection from ice and snow covered surfaces may increase the albedo and contribute an additional 30% in parts of the year (Kromann *et al.* 1986) (see chapter 11 for more details). As the ground surface reflection is important to ocular exposure, a very substantial impact upon ocular health may be expected (Sliney 1986, 1995).

Biological effects

The acute effects of UV-B on humans are erythema ('sunburn') and photokeratitis ('snow blindness'). The chronic effects are elastosis (wrinkles), skin cancer and possibly cataracts. The acute effects appear within hours and fade within days.

The chronic effects take years to develop and are not reversible (WHO 1994b).

Photodermatoses are often UV-A provoked while other types of effects are mainly UV-B provoked.

Erythema

Erythema or sunburn (literally, reddening of the skin) is an acute response resulting from UV irradiation of the skin. The sunburn is the result of the release of inflammatory cytokines leading to skin reddening from the dilation of the small superficial blood vessels, and later edema and thickening of the stratum corneum. The skin adapts to higher UV radiation exposure levels with melanogenesis (tanning). Although this optically reduces the radiation reaching the basal layer, the melanin pigment may play a greater role in reducing the oxidative damage to DNA and other key biological molecules in less superficial layers. The maximal reaction is found 8-24 hours after irradiation and in severe cases blistering may be seen (McKinlay and Diffey 1987). Erythema is not very problematic in itself but repeated heavy sunburns during childhood have been related to the development of the skin cancer, malignant melanoma.

Snow blindness

Snow blindness, the transient damage of the cornea (photokeratitis) and conjunctiva (photoconjunctivitis) resulting from acute exposure to solar UV-B reflected from ice and snow (Hedblom 1961, Sliney 1983, 1995), has been known since ancient times in the Arctic. Because the diffusely reflected UV-B comes from all sides, it is difficult to protect the eyes, even with modern sunglasses with UV-absorbing lenses, since photokeratitis is known to result from UV-B entering from the temporal areas of the lenses (Sliney 1994). Antique Inuit protective masks and slit goggles (Figure 12·1), however, appear to offer effective protection since spatial rather than spectral attenuation is emphasized. Sliney (1994) found that less

Figure 12·1. Traditional Inuit slit goggles from Greenland; the goggles protect the eyes against ultraviolet radiation. Photo: Rikke Claesson.

than 1% UV-B entered a UV detector mounted in a mannequin's eye position fitted with an Inuit bone slit goggle, compared to 10-20% for conventional UV-B-absorbing sunglasses.

The incidence of snow blindness is unknown (WHO 1994a), but it is more common in the spring, when snow is still present and the sun's angle of elevation is such that substantial UV-B is present in the terrestrial solar spectrum. The symptoms are severe pain, and in some cases blepharospasm (uncontrolled blinking). The signs and symptoms last for a day or two. The condition is almost always reversible and it has generally been accepted that the condition is without sequelae. However, repeated severe episodes of snowblindness may well increase the risk of delayed corneal pathologies.

Elastosis

UV-B is the primary cause of destruction of collagen and elastic fibers of the skin leading to inelastic skin and wrinkles (Bissett *et al.* 1987). Such changes are commonly observed in facial skin of Inuit hunters who have rather normal skin in areas protected by clothes. Studies of elastosis in Arctic populations have not been reported in the literature.

Skin cancer

By far the most significant health impact of increased UV-B irradiation of the skin concerns the expected increased incidence of skin cancers as these are strongly associated with UV-B exposure (De Gruijl and van der Leun 1994, IASC 1995). The three most common types of skin cancers are termed basal cell carcinoma, squamous cell carcinoma, and malignant melanoma. These tumors are lethal in <1%, <5% and approximately <30% of the cases, respectively. They develop from mutated cells in the basal cell layer of epidermis, the squamous cell layer, and from the melanocytes (pigment cells) in the basal cell layer. The incidence of skin cancers is strongly dependent upon skin phototype and melanocompetent skin type. Inuit populations are far less likely to develop skin cancers than are those with incomplete photorepair mechanisms.

In Greenlanders, only about 15% of all three types of tumors are found relative to the expected prevalence from the frequency among Danes living in Denmark (Prener *et al.* 1991). The reasons for this lower incidence in Greenlanders are protection against exposure. In part this is due to the dark pigmentation of skin compared to Danes in Denmark. Additionally, whereas Danes living in Denmark engage in sunbathing, in particular combined with travel and vacations in regions of high UV, Greenlanders are protected by more or less complete clothing cover year-round. Wulf (1994) has demonstrated that, among Inuit, pigment protection of the skin against UV-B is about twice as efficient as that in the Scottish population.

The UV-B exposure level of unprotected skin in Arctic regions has not been investigated, and studies of ground radiation in Nuuk, the most populated area of Greenland, has just begun.

Cataract

Animal studies have shown that cortical and posterior subcapsular cataract can be caused by UV-B (Pitts 1970). In humans, cortical cataract has also been linked to chronic UV-B radiation exposure (WHO 1994a).

The action spectrum for cataract formation, together with the absorption spectrum of the cornea, reveals that wavelengths between 295 and 325 nm are most effective (Pitts 1970). Because these wavelengths correspond exactly to the wavelength region that will increase as a result of ozone depletion, special attention should be paid to proper eye protection, keeping in mind that ground reflection with diffuse radiation requires goggles with side shielding.

Increases in UV-B radiation should be considered a problem which could be costly for health care in remote areas (IASC 1995). Other eye diseases such as spheroidal degeneration of the cornea, conjunctiva (climatic droplet keratopathy), and pterygium, may or may not be associated with UV-B exposure. However, pterygium mainly occurs under conditions of climatic extremes and might therefore be of special relevance for UV-B in the Arctic (IASC 1995, WHO 1994a).

Photodermatoses

There are a number of skin pathologies which are caused by UV radiation. Photodermatoses are often provoked by UV-A exposure and may actually be improved by some UV-B irradiation. For example, polymorphic light eruptions (PLE) is a common skin disease which appears most often with heavily itching papules. The rash is primarily UV-A induced and may actually be improved slightly if the ratio of UV-B to UV-A increases, as will occur with ozone depletion. The beneficial effect should be caused by increased skin thickness, increased

pigmentation, and the UV-B suppression of immune function. The frequency of this disorder in the Arctic is not known.

Actinic prurigo (AP), a skin disorder, is described as affecting the Canadian Inuit and may be hereditary. AP is most often provoked by UV-B and will be worsened with an increase in ambient UV-B radiation. This seasonal disease starts in childhood (Orr and Birt 1984).

Immune system

There has been a growing recognition during the last decade that UV irradiation of the skin can alter the immune system. Although very low, sub-erythemal doses have long been reported to enhance the general immune system, the adverse affects of UV exposure – particularly UV-B – are now recognized. UV-B exposure affects the function of the antigen-presenting cells of the skin, thereby affecting the immune system (Schwarz 1995). The immune effect is not limited to the skin as there is also experimental evidence of a systemic immunosuppressive effect of UV-B radiation (Goettsch *et al.* 1994). It is unclear how much exposure is necessary to induce systemic immune suppression (IASC 1995).

The immune function may be easily affected by UV-B irradiation because *trans*-urocanic acid on the skin surface is converted by UV-B to *cis*-urocanic acid, which directly affects the Langerhans cells in the skin (Pasanen *et al.* 1990, Noonan and deFabo 1992).

The suppression of the immune system is primarily a function of UV-B irradiation. The immune defense system may thus be impaired as a result of increased UV-B radiation due to ozone depletion (IASC 1995).

Antioxidant defense

Free oxygen radicals play an important role in causing DNA and cell membrane damage (Halliwell and Gutterridge 1989).

UV-B irradiation of the skin results in formation of reactive oxygen intermediates and elevated oxidative stress. Also, dietary contaminants such as heavy metals may result in this type of reaction. It is therefore possible that dietary contaminants and UV-B radiation may act together to reduce the naturally-occurring antioxidant defense systems of the skin. However, the diet in the Arctic is, in general, rich in antioxidants, which may provide some protection (Halliwell and Gutteridge 1989). Improvements in the defense systems in relation to chronic UV-B exposure have also been described (Shindo *et al.* 1993). The overall result of increased UV-B radiation should be considered harmful (Punnonen *et al.* 1995).

General conclusions

Since Arctic populations are largely well clothed and the skin covered except for the face, one may expect only a slight increase in the risk of skin cancer other than for the face. Of far more serious concern may be ocular exposure.

12.3. Essential elements and other nutrients

Traditional diets of all cultural groups in the Arctic include large amounts of animal foods. These diets are known to be rich sources of most of the required nutrients, and it is evident that the majority of essential minerals, protein and essential fatty acids in the total diet come from the animal species traditionally used for food.

This section discusses the essential trace elements included in the human health core program (Cu, Zn, Se), as well as iron and tin. It also looks at fatty acids, vitamins and protein intakes, all of which may modify the effects of contaminants.

12.3.1. Copper (Cu)

Copper is an essential trace element. It is an important cofactor in oxidative proteins or enzymes (da Silva and Williams 1991). For example, cytochrome-c oxidase, which is critical to respiration, contains copper; similarly, tyrosinase is a copper-based enzyme involved in the oxidative catabolism of tyrosine. Ceruloplasmin is the most important serum copper-transport protein.

Good dietary sources of copper are organ meats, especially liver, followed by seafood, nuts and seeds (US National Research Council 1989). Human milk contains approximately 0.3 mg/L, while cow's milk contains around 0.09 mg/L. An intake of 1.5-3.0 mg Cu per day for adults (between 1.0-2.0 mg per day for children) is considered adequate and safe (Burtis and Ashwood 1994).

Copper deficiency

Although copper deficiency is not frequently reported, it produces a plethora of clinical symptoms (Burtis and Ashwood 1994). Patients exhibit low plasma ceruloplasmin levels, anemia, neutropenia, and scurvy-like bone changes which respond to copper supplements. Deficiency is also illustrated by Menke's disease, a sex-linked disorder of copper metabolism in which a defective copper transport gene is responsible for systemic copper deficiency. Individuals exhibit progressive cerebral degeneration, retarded growth and abnormally sparse and brittle hair ('kinky hair syndrome').

Copper toxicity

Copper toxicity is rare (Burtis and Ashwood 1994). The genetically determined Wilson's disease clearly illustrates that copper is a systemic poison. In this inborn error of metabolism, there is an accumulation of copper in all body tissues due to a defective gene encoding for a protein involved in copper transport. A positive copper balance leads to deposition of copper in the liver where it causes cirrhosis; in the brain, causing mental disturbances, spasticity and tremor; in the cornea, where the deposits are visible as the Kayser-Fleischer ring; and in the kidneys, causing renal tubular loss of amino acids, phosphate, bicarbonate and urate.

Accepted reference intervals

The generally accepted serum Cu levels are between 601 and 1373 μg/L (Minoia *et al.* 1990). In pregnant women, the concentration is generally higher, increasing during pregnancy. Accepted values at term are between 1180 and 3200 μg/L. The blood Cu concentration is known to increase in women using estrogen therapy or substitution. A variation through childhood and youth is recognized, with low levels of 200-700 μg/L between 0-6 months, increasing to 900-1900 μg/L by 6-12 years of age. There are also gender differences, with slightly lower levels in males (700-1400 μg/L) than in females (800-1550 μg/L).

12.3.2. Zinc (Zn)

More than 70 metalloenzymes are known to require zinc as a cofactor. One of these is the zinc- and copper-containing superoxide dismutase (SOD), which is important in the oxidative defense system. Zinc also induces metallothionein, a free radical scavenger. Metallothionein also binds other heavy metals, especially cadmium, and as such acts as a detoxifying agent.

To meet the needs of most healthy people, the recommended zinc intake for adults in North America is 15 mg/d for males

and pregnant women, 12 mg/d for other females and 10 mg/d for children. Zinc supplementation is normally not required. The accepted reference interval for zinc in plasma is 700 to 1500 μg/L. Plasma zinc concentrations exhibit both circadian and post prandial fluctuations, and are low during pregnancy. Fasting morning values of plasma Zn below 700 μg/L suggest marginal zinc deficiency.

Although zinc levels in the body are regulated by homeostatic mechanisms and do not accumulate with continued exposure (Bertholf 1988), the margin of safety for dietary zinc intake is relatively narrow. The effects of excess zinc intake include: impairment of copper status (hypocupremia), microcytosis, neutropenia, immune response impairment and reduction in high density lipoprotein concentrations (HDL).

12.3.3. Selenium (Se)

Populations depending mainly on marine foods have a relatively high dietary intake of selenium, while those consuming food from the terrestrial food chain generally receive a moderate or low amount. Daily recommended dietary intake in most countries is 50-250 μg/d for adults.

Very high blood selenium levels have been reported in some Inuit populations for whom marine mammals are an important food source. In the Thule district in North Greenland, a median blood Se concentration of 1225 μg/L has been reported (Hansen 1990). The highest individual concentration was 3000 μg/L. Chronic selenosis is known in China, and symptoms in susceptible patients were found at blood concentrations above 1000 μg/L (Yang *et al.* 1989).

The Chinese study suggests a selenium intake of 400 μg/person/d as a maximum safe daily intake, corresponding to 560 μg/L in blood. No signs of chronic selenosis have been observed in the population of North Greenland. This may be due to the fact that in the Arctic, selenium is supplied through food of animal origin where the selenium is protein-bound as Se^{2-} in selenomethionein or selenocysteine. In China, the selenium is present in the soil and probably also in drinking water as selenate (Se^{6+}) or selenite (Se^{3+}), which have oxidative properties. As such, the suggested maximum safe intake of 400 μg/person/d is not appropriate for Arctic populations.

Whole blood Se concentrations up to 80 μg/L are significantly correlated with increased activity of the hydroperoxide-reducing enzyme glutathione peroxidase (GSH_{Px}), suggesting that 80 μg/L is close to the saturation limit for this enzyme (Thomson and Robinson 1986). It is not known whether this level in blood is sufficient to meet the requirements of other functional selenoproteins and for a possible cancer prevention effect.

A daily selenium intake of 50-250 μg is regarded as safe and adequate. This corresponds to whole blood Se levels between 115 and 390 μg/L according to the regression between blood selenium concentration (y mg/L) and dietary selenium intake (x μg/d) calculated by Yang *et al.* (1989):

$$\log y = 0.767 \log x - 2.248.$$

There is a good correlation between serum- and erythrocyte-selenium under conditions of low selenium intake. At higher intakes, the serum selenium concentration tends to level off at a concentration of approximately 150 μg/L, while erythrocyte selenium increases with intake. Consequently, the erythrocyte-selenium to plasma-selenium ratio seems to be the best measure of selenium status, especially in the Arctic where there is a very high dietary supply in some population groups.

Plasma selenium concentrations decrease during pregnancy compared to plasma concentrations of non-pregnant women. This is probably a result of the increased total plasma volume. In cord blood plasma, the selenium concentration is about 40% lower than the maternal plasma level, while maternal and cord blood whole blood levels are the same (Hansen *et al.* 1984). This may be partly due to the higher hematocrit value of cord blood as well as the higher binding capacity of fetal hemoglobin.

There is some evidence that low selenium intake may be associated with low sperm motility (MacPherson *et al.* 1994). A review of selenium in relation to fertility is provided in Hansen and Deguchi (1996).

It has also been suggested that selenium is a protective factor in the development of atherosclerotic lesions and ischemic heart disease (IHD). However, essential fatty acids of the n-3 series have also been suggested as protecting against this disease. Because selenium and n-3 fatty acids are both present in large quantities in marine food, it is difficult to conclude definitively the role of selenium in the protection against IHD. It is possible that selenium acts as an important co-factor, contributing to the oxidative defense system (for review see Hansen *et al.* 1994). A recent study (Tsukahara *et al.* 1996) indicates that selenium is of importance for collagen metabolism in young infants.

Epidemiological investigations have indicated a decrease in human cancer death rates (age and sex adjusted) correlated with increased selenium intake. There is also experimental evidence in animals to support the antineoplastic effect of selenium. A possible mechanism of the protective effects of selenium has been postulated involving the inhibition of the formation of carcinogen malonaldehyde, a product of peroxidative tissue damage.

In addition to the apparent protective effect against some carcinogenic agents, selenium is an antidote for the toxic effects of other metals, particularly arsenic, cadmium, mercury (organic and inorganic), and copper. The mechanisms underlining these interactions are not fully understood. The protective effect of selenium on mercury toxicity is discussed in more detail in chapter 7.

12.3.4. Iron (Fe)

While iron is as an essential element, there are also toxicological considerations associated with excess dietary iron. Body iron stores are affected by a variety of factors and are strongly influenced by nutritional iron intake. Serum ferritin correlates well with iron stores in healthy individuals. Milman *et al.* (1992) demonstrated that Inuit hunters in North Greenland had high serum ferritin levels and ample body iron reserves. Eight of 67 individuals investigated showed values greater than 300 μg/L, i.e., increased iron stores. The serum ferritin levels increased with age without the plateau seen in Caucasians which suggests continued accumulation of iron reserves in elderly Inuit associated with consumption of large quantities of iron rich meat from marine mammals and/or genetic differences in the regulatory mechanisms for body iron stores. The high dietary intake of iron may protect against absorption of cadmium also present in marine food. Iron deficiency has, however, been described among Canadian Inuit (Valberg *et al.* 1976).

12.3.5. Tin (Sn)

Although animal experiments indicate that tin is an essential nutrient, naturally occurring tin deficiency is unknown in both animals and humans.

The use of tin cans for food preservation presents little hazard to health unless the tin surfaces are exposed to the air and contain acidic material. Ingested inorganic tin is poorly absorbed and is mainly excreted in the feces. Apart from rare reports of gastrointestinal symptoms, there is little evidence of human toxicity from inorganic tin in foods.

The organo-tin compound tributyltin (TBT) may, however, be of importance to the Arctic marine environment. Low concentrations of TBT originating from antifouling paint used on ships have been shown to have reproductive effects in mollusks (Svavarsson and Skarpheðinsdottir 1995). TBT has also been shown to be highly toxic in experimental animals, acting through the generation of reactive oxygen species (Clerici 1996). The documented effects of TBT on human health are inflammation of the airway, and eye and skin irritation (WHO 1990a, Snoeij *et al.* 1987). The effect of TBT on skin is suggested to involve intercellular modulation of interleukin-1αIL-1α (Corsini *et al.* 1996). An evaluation of the human toxicity of TBT, as a marine contaminant, is warranted if it is shown to be transported through the food chain.

12.3.6. Other nutrients

While this report focuses on contaminants in the circumpolar region, it is important not to lose sight of the larger benefits that traditional diets provide to the health and well-being of Arctic peoples.

Traditional foods, especially marine mammals and fish, contain large amounts of specific essential lipids (n-3 fatty acids), and their consumption has been associated with a lower incidence of ischemic heart disease in Greenland, Japan and Canada (Dyerberg *et al.* 1975, Yamori *et al.* 1985, Young *et al.* 1993). A high ratio of eicosapentaenoic acid (EPA), an n-3 fatty acid, to arachidonic acid (AA), a non-n-3 fatty acid, is considered a valid indication of low risk of ischemic outcomes (Hirai *et al.* 1987). For Inuit living along the shore of Hudson Bay in the Canadian Arctic, death from ischemic illness is very rare and EPA to AA ratios are more than seven times higher than in southern Canadian populations (Dewailly *et al.* 1994b, and pers. comm.).

The contribution of traditional diets to total energy requirements, and intakes of proteins, vitamins, and essential elements will vary by indigenous group, by region, by food items consumed, and by frequency/seasonality of consumption. Total daily energy provided by country food has been reported to range between 47% (Wein 1995 – Hudson Bay, Canada), 29% (Kuhnlein *et al.* 1996 – Baffin Island, Canada) and 19% (Kuhnlein *et al.* 1995a – Central Arctic Canada). The majority of the protein intake, and well in excess of required nutrient intakes, for both Inuit and Dene/Métis comes from their traditional diet (Kuhnlein *et al.* 1995b, 1996). These same diets also provide more than adequate amounts of iron and zinc, vitamins D and E and thiamin, riboflavin and niacin. High levels of vitamin A (as retinol) are found in animal liver and blubber. Only a few essential elements and vitamins (calcium, vitamin C and B_6, folic acid) are found in barely adequate amounts (Doolan *et al.* 1991, Lawn and Langrer 1994, Wein 1994a, 1994b and 1995, Kuhnlein *et al.* 1995b).

Nutrients in the diet often interact directly to influence the uptake or toxicity of environmental contaminants. Zinc, iron, calcium, selenium, cobalt, manganese, pyridoxine (B_6 complex vitamin), ascorbic acid and dietary protein all interact to reduce cadmium toxicity, while zinc, iron, calcium, selenium, cobalt and chromium can all influence the toxicity of lead (dietary deficiencies increase lead toxicity) (Chowdury and Chandra 1987). Dietary calcium has also been inversely associated with blood lead levels in children 1-11 years old. Vitamin D_3 (cholecalciferol), while stimulating the absorption of calcium, phosphate and other essential elements, also increases the absorption of some toxic metals (cadmium, lead) and radionuclides (strontium, caesium) (Moon 1994).

Traditional diets provide a strong nutritional base for the health of Arctic indigenous peoples. While evidence of clinical deficiencies of individual vitamins or minerals has not been documented in Arctic populations, a move away from the consumption of traditional foods, especially animal foods, would most likely lead to significant nutritional deficiencies and poor health.

12.4. National reports

12.4.1. Canada

Introduction

The 93 000 people of Canada's north populate a very large land mass encompassing the Yukon Territory in the west, the central and northern Northwest Territories (NWT), and northern Quebec and Labrador in the east. The 46 500 indigenous peoples living in the north make up 17-90% of the population in the various territories/regions, and include the Inuit, the Indians or First Nations, and the Métis (see chapter 5) (Figure 12·2).

In response to concerns about environmental contaminants and the importance of country foods in the north, the Government of Canada instituted the national Arctic Environmental Strategy, Northern Contaminants Program in 1991. Much of the new data reported here is a product of this program and should help to demonstrate if (and how) the northern people (Figure 12·2) and/or their environment are being adversely affected by these contaminants.

Sources of exposure

General population

The indigenous peoples of the north are much more likely to be exposed to several environmental contaminants than the non-indigenous population of Canada's north, because they consume a much larger proportion of traditional foods, i.e., the products of hunting, fishing or gathering in the north. Harvest surveys indicate country food consumption in the Canadian Arctic ranging from 27 to 292 kg per person per year. The leading foods include caribou, moose, fish, seal, muskox and waterfowl, though some indigenous groups consume over 80 different northern species (CACAR 1997) (for more detailed information see chapter 5). In the eastern Canadian Arctic, the major sources of exposure of coastal peoples are seal and beluga meat for mercury and beluga fat for POPs, and to a lesser extent fish for both of these contaminants. In the western Canadian Arctic, the primary source of POPs for non-coastal peoples comes from freshwater fish. Consumption of large land mammals contributes to body burdens of cadmium and radionuclides.

Local populations

There are very few significant sources of local pollution in Arctic Canada. PCBs have been reported near abandoned Distant Early Warning (DEW) Line sites, but these are not a major contributor to total Arctic environmental loadings. Past and present mining operations in the Northwest Territories, including Yellowknife and Port Radium, may lead to local exposures to some heavy metals. Geological cadmium in the Yukon Territory is probably responsible for the slightly higher concentrations in caribou kidney in the western Canadian Arctic. There is, as yet, no consensus on the reasons for elevated mercury levels in the eastern Canadian Arctic.

Figure 12·2. Indigenous Peoples of the Canadian Arctic.

Long-range transport from industrial and natural sources, acidification of the eastern land mass, naturally high geological levels and hydroelectric flooding have all been postulated.

Levels and trends of contaminants in humans

DDT and metabolites

For the population in southern Canada, the dietary intake of total DDT (including metabolites) has been declining since the 1960s. This has been reflected by a concomitant ten-fold decline in total DDT in human tissues over the last 20 years (Conacher and Mes 1993, Mes 1994).

Levels of total DDT and its metabolites in human tissue in the Arctic are considerably higher than those in southern Canadians, reflecting the greater consumption of high trophic level species for food. Concentrations of DDE are four- to five-fold higher in human breast milk from Inuit in northern Quebec than populations from southern Canada (Table 12·4). People from the north shore of the St. Lawrence River who consume large amounts of fish and gull eggs have intermediate levels of DDE in breast milk. Even higher concentrations of DDE are seen in abdominal fat tissue from Greenland Inuit. The observed differences may be due to increased consumption of contaminated country foods, older age groups in the Greenland Inuit study versus the Quebec Inuit study, or tissue-specific concentration differences (abdominal fat vs. breast milk fat). There is no indication that DDT levels are declining in Arctic populations, however, tissue sampling in the north has only occurred relatively recently and, thus, there are insufficient data for temporal trend analyses.

Table 12·4. Comparative concentrations of DDE in human milk fat (Canada) and omental fat (Greenland).

Location	Year of sampling	Mean, ng/g lipid	Reference
Southern Canada	1992	222	Newsome *et al.* 1995
Southern Quebec	1989/90	340	Dewailly *et al.* 1996a
Lower North Shore, Quebec	1991	823	Dewailly *et al.* 1991
Nunavik, northern Quebec	1989/90	1212	Dewailly *et al.* 1993a
Greenland	1993	3844	Dewailly pers. comm.

Toxaphene

Toxaphene intakes by Arctic populations are entirely dependent on the type and amount of country food consumed (of all tissues tested, toxaphene levels are highest in ring seal blubber and beluga blubber in the eastern Arctic) (Kuhnlein *et al.* 1995a). There are very few data available on toxaphene in the tissues of Arctic populations. A study of three Inuit women from northern Quebec reported levels of toxaphene in breast milk of 221 ng/g lipid (Stern *et al.* 1992). This is a very small sample size for any evaluation and is unlikely to be representative of the larger population. A recent analysis of a 1987-88 dietary survey indicates that for one group of Canadian Inuit women the daily intake of toxaphene frequently exceeds the TDI (Table 12·35).

PCBs

In southern Canada, the estimated adult daily intake for total PCB from diet is 0.008 μg/kg bw/day (Conacher *et al.* 1994). Intakes by Arctic residents consuming large amounts of traditional foods from the aquatic and marine environment are considerably higher. In results from two surveys of Canadian Inuit women, 16% and 4% of the women's daily intakes exceeded the TDI (Table 12·35).

Several studies suggest that PCB levels in breast milk fat are significantly elevated in some Inuit mothers. In breast milk samples collected from Inuit women residing in the Nunavik region of Arctic Quebec (Hudson Bay, Hudson Strait and Ungava Bay), total PCB levels were elevated 4.7-fold in 1988 and 5.6-fold in 1989/90 when compared to a southern Quebec non-indigenous population in the same sampling years

Table 12·5. Comparative concentrations of PCBs in human milk fat.

Location	Year of sampling	Mean, ng/g lipid	Reference
Southern Canada	1992	238[b]	Newsome *et al.* 1995
Southern Quebec	1989-90	520[a]	Dewailly *et al.* 1992
Southern Quebec	1988	770[a]	Dewailly *et al.* 1989
Lower North Shore, Quebec	1991	2160[a]	Dewailly *et al.* 1991
Nunavik, northern Quebec	1988	3600[a]	Dewailly *et al.* 1989
Nunavik, northern Quebec	1989-90	2900[a]	Dewailly *et al.* 1992

a. PCBs as Aroclor 1260. b. Sum of PCB congeners (PCBs as sum of congeners is typically approximately one-half PCBs as Aroclor 1260).

Table 12·6. Comparative concentrations of dioxins, furans and dioxin-like PCBs in human milk fat.

Location	Year of sampling	Mean[a], pg/g lipid	Reference
Dioxins and furans			
Southern Canada	1986-87	15.6	Ryan *et al.* 1993
Southern Quebec	1989-90	9.6	Dewailly *et al.* 1992
Nunavik, northern Quebec	1989-90	19.0	Dewailly *et al.* 1992
Netherlands	1990-92	30.2	Koopman-Esseboom *et al.* 1994a
Dioxins, furans, and dioxin-like PCBs			
Southern Quebec	1989-90	17.9	Dewailly *et al.* 1992
Nunavik, northern Quebec	1989-90	42.3	Dewailly *et al.* 1992
Netherlands	1990-92	46.2	Koopman-Esseboom *et al.* 1994a

a. expressed as 2,3,7,8-TCDD TEQs.

Table 12·7. Comparative concentrations of mirex in human milk fat (Canada) and omental fat (Greenland).

Location	Year of sampling	Mean, ng/g lipid	Reference
Southern Canada	1986-87	15.6	Ryan *et al.* 1993
Southern Canada	1992	1.9	Newsome *et al.* 1995
Southern Quebec	1989-90	1.6	Dewailly *et al.* 1993a
Lower North Shore, Quebec	1991	12	Dewailly *et al.* 1991
Nunavik, northern Quebec	1989-90	16	Dewailly *et al.* 1993a
Greenland	1993	153	Dewailly pers.comm.

(Table 12·5). Women from the north shore of the St. Lawrence River who consume more fish and country food than women from southern Quebec but less than Inuit women had intermediate levels of PCBs in their breast milk.

While levels of total PCBs have declined since the 1980s in breast milk of southern Canadian women (Newsome *et al.* 1995), there is no similar information yet available for Arctic residents. Historical values are also difficult to compare due to changing analytical techniques and quantitation methods (reported PCB concentrations based on an Aroclor 1260 standard are approximately double those based on a sum of specific PCB congeners). This may partly explain the difference seen in Table 12·5 between the 1992 southern Canada PCB level in breast milk of 238 ng/g lipid (sum of congeners) and the 1989-90 southern Quebec PCB level of 520 ng/g lipid (Aroclor 1260).

Dioxins (PCDDs) and furans (PCDFs)

In 40 breast milk samples collected between July 1989 and July 1990 from Inuit women residing in the Nunavik region of Arctic Quebec (Hudson Bay, Hudson Strait and Ungava Bay), 2,3,7,8-TCDD TEQs for PCDDs and PCDFs were moderately elevated when compared to the southern Quebec non-indigenous population (19.0 pg/g lipid vs. 9.6 pg/g lipid) (Table 12·6) (Dewailly *et al.* 1992). However, total PCBs were more than five-fold higher in the Quebec Inuit mothers' breast milk than the same control population. When TEQs for non-*ortho*-substituted PCBs (CBs 77, 126 and 169) were factored in, the 2,3,7,8-TCDD TEQ value increased to 42.3 pg/g for the Inuit milk samples compared to 17.9 pg/g for the southern Quebec samples (Table 12·6). For this Inuit population, but not the southern Quebec population, the majority of the 2,3,7,8-TCDD TEQs are due to dioxin-like PCBs.

A large Netherlands study of breast milk and child development found levels of 2,3,7,8-TCDD TEQ (PCDD/PCDF/coplanar PCBs) in breast milk of 46.2 pg/g lipid. These levels are very similar to the levels in Inuit breast milk (Table 12·6). In contrast to the Inuit results, the majority of the 2,3,7,8-TCDD TEQ in this Netherlands study are from the dioxin and furan component. Subtle clinical, immunologic and neurodevelopmental alterations associated with breast feeding have been reported in the infants/children of the Netherlands cohort (Koopman-Esseboom *et al.* 1994a, Huisman *et al.* 1995, Weisglas-Kuperus *et al.* 1995). A preliminary report on a study of Canadian Inuit also shows an association between increasing PCB/dioxin/furan levels and immune system deficits (Dewailly *et al.* 1993b).

Mirex

While mirex has never been registered for use in Canada as a pesticide, it did enter Canada as a fire retardant (dechlorane) from the mid-1960s to mid-1970s, and via water currents from the US side of the Great Lakes basin. Mirex levels in breast milk are above average for communities consuming high amounts of fish and marine bird eggs (Dewailly *et al.* 1991). Levels in the breast milk of Inuit from Nunavik, northern Quebec, are ten times higher than those in southern Quebec residents (Table 12·7). Intermediate levels of mirex can be seen in the lower North Shore St. Lawrence population which consumes more fish and seabird eggs than the general population (Dewailly *et al.* 1991). Even higher concentrations of mirex are seen in abdominal fat tissue from Greenland Inuit.

Chlordane

Chlordane is not currently registered for use in Canada and enters the Arctic ecosystem primarily via long-range atmospheric transport. Chlordane and the compounds related to it accumulate in fat and are found in human tissues although generally only in small amounts. Tissues often contain relatively larger amounts of *trans*-nonachlor and the metabolite oxychlordane. The breast milk of Inuit mothers from Nunavik (northern Quebec) had chlordane levels ten times higher than levels seen in southern Canadian residents (Table 12·8). Even higher relative levels of *trans*-nonachlor (80-fold) and oxychlordane (64-fold) are seen in abdominal fat from Greenland Inuit, but this may be due to several factors as indicated previously. In results from two surveys of Canadian Inuit women, 48% and 75% of the women's daily intakes exceeded the TDI. Only 6% of the Dene women's intakes exceeded the TDI (Table 12·35).

Table 12·8. Comparative concentrations of chlordane and related compounds in human milk fat (mean, ng/g lipid).

Location	Year	Chlordane (α and γ)	*Cis*-nonachlor	*Trans*-nonachlor	Oxy-chlordane	Reference
Southern Canada	1992	0.37	2.89	17.5	13.4	Newsome *et al.* 1995
Southern Quebec	1989-90	n.d.	–	–	–	Dewailly *et al.* 1993a
Lower North Shore, Quebec	1991	n.d.	–	–	–	Dewailly *et al.* 1991
Nunavik, northern Quebec	1989-90	3.7	–	–	–	Dewailly *et al.* 1993a
Greenland	1993	11.6	3.12	1463	862	Dewailly pers. comm.

n.d.: not detected. – : not analyzed/not reported.

Table 12·9. Comparative concentrations of HCHs in breast milk (Canada) and omental fat (Greenland) (ng/g lipid).

Location	Year	α-HCH	β-HCH	γ-HCH	Reference
Southern Canada	1992	0.31	22.6	1.03	Newsome *et al.* 1995
Greenland	1993	–	108.9	–	Dewailly pers. comm.

Hexachlorocyclohexanes (HCHs)

Canada still has some registered uses for lindane (α-HCH). Like other POPs, most human exposure to HCHs comes from food consumption. Because of its persistence, β-HCH is found at the highest level of the three isomers reported in the southern Canadian population (Table 12·9). There are no data for levels in Arctic populations except for one report from Greenland where β-HCH levels in abdominal fat samples were five times higher than southern Canadian levels in breast milk fat (Table 12·9). As previously indicated for DDT, differences in contaminant levels in the samples from Greenland and Canada may be explained by a number of factors.

POPs in newborn cord blood

The exposure of the developing fetus to maternal contaminants through the placenta is an important route of exposure during many critical stages of development. Data has recently become available on POPs in newborn cord blood from several Canadian Arctic and southern populations (Dewailly *et al.* 1996b, Walker *et al.* in prep.). Inuit populations have a greater range of POPs at detectable levels and higher geometric mean concentrations of POPs. This is not unexpected as Inuit consume more marine fats, which have elevated POP residues, than Dene/Métis peoples in the central Arctic (Kuhnlein *et al.* 1995b). Any comparison of maternal and newborn cord blood concentrations of contaminants needs to consider that the important determinants of contaminants level are age of the mother, number of previous children, and number of children breast fed, as well as the amount and type of country food consumption. While these comparisons are as yet incomplete, it is possible to compare some of the contaminants showing the highest levels and greatest differences between the groups to verify whether previous information on country food consumption supports the contaminant patterns seen.

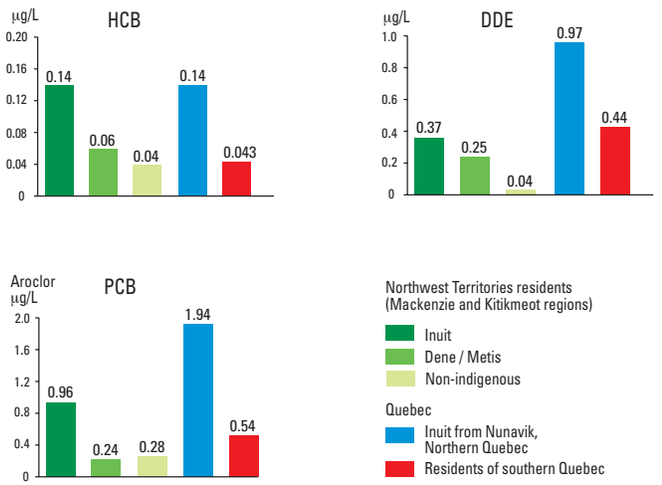


Figure 12·3. Geometric mean levels of HCB, DDE and PCBs in cord blood of newborns in different Canadian population groups.

Based on geometric means, the contaminants present in the highest concentrations in newborn cord blood are hexachlorobenzene (HCB), DDE and PCBs (Figure 12·3). Levels of PCBs and DDE are twice as high in Nunavik (northern Quebec) Inuit than in Inuit living in the NWT. HCB levels are similar in Inuit in both areas. The difference between Inuit from Nunavik and Inuit from the NWT warrants further study and analysis. Figure 12·3 also shows a comparison of Inuit with other population groups. The concentrations of PCBs and DDE are considerably higher in the Inuit (Nunavik and NWT) than in the other population groups (Dene/Métis and non-indigenous in the NWT; and residents of southern Quebec). Levels of HCB in Inuit (both NWT and Nunavik) are two to three times the levels seen in the other population groups.

Analyses were completed for 14 PCB congeners in newborn cord blood from Nunavik and southern Quebec populations. Similar PCB congener patterns can be seen in these two populations, although the levels for each congener in the Nunavik population survey are consistently higher (Figure 12·4).

PAHs

There are no data available on PAH levels in breast milk, fat or blood of Canadian Arctic residents.

Mercury

From 1970 to 1995, the Medical Services Branch (MSB) of Health Canada, within its Environmental Contaminants Program, has carried out mercury analyses of hair and blood

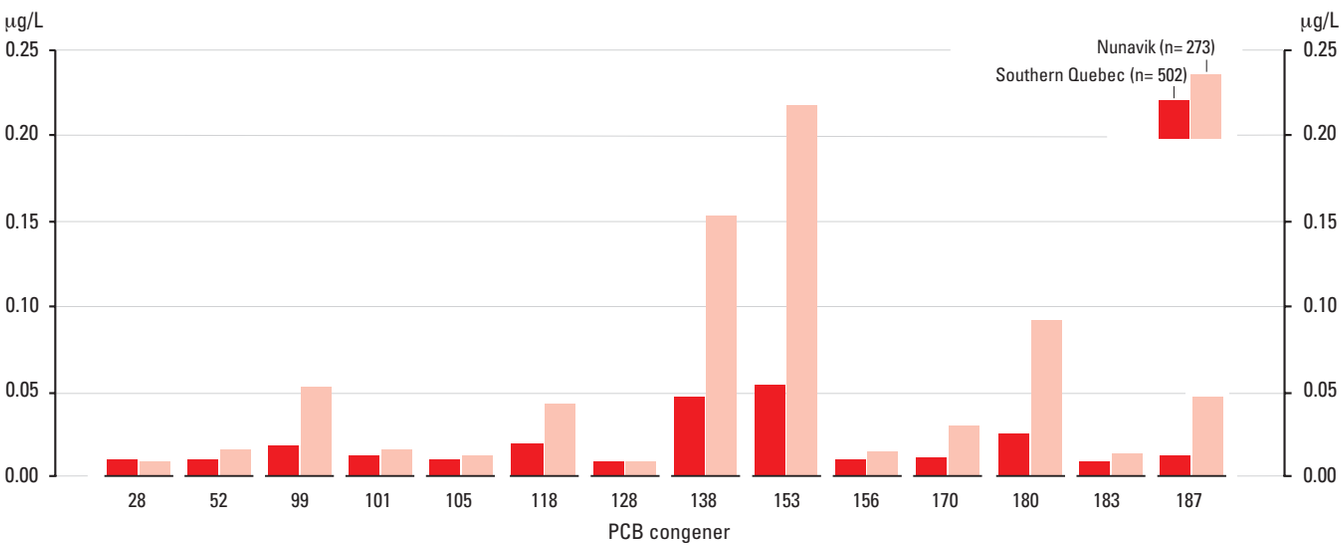


Figure 12·4. Concentrations of PCB congeners in cord plasma from Nunavik and southern Quebec newborns (adapted from Dewailly *et al.* 1996b).

Table 12·10. Individual blood methylmercury levels for NWT, Canada, by cultural group (1972-1989). Source: Wheatley 1995.

Methylmercury level, μg/L	Dene		Inuit	
	No. of individuals	%	No. of individuals	%
Less than 20	572	80.45	647	42.82
20 to 99	137	19.27	844	55.86
100 to 199	2	0.28	16	1.06
200 to 299	0	0	4	0.26
Total	711	100.00	1511	100.00

samples obtained from 38 571 residents of 514 native communities across Canada as part of clinical risk assessment (Wheatley 1994). As shown in Table 12·10, of 711 Dene and 1511 Inuit from the NWT who were tested, nearly three times as many Inuit (57.2%) exceeded 20 μg/L as Dene (19.6%) (Wheatley 1995, Wheatley and Paradis 1996a).

Table 12·11 sets Canadian findings in the context of findings in other circumpolar countries and over time. In general, most the mercury data reported by Wheatley and Paradis (1995, 1996a, 1996b) indicate that Inuit from the eastern NWT and Nunavik, and northern Quebec Cree, have higher levels of mercury than other circumpolar peoples. One recent data set from Greenland indicates high cord and maternal blood mercury levels are also found in this population (Hansen *et al.* 1990a, 1990b). Some archeological hair sample results from northern Baffin Island (dated to originate from the years 400 and 1150) (Wheatley and Wheatley 1988) and from Greenland (1485) (Hart-Hansen *et al.* 1991) are also included in Table 12·11 (converted to blood level equi-

Table 12·11. Current and historical blood mercury levels.

Location	Year(s) collected	n	Mean[a], μg/L	SD[b]	Range, μg/L	DL[c] μg/L	Reference	Comments
Canada (indigenous peoples)	1970-1995	38571	14.13	22.63	1-660	1	Wheatley and Paradis 1995[d]	Blood, cord blood, hair
Quebec								
Nunavik (Inuit)	1974-1982	1114	46.76	33.08	2.67-267.33	1	Wheatley and Paradis 1996a[d]	Blood, cord blood, hair
	1977-1982	142	48.21	43.98	4.20-254.50	1		Blood
	1978-1982	125	33.80	20.09	6.00-101.80	1		Cord blood
	1974-1982	847	48.42	32.10	2.67-267.33	1		Hair
Nunavik (Inuit)	1992	252	14.06		0.8-96		Dewailly *et al.* 1994b	Blood (men)
	1992	240	15.97		2.0-112			Blood (women)
	1993-1995	299	12.09		1.0-99		Dewailly *et al.* 1996b	Cord blood
Northern Quebec (Cree)	1971-1982	4670	34.31	40.56	1.50-649.40	1	Wheatley and Paradis 1996a[d]	Blood, maternal blood, cord blood, hair
	1971-1985	1129	42.94	52.32	1.50-649.40	1		Blood
	1971-1982	600	22.69	23.82	1.50-224.00	1		Cord blood
	1972-1982	2940	33.38	37.29	1.67-571.00	1		Hair
Northwest Territories								
Nunavut (Inuit)	1972-1989	1339	29.98	23.99	1.00-225.67	1	Wheatley and Paradis 1996a[d]	Blood, maternal blood, cord blood, hair
	1972-1989	286	19.52	22.33	1.00-200.00	1		Blood
	1978-1988	61	17.17	15.48	1.50-86.00	1		Maternal blood
	1978-1986	31	40.53	32.74	6.50-130.40	1		Cord blood
	1973-1987	961	33.57	23.37	1.00-225.67	1		Hair
Nunavut (Inuit)	1994-1995	67	3.46		0.6-12.84	1	Walker (in prep.)	Maternal blood
	1994-1995	62	5.72		0.6-27.88	1		Cord blood
Western NWT (Dene)	1976-1983	980	16.24	16.54	1.00-138.00	1	Wheatley 1995[d]	Blood, cord blood, hair
	1982-1983	76	10.66	11.13	1.30-66.50	1		Blood
	1978-1982	5	14.36	12.32	4.30-35.00	1		Cord blood
	1976-1986	899	16.72	16.86	1.00-138.00	1		Hair
Western NWT (Dene)	1994-1995	51	1.74		0.4-5.62	1	Walker (in prep.)	Maternal blood
	1994-1995	47	1.91		0.4-8.83	1		Cord blood
Non-indigenous	1994-1995	121	1.25		0.20-4.21	1		Maternal blood
	1994-1995	121	1.66		0.20-12.84	1		Cord blood
Yukon	1977-1978	299	7.10	6.15	1.50-67.00	1	Wheatley 1979[d]	Blood, cord blood, hair
	1977-1978	83	7.04	4.01	2.10-21.70	1		Blood
	1977	31	4.12	1.87	1.50-7.80	1		Cord blood
	1977	185	7.63	7.19	1.67-67.00	1		Hair
Comparison groups								
Ontario	1992-1993	176	2.80	2.30	< 2.0-17.0	2	Kearney *et al.* 1995	Whole blood (fisheaters)
Greenland	1987	35	12.00		4-23		Milman *et al.* 1994	Serum
	1987	32	10.00		3-24			Serum
	1982-1988	37	44.13	24.07	6.02-96.29	1	Hansen *et al.* 1990b	Maternal blood
	1982-1988	37	88.25	48.34	10.03-178.54	1		Cord blood
Faeroe Islands	1986-1987	53	12.00		2.6-50.0		Grandjean *et al.* 1992	Whole blood
	1986-1987	53	2.10		< 0.1-8.2			Serum
	1986-1987	997	24.20		13.0-40.2			Cord blood
Historical levels from mummified remains								
Greenland	ca. 1485	1	10.0				Hart-Hansen *et al.* 1991	Hair
N. Baffin (Dorset)	ca. 400	3	8.00		3-12	1	Wheatley and Wheatley 1988	Hair
N. Baffin (Thule)	ca. 1150	6	4.00		2-7	1		Hair

a. Measurements of mercury were made in a variety of tissues but all tissue levels are normalized as concentrations in blood. b. SD: Standard deviation. c. DL: Detection limit. d. Some of these data have been specifically analyzed for this report by Wheatley *et al.*, and are also described in Wheatley and Paradis 1995, 1996a.

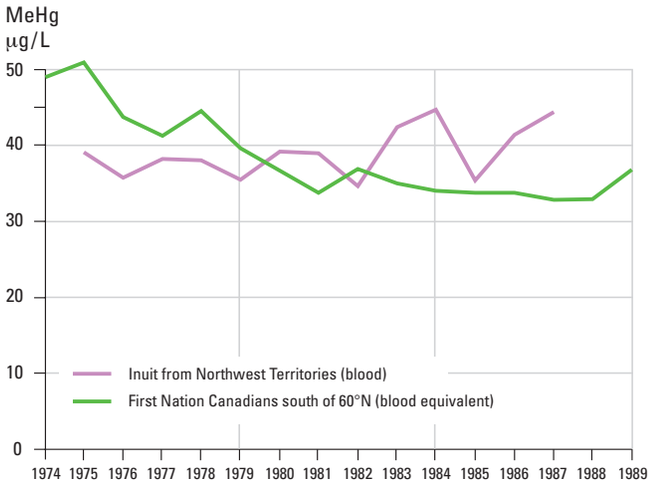


Figure 12·5. Time trends of mean methylmercury concentrations (adapted from Wheatley and Paradis 1996a).

valents to facilitate comparison). All are low compared with current human methylmercury levels in the same geographic areas. Figure 12·5 provides trend data over 15-20 years for individuals and communities south of 60°N (First Nations) and in the NWT (Inuit) (Wheatley and Paradis 1996a). The trend in First Nations communities south of 60°N appears to be downward. Whether this is because of falling mercury levels in fish or because less fish are being eaten by the people has yet to be determined. With the NWT data, there is no obvious trend. There is more recent data from the NWT and Nunavik which does suggest that some Dene and Inuit groups may have decreased blood mercury levels (means 1.74 to 15.97 µg/L from 1992-95 vs. 10.66 to 48.21 µg/L before 1992, Table 12·11). Dietary, geographic, regional, or seasonal differences may explain the apparent decrease.

In a limited number (n = 36) of cord blood samples collected from Inuit and Dene from 1978 to 1986 from various regions of the NWT, 69% had methylmercury levels equal to or greater than 20 µg/L (mean 36.9 µg/L, range 4.3-130.4) (Wheatley and Paradis 1996a). This is in contrast to more recent data (1993-95) from the NWT and Nunavik, northern Quebec, which found lower levels in cord blood of Inuit newborns (means 5.72 and 12.09 µg/L, respectively, Table 12·11). The concentration of mercury in maternal blood was also markedly lower in the 1994-95 Walker *et al.* study (in prep.) than was seen in the 1978-86 study (Table 12·11). The lower concentrations seen in 1994-95 cord and maternal blood samples may be due to dietary differences over time (1980 vs. 1994), geographic differences (eastern vs. western NWT) or seasonal differences.

In general, levels of methylmercury were higher in the northern and eastern Inuit communities where the consumption of fish and marine mammals is extensive (Wheatley and Paradis 1995). Two recent studies (1987, 1992) indicate that 29% and 37% of Inuit women in these communities had daily dietary intakes of mercury which exceeded the TDI (Table 12·35). The source of methylmercury (geological or anthropogenic) in the Canadian Arctic food chain and in traditional foods, such as marine mammals consumed by Inuit, continues to be a subject of debate (Wheatley and Wheatley 1988, Muir *et al.* 1992). There is also ongoing controversy about possible protective mechanisms involving selenium.

Lead

In the most recent Canadian market basket survey, the estimated mean dietary intake of lead for a sixty kilogram adult in 1986-1988 was 24 µg per day (Dabeka and McKenzie 1995). The estimated daily intakes for adults can range worldwide from 15-316 µg/d (WHO 1995). In the absence of occupational exposure, the average blood lead concentration in adults is 100-200 µg/L, depending on the degree of industrialization (air pollution, automobile exhaust, etc.). This value is far in excess of the estimated blood lead level in preindustrialized humans of 0.16 µg/L (Flegal and Smith 1992).

Results of the recent Santé Quebec Health Survey of Inuit from Nunavik, showed that the mean blood lead concentrations were approximately 86 µg/L. Blood lead levels could be positively correlated to smoking and consumption of marine mammals (Dewailly *et al.* 1994b). In a 1987 survey of northern Greenlandic Inuit hunters and their families, the median blood lead value for males was 96 µg/L (median age 40 years) and for females, 56 µg/L (median age 36 years) (Milman *et al.* 1994) (Table 12·12). The major source of lead exposure was estimated to be food items of marine origin (ringed seal, narwhal, walrus and beluga) and there was a positive correlation between increasing blood lead levels and age. In the Faeroe Islands, 52 women (20-50 years old) who consumed fish and pilot whale meat had a median blood lead level of 20 µg/L (Grandjean *et al.* 1992). In a 1989 sample of Michigan fishermen (n = 115) who consumed on average 38 fish meals per year, blood lead levels were 55 µg/L compared to 38 µg/L in the controls (n = 95, 4.1 average fish meals per year) (Hovinga *et al.* 1993) (Table 12·12). Blood lead values for both sample sets were directly influenced by amount of smoking. A study of fisheaters from the Canadian side of the Great Lakes also indicated that women who consumed Great Lakes fish had slightly higher blood lead compared to non-fisheaters (24 vs. 19 µg/L, Kearney *et al.* 1995, Table 12·12).

Initial results of cord blood screening for lead from Nunavik (Table 12·12) have found levels approximately three-fold higher than comparison samples from Toronto and Quebec City (52 µg/L vs. 17 and 18 µg/L, respectively) (Dewailly 1994a).

Table 12·12. Blood lead levels.

Location	Year(s) collected	n	Reported mean, µg/L	SD	Range, µg/L	DL[a], µg/L	Reference	Comments
Faeroe Islands	1986-87	52 ♀♀	20	±5.9%	8-36		Grandjean *et al.* 1992	Whole blood
Greenland	1987	35 ♀♀	56		8-326		Milman *et al.* 1994	Whole blood
Greenland	1987	35 ♂♂	96		31-240		Milman *et al.* 1994	Whole blood
Nunavik (Inuit-adult)	1992	492	86		4-23		Dewailly *et al.* 1994a.	Whole blood
Nunavik (Inuit-newborn)	1994	59	52	±6.8%	8-267	2	Dewailly *et al.* 1994a.	Cord blood
Northwest Territories (Inuit)	1994-95	62	29		6-157	2	Walker (in prep)	Cord blood
Dene/Métis	1994-95	47	20		2-62	2	Walker (in prep)	Cord blood
Non-indigenous	1994-95	121	16		2-64	2	Walker (in prep)	Cord blood
USA, Michigan	1989	115 ♂♂	55		10-170	10	Hovinga *et al.* 1993	Fisheaters, whole blood
USA, Michigan	1989	95 ♂♂	38		10-100	10	Hovinga *et al.* 1993	Controls, whole blood
USA, California	1984	1728	49		5-150		Satin *et al.* 1991	Cord blood
Canada, Cornwall	1992-93	32	24		5-56	2	Kearney *et al.* 1995	Fisheaters, whole blood
Canada, Cornwall	1992-93	10	19		5-62	2	Kearney *et al.* 1995	Non-fisheaters, whole blood

a. DL: Detection limit.

A similar cord blood study from the NWT found slightly lower lead levels in Inuit cord blood (29 µg/L), but even lower levels were seen in Dene/Métis and non-indigenous groups (20 and 16 µg/L, respectively) (Walker *et al.* in prep).

Cadmium

In the Canadian Arctic, organ meats (liver and kidney) from terrestrial ungulates (hoofed mammals), particularly caribou and muskoxen, have been identified as the principal dietary cadmium sources. Caribou kidney samples usually contain the highest concentration of cadmium but only slightly higher than Ontario and Quebec moose and white-tailed deer kidney samples. Cadmium content of muscle or organs other than kidney or liver has been determined to be up to 100 times lower (Gamberg and Scheuhammer 1994).

Few, if any, comparative studies have been conducted in the Arctic dealing with the relationship between cadmium levels in maternal vs. fetal blood. Although metallothionein induction has been detected in placental tissue, cadmium analysis in paired maternal and cord blood samples collected from three cities in Taiwan suggests that the placenta acts as a partial barrier. In 159 samples collected in 1988, the mean cadmium level in cord blood was approximately 60% that of maternal blood (0.78 vs. 1.30 µg/L, respectively) (Soong *et al.* 1991). Similar studies looking at the relationship between cadmium levels in maternal vs. fetal blood are underway in the NWT (Walker *et al.* in prep).

The influence of dietary and environmental sources of cadmium exposure appear to be minor compared to the contribution from smoking (Benedetti *et al.* 1994). Sampling in Nunavik, northern Quebec in 1992 indicated that blood cadmium values for non-smoking Inuit and non-smoking urban Caucasians (Quebec City) are not appreciably different (Bennedetti *et al.* 1994). However, the mean blood cadmium level in Inuit smokers was approximately 20-fold higher than the blood cadmium level of a smaller subset of non-smokers (5.4 µg/L vs. 0.27 µg/L). A recent study of Inuit women reported mean cadmium levels of 1.8 µg/L, however, this group is likely made up of both non-smokers and smokers (Table 12·13). Lower levels of cadmium were seen in the Dene/Métis and non-indigenous mothers surveyed (0.7 µg/L and 0.6 µg/L, respectively) (Walker *et al.* in prep).

Radiocaesium

The radiocaesium isotopes ^{137}Cs and ^{134}Cs are considered to be the artificial radionuclides of greatest concern in the Arctic environment. The most significant route of exposure is from the ingestion of caribou meat followed by ingestion of freshwater fish. In 1987/88, the levels of ^{137}Cs in caribou herds of the Canadian Arctic varied from 50 to 700 Bq/kg (EHD 1991, Thomas *et al.* 1992). Fish from freshwater lakes in the Arctic contain up to 20 Bq/kg of ^{137}Cs (Lockhart *et al.* 1992). For comparison, an acceptable level for ^{137}Cs in meat is 1000 Bq/kg (Codex Alimentaries Commission 1989).

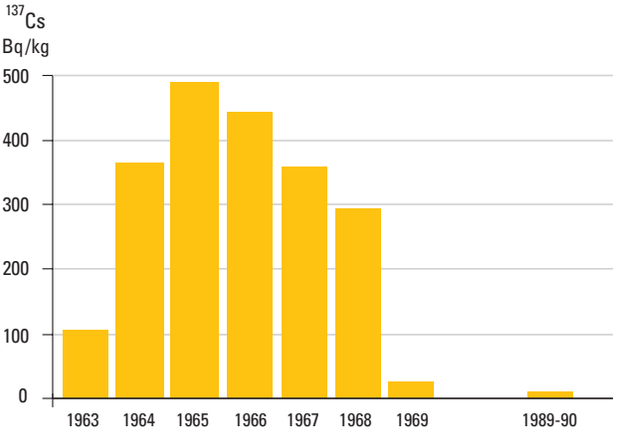


Figure 12·6. Body burdens of radiocaesium (yearly averages) across the Canadian Arctic.

Because of the radiation it emits, radiocaesium can be readily measured in humans by means of a portable whole-body counter. During 1989/90, such measurements were carried out on over 1100 people in five Arctic communities where caribou hunting is prevalent (Tracy 1993, Tracy *et al.* 1996). The highest concentration of ^{137}Cs in human tissue (mainly muscle) was 110 Bq/kg, corresponding to a radiation dose of 0.40 mSv/y. This can be compared with natural background radiation which is about 2 mSv/y. In the 1960s, some individual levels were as high as 2000 Bq/kg giving a dose of up to 5 mSv/y. Figure 12·6 shows the downward trend in radiocaesium body burdens from the mid-1960s to 1989/90. Each bar represents the average body concentration across all Canadian Arctic communities for a given year.

Radiostrontium

^{90}Sr is not considered a problem in the Canadian Arctic food chains; in fact, the chief route of exposure for Arctic residents to ^{90}Sr would be from food imported from agricultural areas in the south. The primary concern over this radionuclide is its effects on the developing bones of children. ^{90}Sr has been measured in autopsy samples of human vertebrae across Canada. From a maximum of 150 Bq/kg calcium in 1964, the levels across Canada declined to 50 Bq/kg calcium by 1978 (Meyerhof *et al.* 1979).

Radioiodine

Because of their short half-lives, ^{131}I and ^{132}I are considered to be a problem only in freshly contaminated foods (i.e., milk, vegetables). They are not concentrated in meat and fish which are more typical of Arctic food chains.

Table 12·13. Current and historical blood cadmium levels.

Location	Year(s) collected	n	Reported mean, µg/L	Range, µg/L	DL[a], µg/L	Reference	Comments
Greenland	1987	32 ♀♀	2.2	0.8-6.5		Milman *et al.* 1994	Whole blood
Greenland	1987	35 ♂♂	2.3	0.7-6.9		Milman *et al.* 1994	Whole blood
Nunavik (Inuit)	1988	7	0.27	0.31-0.45		Benedetti *et al.* 1994	Non-smokers, whole blood
Nunavik (Inuit)	1988	117	5.4	4.7-6.2		Benedetti *et al.* 1994	Smokers, whole blood
Northwest Territories (Inuit)	1994-95	67	1.8	0.07-7.3		Walker (in prep)	Maternal, whole blood
Northwest Territories (Dene/Métis)	1994-95	51	0.7	0.01-5.4		Walker (in prep)	Maternal, whole blood
Northwest Territories (non-indigenous)	1994-95	121	0.6	0.01-8.6		Walker (in prep)	Maternal, whole blood
USA, Michigan	1989	115 ♂♂	0.6	0.1-5.0	0.1	Hovinga *et al.* 1993	Fishermen, whole blood
USA, Michigan	1989	95 ♂♂	0.41	0.1-2.5	0.1	Hovinga *et al.* 1993	Controls, whole blood
Canada, Ontario	1992/93	65	3.94	1.0-14.6	0.2	Kearney *et al.* 1995	Smokers, whole blood
Canada, Ontario	1992/93	59	0.14	< 0.2-0.9	0.2	Kearney *et al.* 1995	Non-smokers, whole blood

a. DL: Detection limit.

Radiolead and radiopolonium

Of all radionuclides today, natural or artificial, radiolead (^{210}Pb) and radiopolonium (^{210}Po) make the greatest contribution to human radiation doses in the Canadian Arctic. Polonium-210 reaches levels of 200 to 500 Bq/kg in caribou livers and kidneys. Levels in muscle are much lower; about 10 to 30 Bq/kg. Lead-210 tends to concentrate in bone; values in soft tissue are about an order of magnitude lower than for polonium (Thomas *et al.* 1994, Tracy 1993). Polonium-210 may also be taken up by marine shellfish (Hunt and Allington 1993).

Blanchard and Moore (1970) have reported ^{210}Po concentrations of up to 10 Bq/kg in the livers of Alaska residents who eat caribou. Up to 5 Bq/kg of ^{210}Pb were found in the rib bones of these people. Some residents in northern communities may be receiving up to 10 mSv per year from ingested ^{210}Po, as compared to a normal background radiation dose of about 2 mSv per year (Tracy and Walsh 1995). It is likely that this has been the situation in the Canadian Arctic for several thousand years.

Radium

Radium (^{226}Ra) is present in trace amounts in all rocks and soils. Uranium mining and processing operations may lead to enhanced local concentrations of ^{226}Ra in the environment as has been observed at trans-shipment points along the Great Bear and Mackenzie Rivers.

Background levels of radium in the human body, including Arctic residents, are about 1 Bq per person (ICRP 1975).

Radon

There are relatively few measurements of radon (^{222}Rn) in Canadian Arctic dwellings, however, radon is less likely to be a problem in Arctic Canada than in southern Canada. Permafrost and snow cover tend to inhibit the emanation of radon from the soil. The northern practice of building homes on piles, rather than on a full concrete basement, also retards the build-up of radon in indoor air.

Conclusions

The social, cultural, spiritual and physical health of Canadian Arctic indigenous peoples depends on the collection and consumption of country (traditional) foods. A diet based on traditional foods is of high nutritional benefit. Marine mammals and fish are rich sources of n-3 fatty acids which are thought to protect against cardiovascular disease. The consumption of local fish, meat, wild greens and berries provides the necessary dietary intake of most vitamins, essential elements and minerals.

A decrease in the use of traditional food has been documented for some indigenous peoples in Canada for a variety of reasons, especially among younger generations. Compared to a traditional diet, a diet based on market foods has less protein and iron, more fat (especially saturated fat) and leads to a much higher carbohydrate intake. Dietary shifts are also associated with 'lifestyle' changes such as a more sedentary lifestyle because less time is spent hunting. A loss of traditional knowledge can also result in a shift in diet, as hunting and food preparation are integral parts of indigenous people's culture. In addition, dietary shifts may result in an increase in the incidence of certain diseases such as diabetes and cardiovascular disease.

The predominating scientific evidence, globally, suggests that the greatest health impacts of POPs are on the fetus (through transplacental exposure), and newborns (through breast feeding). The fetus is highly vulnerable to the effects of contaminants and will be exposed during this critical period of development because maternal POPs will cross the placenta barrier. Two- to four-fold higher concentrations of various POPs in Inuit cord blood demonstrate that some Canadian Arctic populations who consume large amounts of more heavily contaminated country foods have higher levels of POPs in their blood and these are being passed on to the developing fetus. The concentrations of POPs in Inuit breast milk are two- to ten-fold higher than in southern Canadian breast milk and have raised justifiable concern among mothers in the Arctic. The health benefits to newborns of breast feeding are substantial, e.g., mother–child bonding, immunological benefits transferred from mother to child, nutritional value and reduced risk of bacterial contamination from poorly prepared formulas. Studies that have evaluated the potential effects of the presence of PCBs in human milk on newborns are limited and inconclusive. Breast feeding should continue as the benefits of breast feeding far outweigh the currently known risks attributed to infant exposure to contaminants through breast milk.

Elevated levels of toxaphene, chlordane and mercury, coupled with current intake scenarios, suggest some Inuit groups are exposed to levels of these three contaminants significantly above the Tolerable Daily Intake (TDI). The Dene of the western NWT have smaller exceedances of the TDI for toxaphene and chlordane (CACAR 1997). There is insufficient information to conclude whether the TDI for dioxins and furans and dioxin-like PCBs is being exceeded in Canadian Arctic populations. Also, there is as yet little conclusive scientific information directly linking harmful human effects to these low levels of exposure. The risks associated with a shift in dietary preference need to be considered along with the risks associated with the presence of contaminants in Arctic wildlife consumed as traditional food. Weighing the uncertainty in some of the TDI values (e.g., toxaphene) against the benefits of traditional food gathering and consumption, it is generally recommended that consumption continue. However, consideration needs to be given to reducing intakes of some highly contaminated foods and increasing consumption of other equally nutritious traditional foods that have lower contaminant levels (Dewailly *et al.* 1996e).

Levels of lead in adults and children are below a level of concern, however, there is a need to continue monitoring because of its extreme effects on fetal and infant neurobehavioral development.

Cadmium levels in smokers are elevated and may have an impact on kidney function. A recent risk assessment for cadmium suggests that general population effects (tubular dysfunction in the kidneys) may occur at lower levels than those previously considered safe.

The level of human exposure to anthropogenic radionuclides in the Canadian Arctic has declined dramatically since the cessation of above-ground weapons testing and is now very similar to levels in the temperate zone. Due to contamination of the terrestrial food chain (i.e., particulates → lichen → caribou), some indigenous groups may be exposed to higher levels of some natural radionuclides.

Determining specific adverse human health effects in Arctic peoples due to the presence of contaminants in traditional foods and human milk is extremely difficult for methodological and ethical reasons. Results are also difficult to interpret because of a wide range of confounding factors (socio-economic, lifestyle and gender/age related). For example, Arctic residents are at significantly higher risk of exposure to cadmium through cigarette smoking than from dietary sources. Smoking is also a major risk factor for both coronary heart disease and lung cancer. These kinds of confounding factors cannot be ignored in an assessment of the

Table 12·14. Ongoing human health related research projects in Canada of relevance to the AMAP process.

Title	Principal investigator	Project status
Maternal and cord blood sampling in Baffin and Keewatin Regions of the NWT	J. Walker and Regional Health Boards	Ongoing
Advice on consultation, health risks, and health research on contaminants in the North	A. Gilman / D. Riedel / J. Van Oostdam	Ongoing
Yukon First Nations. Assessment of dietary risk: benefit	Centre for Nutrition and the Environment of Indigenous Peoples (CINE)	New
Vitamin A and calcium in traditional and market foods, NWT	CINE	New
Cord blood study - Nunavik	E. Dewailly / P. Ayotte	Ongoing
Transplacental exposure to PCBs and infant development – Nunavik	G. Muckle	Pilot complete
Toxaphene- and chlordane-induced liver toxicity among Inuit	E. Dewailly / P. Ayotte	New
Variance in food use in communities	CINE	Publication in prep.

relative risk of exposure to contaminants through contaminated food, however, they do not diminish governmental responsibility to reduce environmental levels of contaminants in the Arctic. Several ongoing studies in the Canadian Arctic will help fill important data gaps relating to exposures and effects (Table 12·14).

Considering what is known about the presence of POPs and mercury in the Canadian Arctic, and the current exposures of some communities, it would be prudent to continue to aggressively seek international agreement to reduce or eliminate the manufacture and use of most of these substances.

12.4.2. Denmark/Greenland and The Faeroe Islands

Introduction

Greenland and the Faeroe Islands, with populations of approximately 55 000 and 45 000, respectively, are parts of the Kingdom of Denmark. Although part of Greenland and all of the Faeroe Islands are subarctic, they are included in the AMAP area. Living conditions and exposure are different in the two areas and are, therefore, dealt with separately (cf. chapter 5). Figure 12·7 shows the main AMAP study area in Greenland, the Disko Bay region on the west coast of Greenland. The Faeroe Islands were not included in AMAP human health monitoring activities carried out during 1994-96.

12.4.2.1. Greenland

Sources of exposure

General population

The general population of Greenland is predominantly exposed to environmental toxins through the traditional diet of marine mammals, i.e., seals, walrus, whales and polar bear, and to a lesser extent through fish consumption. Marine mammals are at the top of the marine food chain and, due to biomagnification, have the highest concentrations of contaminants of any marine trophic levels. Concentrations of methylmercury and a number of persistent organochlorine compounds are high in the flesh, blubber and organs of these popular food items. Contaminant concentrations vary according to species and age of the animals consumed, and most likely also according to their natural habitat. Human exposure depends largely upon dietary habits.

Local population groups

Although point sources are uncommon in Greenland, two cases warrant mentioning. The first is 'The Black Angel' lead and zinc mine situated near the small village of Ukussissat in northern West Greenland. The mine was closed in 1990 after 20 years of metal ore production which significantly contaminated the local environment with zinc, lead and cadmium. The contamination has been monitored and is now decreasing, but there remain unsubstantiated claims from the local residents of fish and seals with sores or exhibiting strange behavior. There have also been claims of increased morbidity in the local population.

The second involves radionuclide contamination following the crash of a US Air Force B-52 bomber carrying nuclear weapons. The bomber, carrying four bombs, crashed on the sea ice near Thule Air Base in 1968 and exploded, scattering plutonium, americium and tritium over the sea ice and into the ocean (see chapter 8, section 8.3.1.2.3). A large clean-up operation followed, and the Danish workers employed in the clean-up have claimed that they have suffered from a number of symptoms and diseases due to exposure to radioactivity during this work. These include claims that mortality, in particular due to cancer, is increased among the former Thule workers, and that their fertility is reduced. Several well conducted epidemiological investigations have failed to substantiate these claims (Juel 1992). However, based on the persisting doubt about health effects on the exposed workers, a Nordic expert group was established in 1995 to assess all available information concerning the Thule accident. The main conclusion of the report of this group was that there is no evidence of negative health effects, but that, based on the long latency period of certain types of cancer, the registry investigations of cancer should continue (Lund 1996).

Recent mining activities, such as the lead and zinc mine at Mestersvig in East Greenland, and the small-scale uranium

Figure 12·7. Human health study locations in Greenland and the Faeroe Islands; Disko Bay was the main area studied under the AMAP monitoring program (1994-96).

mine near the small town of Narsaq in south Greenland, are not known to have caused any detectable human exposure.

Waste disposal and incineration in many towns and villages in Greenland does not appear to be well controlled. Although it has not been documented, it is possible that some town or village dumps cause significant contamination of the local environment.

Dietary habits

Dietary habits pertaining to subsistence foods have been investigated throughout Greenland in a recent survey (Bjerregaard *et al.* 1995). In a food frequency questionnaire, people were asked to rate their consumption of various types of subsistence food. Inuit men consumed subsistence food 33 times per month on average, compared to 27 times per month for women. The frequency of consumption increased with age, from 22 times per month in the 18-24 year old age class to 42 times per month in the 60 years and older age class.

As expected, the consumption of subsistence food showed a pronounced regional variation, with the lowest consumption in towns and the highest consumption in villages. Geographically, the lowest frequency of consumption of subsistence food was reported in the capital (Nuuk) and in towns on the southwestern and southeastern coasts (23 times per month). In towns on the northwestern coast, and in villages on the southwestern and southeastern coasts, frequency of consumption was intermediate (31-36 times per month). The highest consumption was reported in the villages on the northwestern coast and in the northernmost district (Thule) (46-50 times per month).

Seal meat is the most commonly eaten type of subsistence food, followed by fish and whale meat, although there are distinct regional patterns. In the northern and eastern villages, consumption of seal meat is markedly higher than consumption of any other food item. In the towns on the south and east coast, fish consumption predominates.

In conclusion, marine food items, in particular meat and organs of marine mammals, are consumed in the Inuit population of Greenland to an extent that is presumed to result in a relatively high exposure to environmental contaminants.

Levels and trends of contaminants in humans

Persistent organic pollutants

While there are few studies of the levels of persistent organic pollutants (POPs) in Greenlanders, results from human studies in the eastern Canadian Arctic and studies on the Greenlandic fauna indicate that concentrations are likely high.

A few studies from the late 1970s on fat taken from biopsies from Greenlanders showed higher DDT and DDE levels in Greenlanders than in the population of Denmark, but lower DDE levels than in the United States, eastern Europe and India (Clausen and Berg 1975, Jensen and Clausen 1979). Concentrations of lindane, aldrin-like residue, dieldrin, heptachlor-like residue, heptachlor epoxide, and PCBs were similar in Greenlanders and Danes. Unfortunately, it was not specified where the specimens were taken from in Greenland.

A recent study, in Nuuk and Ilulissat, of POPs in fat taken at autopsy, showed very high levels in Greenland compared with Canada, Finland and the United States. The mean concentrations in Greenland are shown in Table 12·15.

Under the AMAP human health monitoring program in Greenland, at the time of writing, 40 maternal blood samples and 29 cord blood samples had been analyzed for POPs, including 24 sample pairs. Details, also including results of more recent analyses, can be found in Annex Table 12·A12. It should be noted that concentrations on a lipid basis are similar in mothers and babies. Concentrations for Greenland samples are very high compared with samples from southern Canada, but are low compared with another population survey from Greenland (Dewailly *et al.* 1996d).

Table 12·15. Levels of organochlorines in the omental (intra abdominal) fat from autopsies of Inuit from Greenland (N = 42, sampled in 1993) (Dewailly *et al.* 1995, Dewailly pers. comm.).

Contaminant	Arithmetic mean, µg/kg lipid	95% confidence interval
β-HCH	108.9	91.0-126.7
α-chlordane	1.09	0.68-1.51
γ-chlordane	10.5	8.2-12.9
Cis-nonachlor	311.6	260.0-363.1
p,p'-DDE	3843.8	3197.2-4490.4
p,p'-DDT	143.3	116.8-169.8
Hexachlorobenzene	676.1	554.7-797.5
Mirex	152.6	123.5-181.7
Oxychlordane	862.4	679.9-1044.8
Trans-nonachlor	1463.1	1205.5-1720.7
PCBs[a]	15700	13500-17900

a. PCBs measured as Aroclor 1260.

Mercury

The concentration and distribution of mercury in humans in Greenland has been thoroughly studied over the last 15 years. Surveys have been performed in adults, pregnant women and newborn babies in most parts of Greenland, including the central west coast, with the highest population concentration, and the hunting districts in northwestern, northern and eastern Greenland. In all four regions studied, the determining factor for mercury exposure was the daily intake of meat from marine mammals. At a regional level, the median blood mercury concentrations were directly proportional to the registered mean number of seals caught (and consumed) indicating that mercury concentration in meat is probably similar in all regions of Greenland (Hansen 1990).

In adults, median as well as maximum whole blood concentrations of mercury are lowest in the southwest, higher in the northwest and on the east coast, and very high in North Greenland (Figure 12·8). In North Greenland, 16% of the adult population studied had blood mercury concentrations exceeding 200 µg/L. WHO regards this level as the minimum toxic blood concentration in adults. More than 80% of the population in North Greenland exceeded 50 µg/L

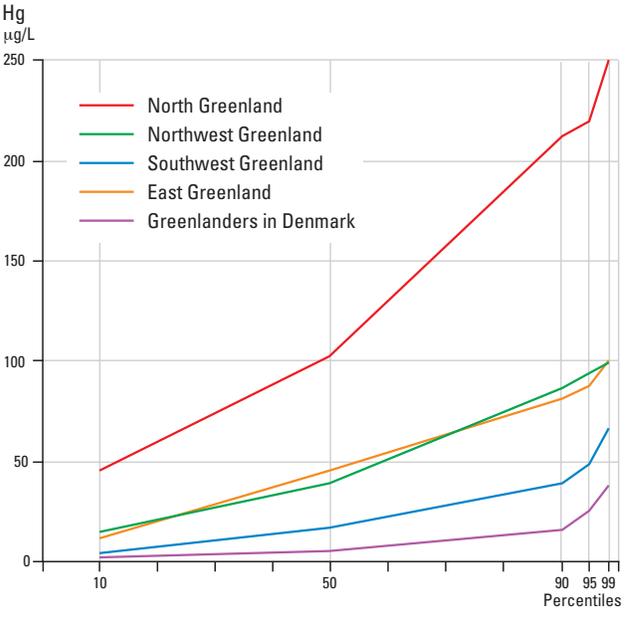


Figure 12·8. Distribution (in percentiles) of whole blood mercury concentrations in four regions in Greenland and in Greenlanders living in Denmark (Hansen 1988).

(Hansen and Pedersen 1986). This concentration is the maximal acceptable blood mercury concentration for pregnant women (WHO 1990b).

In studies of fetal exposure, blood samples were collected from pregnant women prior to delivery and from the umbilical cord at birth. Mercury concentrations in maternal and cord blood were linearly correlated, but concentrations were somewhat higher in cord blood. Samples were collected from 1982 to 1990. No temporal trend was observed. The regional variation was similar to that of the adults. A preliminary analysis of the data showed a negative correlation between blood mercury concentration and birth weight (Foldspang and Hansen 1990), but a reanalysis of data from the whole study showed only a minor and non-significant negative association. Smoking habits and population group were major determinants of birth weight and gestational length.

Under the AMAP human health monitoring program in Greenland, 20 paired maternal and cord blood samples were analyzed for mercury. For the paired samples, the mean concentrations (arithmetic mean) in mothers and newborns were 24.2 µg/L and 53.8 µg/L, respectively, with medians of 16.9 and 56.7 µg/L. Data for all samples are given in Annex Table 12·A13. These concentrations were very high compared with samples from Denmark. Compared with earlier results from Greenland, the mercury concentrations in maternal blood were lower than previously found in southwest Greenland, probably a result of decreased consumption of marine food by pregnant women.

Lead

Lead levels were determined in individuals living in four representative areas of Greenland and were compared with levels in Greenlanders living in Denmark (Figure 12·9). The differences among the four areas were relatively small and no difference was found between levels in Greenlanders and Danes living in Greenland. The blood lead levels in Greenlanders were also comparable to those of inhabitants of industrialized areas in western Europe. Umbilical cord blood lead levels were slightly lower than those of the mothers (Hansen and Pedersen 1986, Hansen 1988).

Lead concentration in blood was not related to dietary habits, i.e., consumption of marine mammals, or to smoking habits, except in pregnant women for which a moderate relationship to smoking was demonstrated. In East and North Greenland, blood lead levels increased with age. This effect was most pronounced in women.

Blood lead concentrations in Greenlanders are decreasing with time, following the same downward trends noted in Europe and North America where declines are directly related to the introduction of lead-free gasoline. Furthermore, it has been demonstrated by analyses of lead in samples of inland ice that concentrations in Greenland reflect the consumption of leaded gasoline in both Europe and North America. Therefore, it seems well substantiated that the surprisingly high lead exposure in Greenland is caused by long-range atmospheric transport of lead from urbanized centers at lower latitudes.

Under the AMAP human health monitoring program in Greenland, 20 paired maternal and cord blood samples have been analyzed for lead. For the paired samples, the mean concentrations (arithmetic mean) were 45.6 µg/L in mothers and 34.9 µg/L in the newborn, with medians of 34.2 and 31.9 µg/L, respectively. Data for all samples are given in Annex Table 12·A13. These concentrations are lower than those previously found and are in agreement with the decreasing trends observed in other parts of the world.

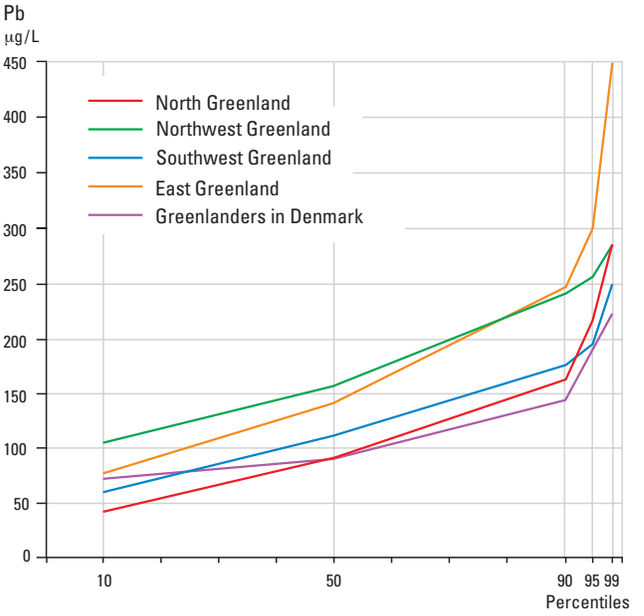


Figure 12·9. Distribution (in percentiles) of whole blood lead concentrations in four regions in Greenland and in Greenlanders living in Denmark (Hansen 1988).

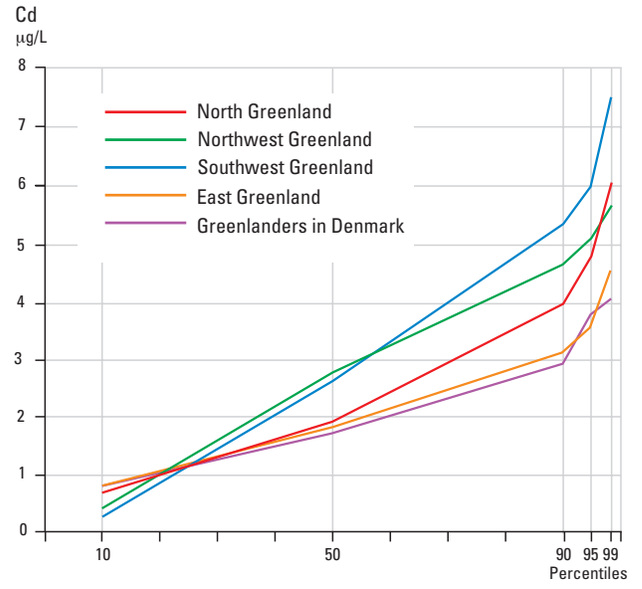


Figure 12·10. Distribution (in percentiles) of whole blood cadmium concentrations in four regions in Greenland and in Greenlanders living in Denmark (Hansen 1988).

Cadmium

Cadmium has been studied together with mercury and lead in the same four regions of Greenland, however, cadmium was only measured in the adult population. Figure 12·10 shows that the median blood cadmium concentration in East and North Greenlanders was the same as levels in Greenlanders living in Denmark, and lower than levels in people living in West Greenland.

The major determinant of blood cadmium concentration is smoking, such that a dietary effect can only be evaluated in non-smokers. The median cadmium concentration in non-smokers was higher in East and North Greenland, which is in accordance with the higher intake of marine mammals in these regions. In smokers, however, as in the general population, concentrations were higher in West Greenland. This cannot be explained as a result of age or sex differences, as neither age nor sex were associated with blood cadmium. Neither can it be explained as being due to different proportions of smokers in the sample groups. While quantitatively

different smoking habits may be an explanation, a population survey has not demonstrated differences in numbers of cigarettes smoked in the four regions (Hansen and Pedersen 1986, Hansen 1988, Bjerregaard *et al.* 1995).

Under the AMAP human health monitoring program in Greenland, 42 maternal blood samples were analyzed for cadmium. The mean concentration (arithmetic mean) was 1.29 µg/L and the median was 0.9 µg/L. For details see Annex Table 12·A13. Concentrations were considerably lower than previously observed.

Selenium

Selenium has also been studied, but only in adults in East and North Greenland. Selenium levels were high in Greenlanders, most notably in North Greenlanders (Figure 12·11) (Hansen 1988). Similar to the findings for mercury, selenium concentrations were closely related to the amount of marine mammals eaten. The very large difference in selenium concentration between East and North Greenland is not readily explained,

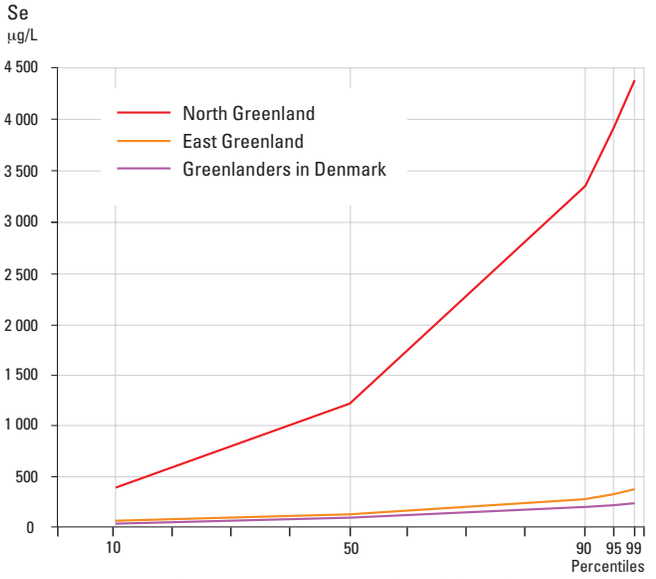


Figure 12·11. Distribution (in percentiles) of whole blood selenium concentrations in East and North Greenland and in Greenlanders living in Denmark (Hansen 1988).

but may be caused by differences in selenium contents of various species of marine mammals. Selenium levels were also high in umbilical cord blood, but somewhat lower than in maternal blood, indicating that selenium does not pass the placental barrier as readily as mercury (Hansen and Pedersen 1986). In North Greenland, but not in East Greenland, there was a positive association between mercury and selenium concentrations in individuals. The absence of this correlation in East Greenland may indicate that the dietary sources of mercury and selenium are different between the regions (Hansen 1988). Consumption of whale skin (*muktuk*), which is very high in selenium, is very common in North Greenland but not as common in East Greenland.

Under the AMAP human health monitoring program in Greenland, 26 paired maternal and cord blood plasma samples were analyzed for selenium. For the paired samples, the mean concentrations (arithmetic mean) were 67.0 µg/L in mothers and 39.1 µg/L in the newborn, with median concentrations of 67.8 and 37.8 µg/L. This is lower than previously observed and is another indication of decreased consumption of marine foods.

Other metals

Under the AMAP human health monitoring program in Greenland, 26 paired maternal and cord blood plasma samples were also analyzed for zinc and copper. For the paired samples, mean zinc concentrations (arithmetic mean) were 545 µg/L in mothers and 870 µg/L in the newborn (medians 549 and 797 µg/L, respectively). The mean concentrations of copper (arithmetic mean) were 2237 µg/L in mothers and 403 µg/L in the newborn (medians 2236 and 370 µg/L, respectively). Data for all samples are given in Annex Table 12·A13.

Other epidemiological factors

The Greenlandic mortality pattern is characterized by a high incidence of injuries, i.e., accidents and suicides, a high mortality from primarily natural causes, and a relatively low mortality from ischemic heart disease (Bjerregaard 1991). The disease pattern and causes of death are recorded with a high degree of uncertainty which, together with the small and scattered population, makes epidemiological inference difficult. To date, no diseases or symptoms have been registered which can be unequivocally related to environmental contaminant exposure. However, the measurement of more subtle neurological and reproductive outcomes have not yet taken place.

The traditional Greenlandic diet is central to the issue of environmental exposure to toxins. Counter to the presumed negative effects of environmental toxins, the traditional diet provides compounds with known or supposed benefits to health. Selenium is present in high concentrations in the traditional diet. It is an antioxidant and acts as an antagonist to methylmercury. N-3 fatty acids of marine origin have been the subject of numerous studies and are thought to be at least part of the explanation for the low occurrence of atherosclerosis and ischemic heart disease in Greenlanders. Furthermore, monounsaturated fatty acids from seal and whale blubber may have the same beneficial effects on the pathology of atherosclerosis and hypertension as have been demonstrated for oleic acid, another monounsaturated fatty acid.

The traditional Greenlandic diet is also a very important part of the Greenlandic culture. Eating Greenlandic food is an essential part of the Greenlanders' identity. This is true both in the hunting districts, where even young people prefer to regularly eat seal meat, and in the capital where a traditional festive meal, large or small, always includes Greenlandic specialties such as *muktuk*, dried whale meat, dried fish, blubber, etc.

12.4.2.2. Faeroe Islands

Levels and trends of contaminants in humans

Umbilical cord blood samples from 1023 consecutive births in the Faeroe Islands showed a median blood mercury concentration of 24.2 µg/L; 250 of the samples (25.1%) had blood mercury concentrations that exceeded 40 µg/L (Grandjean and Weihe 1992). Median mercury concentrations in maternal hair were 4.5 µg/g; 130 samples (12.7%) contained concentrations that exceeded 10 µg/g. Frequent consumption of whale meat during pregnancy and, to a much lesser degree, frequent consumption of fish, were associated with high mercury concentrations in cord blood and hair. Increased parity or age were also associated with high mercury concentrations in cord blood and hair. Blood mercury levels were slightly lower if the mother had occasionally consumed alcoholic beverages. Mercury in blood correlated moderately with blood selenium (median 110 µg/L). Increased selenium concentrations were associated with intake of whale meat, alcohol abstention, delivery after term, and high parity. Lead in cord blood was low (median 17 µg/L), particularly if the mothers abstained from smoking.

A questionnaire completed by 331 Faeroese adults revealed a daily consumption of 72 g fish, 12 g whale muscle and 7 g of blubber. Fish and pilot whale constituted 44%

and 9.5% of Faeroese dinner meals, respectively (Vestergaard and Zachariassen 1987). Most of the fish consumed in the Faeroe Islands is cod with an average mercury concentration of about 0.07 μg/g (Hygiene Institute, Tórshavn, pers. comm.). Almost all of the mercury in fish is methylated. Muscle tissue of Faeroese pilot whales contained an average mercury concentration of 3.3 μg/g, about half of which was methylmercury. Higher concentrations occurred in the liver, mainly in the form of inorganic mercury (Juhlshamn *et al.* 1987).

Based on the data from the questionnaires, and the total mercury concentration in whale and fish, an average daily mercury intake from these foods for individuals over 14 years of age can be calculated as being about 36 μg. If the steady-state blood level (in μg/L) is numerically equal to the average daily intake of a 70 kg person (expressed in μg methylmercury), the expected blood level would be about 36 μg/L. However, as only two-thirds of the total mercury in food is methylmercury, the expected average blood levels would be about 25 μg/L. This level is in agreement with the actual findings in the Faeroe Islands.

In a recent study of organochlorine concentrations in pilot whales, it was found that the overall average PCB concentration in blubber is very high, i.e., about 30 μg/g lipid, with total DDT being about 20 μg/g lipid (Borrell and Aguilar 1993). Expressed on a lipid weight basis, the organochlorine concentrations are about the same in muscle tissue. However, the fresh weight concentration of PCB averages only about 0.6 μg/g and total DDT is about half as much (Borrell and Aguilar 1993). Based on these findings, the average daily intake of PCB of the Faeroe Islands is estimated at above 200 μg, with considerable inter-individual variation. For comparison, the average daily PCB intake in Scandinavia is about 15-20 μg. Thus, the difference in average PCB exposure between these two population groups corresponds to one order of magnitude.

Samples of human milk were collected on the third and fourth day after delivery from women who had given birth in the hospital in Tórshavn (Table 12·16). Samples were collected over a one month period. Four pooled samples were made from 22 samples such that each mother contributed the same amount of fat to the pool. The four groups were separated as follows: Group I: low mercury concentration in the milk (<1 μg/L); Group II: high mercury concentration in the milk (>4 μg/L); Group III: no more than one fish dinner per week during pregnancy; and Group IV: at least five fish dinners per week during pregnancy. The frequency of pilot whale dinners was very low in Group I, very high in Group II, and at the same intermediate level in Groups III and IV. However, the extent of blubber consumption was not quantified.

Table 12·16. Total PCB concentrations in four pooled human milk samples from Tórshavn, Faeroe Islands.

Pool [a]	Whole weight, μg/L	Lipid adjusted, μg/g	% lipid
I	47.1	2.2	2.22
II	80.3	3.5	2.31
III	46.6	1.8	2.54
IV	58.3	2.8	2.11

a. For explanation, see text

Umbilical cord tissue from all children born on the Faeroe Islands in 1986/87 was preserved. Preliminary data on 24 cord samples suggest an overall average PCB concentration of about 1.4 μg/g lipid. This concentration is based on the sum of the concentrations of PCB congeners 153, 138 and 180, multiplied by 1.7 (Grandjean and Weihe 1992).

12.4.2.3. **Conclusions**

In the absence of local point sources of pollution, the major routes of exposure to environmental contaminants (methylmercury, persistent organic pollutants) in Greenland are consumption of meat and organs from marine mammals. Smoking is the dominant source of cadmium exposure. The population of Greenland is exposed to lead at approximately the same level as the populations of Western Europe and North America although the pathway of exposure is not known with certainty.

Exposure to contaminants of concern through the diet and through smoking is high. Exposure to methylmercury and POPs is at a level where negative consequences for health may be expected. While there are no examples of overt toxic effects from environmental pollutants in the Greenlandic population, the subtle effects that may occur are very difficult to detect, can be easily overlooked, or can be masked by other factors.

More detailed dietary information is needed concerning the species, organs, and amounts eaten by Greenlanders. In addition, information about the relationship between diet and body burden of pollutants is needed. Ongoing studies in the Disko Bay area will supply some of this information. It is also important to follow the temporal trends in exposure to environmental pollutants. A survey program of pregnant women and their newborn babies was initiated in 1994 and continues as part of the AMAP human health monitoring program. Table 12·17 outlines ongoing studies in Greenland and the Faeroe Islands that are relevant to the AMAP process.

At present, most health professionals with knowledge of Greenland agree that the possible negative effects of pollutants in the diet are far outweighed by the positive health-related and social benefits of the traditional diet. With information from dietary surveys and AMAP human health surveillance, and information on species- and organ-specific concentrations of contaminants in consumed animals, it will be possible to establish more carefully constructed dietary guidelines for vulnerable population subgroups, e.g., pregnant women and children. It is also agreed that action should be taken to reduce the high prevalence of smoking.

Table 12·17. Ongoing human health related research projects in Greenland and the Faeroe Islands of relevance to the AMAP process.

Title	Principal investigator	Project status
Birth weight and mercury exposure	J.C. Hansen, Centre for Arctic Environmental Medicine (CAM)	In progress
Exposure of pregnant women in the Disko Bay area to environmental contaminants	P. Bjerregaard, Danish Institute for Clinical Epidemiology (DICE)	In progress
Environmental risk factors in West Greenland	P. Bjerregaard, DICE	In progress
Dietary survey in Disko Bay area	T. Pars, DICE	In progress
Health interview survey in Greenland	P. Bjerregaard, DICE	In progress
Neurotoxic effects of prenatal exposure to mercury in the Faeroe Islands	P. Grandjean, P. Weihe	In progress
Neurotoxic effects of prenatal exposure to PCBs in the Faeroe Islands	P. Grandjean, P. Weihe	In progress
Neurotoxic effects of prenatal mercury exposure in North Greenland	P. Weihe	In progress

12.4.3. Iceland

Introduction

The total population of Iceland is 266 783. The Icelandic population is culturally and socially homogeneous with immigrants accounting for only 2% of the population (cf. chapter 5).

Sources of exposure

Since heavy industry and agriculture are limited in Iceland, the main source of exposure to pollutants is likely dietary. Although Icelanders are typically western in most aspects, their diet has many subarctic characteristics. Icelanders consume more fish than other nations in Europe and the diet is unusually rich in protein and fat. Whale meat was a seasonal component of most people's diet until 1986, and seal and seabirds are harvested in many rural areas.

Levels and trends of contaminants in humans

There is very little data on levels of contaminants in people. Blood lead levels were measured in 37 individuals living in Reykjavik in 1992 (Thordardottir and Jóhannesson 1993). The levels were about 50 μg/L (range 25-88 μg/L) and were three- to five-fold lower than had been found in a similar study in 1978 (Pormar and Jóhannesson 1979). Between 1986 and 1990, lead levels in ambient air in Reykjavik decreased five-fold (Gísladóttir 1992). Cadmium was analyzed in the renal cortex of 30 accident victims and was below 40 μg/g in all samples (Sólbergsdóttir and Jóhannesson 1992). Mercury, arsenic, cadmium, selenium and zinc were analyzed in human hair from 58 individuals in 1981 (Jóhannesson *et al.* 1981). The levels were all found to be low or normal. Volcanic and geothermal activity did not appear to increase the body burden of mercury in the Icelandic population.

The levels of PCBs and organochlorine pesticides in 22 samples of breast milk collected in Reykjavik in 1993 were similar or higher than reported elsewhere (Jensen 1990). The mean levels in milk fat were: 47 ng/g HCB, 360 ng/g ∑DDT, 830 ng/g ∑PCB, and 206 ng/g CB 153 (Olafsdottir pers. comm.). The levels of PCBs and organochlorine pesticides in adipose tissue and brain from 15 postmortem samples were determined in 1994. The levels were again found to be similar or higher than reported elsewhere (Luotamo *et al.* 1991, Willams and LeBel 1990). The mean levels in adipose tissue were: 100 ng/g lipid HCB, 1100 ng/g lipid ∑DDT and 550 ng/g lipid CB 153. A study of metals and organochlorines in maternal blood is ongoing in collaboration with the other AMAP nations. Organochlorine levels in a pooled serum sample from 22 males are shown in Table 12·18.

Table 12·18. Organochlorines in human serum in Iceland. A pooled sample from 22 males, age 31-61, mean = 45.3±7.4 years.

Contaminant	μg/L
HCB	0.623±0.019
DDE	2.34 ±0.13
CB 74	0.120±0.005
CB 99	0.156±0.007
CB 101	0.125±0.008
CB 105	0.065±0.019
CB 118	0.162±0.017
CB 138	0.592±0.026
CB 153	1.22 ±0.05
CB 170	0.320±0.027
CB 180	0.744±0.045
CB 183	0.115±0.009
CB 187	0.261±0.016
CB 194	0.136±0.025

Figure 12·12. Human health study locations in Arctic Norway, Russia and Finland; red dots indicate the main communities studied under the AMAP monitoring program.

12.4.4. Norway, Russia and Finland

The national reports of Norway and Russia, and to a certain extent Finland, are partly integrated because of the proximity of parts of their Arctic regions where related human health studies have been carried out under AMAP (Figure 12·12). The most severe ecological problems in the area are related to the industrial pollution on the Kola Peninsula of Russia, close to the Norwegian-Russian-Finnish border, and in the Norilsk region of Siberia.

12.4.4.1. Norway

Introduction

The Arctic part of mainland Norway, defined as north of the Arctic Circle, has a subarctic climate because of the Gulf Stream influence. The three northernmost counties in Norway are Nordland, with approximately 240 000 inhabitants, of which nearly 160 000 live north of the Arctic Circle; Troms, with approximately 145 000 inhabitants; and Finnmark with approximately 73 000 inhabitants (figures from Statistisk Sentralbyrå, Oslo, 1994). They are treated as one region, referred to as Northern Norway, in matters concerning social affairs, economic questions and health care. The climate is, however, not significantly different from much of the rest of the west coast of Norway. There is only one significant source of industrial pollution within mainland northern Norway, namely the iron-producing Syd-Varanger Company in Kirkenes. The facilities at this point source were closed down permanently in April 1996.

Sources of exposure

The general population of northern Norway has three main exposure sources:

1. Dietary intake by coastal populations of global contaminants, primarily persistent organic pollutants and mercury in marine foods. The main source is fish, and to a lesser extent marine mammals, making an important difference in the dietary patterns to the comparable groups in Greenland and Canada.
2. The regional air pollutants, mainly SO_2 and metals such as iron, copper and nickel, derived from the extensive industrial emissions on the Kola Peninsula close to the Norwegian border.
3. The radiation contamination following the Chernobyl accident which affected the south of Nordland, and, more

historically, the nuclear testing program on Novaya Zemlya affecting the inland areas of Finnmark. This problem is discussed in more detail in chapter 8.

Water

The quality of the water supply to Norwegian communities is routinely monitored by the local health authorities. There are standard reference limits for metals and infectious agents, and the water quality, with very few exceptions due to seasonal variations, is of no medical concern.

Air

The air pollution in northern Norway is of special concern in the Norwegian-Russian border area. Monitoring of air pollution on the Norwegian side started in Sør-Varanger in 1974, and on the Russian side in 1985. The Joint Norwegian-Russian Commission on Environmental Cooperation was formed in 1988, with a special Norwegian-Russian Health Group established in 1991. In 1989, the total SO_2-contamination from the Nikel and Zapolyarnyy nickel plants was 272 000 tonnes, three times the total SO_2-pollution originating from Norway in the same year. A total of 1100 tonnes of metals were also emitted, including 510 tonnes nickel, 310 tonnes copper and 18 tonnes cobalt. Air measurements at Norwegian border stations have exceeded 3000 $\mu g/m^3$ SO_2. On the Russian side, concentrations exceeding 500 $\mu g/m^3$ are instantly followed by a decrease in production to reduce emissions. Different aspects of human health are now investigated in this area. The production of nickel has decreased in the last years, due to the economic changes in Russia. There is now political agreement for the renovation of the nickel plant in Nikel.

Occupational

The only major industrial pollution source on mainland northern Norway was the Syd-Varanger Company which was concerned with iron production (Sivertsen and Scholdager 1991). Recently, all operations at this plant have been terminated. The emissions of SO_2 from this plant were minor compared to those from the neighboring Russian nickel industry. However, a substantial release of dioxins was recognized during the last years of operation. Investigations are underway to assess any associated human health risks.

12.4.4.2. **Russia**

Introduction

The Russian Arctic territories cover by far the largest segment of the circumpolar region, extending from the Kola Peninsula in the west to Chukotka in the east. The population of the Russian Arctic, as defined by Russia and according to the 1989 census, is approximately 2 million, including 66 000 indigenous minorities and over 22 000 indigenous Yakuts. Additional areas of the Russian North, with conditions similar to the Arctic, include another 1.7 million persons, of whom 200 000 are members of indigenous minorities and other indigenous groups (Klopov pers. comm.) (see also chapter 5). The region includes two particularly heavily polluted industrial areas, namely the part of the Kola Peninsula adjacent to the Norwegian-Finnish border, and the Norilsk region in Siberia. In both regions, nickel production industries constitute a major source of pollution. The unique feature of the Russian Arctic, compared to Norway and Finland, is that the Arctic climate reaches to areas far south of the Arctic Circle in most of Siberia and the Russian far east. The AMAP human health monitoring program study areas within the three countries are shown in Figure 12·12.

Sources of exposure

The Russian Arctic is the most industrialized of the Arctic regions. In the former Soviet Union (FSU), it was official policy to industrialize the region, and this resulted in an extensive influx of people from other parts of the FSU. Political and social dominance by the new residents occurred throughout the territory. The migrants were, and continue to be, mostly concentrated in urban centers and employed in industrial activities, such as mining of coal and metal ores, and oil and gas production and refining (Table 12·19). The indigenous populations largely continue their traditional activities, such as reindeer herding, fishing and hunting. As a consequence, the immigrant populations are primarily exposed to contaminants through their work, while indigenous people are mainly exposed through the food chain.

Table 12·19. Industrial cities of the Russian Arctic (reproduced from Revitch 1995).

Region/city	Population	Main industrial activity
Murmansk province		
Murmansk	473000	Harbor, ship repair
Severodvinsk	66000	Ship building
Kandalaksha	54000	Aluminum industry
Apatity	89000	Extraction of apatite
Kirovsk	43000	Extraction of apatite
Monchegorsk	68000	Nickel industry
Olenegorsk	47000	Extraction of iron
Kovdor	31000	Extraction of iron
Zapolyarnyy	23000	Nickel industry
Nikel	22000	Nickel industry
Komi Republic		
Vorkuta	117000	Extraction of coal
Inta	61000	Extraction of coal
Yamalo Nenetsky Autonomous Okrug		
New Urengoy	105000	Gas extraction
Nadim	52000	Gas extraction
Taimyr Autonomous Okrug		
Norilsk	169000	Nickel, copper, cobalt and other non-ferrous industry

Water

The quality of drinking water sources to towns of the Russian Arctic are, with some exceptions, generally good, because urban populations are supplied from centralized water facilities (Klopov 1995 pers. comm.). However, because the supply pipelines are often very old, and water is transported over long distances, the water often remains in the pipes for long periods prior to its use as tap water, resulting in contamination with metals, especially iron. In rural areas, the water often comes from natural freshwater reservoirs, such as rivers, lakes and melting of snow in the winter. This increases the risk of environmental contamination, especially by infectious agents. The consequence is a high occurrence of gastrointestinal diseases, especially in children, in these areas. Upper limits for some chemical substances in fresh tap water are given in Table 12·20.

Table 12·20. Russian upper limits for selected elements in tap water, mg/L.

Fluoride	1.5
Manganese	0.1
Arsenic	0.05
Mercury	0.005
Selenium	0.001
Cadmium	0.01
Nickel	0.1
Copper	0.1
Lead	0.03
Zinc	1.0

Air

During the period since the communist revolution in 1917, the Russian Arctic has been extensively industrialized. Today there are 34 cities of industrial importance in the territory (Revitch 1995) and their emissions are significant point sources of pollution. Norilsk is by far the biggest source of pollution, with a total release of over 2 million tonnes/y of different pollutants, although primarily SO_2. This is the cause of the highest level of air pollution ever registered, with SO_2 levels exceeding up to 22 times the maximal permissible level during some periods.

Occupational

Nasal and lung cancer are recognized as chronic occupational diseases in the nickel industry (Norseth 1994). Potential reproductive and developmental health effects for women of fertile age who work in nickel plants and their offspring are currently being assessed. As in other industrial countries, industry-specific occupational diseases occur in connection with the northern Russian industries identified in Table 12·19.

On the Kola Peninsula, Chashschin *et al.* (1994) have completed a cross-sectional study of 821 male and 758 female workers in a nickel hydrometallurgy refining plant. Even with serious limitations in the statistical and sampling details of the pregnancies and newborn babies, the results suggest some adverse reproductive and developmental effects. This constitutes a real concern that requires a more comprehensive and quantitative epidemiological investigation. Such work has now been initiated and involves extensive international cooperation.

12.4.4.3. Russia and Norway – Levels and trends of contaminants in humans

Methods

Under the AMAP human health monitoring program for Northern Norway, it was decided to solicit anamnestic information and collect biological samples from 50 continuous delivery patients in Hammerfest and Kirkenes, and from 15 delivery patients in the Tromsø Regional University Hospital who lived in Målselv. The town of Bergen in the southwest of Norway was used as a reference. In Russia, the same protocols were carried out in Nikel, Murmansk, Apatity, Monchegorsk, Arkhangelsk, Salekhard and Norilsk. The patients were asked to complete a written questionnaire addressing the following: age, parity, ethnic background, places of residence exceeding six months, schooling, occupation, smoking habits, alcohol, medication, serious diseases and dietary habits, especially concerning certain types of food. The following information was collected about the births: naegele term, date of birth, length of baby, weight of baby, weight of placenta, APGAR score, congenital malformations, gestational age, and individual comments by the doctor or midwife. The completion of the informed consent form and collection of anamnestic information was done before the delivery process started, to minimize stress. Standardized procedures that minimized inadvertent contamination were employed to collect blood and urine from mothers, cord blood, urine from the neonates, and placental tissue.

Persistent organic pollutants

A Norwegian survey of PCBs, PCDDs and PCDFs in human milk during 1985/86 included the town of Tromsø in the Norwegian AMAP area (Clench-Aas *et al.* 1988). Elverum, an inland community in the south, and the southern industrialized town of Porsgrunn, were also surveyed. No significant differences in the concentrations of PCBs were found between the three locations, nor in the levels of PCDDs and PCDFs expressed as TCDD equivalents (TEQs). However, an indication of regional differences was found for some PCDD and PCDF congeners, consistent with suspected sources of these compounds. All the values were close in magnitude to data reported for other western countries.

A more comprehensive study was carried out in the same areas in 1992/93 (Becher *et al.* 1995), also including locations in Lithuania. There was no significant change in the concentrations of PCBs between the earlier (1985/86) and more recent (1992/93) studies, with means for total PCB in human milk of 534 μg/kg lipid and 496 μg/kg lipid, respectively.

In an investigation of human milk from mothers living in Oslo (n = 28), Skaare *et al. (*1990) found a mean concentration of total PCB of 488 μg/kg lipid. Becher *et al.* (1995) reported total TEQs, including dioxin-like PCBs, between 31 and 42 pg TEQ/g lipid in Norway, compared to 45-49 pg TEQ/g lipid in Lithuania. Dioxin-like PCBs were found to contribute two to three times more to the total TEQs than the PCDDs and PCDFs. Major congeners among the dioxin-like PCBs were CBs 126, 156, 114, 118, and 170. Comparison with the 1985/86 study, indicates a decrease in the mean TEQ levels of about 37% over a 7-year time span for PCDDs/PCDFs, while the levels of total PCBs have remained unchanged or have only slightly decreased. This interesting temporal trend needs to be documented further in future studies.

In 1993, Polder *et al.* (1996) carried out a study on mothers milk in Murmansk and Monchegorsk on the Kola Peninsula. Total PCB in mothers milk from Murmansk was in the range of 250-635 μg/kg lipid, with a mean value of 484 μg/kg lipid (range 7.5-19 μg/L and mean 12.6 μg/L). The results from Monchegorsk were in the range of 262-854 μg/kg lipid, with a mean value of 535 μg/kg lipid (range 7.8-25.6 μg/L and mean 16.1 μg/L). Specific congener concentrations were marginally higher for CBs 74, 99, 118, 138 and 153, while other congeners, such as CB 180 and 187, were lowest. This pattern corresponds well to the known dominance of dietary intake as the major exposure pathway for the general population and the similarity of dietary habits within a country. The conclusion was that the exposures in Monchegorsk and Murmansk are relatively low. No significant geographical differences in levels of PCBs and PCDDs/PCDFs between Murmansk and Monchegorsk were found.

In central Russia, there has been an investigation of breast milk and placentas from mothers employed in a transformer plant in Serpuhov (Moscow County) (Pleskatchevskaya and Bovonikova 1992). The range of the total PCB levels found in milk was 14-105 μg/L (467-3500 μg/kg lipid).

In the Russian AMAP human health monitoring study, PCB levels were analyzed in plasma from women in Salekhard and Norilsk (Klopov pers. comm.). The arithmetic mean concentration of PCB in plasma of women 18-24 years old was 7.5 μg/L in Norilsk and 6.8 μg/L in Salekhard. A mean concentration of 9.9 μg/L has been reported in a comparable age group of an Inuit population in Canada (Dewailly *et al.* 1994a). By comparison, the mean plasma PCB concentration in a group of women 25-44 years old was 13.8 μg/L in Norilsk and 16.1 μg/L in Salekhard (19.5 μg/L in the Inuit population in Canada). The mean concentration of PCB in cord blood samples was 2.1 μg/L in Norilsk and 1.6 μg/L in Salekhard (2.8 μg/L in the Inuit population in Canada). The mean concentration of PCB in breast milk of the Norilsk women was 799 μg/kg lipid, while in Salekhard it was 847 μg/kg lipid. The major food source of PCB is freshwater fish, which constitutes a significant portion of the diet.

Very high levels of DDT have been reported during 1970-1980 in human blood samples from the southern regions of the former Soviet Union (Klopov pers. comm.). The reason for this might be the huge quantities of pesticides used in agriculture practices. In certain regions of Moldavia, this has been suggested to be a reason for a high incidence of miscarriages and congenital malformations (Klopov pers. comm.). The mean concentrations of *p,p'*-DDE in maternal plasma of delivering women in Norilsk and Salekhard were found to be rather low: 0.67 and 0.38 μg/L, respectively, compared to 11.3 μg/L in the Inuit population of Quebec, Canada (Dewailly *et al.* 1994a). In cord blood, the concentrations showed the same pattern: Norilsk 0.23 μg/L and Salekhard 0.28 μg/L, compared to 1.63 μg/L in northern Quebec. The mean *p,p'*-DDE in breast milk showed levels of 1291 μg/kg lipid (Norilsk) and 978.3 μg/kg lipid (Salekhard), compared to 1212 μg/kg lipid (Nunavik in Canada).

PAH levels in Russian human populations have not been previously reported. These substances were analyzed in mothers' blood, cord blood and placenta from both Norilsk and Salekhard. Naphthalene, phenanthrene, anthracene, fluoranthene, pyrene, benzo[a]pyrene, benzo[e]pyrene and 2-methyl-naphthalene were determined (Klopov 1995 pers. comm.). For naphthalene, the mean concentration in mothers' blood was 9.5 μg/L in Norilsk and 10.7 μg/L in Salekhard; in cord blood it was 2.5 and 5.8 μg/L, respectively; and in placental tissue it was 4.9 and 3.9 μg/kg, respectively. The results point to the placenta as a partial barrier of PAH compounds, the extent of which appeared compound specific. Benzo[a]pyrene was not found in quantifiable levels in cord blood, while anthracene and benzo[e]pyrene were higher than in maternal blood.

Under the Norwegian-Russian AMAP human health monitoring study, ten samples of breast milk were collected from first-delivery mothers in Arkhangelsk and Severodvinsk, both located in Arkhangelsk County. These samples were analyzed for chlorinated organic compounds, and the results are summarized in Table 12·21. The observed concentrations match those found in Murmansk, Monchegorsk and Lithuania. Other than for total chlordanes, and possibly also total PCBs and total di-*ortho* PCBs, concentrations of chlorinated compounds in breast milk are considerably higher in samples from Russia and Lithuania than in samples from Norway. The relative enhancement of total mono-*ortho*-PCBs in the Russian samples is especially interesting, since these have significant associated TEQ values because of their resemblance to dioxins.

Considering the data in Table 12·21, a final observation concerns the DDE to DDT ratio values. This ratio provides information about current use of *p,p'*-DDT, or other possible current exposure, such as through imported foods. The relatively low DDE/DDT values found for the Russian breast milk samples suggest a higher current exposure to DDT here than in Norway or Lithuania.

Metals

Mercury

Under the joint Norwegian-Russian AMAP human health monitoring study of Norway, Kola and Arkhangelsk, the mean maternal blood mercury levels (see Annex Table 12·A13) were marginally higher in the Norwegian population ($p < 0.01$); and for both the Russian and Norwegian groups as a whole, they corresponded to reference values for moderate to no fish consumption (Odland *et al.* 1996, 1997). When broken down by community, the following mean values (in μg/L) are found: 1.6 (Arkhangelsk, n = 50); 2.3 (Nikel, n = 50); 2.5 (Hammerfest, n = 57); and 3.4 (Kirkenes, n = 40), with the difference between Hammerfest and Arkhangelsk being statistically significant ($p < 0.0001$).

Consistent with the previously observed correlation between fish intake and blood mercury levels (Brune *et al.* 1991), the mean for the fishing town of Kirkenes was the highest. The very low levels for Arkhangelsk suggest that methylmercury does not enter the food chain. It is clear that the levels observed in Kirkenes, Hammerfest, Nikel and Arkhangelsk do not pose any health risk.

Cadmium

The comparisons of the results from Norway, Kola and Arkhangelsk (Annex Table 12·A13) indicate that the cadmium concentrations in mothers' blood are marginally lower in the Russian group as a whole ($p < 0.05$); both sets of data are, however, well below the baseline reference values of <2.0 μg/L (Odland *et al.* 1996, 1997). The data obtained from the Siberian locations and Tromsø conform to this trend. This comparison could not be made for the cord blood levels, since many of the values in both groups were below the detection limit. As expected, smokers (10% of the total) had the highest blood cadmium concentrations (median of 2.1 μg/L).

The cadmium concentrations in blood of both the Russian and Norwegian populations studied are very low, indicating insignificant environmental exposure. A significant difference between the non-smoking mothers and the mothers smoking more than ten cigarettes per day ($p < 0.0001$) was observed. Interestingly, some of the mothers smoking more than ten cigarettes per day have children with blood concentrations below the detection limit of 0.1 μg/L, suggesting a possible threshold for the passage of cadmium through the human placenta. Differences between different mammals are known, with free passage of cadmium through the Wistar rat placenta, while the hamster placenta is totally impermeable to this metal (Tsuchiya *et al.* 1987, Hanlon *et al.* 1989). It seems that the human placenta serves as a selective barrier to cadmium with an average attenuation of 40-50% (Lauwerys *et al.* 1978, Roels *et al.* 1978, WHO 1992a). Because many of the cord blood cadmium concentrations were below the detection limit, it may be that individual variability related to physiological phenomena such as metallothionein induction in the placenta is operative.

Table 12·21. Levels of organic contaminants in breast milk from northern Russia, northern Norway and Lithuania in the period 1993-1996. Mean values, ng/g lipid.

	n	HCB	∑HCH	∑chlordane	*p,p'*-DDE	*p,p'*-DDT	∑DDT	Ratio DDE/DDT	∑PCBs	∑mono-*ortho* PCBs	∑di-*ortho* PCBs
Severodvinsk 1996	10	95	467	35	1118	176	1336	6.4	517	123	394
Arkhangelsk 1996	10	99	558	27	1687	344	2088	4.9	516	116	400
Murmansk 1993	10	129	858	59	1269	178	1615	7.1	484	95	389
Monchegorsk 1993	10	111	745	33	892	145	1155	6.2	535	132	403
Tromsø 1993	10	39	34	42	310	19	328	16.3	383	54	329
Lithuania 1993	12	264	417	20	861	43	1002	20.0	529	115	414

Mothers' blood and cord blood from Norilsk and Salekhard have also been analyzed for cadmium by Russian colleagues (Klopov 1995 pers. comm.). Comparable findings to those described above are observed, with a very high correlation between cadmium levels and smoking habits. The mean blood cadmium concentration of smoking mothers in Norilsk was 5.7 μg/L, while in non-smoking mothers it was 1.3 μg/L. The values in women from Salekhard were 5.7 μg/L for smoking women and 0.8 μg/L for non-smoking mothers. Mean cadmium levels in cord blood were 2.8 μg/L in samples from Norilsk and 2.5 μg/L in the Salekhard samples. Note that these levels are somewhat higher than those found in the AMAP study (reported in Annex Table 12·A13). Harmonization of analytical methods is, therefore, prudent.

Lead

Under the joint Norwegian-Russian AMAP human health monitoring study in Norway, Kola Peninsula and Arkhangelsk, the mean lead levels in mothers and neonates in Russia as a whole (Annex Table 12·A13) are significantly higher than in Norway ($p < 0.0001$); both sets of data conform to the expected reference values (Odland *et al.* 1997). The recently obtained results from Siberia and Tromsø concur. A linear relationship between maternal blood and cord blood lead is clearly observed (e.g., for the town of Nikel, $r = 0.96$, $p < 0.02$, $n = 24$).

Even though the Russian values are significantly higher as a whole, both population groups have mean blood lead concentrations within the reference interval of 0-40 μg/L. Russian cars are still using leaded gasoline, but the traffic density is not as high as in western cities, even in a town like Monchegorsk with a population of about 60 000 inhabitants. If the currently observed blood lead concentrations of the Russian women are compared with western measurements from 20 years ago (Environmental Health Perspectives 1990, 1991), much lower concentrations are found in the Russian women. Overall, the Norwegian blood lead results constitute some of the lowest concentrations reported in the literature. Improvement in preventing contamination during collection, sampling, handling and storage of the specimens might be one reason for this. Another, of course, is that following introduction of lead-free gasoline, the effect of reduced emissions are being observed in humans.

Whole blood lead concentrations were determined in the children aged 2 to 13 from three Russian towns (Lovozero, Krasnochelie and Apatity). Lovozero is the Russian center for the Saami population with approximately 2000 inhabitants. Krasnochelie is a very isolated community on the Ponoi River, far east on the Kola Peninsula, with a mixed population of Saami, Komi, Nenets and Russians, totaling 850 people. Apatity is one of the bigger cities of Murmansk County, with approximately 89 000 inhabitants. The results

Table 12·22. Comparison of blood lead levels in children in Krasnochelie, Lovozero and Apatity on the Kola Peninsula of Russia, μg/L.

	n	Mean	SD	Range
Lovozero	50	58	22.8	21-114
Krasnochelie	13	83[a]	37.3	21-157
Apatity	22	54	22.8	27-81

a. Statistically different from the other two values ($p < 0.001$).

are shown in Table 12·22. The blood lead concentrations of the children in Krasnochelie are significantly higher than the concentrations in both Lovozero and Apatity ($p < 0.001$). Blood lead concentrations greater than 100 μg/L are of medical concern in children, and these occur mostly in Krasnochelie, but also in Lovozero. Other investigations have also revealed a trend of higher blood lead levels in small, remote communities than in bigger cities (Nieboer pers. comm.). One possible explanation might be the lead content of wild meats due to the use of leaded ammunition, especially lead shot. A more thorough dietary evaluation is necessary to further elucidate this important finding.

Nickel

Both the neonatal and maternal mean urinary nickel levels are considerably higher among the Russian communities studied ($p < 0.0001$) (Odland *et al.* 1997) (Figures 12·13 and 12·14). Those reported for the Norwegian group fall within the reference interval of 0.5-6.1 μg/L (Sunderman *et al.* 1988,

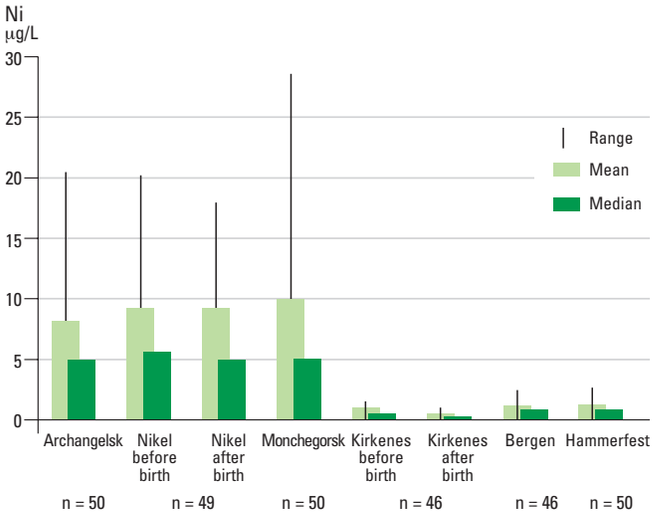


Figure 12·13. Nickel levels in urine from pregnant and delivering women in different areas of Russia and Norway.

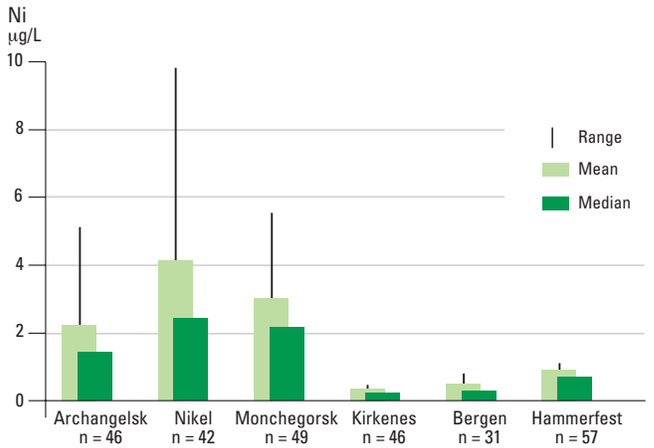


Figure 12·14. Nickel levels in urine from newborn children in different areas of Russia and Norway.

Sunderman 1993), while the Russian mean values for mothers are higher than this interval. Breaking down the urinary nickel concentrations for mothers by communities, the values for the Russian towns of Nikel and Arkhangelsk are significantly higher than those for the Norwegian centers ($p < 0.0001$) and different from each other ($p < 0.01$). In terms of the neonatal first voids, there were no statistical differences between individual communities, whether in Russia or Norway. There appears to be no apparent relationship between the maternal and corresponding neonatal urinary nickel concentrations ($r = 0.11$, $p > 0.5$, $n = 224$).

The reason for the higher mean urinary nickel levels observed in mothers and their newborns in the Russian communities is not obvious. When the observed concentrations are corrected using urinary creatinine levels, the difference

Table 12·23. A comparison of cadmium, lead, mercury and nickel in whole blood (WB), cord blood (CB), urine of mothers (UM), and first-void urine of neonates (UC), and ^{137}Cs in placenta (P) in selected Norwegian and Russian communities.

Element	Sample type	Concentration – mean and standard deviation: Russian communities	n	Norwegian communities	n	Level of significance, p
Cadmium, μg/L	WB	0.4±0.2	206[b]	0.5±0.6	120[b]	<0.05
	CB	<DL[a]	73[c]	<DL[a]	69[c]	
Lead, μg/L	WB	33.2±16.6	206[b]	12.4±6.2	120[b]	<0.0001
	CB	20.7±10.4	71[c]	12.4±6.2	68[c]	<0.0001
Mercury, μg/L	WB	1.9±0.9	60[d]	2.5±0.8	25[d]	<0.01
Nickel, μg/L	UM	8.9±9.7	98[e]	0.9±0.7	142[e]	<0.0001
	UC	2.5±3.1	64[f]	0.6±0.2	77[f]	<0.0001
^{137}Cs (Bq/kg, 7-h counting, DL 1.3 Bq/kg)	P	1.9±1.4 (range 1.3-7.9)	41[g]	5.5±11.0 (range 1.3-83.2)	74[g]	

a. Values < DL = 0.1 μg/L for statistical purposes; DL: detection limit. b. Communities include: Bergen, Hammerfest and Kirkenes (Norway); Murmansk, Monchegorsk, Apatity, Arkhangelsk and Nikel (Russia). c. Communities include: Kirkenes and Bergen (Norway); Nikel and Arkhangelsk (Russia). d. Communities include: Hammerfest (Norway); Nikel and Arkhangelsk (Russia). e. Communities include: Kirkenes and Bergen (Norway); Nikel and Arkhangelsk (Russia), samples from middle of pregnancy and post partum, values are unadjusted. f. Communities include: Kirkenes and Bergen (Norway); Nikel and Arkhangelsk (Russia); values are unadjusted. g. Communities include: Saami population of Finnmark, Kirkenes, Hammerfest, Sømna, Namdal, Bergen and Valdres (Norway); Arkhangelsk and Nikel (Russia); Gomel (Belo-Russia); and Kiev (Ukraine).

between the Russian and the Norwegian communities is reduced considerably, but nevertheless remains significant ($p < 0.001$). As discussed (Nieboer *et al.* 1992), specific gravity corrections might have been more suitable, but unfortunately these were not measured. The creatinine normalized data does, however, suggest some additional environmental contribution for the residents of Nikel, compared to Arkhangelsk. This is not unexpected, and has also been observed for the nickel mining/refining town of Sudbury, Canada (Hopfer *et al.* 1989). For random, but corrected urine samples from Sudbury residents, urinary nickel levels averaged 3.2± 2.8 μg/L (range 0.4-10.0 μg/L), compared to 2.0±2.0 μg/L (range 0.7-5.4 μg/L) for residents of Hartford, Connecticut, USA, a city without an obvious nickel point source (Hopfer *et al.* 1989, Nieboer *et al.* 1992). Clearly, the values observed for Kirkenes and Bergen are lower than those reported for Hartford, presumably reflecting less industrialization and urbanization. Interestingly, the average nickel levels in tap water reported by Hopfer *et al.* (1989) were 0.4±0.2 μg/L in Hartford and 109±4.6 μg/L in Sudbury. The industrial emissions of nickel in Sudbury are clearly reflected in the nickel levels in the water supply source. Recent data for tap water from Nikel and Zapolyarnyy also show considerable elevation (up to 90 μg/L) compared to the towns of Apatity and Kirovsk (1-8 μg/L). Drinking water as a source of nickel will need to be explored further to resolve the relatively high levels observed in the Arkhangelsk community. Leaching of nickel from stainless steel items is also known (WHO 1991b). Another possibility for individual exposure is nickel release from oral prostheses, which contain significant amounts of this metal in Russia. Although the average western diet contains about 150 μg per day of nickel, there are certain food items that are relatively rich in this metal, such as chocolate (WHO 1991b). Special dietary sources in the Russian communities might, therefore, also be investigated.

Since the measurement of toxic metals in first-voided urine specimens from newborns have apparently not been attempted before, no comparison with literature values is possible. The finding of significant levels of nickel in these samples attest to the free-flow of nickel across the placental barrier.

Sunde and Alexander (1992) have completed urinary nickel measurements for inhabitants of the Sør-Varanger region of Norway (Pasvik and Bugøynes districts) near the Norwegian-Russian border, in April 1990. All participants were over 40 years of age, with equal distribution of sex (n = 22 from the district of Pasvik, n = 24 from the district of Bugøynes). The median urine nickel concentration in Pasvik, nearest to Nikel, was 1.3 μg/L, while the median value in the reference group in Bugøynes was 1.0 μg/L. Since these concentrations are from a group of adult citizens living in the same community as the women and children participating in the Kirkenes (AMAP) study, it is not surprising that the latter exhibited comparable concentrations.

The Russian-Norwegian Health Group has, in the period 1993-96, organized a comprehensive study of health conditions of the adult population of selected communities in Finnmark in Norway and the Kola Peninsula in Russia. The investigation has focused on health conditions in the areas contaminated by the nickel industry of the Kola Peninsula. The results are under assessment at the time of writing of this report (T. Smith-Sivertsen pers. comm.). Preliminary results, thus far, suggest that the urine nickel levels for inhabitants of Sør-Varanger, near the Norwegian-Russian border, are not increased compared to reference populations. However, urban-dwelling does appear to be a risk factor for increased nickel exposure. The conclusions of this study, to date, are that the individual nickel exposure attributed to air pollution from the neighboring Russian nickel industry is of minor importance.

Compared to nickel workers, for whom urinary nickel concentrations are typically in the range 10-100 μg/L, the exposure to nickel in the Russian communities studied might be considered to be mild to moderate. Although the health consequences of this implied level of exposure are not believed to be of concern, the observations reported should be subjected to closer scrutiny. The use of 24-h urines, or specific gravity adjusted spot specimens are advised in future studies in order to reduce the natural variation due to dilution effects (Nieboer *et al.* 1992). In addition, non-pregnant females should be studied, since pregnancy and child birth involve complex physiological processes and trauma that may influence urinary nickel levels (Nomoto *et al.* 1983).

A comparison of the different toxic metals between Russian and Norwegian mothers and newborns is shown in Table 12·23.

Copper

Copper values in maternal sera show a very similar pattern for Bergen, Hammerfest, Tromsø, Kirkenes, Nikel (Group 1, n = 50, sampling period 1991; Group 2, n = 50, sampling period 1993/94) and Arkhangelsk (Annex Table 12·A13) (Odland *et al.* 1996). In the Norwegian towns of Bergen and Hammerfest, the mean serum copper concentrations were 2140 μg/L (n = 50, range 1029-3120 μg/L) and 2090 μg/L

(n = 57, range 1550-3190 μg/L), respectively. Reference intervals for serum copper for pregnant women at term are 1180-3200 μg/L (Burtis and Ashwood 1994). In Kirkenes, near the Norwegian-Russian border, the mean value was 2200 μg/L (n = 40, range 1582-2930 μg/L). The two groups from the industrial town of Nikel, sampled in 1991 and 1993/94 have mean values of 2123 μg/L (range 1182-3088 μg/L) and 2160 μg/L (range 1843-2491 μg/L), respectively. Arkhangelsk mothers show mean serum copper concentrations of 2310 μg/L, with range 1582-3629 μg/L. Visual inspection of these data suggested a normal distribution. Comparing the combined data sets within each country, there is no significant difference between the Norwegian (n = 162) and Russian (n = 100) results ($p > 0.1$). Arkhangelsk females have significantly higher values than Hammerfest females ($p < 0.005$), while there is no significant difference between the Nikel and Kirkenes groups ($p > 0.5$). In both the Russian and Norwegian female subjects, the reported serum copper concentrations fall into the mid-range of the international reference interval for pregnant women at term.

It is concluded that the diets of the Russian and Norwegian female subjects studied are adequate for maintaining a proper copper balance.

Zinc

Zinc levels in sera of pregnant women in Bergen, Hammerfest, Tromsø, Kirkenes, Nikel (Group 2) and Arkhangelsk are summarized in Annex Table 12·A13 (see also Odland *et al.* 1996). The highest mean serum zinc concentrations are in the group from Bergen, with a mean value of 770 μg/L. Serum zinc levels for the Russian groups of women were generally lower than those observed for the Bergen individuals, reaching statistical significance ($p < 0.001$). Except for the Bergen group, the mean concentrations fall below the accepted range for all the other groups studied. The international reference intervals are 700-1500 μg/L for serum, with plasma levels 5 to 15% lower (namely 587-1215 μg/L) because of osmotic fluid shifts from the cells due to anticoagulant use (Burtis and Ashwood 1994).

An explanation for the relatively low serum zinc levels is not immediately apparent. During pregnancy, Norwegian women usually use mineral supplements and thus the low levels seen in the Hammerfest group is surprising. Furthermore, since the iron, selenium and copper balances were in the normal range for both the Russian and Norwegian subjects, and since meat and/or seafood are good sources of these elements, as well as of zinc, dietary deficiency is not likely. Zinc is relatively poorly absorbed from whole grain products and their relative importance in the diet might furnish one explanation. Further research is required to verify the apparent low zinc status of some of the women studied, including the use of a detailed food frequency questionnaire.

Selenium

Selenium in maternal whole blood and cord blood have been compared for a number of Russian and Norwegian communities, excluding the Siberian and Tromsø locations (Odland *et al.* 1996). The patterns for maternal blood and cord blood are very similar; in Norway (excluding Tromsø), 111 (n = 114, range 63-158) and 118 μg/L (n = 69, range 79-229), respectively, and in Russia (excluding Siberian locations) 126 (n = 219, range 63-205) and 111 μg/L (n = 86, range 79-158), respectively. Comparing the different locations (Annex Table 12·A13), the highest whole blood selenium values for mothers are found in Apatity (139±15 μg/L), and the lowest in Norilsk (80.6±11.9 μg/L) in Russia and in Tromsø (93.2± 18.2 μg/L) in Norway. All results for the Norwegian and Russian females were in the normal range. A comparison according to smoking habits gave no differences in blood selenium levels between non-smokers and women smoking more than ten cigarettes per day. The average serum selenium levels in the mothers ranged from 79 μg/L (Nikel, Group 2) to 95 μg/L (Kirkenes) and were higher in the Norwegian females ($p < 0.0001$).

The serum or whole blood selenium concentrations found are within the international reference intervals for adults (Minoia *et al.* 1990, Aro *et al.* 1995, Wang *et al.* 1995a, 1995b) of 56-105 μg/L in serum or plasma and 76-140 μg/L in whole blood. Levels at birth, as measured in cord blood, are comparable to those observed for the corresponding mothers. This is in agreement with previous observations in Finland (Aro *et al.* 1995). Although the maternal and cord blood serum selenium levels were marginally lower ($p < 0.001$) in the Russian samples, the reverse trend is observed for whole blood ($p < 0.001$). However, in the latter comparison, samples were included from Apatity, Monchegorsk and Murmansk, which have considerably higher average concentrations (139, 129 and 134 μg/L, respectively). The finding of normal selenium status is perhaps somewhat surprising in the light of concern about low selenium intake/status in Finland and Sweden (Wang *et al.* 1995a, 1995b).

Iron

The women in Nikel, Russia have the highest mean serum iron levels in the essential element status study, namely 715 μg/L (standard deviation 603), while Bergen and Hammerfest women have mean values of 497 and 486 μg/L (standard deviation 318 and 424, respectively); these concentrations are significantly different ($p < 0.05$) (Odland *et al.* 1996). A similar pattern exists for serum ferritin. Women from Arkhangelsk have the highest mean serum ferritin value (37.1 μg/L, standard deviation 47.7). The lowest values were found in Bergen, with a mean value of 23.4 μg/L (standard deviation 14.6). On average, ferritin levels in the Russian group are higher ($p < 0.05$). The Norwegian reference interval for serum iron is 503-1396 μg/L. There are, however, so many confusing and complex factors influencing the absorption, distribution and excretion of iron that serum iron levels are not a suitable measure of the iron stores in the body. By contrast, serum ferritin in healthy women is a very appropriate indicator of such stores (Burtis and Ashwood 1994, Borch-Iohnsen 1995). Reference values for serum ferritin have quite a wide range, and the current Norwegian reference interval is 7-133 μg/L depending on age, sex and pregnancy. Concentrations indicative of depleted iron stores are < 10 μg/L for age ≤ 14 years and <12 μg/L for age ≥ 15 years (Isselbacher *et al.* 1994).

For the females examined, the ferritin levels found were within the expected range, and thus the iron status of the women studied may be considered normal. This conclusion is supported by the apparent absence, according to questionnaire results, of disease factors that can enhance serum ferritin concentrations such as liver disease and chronic infection (Isselbacher *et al.* 1994). Interestingly, the lowest ferritin concentrations were observed for the Norwegian towns of Bergen and Hammerfest; they are not statistically different ($p > 0.2$) compared to the groups from Nikel and Kirkenes, but are statistically lower ($p < 0.05$) relative to Arkhangelsk. Overall, the Norwegian ferritin concentrations were lower ($p < 0.05$). This is difficult to interpret since iron supplements for pregnant women in Norway may be considered routine. The serum iron levels follow the same general trend and are also within the accepted reference interval.

Table 12·24. A comparison of essential elements in whole blood (WB) or serum (S) of females in selected Norwegian and Russian communities, with accepted reference intervals.

Element	Reference interval, µg/L	Sample type	Concentration – mean and standard deviation: Russian Communities	n	Norwegian Communities	n	Level of significance, p
Copper[a]	1180-3200 (end-of-term values)	S	2219±389	112	2149±350	147	>0.1
Ferritin[a]	7-133	S	33±41	100	24±19	154	<0.05
Selenium[a]	55-103	S	81±15	124	94±13	154	<0.0001
Selenium[b]	79-142	WB	123±19	215	112±17	123	<0.0001
Zinc[a]	700-1500	S	524±98	112	648±118	147	<0.0001

a. Communities include: Bergen, Hammerfest and Kirkenes (Norway); Nikel and Arkhangelsk (Russia).
b. Communities include: Bergen, Hammerfest and Kirkenes (Norway); Apatity, Arkhangelsk, Monchegorsk and Murmansk (Russia).

A comparison of the different essential elements in Russian and Norwegian mothers and newborns are shown in Table 12·24.

Clinical chemical parameters

Other clinical chemical values selected for screening were: cholesterol, high density lipoprotein (HDL), triglycerides, thyroxine (FT4), thyroid-stimulating hormone (TSH), alanine aminotransferase (ALAT), aspartate aminotransferase (ASAT), bilirubin and creatinine (Odland *et al.* 1996). What is of special interest is that the lipid values (cholesterol, triglycerides and HDL concentrations) were generally higher in the Russian population, although not significantly so ($p > 0.05$); they fall within the Norwegian or international concentration reference intervals.

Radionuclides

Over the past 30 years, there has also been considerable concern about human health effects of radiation in the Norwegian-Russian Arctic areas (Paakkola 1991). These questions are addressed in more detail in chapter 8. In terms of human exposure, whole-body counting has been carried-out in certain areas, but no sampling of material from pregnant women or neonates has been done. Cs-137 has been recognized as the most important anthropogenic radionuclide in the nutritional chain (WHO 1983). The major reasons for concern in Arctic Fennoscandia and Russia are: the nuclear testing program on Novaya Zemlya in the 1950s and 1960s; the Chernobyl accident in 1986, with resulting contamination of large areas of the Ukraine, Belo-Russia, Russia, Sweden and Norway; the continued operation of aged nuclear power stations on the Kola Peninsula; and the radioactive fuel- and waste-treatment and storage from both civilian and military sources on the Kola Peninsula and Arkhangelsk Region (Paakkola 1991, Moberg and Reizenstein 1993). In Narjan Mar, in Arkhangelsk County, close to Novaya Zemlya, unverified medical data claim that there are very high incidences of spontaneous abortions, congenital malformations and cancer (Tkatchev *et al.* 1994). A group from the University of Tromsø have investigated the data files in the hospitals of Narjan Mar during the summer of 1996 (Lund, Special AMAP Report 1996, unpubl.). Their conclusion was that the basic information used by Tkatchev and colleagues in their assessment is reliable, and constitutes a valid basis for epidemiological research activities with improved design in this area. The specific radionuclide component of the Russian-Norwegian AMAP human health program was initiated to explore whether ^{137}Cs in whole placenta is a suitable potential indicator for reflecting the radiation exposure and burden to the fetus during pregnancy.

Cs-137 is one of the most significant fission products (6-7% of the fission yield). It has a radioactive half-life of 30.2 years and its beta-particle decay (average energy of 0.19 MeV) is accompanied by gamma radiation of modest energy (0.66 MeV). Cs-137 transfer to the diet is generally from grain products, meat and milk. The lichen-caribou-human food chain represents a particularly important transfer pathway in the Arctic. The body elimination rate for ^{137}Cs, expressed as its biological half-life, varies with age and physiology: 19 days (infants), 57 days (children), 49 days (pregnant women), 84 days (women), and 105 days (men) (WHO 1983). The concentration ratios between maternal tissue, placenta, and fetal tissue are 1 : 1 : 1 and this is the basis for using the determination of ^{137}Cs in placenta for dose estimation purposes (Stather *et al.* 1992).

Measurements of ^{137}Cs in placenta show a more homogenous pattern in the Russian than in the Norwegian material, with ranges 1.3-7.9 Bq/kg ww and 1.3-83.2 Bq/kg ww, respectively (Odland *et al.* 1997). In terms of specific communities, no detectable values were found in Arkhangelsk, only two low values in Nikel, and only one low value in Kiev; the limit of detection being 1.3 Bq/kg. In Gomel, Belo-Russia, detectable values were found in six out of seven placentas (range 1.3-7.9 Bq/kg). In Norway, no detectable values were found in Bergen, however, all values were detectable in Sømna (range 1.38-7.26 Bq/kg) and Valdres (range 2.81-15.92 Bq/kg). Ten women from the Saami population of Finnmark also all had detectable values, ranging from 1.3 to 14.6 Bq/kg. For Kirkenes and Hammerfest, near the Russian border, a more mixed picture is seen, with detectable values in two out of ten and five out of eight placentas, respectively (ranges 1.3-3.9 and 1.3-6.13 Bq/kg). For Trøndelag, the results were also variable, below the detection limit in 6 out of 11 placentas but with high values in two placentas (46.3 and 83.2 Bq/kg).

The preliminary results of this study are somewhat surprising. Cs-137 levels found in the Norwegian samples have a wider range than those for the Russian samples, with two very high outliers from Namdal, Trøndelag (46.3 and 83.2 Bq/kg). The two patients with whom these high values were associated revealed in their answers to the questionnaire that a substantial amount of their total diet was of local, natural origin. Namdal lies in the middle of the Norwegian zone that received the highest accumulation of radiation products from the Chernobyl accident, indicating a connection between this event and accumulation of ^{137}Cs in the food chain in the polluted areas. The other known Norwegian areas of concern after the accident, Sømna and Valdres, show a very homogenous pattern (mean 4.61 Bq/kg, standard deviation 4.21, range 1.38-7.26; and mean 5.94 Bq/kg, standard deviation 5.22, range 2.81-15.92, respectively). These values are very different from those for Bergen, which is well away from the zone affected by Chernobyl, where all placentas had values of ^{137}Cs below the detection limit. The Saami population of Finnmark, outside the main zone in Norway affected by Chernobyl, but with the largest amount of local traditional food in their diet, have a similar pattern of placental ^{137}Cs levels to that found in the Norwegian population in the Chernobyl

zone; mean 7.14 Bq/kg, standard deviation 5.21, range 0.87-14.5. The Russian populations studied, namely Nikel and Arkhangelsk, and the Ukrainian city Kiev, just south of Chernobyl, show placental ^{137}Cs values at or below the detection limit. The wind direction immediately after the accident protected Kiev from contamination. Arkhangelsk, south of Novaya Zemlya, the northern center of the Russian atomic bomb testing program, had no detectable values of ^{137}Cs in the local placentas. Gomel, Belo-Russia, is in the Chernobyl affected zone of the FSU. Here, the results show detectable values in six out of seven placentas (mean 4.49 Bq/kg, standard deviation 4.2, range 2.3-7.9). Overall assessment of the data suggests good correlations between the levels of ^{137}Cs in placenta (Bq/kg ww) and the dietary intake of local food. Proximity of communities to the Chernobyl contamination zone is also important.

A similar investigation of plutonium concentrations in placentas has been performed by Lund (pers. comm.). The highest concentration was close to 1 mBq/kg fresh placenta, an order of magnitude higher than levels previously reported from England. This study indicates that placentas could also be used for biological monitoring of external plutonium contamination.

Lund and Galanti have investigated the incidence of thyroid cancer in Scandinavia in relation to ^{131}I in fallout, based on information from the Norwegian and Swedish Cancer Registers (E. Lund pers. comm., AMAP special report unpubl.). This investigation showed that there was evidence of a higher risk of thyroid cancer before the age of 25 years among children born between 1951 and 1962 (the cohorts exposed during childhood to radioactive fallout from historical weapons testing) compared to those born in 1963 or later, when data from both countries were analyzed together. The results of this study are compatible with the hypothesis that radioactive fallout from atomic weapons tests on Novaya Zemlya had an impact on thyroid cancer risk in Norway and Sweden. The results are, however, not easily explained by radioactive fallout alone. The higher risk of thyroid cancer at young age among persons who were born, or were young children, between the 1950s and early 1960s, compared with those born later, awaits an alternative plausible explanation.

12.4.4.4. Finland

Introduction

Finnish Lapland is the northernmost province of Finland, with about 200 000 inhabitants living in an area of 93 000 km^2. Of the population, ca. 60% live in the towns of Rovaniemi, Kemi, Tornio and Kemijarvi and their surroundings. There are no major sources of pollution in the northern parts of Finnish Lapland. The most northern pulp industry is in Kemijarvi, near Salla which is one of the study communities under the Finnish AMAP human health monitoring program.

Sources of exposure

The most important pollution exposures, with respect to human health, are to sulfur oxides and metals, which originate from the Kola Peninsula. Acidification enhances the release of metals from the soil. Soil buffering capacity is poor because they lack cations, such as calcium, potassium, magnesium and ammonium. Mercury is the most important toxic metal because it bioaccumulates in fish, one of the most important parts of the diet in Lapland.

Water

Most of the small communities involved in the Finnish human health study have their own wells or are using surface water for drinking water supply. In the municipality of Inari, both the water in wells and the surface waters have a low mineral content (Soininen pers. comm.). In some cases, well water can contain a large amount of iron.

Air

The prevailing winds in Lapland are from the southwest, and only from the opposite direction 7% of the time. Hence, the air pollution from the Murmansk region does not generally affect northern Finland and the average SO_2 air concentrations are rather low. However, when the plumes from the nickel-smelters on the Kola Peninsula are transported westwards, hourly concentrations of several hundred micrograms per cubic meter may occur in the northeastern parts of Lapland (Juntto 1992). The same concern is relevant for other pollutants originating from the smelters, namely carbon dioxide, particles and metals.

Levels and trends of contaminants in humans

The populations included in the Finnish human health monitoring program are from the Saami communities of Inari, Enontekiø and Utsjoki, and from the eastern Lapland communities of Pelkosenniemi, Salla and Savukoski. The eastern Lapland communities were selected because of possible effects of pollutants originating from the Kola Peninsula.

Persistent organic pollutants

Organochlorine pesticide and PCB residues were analyzed in 183 human milk samples obtained in 1984/85 from 165 women living in different parts of Finland (Mussalo-Rauhaama *et al.* 1988). The *p,p'*-DDE concentrations were above the detection limit in 99.5% of the samples, *p,p'*-DDD and *p,p'*-DDT in 57.9%, isomers of HCH in 30%, *cis*-chlordane in 4.9%, oxychlordane in 3.3%, *trans*-nonachlor in 6%, heptachlor in 12%, and heptachlor epoxide in 6.6% of samples. Mirex was not found in any of the milk samples, whereas the signals of toxaphenes were detected, but could not be quantified. The mean fat-adjusted residue levels above the detection limit in Finnish human milk samples of primipara mothers were 660 μg/kg lipid for total DDT compounds, 80 μg/kg lipid for HCB, 930 μg/kg lipid for PCBs, 410 μg/kg lipid for chlordane compounds, 200 μg/kg lipid for isomers of HCH, and 100 μg/kg lipid for heptachlor epoxide. The corresponding geometric means were 460, 60, 570, 20, 20, and 10 μg/kg lipid, respectively. The age of the mothers correlated positively with the DDE concentrations in human milk. The residues of organochlorine compounds in human milk did not differ between mothers living in industrial regions and other mothers. Small, but insignificant differences between regions were found. No relation was found between the organochlorine contents and fish consumption, smoking habits, weight loss or social group of the donors. Additional AMAP data are currently being gathered.

Metals

Mercury. Mussalo-Rauhamaa *et al.* (1996) have described trends in the concentrations of mercury, copper, zinc and selenium in hair and serum of inhabitants of northeastern Finnish Lapland in 1982-1991. The mean concentration of mercury in the hair of the 19 individuals in the study from northeastern Lapland in 1991 was 1.5 mg/kg (range 0.2-6.2 mg/kg). The men consumed more reindeer meat and fish than the women, but no significant difference in mercury content between the sexes was observed. The mean mercury concentrations in hair from people living in the Russian-Finnish border region were slightly higher, but not significantly different. The comparison of people in Lapland from 1982 to 1991 showed slightly lower values in 1991, but the differences were not significant.

Cadmium. An investigation of blood cadmium in 230 male reindeer herders was conducted in Finnish Lapland in 1991 (Nayha *et al.* 1991). The mean age of the studied group was 43 years, and 31% of them were smokers. The mean cadmium concentration was 1.1 μg/L (range 0.1-37.1 μg/L) and increased from southwest to northeast. A statistical analyses, accounting for smoking and age factors, showed geographical area to be a significant factor. The health-based upper limit for blood cadmium (5 μg/L) was exceeded in 4.8% of the men and in 10% of those living in the northeastern areas. Blood cadmium was twice as high in men who ate reindeer meat at least twice a week than those who did so less often. The source of the cadmium was assumed to be emissions from the nickel smelters on the Kola Peninsula. Subsequent Norwegian/Russian investigations have not confirmed this conclusion.

Copper and zinc. In the study of Mussalo-Rauhamaa *et al.* (1996), inhabitants of Lapland had high hair copper levels, up to 671 mg/kg, which may indicate external contamination or exceptionally high intake. Well water in Finland is usually soft, which may cause corrosion of copper water pipes. No tendency for any increase or decrease in copper or zinc concentrations in the hair of inhabitants of Ivalo was found during the interval 1982-1991, nor was there any relationship between hair and serum copper concentrations.

Selenium. Finland is one of the several low selenium areas in the world (Mussalo-Rauhaama and Lehto 1989, Mussalo-Raumaha *et al.* 1996). In studies carried out between 1941 and 1984, the mean intake of Se ranged from 20 to 50 μg per day in Finnish adults, depending on the ratio of imported grain to domestic grain. Lapps, however, have exhibited high selenium intake, thought to be the result of their use of the lichen-reindeer food chain. The recommended daily selenium intake is 50-200 μg/day for adults and children over seven years of age. Since 1984, Se as sodium selenate has been added to fertilizers: 16 mg/kg for soil where cereal grains are grown, and 6 mg/kg for soil where grasses and silage are grown. The principal goal was to increase the Se concentration in cereals to a level ten times higher than that found without supplementation. The latest reports from central parts of Finland show average selenium concentrations in serum between 100 and 150 μg/L for adult males. It is concluded that the average serum selenium levels are now comparable to those in the British and Canadian populations, and to those of Finnish Lapps. The supplementation of fertilizers with selenium is continuing.

At the present time, no specific AMAP human health monitoring program data for Finnish pregnant women or children are available. In the study of Mussalo-Raumahaa *et al.* (1996), the mean concentration of selenium in the sera of 11 persons living in Ivalo was 97 μg/L (range 74-116 μg/L) in 1982 and 129 μg/L (range 67-204 μg/L) in 1991, whereas the mean value for serum selenium in the Nellim district in 1991 was 159 μg/L (range 119-174 μg/L) and from the Sevettijarvi-Naatamø district 120 μg/L (range 64-202 μg/L). There was a significant tendency for an increase in selenium concentration in the sera of Lapps ($p < 0.05$, Wilcoxon signed rank test). In 1982, the serum selenium concentration in males in Ivalo was higher than the concentration in females, but this difference was not found in 1991. No association was observed between selenium content and the consumption of fish, meat, dairy products or grain products, neither in 1982 nor in 1991. Since 1990, the selenium concentrations of serum in the normal population in northern Finland has been consistently higher.

Radionuclides

Finnish data from 1970-1980 show a strong association between the intake of reindeer meat and whole-body concentrations of ^{137}Cs in the Lapps of Lapland (Rahola *et al.* 1993). As discussed for the Norwegian/Russian data, body burdens of ^{137}Cs correlate with dietary habits. The average ^{137}Cs body-burden in Inari male Lapps in 1976 was 8473 Bq (229 nCi), 3663 Bq (99 nCi) and 777 Bq (21 nCi) for reindeer herders, fishermen, and Lapps of other occupations, respectively. whole-body counts for ^{210}Po in Lapps were about 12 times higher than in residents of southern Finland. Further information on radionuclides is contained in chapter 8.

12.4.4.5. Conclusions for Norway, Russia and Finland

This section constitutes a preliminary overview of the status in Arctic regions of Norway, Russia and Finland of the toxic and essential elements and organic contaminants included in the AMAP human health studies. The focus has been on the concentrations in blood, urine or placenta from women pre- and post-delivery, and in cord blood and urine from their newborn babies. For cadmium, lead and mercury, the levels found are mostly within baseline reference intervals, in all the populations examined, including groups from the Russian town of Nikel which is regarded as seriously polluted. The urine nickel levels were significantly higher in the Russian populations, but appear to be independent of the nearness to a nickel refinery. Other sources of nickel exposure need to be explored. The ^{137}Cs levels in placenta seem to correlate with the location of the collection site relative to the Chernobyl fallout zone, with the highest counts observed for individuals living in areas with high consumption of traditional foods. It may be concluded that, with the exception of zinc, the essential element status in the Arctic centers studied appears to be satisfactory. The relatively low serum zinc levels observed in both the Russian and Norwegian communities needs to be confirmed as part of a more comprehensive zinc nutrition survey. In the available studies reporting levels of toxic metals in biological material from Finland, no concentrations have been found near any 'lowest-observed-adverse-effect-levels'. The concentrations reported here for chlorinated organic compounds in breast milk from Finland are intermediate in magnitude to those found in samples from Norway (lower levels) and Russia (higher levels), except for total PCB and total chlordanes, which appear to exceed the

Table 12·25. Ongoing human health related research projects in Norway and Russia of relevance to the AMAP process.

Title	Principal investigator	Project status
Comparative studies of obstetric patients and their outcomes in Norway and Russia	J.Ø. Odland	In progress
Health status of adult populations in the Norwegian-Russian border zone	E. Lund	In progress
Organic contaminant status in obstetric patients in northern Norway, Kola Peninsula and Arkhangelsk	J.Ø. Odland	In progress
Epidemiologic survey of cancer incidence in Arkhangelsk/Narjan Mar	E. Lund, A. Tkachev	In progress
Reproductive and developmental health among women working in nickel refineries on the Kola Penninsula	E. Nieboer, V. Tchachtchine	In progress
Influence of environmental and foodweb pollution on levels of contaminants in human media and on the health of groups at special risk in the Taimyr Autonomous Okrug	V. Klopov	In progress

Russian values. Further comments concerning levels in blood are made in section 12.5 of this report in the context of an international Arctic data comparison.

Table 12·25 outlines ongoing human health studies in Norway and Russia that are relevant to the AMAP process.

12.4.5. Sweden

Introduction

Due to the influence of the warm Atlantic winds, the area of Sweden north of the Arctic Circle, the northern part of Norrbotten County, has a relatively mild climate. In the most northern area, the average temperature in December is about –15°C. Norrbotten county, covering more than one-fifth of the total area of Sweden, contains about 3% of the total population. The population of the Arctic area is approximately 64 000. The total Saami population in Sweden is about 17 000, of which 3000 are dependent on reindeer herding. However, in 1990, only 523 people were registered as reindeer owners in Norrbotten. The Saami villages are along the western mountain range from the middle of Sweden up to the far north (Figure 12·15).

The two main industrial sites in the Swedish Arctic area which may have caused environmental pollution are the large iron mines in Kiruna and Malmberget. However, there has not been any biological monitoring of xenobiotics in these areas. Monitoring of metals has recently been undertaken south of Norrbotten, in Västerbotten County, in an area polluted with metal emissions from the Rönnskär smelter (65°N). Lead, cadmium, mercury, arsenic, selenium, calcium and iron levels were measured in mothers and newborns during a two-year period. Results from these studies are provided in this section. Because the emissions are lower further north, lower levels are expected in the Arctic area. In order to provide data which is comparable to that of other countries in the circumpolar region, monitoring of metals, organochlorine compounds, and caesium, in mother-newborn pairs has begun in Kiruna (68°N). Metal and organochlorine levels in blood were analyzed by a Canadian laboratory and are reported in section 12.5.

Figure 12·15. Map of Sweden showing locations mentioned in the text; Arctic areas comprise the two northernmost counties, Norrbotten and Västerbotten.

In the Västerbotten Intervention Program, sponsored by the county council, human blood samples are being collected together with results from glucose tolerance and cholesterol tests. Dietary and socio-economic surveys are also underway. All inhabitants living in Västerbotten, including the Saami population in the mountain region, are invited to provide samples on their 30th, 40th, 50th and 60th birthdays. The specimen bank (at the Department of Pathology/Nutritional Research, Umeå University) currently contains data from 45 000 people. These may be used for various kinds of epidemiological studies, e.g., within the fields of research on cardiovascular disease and cancer, and environmental studies. They may also be of use for AMAP human health studies.

Sources of exposure

Food

Between 1984 and 1987, several hundred samples of meat, liver and kidney from Swedish pigs and cattle were analyzed by the Swedish National Food Administration (NFA) for lead, cadmium, arsenic and mercury (Jorhem *et al.* 1991). Analytical procedures included extensive quality assurance. The samples were taken from pigs and cattle slaughtered at all seven abattoirs approved for export to the USA. All of these abattoirs are situated in southern Sweden, which is the main area for meat production. Meat produced there is also distributed in northern Sweden since the farming in Norrbotten is geared towards milk production. Levels are provided in Table 12·26.

Table 12·26. Lead, cadmium, mercury and arsenic concentration (mg/kg ww, mean ± SD) in pig and bovine meat in Sweden in 1984-87 (Jorhem *et al.* 1991).

	Pig meat		Bovine meat	
Metal	Mean±SD	n	Mean±SD	n
Lead	< 0.005±0.009	426	< 0.005±0.001	34
Cadmium	0.001±0.004	426	0.001±0.001	34
Mercury	0.009±0.005	390	0.005±0.005	30
Arsenic	0.024±0.023	338	< 0.015±0.009	29

The lead levels were among the lowest reported in the literature and the cadmium levels were in the lower range of the levels reported from the Netherlands during the same period. No clear time trends were seen, nor was there any difference between the samples from different abattoirs, or any seasonal variation. The arsenic levels found in pig meat were considerably higher than levels reported in 1986 from the Netherlands and the former Federal Republic of Germany. The arsenic concentrations in bovine meat were similar to those previously reported and mercury concentrations in both pig and bovine meat were similar to those reported from several other countries. A decrease in arsenic and mercury levels was found in pig tissues during the study period. This may have been due to a decrease in the use of fish meal in pig feed.

Levels of metals and certain organochlorines were measured in reindeer meat in the Kiruna area during 1983-1994

as part of the Swedish monitoring program in terrestrial biota. The overall geometric mean values of lead, cadmium, and mercury in muscle were 0.007, 0.005, and 0.001 mg/kg, respectively.

In a 1989/90 study of reindeer, 27% (n = 10 603) of the animals had to be rejected because the caesium activity in the reindeer was above 1500 Bq/kg (Albanus *et al.* 1991). Levels were higher in reindeer from Västerbotten than Norrbotten (the Arctic region). In domestic animals, levels were generally below 300 Bq/kg. The ^{137}Cs levels in dairy milk were below 5 Bq/L during 1991. The activity has never exceeded 5 Bq/L in drinking water. The ^{137}Cs activity has never been elevated in crops, vegetables and cultivated berries, and has been below 500 Bq/kg in wild berries. While high caesium activity was found in certain mushrooms, this is not considered to be a health risk to the general population because the consumption is normally low. Fish sold on the market generally had low caesium activity, although high levels prevail in fish from lakes in the most polluted areas. However, these areas are not within the Arctic region. In dairy milk, there is currently very little caesium originating from historical nuclear testing.

Permissible levels in food. Maximum permissible levels (MPL) of lead in food vary from 0.05 mg/kg in infant food, eggs, and meat, to 0.3 mg/kg in lettuce, wine, and canned food, except corned beef (1 mg/kg). The MPL for mercury is 0.5 mg/kg, with certain exceptions, e.g., the MPL for certain fish, such as tuna, is 1 mg/kg. The MPL for tin is 50 mg/kg in infant food and 150 mg/kg in all other food. Lower MPLs were suggested for PCBs in 1993; from 0.02 mg/kg fat in milk-based products to 0.1 mg/kg fat in eggs and meat, and 0.1 mg/kg whole product in fish (NFA 1993, 1994). After the Chernobyl accident, the Swedish NFA introduced a limit value of 300 Bq ^{137}Cs/kg in food (Slorach 1992). This limit value has since been changed to 1500 Bq/kg for reindeer, game, freshwater fish, mushrooms, wild berries and nuts. The limit value within the European Union is 600 Bq/kg. In order to restrict the intake of methylmercury, the Swedish NFA recommends that pregnant and nursing women do not eat pike, perch, pike-perch, burbot, eels or halibut. Other members of the population are advised to limit their fish consumption to once a week. Pregnant women should also restrict their consumption of liver paté. In order to restrict the intake of such contaminants as DDT, PCBs and dioxins, the NFA recommends that regular consumption of liver from cod and burbot be avoided. Baltic herring, wild salmon, and sea trout from the Baltic Sea and the Gulf of Bothnia should not be eaten more than once a week on average; girls and women of reproductive age should limit their consumption to once a month.

Water

The quality of drinking water in Sweden is generally good. Most of the inhabitants obtain their drinking water from community water supplies.

Air

The relationship between air quality and meteorology during winter conditions in the northern parts of Sweden has been studied by the National Defense Research Establishment in Umeå (Johansson *et al.* 1994a). Several air quality models have been tested against the measurements and a new improved model has been developed in order to take into account conditions with low solar altitude, high albedo and low temperatures. Monthly mean and maximum daily mean concentrations of NO_2, SO_2 and black smoke were measured during January to March in 1990 in Jokkmokk, a small community situated at the Arctic Circle. In January, the mean NO_2 concentration was 19 $\mu g/m^3$, 57% of the concentration in Stockholm. The SO_2 concentration of 11 $\mu g/m^3$ was comparable to the values from the Swedish urban air pollution monitoring network. Mean concentrations of black smoke were 27 $\mu g/m^3$.

The concentration of black smoke in Jokkmokk was higher than at any other site in the urban air network (55 stations). The only comparable values (20-24 $\mu g/m^3$) were measured in a few communities in northern Sweden. Large cities, such as Stockholm (more than one million inhabitants) and Göteborg (about 500 000 inhabitants), had considerably lower concentrations of black smoke, with levels of 10 $\mu g/m^3$ and 7 $\mu g/m^3$, respectively. The high concentrations of black smoke were likely due to the common use of wood heating in small communities in the inland parts of northern Sweden. About 50% of the households in Jokkmokk have the facilities to burn wood.

Occupational

Adverse health effects caused by occupational exposure have been reported from the iron mine in Kiruna (Sihm Jörgensen 1986). One hundred and forty-four cases of silicosis were diagnosed and reported to the National Social Insurance Board between 1931 and 1977. Only silicosis stage I has been diagnosed since 1960. The mean concentration of quartz in air decreased from 2.4 mg/m^3 in the 1930s to less than 0.05 mg/m^3 in the 1980s. The expected new cases of silicosis among those who begin to work underground today is less than 1 in 500. Similar figures on silicosis have been reported from the iron mine in Malmberget, 100 km south of Kiruna.

Lung cancer mortality among underground workers almost doubled between the two study periods of 1950-1970 and 1971-1980, and the rate of risk increased from 9.2 to 17.3 cases per million person-years and work level month (WLM, index). This was probably due to an increased accumulated radiation dose. The concentration of radon daughters in the 1950s and 1960s was on average 3.3 Bq/L, i.e., three times the concentration in the early 1970s, however, due to the long latency period for lung cancer, the mortality is increased in the later period. An increased risk of lung cancer has also been reported in underground workers from the iron mine in Malmberget (Radford 1984).

Other

Lifestyle factors such as smoking, and wine and alcohol consumption, may increase the uptake of cadmium and lead. In a study of pregnant women in Västerbotten, blood cadmium levels were twice as high in smokers than in non-smokers. Smoking ten cigarettes or more per day during pregnancy resulted in a 10% decrease in birth weight (unpubl. data).

Levels and trends of contaminants in humans

Persistent organic pollutants

Data on levels of persistent organic pollutants (POPs) in the Swedish population north of the 65°N have not been reported. In addition, there is only limited data available on levels of these pollutants in other organisms from the Swedish Arctic region. Persistent organic pollutant levels in Arctic char from lake Abiskojaure have been collected as part of the Swedish AMAP study on freshwater biota (see chapter 6). Environmental monitoring of persistent pollutants in Sweden has, to date, focused on other (non-Arctic) regions since levels are generally higher in the south of the country and in the central Baltic Sea. Contaminant levels are higher in aquatic organisms than in terrestrial organisms.

It is now clearly established that environmental levels of several persistent organochlorines are declining in Sweden. In the northern areas, close to or above the Arctic Circle, levels in Arctic char, pike, burbot, white-tailed eagle, falcon and osprey are lower compared with southern locations, and are decreasing. Levels in humans are also following the downward trends (Noren 1993). In breast milk sampled in Stockholm between 1967 and 1989, a decrease in the levels of certain pesticides and polychlorinated biphenyls (PCBs) was found. The changes were related to the prohibitions and restrictions applied to the use of these compounds (Table 12·27). Downward time-trends were also seen for polychlorinated dibenzo-*p*-dioxins (PCDDs), polychlorinated dibenzofurans (PCDFs) and specific congeners of PCBs, including non-*ortho* and mono-*ortho* coplanar PCBs. Between 1972 and 1989, average levels of CB 153 in breast milk decreased from 220 to <150 ng/g lipid, CB 138 decreased from 190 to 120 ng/g lipid, CB 180 decreased from 90 to 70 ng/g lipid, and CB 118 decreased from 60 to <30 ng/g lipid. Levels of oxychlordane and *trans*-nonachlor also dropped from 0.02 to 0.012 μg/g lipid. Calculations using toxic equivalent factors relative to 2,3,7,8-TCDD revealed that

Table 12·27. Prohibitions and restrictions applied to certain organochlorine compounds in Sweden (Norén 1993).

Year	Compound	Restriction
1966	Aldrin, dieldrin	Banned for cultivation of carrots, cabbage and onions.
1970	Aldrin, dieldrin	All usage prohibited
	DDT	Prohibited as an insecticide in homes, gardens and agriculture. Exemption for conifers until 1974.
	Chlordane	Prohibited. Minor usage only.
1972	PCBs	Restrictions on use. Presently used only in closed systems (capacitors and transformers). All PCBs should be replaced by other compounds by 1995.
1977	2,4,5-T	Prohibited.
1978	Chlorinated phenols	Prohibited with an exemption for industrial use for textiles and leather. All usage forbidden in 1980 (potential risk of dioxins).
1984	1,2-dichlorobenzene	Prohibited.
1987	Lindane	Agricultural usage forbidden. All usage for domestic purposes prohibited in 1988.

PCB constituted the major part of the toxic equivalents in human milk. Levels of PCB, total or isomer specific, have also been reported in human milk from Umeå (Lindström 1988). In 1986, an average level of 580 μg/kg lipid was reported (n = 10).

PCDDs were first reported in human tissue in Sweden in the early 1980s. In a 1986-88 study, levels in human milk from Umeå, Sundsvall, Uppsala, Borlänge and Göteborg showed similar mean values, of about 20 pg TEQ/g milk fat (Lindström 1988).

A risk assessment of PCBs, initiated by the Nordic Council of Ministers, was published in 1992 (Ahlborg *et al.* 1992). The expert group concluded that it was not yet possible to establish a tolerable exposure level for PCBs, due to incomplete scientific information. However, it could not be ruled out that the current exposure of the Nordic population approximates the level which may give small, but measurable effects on children's behavior and intellectual performance, following fetal exposure or exposure via breast milk.

Metals

During a two-year period, lead and cadmium levels were measured in blood, selenium levels were measured in plasma, and inorganic arsenic levels were measured in urine on three separate occasions during pregnancy in women living in a smelter area and in women living in a control area in Västerbotten (65°N). Umbilical cord blood metal levels were also measured (Table 12·28). The rate of participation was almost 90%. Analyses were carried out using flameless atomic absorption spectroscopy after acid digestion. Quality control analyses were in good agreement with reference values.

Table 12·28. Lead and cadmium in whole blood (μg/L) and selenium in plasma (μg/L) of delivery patients in a smelter and control area in northern Sweden.

	Smelter area		Control area		
Sample	Mean±SD	n	Mean±SD	n	p
Cadmium					
Maternal blood[a]	1.1±0.5	64	1.1±0.5	43	
Cord blood	0.7±0.4	60	0.7±0.3	40	
Lead					
Maternal blood	32±10	241	28±8	144	<0.001
Cord blood	28±8	222	22±8	131	<0.001
Selenium					
Maternal blood	52±10	232	63±15	147	<0.001
Cord blood	45±11	196	48±10	133	<0.01

a. Smokers: blood cadmium levels were about 50% lower in non-smokers

Mercury. Total mercury (Hg) concentration in hair, sampled at delivery, was determined in 122 women living in the smelter area, and in 75 women from the control area (Oskarsson *et al.* 1994). The average Hg concentration in hair was 0.27 mg/kg (range 0.07-0.96 mg/kg), which is somewhat lower than previously reported for pregnant women in Sweden, and very low compared to levels in fish-eating populations in other parts of the world.

Lead. The blood lead levels were low in both the smelter and the control areas, although women in the smelter area and their newborn children had significantly higher lead levels than the controls. During pregnancy, there was a 20% and 15% increase of blood lead concentrations in the smelter area and reference area, respectively. Umbilical cord blood concentrations were significantly correlated with maternal blood lead levels and were 80-87% of the maternal levels recorded at delivery. Blood lead levels in pregnant women were influenced by place of residence, employment at the smelter, smoking, and wine consumption. Because blood lead concentrations increased during pregnancy, despite increased blood volume and unchanged or decreasing environmental lead levels, mobilization of lead from bone during pregnancy was considered a possibility (Lagerkvist *et al.* 1996).

In 1977, maternal and cord blood lead levels were measured in Kiruna and Västerbotten (Zetterlund *et al.* 1977). The mean blood lead concentrations in mother-infant pairs were 61 and 44 μg/L in mothers and infants, respectively, from Kiruna, and 92 and 80 μg/L in mothers and infants, respectively, from Västerbotten. In 1991, the mean blood concentrations in the smelter area in Västerbotten were 32 μg/L in mothers and 28 μg/L in infants (Table 12·28). Current levels in Kiruna are also expected to be lower than 1977 levels.

The use of leaded gasoline decreased by 50% between 1980 and 1991 in Sweden (Jorhem 1994). During that time, the National Food Administration (NFA) monitored the lead levels in blackcurrants grown at different distances from the E4 highway in Umeå (northern Sweden). Results showed a decrease in lead levels with increased distance from the road, as well as a decrease over time. At a distance

of 14 m from the road, the lead levels in blackcurrants decreased from 0.14 mg/kg ww in 1980 to 0.054 mg/kg ww in 1989. At a distance of 134 m from the road, the corresponding values were 0.060 mg/kg ww in 1980 and 0.024 mg/kg ww in 1991.

The decrease in lead levels in blackcurrants is most likely due to the decreased use of organic lead additives in gasoline in Sweden, which decreased from 712 tonnes in 1980 to 380 tonnes in 1991. However, over the same period, the traffic intensity in the area studied increased from 6400 vehicles/24 h to 8000 vehicles/24 h. Since 1986, no sample in the NFA monitoring program has exceeded the 1993 limit value of 0.1 mg/kg.

Biological monitoring of blood lead has also shown a decrease in lead levels over time. Over the two-year study period, a small but significant decrease in blood lead levels was seen in the pregnant women with low blood lead levels in Västerbotten (Lagerkvist *et al.* 1996). Therefore, lead exposure is not likely to be a major health concern in Sweden.

Cadmium. In the Västerbotten study, there were no significant differences in blood cadmium levels between exposed women and controls, with low blood cadmium levels (≤ 1 μg/L) in both groups (Table 12·28). The most important cadmium exposure was a result of smoking. Blood cadmium levels were twice as high in smokers as in non-smokers in both areas. Cadmium levels in the newborns were about 70% of their mothers' level.

The mean daily cadmium intake from the Swedish diet is 10 μg (Slorach *et al.* 1983). An intake of 25 μg/d has been calculated to lead to cadmium-related kidney diseases in 0.1% of the exposed population. An intake of 50 μg/d increases the frequency to 1% (Friberg *et al.* 1986). The risk of kidney damage has been calculated to be two to five times higher in people with low iron stores. Diabetics may also be particularly susceptible to kidney damage from exposure to cadmium (Elinder and Järup 1996). Cadmium exposure is probably lower in northern Sweden than further south.

Selenium. Plasma selenium levels were rather low in Västerbotten, and decreased significantly during pregnancy in both the smelter and the control areas ($p < 0.001$). Selenium levels in newborns were significantly correlated to maternal levels and were 25% lower than their mothers' levels (Table 12·28). Selenium levels were lower in women living in the smelter area than those living in the control area, probably due to different dietary habits.

Arsenic. There were no significant differences in arsenic levels in urine between the two areas, nor were there any differences during the different stages of pregnancy. At week 10 of pregnancy, the geometric mean values were 6.7 and 6.0 μg/g creatinine in the smelter and control areas, respectively. Arsenic levels were comparable to previously reported values in Sweden (Vahter and Lind 1986).

Radionuclides

In January-March 1987, approximately one year after the Chernobyl accident, caesium levels were analyzed in the breast milk of women living in the most contaminated areas of Sweden (NFA 1988). The levels were generally low, 1-5 Bq/L, with one outlier of 11 Bq/L. Whole-body measurements of women who were breast-feeding showed that the caesium levels in breast milk were about 15% of the whole-body level.

Whole-body content of ^{137}Cs was measured in both 1991 and 1992 in the population of northern Sweden, with special interest taken in the Saami population (Johansson *et al.* 1994a, 1994b). Measurements were performed in three areas with different contamination levels, using a whole-body counter installed in a mobile container. Groups of 15-year old students were randomly chosen, one group representing the total population of the area, and the other groups representing members of the Saami communities.

The average whole-body content of ^{137}Cs in the general population varied between 1.1 and 2.0 kBq, and between 3.4 and 25 kBq for the Saami population, dependent on Chernobyl-related contamination level. The ratios between whole-body content and ^{137}Cs deposition found in these measurements were consistent with earlier studies of ^{137}Cs from Chernobyl in other areas of Sweden. However, previously published data based on measurements from historical nuclear weapons test fallout, show a ratio 20-40 times higher. This may be due to the differences in environmental behavior between caesium from Chernobyl and caesium from nuclear weapons test fallout. It may also be due to effective countermeasures following the Chernobyl accident.

Other potential hazards

In 1991, the Swedish NFA recommended that pregnant women restrict their consumption of liver and liver products, except liver paste/paté, because high vitamin A levels had been reported in liver from pigs, cattle and chickens (Ilbäck *et al.* 1991). The levels were especially high in imported beef liver, with levels of more than 40 mg retinol/100 g. Although the NFA considered the risk of congenital malformations to be small with ordinary consumption of Swedish food, the recommendations were issued as a safety precaution until further information was available. The recommended daily intake for pregnant women is 1 mg.

Conclusions

According to recent studies in Sweden in areas south of the Arctic area, metal levels in humans are low and decreasing. An ongoing study in Kiruna will show if the same holds true in the Arctic area. Because exposure to persistent organic compounds may be a problem, the Swedish NFA has issued special recommendations to girls and women of reproductive age concerning the consumption of certain fish. Studies on levels of POPs in human milk and blood are also currently underway in Kiruna.

Table 12·29 outlines ongoing human health related studies in Sweden that are relevant to the AMAP process.

Table 12·29. Ongoing human health related research projects in Sweden of relevance to the AMAP process.

Title	Principal investigator	Project status
Background levels of metals and POPs in humans in Kiruna	B. Jakobsson Lagerkvist, G. Lindström	In progress
Caesium 137 in the population of northern Sweden	G. Ågren	In progress
Ischemic heart disease in Kiruna – Risk factors and sequelae	T. Messner	Finished
Västerbotten County health survey: Human specimen bank	G. Hallmans	In progress
Ischemic heart disease in Norbotten	T. Messner	In progress
Swedish Monitoring Programme in terrestrial biota[a]	T. Odsjö	In progress
Swedish Monitoring Programme in freshwater biota[a]	A. Bignert	In progress

a. The monitoring programs are not directly health related, but may be useful when considering background levels and exposure.

12.5. International study: Data comparison

Introduction

Under the AMAP Monitoring Program, all circumpolar countries agreed to monitor certain contaminants in specific human tissues. During the meeting of the AMAP human health group, held in Copenhagen, September 26-27, 1994, Canada agreed to coordinate and lead a special project for the monitoring of contaminants in maternal blood covering the entire circumpolar region, using a standard sample collection procedure. It was also agreed that all analyses would be conducted by one laboratory to ensure direct comparability of results. Seven of the eight circumpolar jurisdictions have implemented the maternal blood monitoring protocol. Discussions with the United States (Alaska), the only non-participant, are ongoing. Samples from six of the seven countries have been submitted and analyzed by the 'Centre de Toxicologie de Quebec' in Quebec City, Canada. The study was funded by the Canadian Department of Indian Affairs and Northern Development and by Health Canada.

The objective of this project was to obtain sufficient samples during 1995/96 to assess geographical variations of contaminants (organochlorines and heavy metals) throughout the Arctic. Each participating country agreed to send 30 maternal blood samples (plasma and whole blood). Maternal blood was selected instead of cord blood because it could provide sufficient sample volumes for duplicate analyses (one for the international reference laboratory in Quebec, Canada, and one for national laboratories in Denmark and Norway).

Methods

Forty ml of blood were obtained from each mother. Each sample was analyzed for 14 PCB congeners (IUPAC Nos. 28, 52, 99, 101, 105, 118, 128, 138, 153, 156, 170, 180, 183, 187) and 13 pesticides (aldrin, β-hexachlorocyclohexane, *cis*-chlordane, *trans*-chlordane, *cis*-nonachlor, *p,p'*-DDT, *p,p'*-DDE, dieldrin, heptachlor epoxide, hexachlorobenzene, mirex, oxychlordane and *trans*-nonachlor). PCBs and pesticides were measured in plasma using gas chromatography with electronic capture detectors according to the method of Ferron (1994). Lead, cadmium and selenium were measured in whole blood using atomic absorption with a graphite furnace (Stoeppler and Brandt 1980, Parsons and Slavin 1993). Mercury (both total and inorganic) was measured in whole blood samples using flameless atomic absorption according to Ebbstadt *et al.* (1975).

Each participating country obtained patient consent for the sampling and completed a questionnaire for each mother covering, as a minimum, date of sampling, country/region of residence and age of the mother.

Results and discussion

The results presented here are those of the Canadian reference laboratory. The results for 11 of 13 pesticides and PCBs (as Aroclor 1260 and congener-specific) are summarized in Tables 12·30 and 12·31, respectively, for Canada, Greenland, Sweden, Norway, Iceland and Russia (Finnish and Alaskan data were not available as of September 1996). In Table 12·32, the concentrations of PCBs and DDE in umbilical cord blood for Canadian Inuit (from Nunavik), Canadian general population and Greenland Inuit populations are also presented because they were analyzed in the same reference laboratory and can be used for international comparison. Table 12·33 provides data on levels of four metals in maternal blood for Canada, Greenland, Sweden, Norway, Iceland and Russia.

Table 12·30. Maternal plasma concentrations of pesticides (geometric means, μg/kg lipid): Circumpolar study 1994-1996.

	Country[a]					
Pesticide	Canada[b] (n=67)	Greenland[c] (n=117)	Sweden[d] (n=40)	Norway[e] (n=60)	Iceland (n=40)	Russia[f] (n=51)
Aldrin	1.0	1.1	1.0	1.3	1.3	1.8
β-HCH	9.3	18.5	9.2	8.1	32.1	222.5
cis-chlordane	1.0	1.1	1.0	1.3	1.3	1.6
trans-chlordane	1.1	1.3	1.0	1.3	1.3	1.4
cis-nonachlor	6.6	20.9	1.2	1.8	2.7	5.3
p,p'-DDE	133	407	84.0	79.4	113.2	411.9
p,p'-DDT	7.9	15.0	2.4	3.0	4.0	48.3
Hexachlorobenzene	55.1	97.6	15.6	23.1	41.2	62.8
Mirex	4.5	9.1	1.1	1.4	1.9	1.4
Oxychlordane	27.8	60.8	1.9	3.7	6.6	3.3
trans-nonachlor	30.5	110	3.8	6.8	12.2	11.5

a. Finnish and Alaskan data were not available in September 1996.
b. Inuit women from west/central NWT. c. Women from Disko Bay region.
d. Women from Kiruna. e. Women from Hammerfest and Kirkenes.
f. Women from Nikel.

Table 12·31. Maternal plasma concentrations of PCBs (geometric means, μg/kg lipid): Circumpolar study 1994-1996.

	Country[a]					
	Canada[b] (n=67)	Greenland[c] (n=117)	Sweden[d] (n=40)	Norway[e] (n=60)	Iceland (n=40)	Russia[f] (n=51)
PCBs (as Aroclor 1260)	439	1577	606	458	590	570
CB 28	1.4	2.6	2.5	2.9	4.1	3.4
CB 52	1.7	3.8	2.0	1.8	2.2	2.3
CB 99	11.5	29.1	6.4	6.7	9.5	20.8
CB 101	1.8	4.3	1.5	1.4	1.9	2.4
CB 105	2.2	7.3	1.9	2.2	3.7	8.2
CB 118	8.8	33.7	11.4	10.5	16.2	31.3
CB 128	1.1	1.5	1.0	1.3	1.3	1.5
CB 138	29.6	118	47.4	35.1	45.7	49.8
CB 153	54.7	185	69.3	53.0	67.8	59.8
CB 156	5.0	15.4	8.6	6.3	8.0	9.0
CB 170	9.7	34.4	18.6	12.1	16.4	10.0
CB 180	26.6	82.5	34.1	25.3	34.4	20.5
CB 183	2.5	12.5	5.9	3.7	5.2	3.7
CB 187	10.2	41.3	11.0	10.3	13.3	8.1
∑14 PCB congeners	167	571	222	173	230	231

a. Finnish and Alaskan data were not available in September 1996.
b. Inuit women from west/central NWT. c. Women from Disko Bay region.
d. Women from Kiruna. e. Women from Hammerfest and Kirkenes.
f. Women from Nikel.

Table 12·32. Umbilical cord blood concentrations of PCBs and DDE (geometric means, μg/kg lipid): Circumpolar study 1994-1996.

	Canada (Inuit)[a] (n=319)	Canada, general population (Caucasian) (n=502)	Greenland (n=102)
PCBs (as Aroclor 1260)	780	211	1388
PCBs (∑14 congeners)	309	115	504
p,p'-DDE	384	173	424

a. Nunavik (northern Quebec, Canada).

Table 12·33. Concentrations of four elements in maternal blood (geometric means, μg/L whole blood): Circumpolar study 1994-1996.

	Country[a]					
Element	Canada[b] (n=67)	Greenland[c] (n=117)	Sweden[d] (n=40)	Norway[e] (n=60)	Iceland (n=40)	Russia[f] (n=51)
Mercury (total)	3.5	19.8	1.6	2.3[g]	2.9	23
Lead	36.1	51.4	19.7	12.4[i]	16.2	22.8[i]
Cadmium	1.76	1.3	0.11	0.53[i]	0.37	0.12[i]
Selenium	121.6	64.3[h]	79.0	105.8[i]	96.4	106.6[i]

a. Finnish and Alaskan data were not available in September 1996.
b. Inuit women from north-central NWT. c. Women from Disko Bay region; analyses by Deptartment of Arctic Environmental Research, Copenhagen.
d. Women from Kiruna. e. Women from Hammerfest. f. Women from Nikel. g. n=27 for mercury samples only. h. Plasma concentration.
i. Analyses by National Institute of Occupational Health, Oslo.

Comparison of maternal and cord blood contaminant concentrations needs to take account of several important determinants of contaminant levels, i.e., age of mother, number of previous children, number of children breast-fed, length of breast feeding, and type and amount of traditional food consumption. Even though evaluations of these determinants relative to contaminant levels are not yet available, it will be instructive to see how the overall contamination patterns vary among the countries. Evaluation of these data incorporating the above determinations should be completed in the near future.

Persistent organic pollutants

The levels of HCB, mirex and three chlordane metabolites were markedly higher in Greenlandic maternal blood samples than those from the other participating countries (Table 12·30). Levels of DDE in Greenlandic and Russian samples were similar and three to five times higher than levels in the other four countries, however, the DDE/DDT ratio for Russian samples was markedly lower than that for samples from all other countries (cf. Figure 12·16), suggesting current continuing use of DDT.

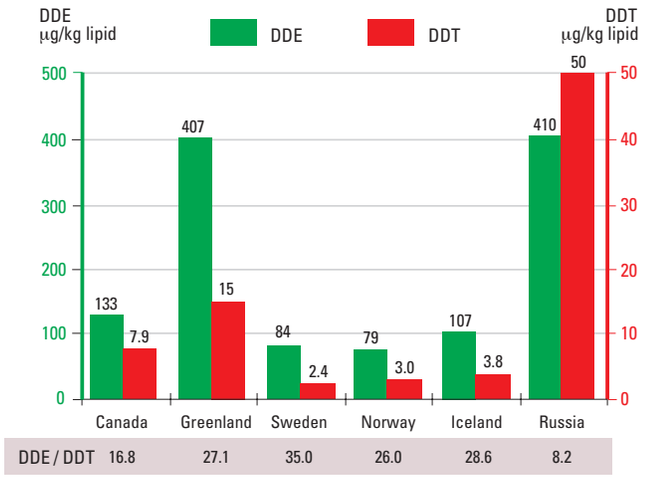


Figure 12·16. Mean maternal plasma lipid concentrations of DDE and DDT; figures below the graph show the DDE/DDT ratios.

Interestingly, β-HCH levels in Russian blood were 8-28 times higher than those in the other countries. In addition, the levels of DDT were 3-20 times higher. These findings appear to suggest that there are either significant uses of both HCH and DDT in the Nikel area, or that there are significant amounts of these pesticides in the food products consumed in this area. These findings are similar to those reported by Polder *et al.* (1996) in human breast milk from the Kola Peninsula.

Pesticide levels in Canadian samples tended to be lower than those from Greenland, but similar in terms of their relative abundance, however, concentrations of *cis*- and *trans*-nonachlor, DDT/DDE, oxychlordane, mirex and hexachlorobenzene were greater than the levels reported in the Swedish, Norwegian and Icelandic samples.

The highest concentrations of PCBs are in the maternal blood samples from Greenland (Table 12·31). This is true both for PCBs measured against an Aroclor 1260 standard and based on a sum of 14 congeners. Maternal samples from other countries contain fairly similar levels of PCBs. A review of the relative amounts of the top seven congeners (153 > 138 > 180 > 187 > 170 = 118 > 99) indicates that the congener patterns are similar in all countries except Russia where there are relatively lesser amounts of congeners 153, 170, 180 and 187 and relatively more of congeners 99 and 118 calculated as a percent of the total amount of PCB (sum of 14 congeners).

With the exception of the β-HCH and DDT/DDE levels found in the Russian samples, the patterns of POPs found in these maternal blood samples are consistent with the relative amounts of traditional food consumed, especially where marine mammals make up a larger amount of the diet. The greater reliance of indigenous people on marine species and the highest concentrations of contaminants in the species consumed (see chapter 5) are found in Greenland, followed by Canada. Mothers sampled in Sweden, Iceland and Norway consumed marine fish species and terrestrial mammals such as reindeer, sheep and cattle, but very few marine mammals. Hence, levels in these countries are very similar and virtually indistinguishable from values found at lower latitudes.

Whole blood values for selected POPs for all six countries, showing data from different populations in Arctic Russia and Canada, are presented in Figure 12·18. Although the whole blood concentration values are lower than those expressed on a lipid weight basis (Table 12·30), the relative amounts and the circumpolar patterns are similar.

Table 12·32 presents recent data on Canadian and Greenland cord blood concentrations for PCBs and *p,p'*-DDE. As in the maternal samples, the highest concentrations are in the cord blood samples from Greenland. The Inuit from Nunavik have intermediate levels of contaminants and the general population of southern Canada (Caucasian) has the lowest levels of contaminants. Samples from other countries were not collected as part of the international comparison exercise.

Congener profiles in cord blood for each of the above population groups are plotted together with the congener profile for maternal blood from Greenland in Figure 12·17. A similar congener profile is seen in all the samples and the same geo-

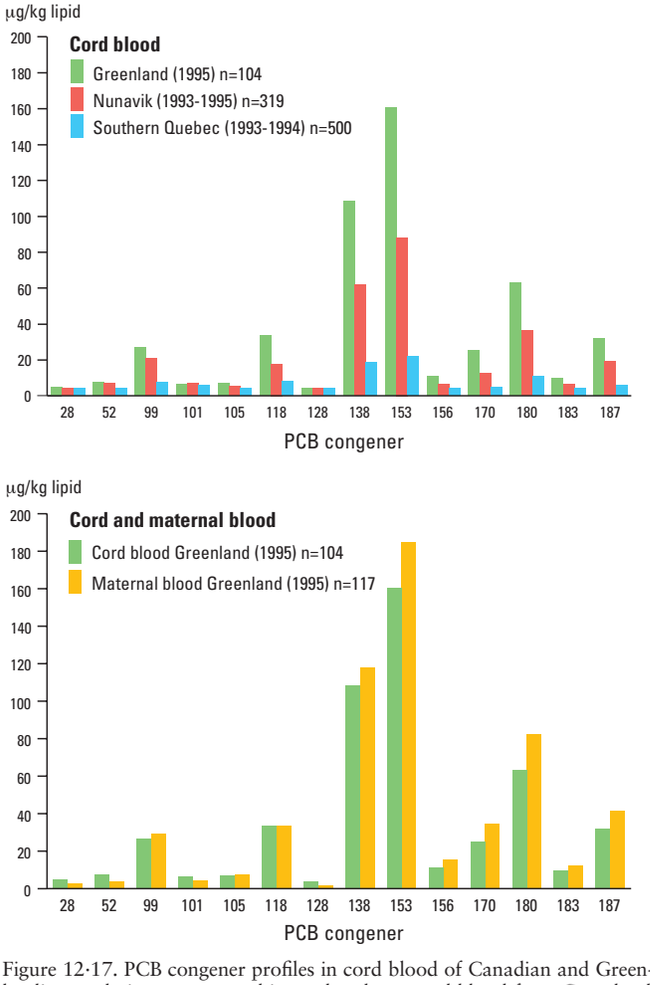
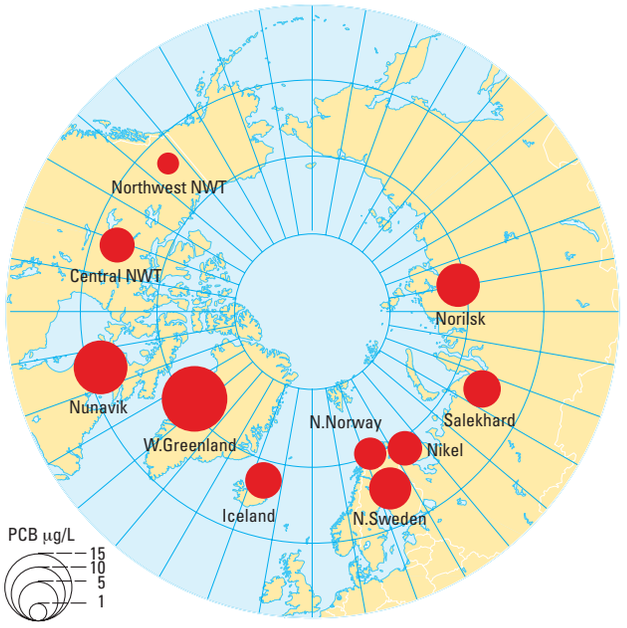
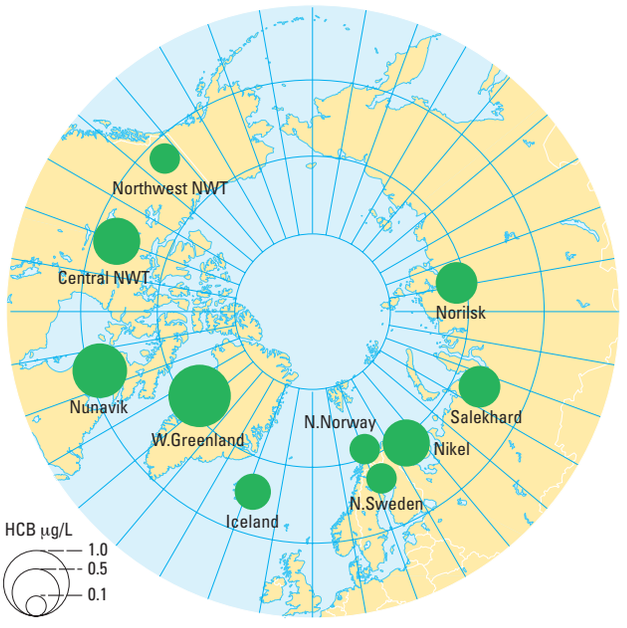


Figure 12·17. PCB congener profiles in cord blood of Canadian and Greenlandic population groups, and in cord and maternal blood from Greenland.





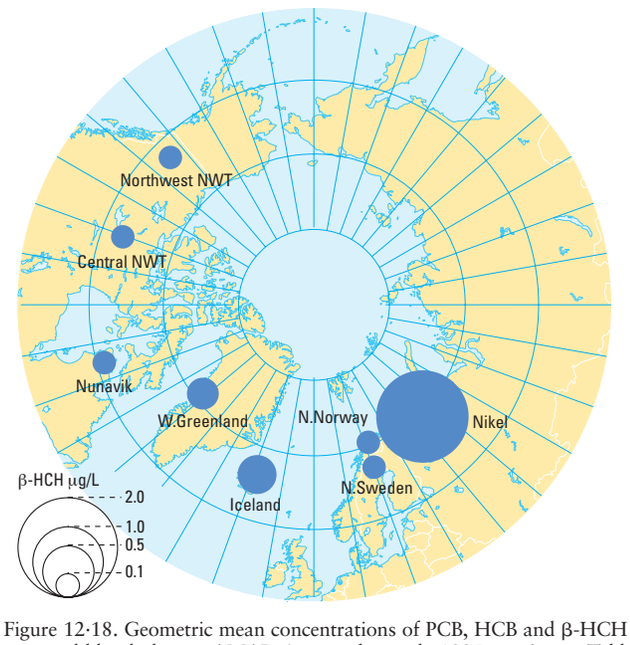
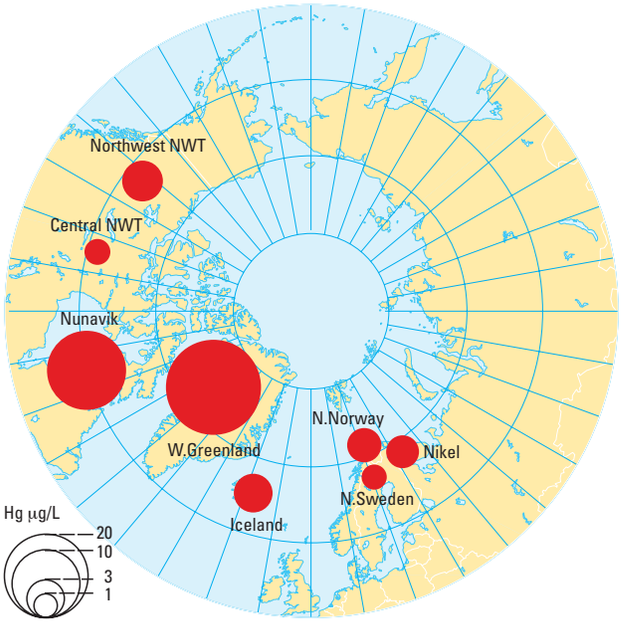
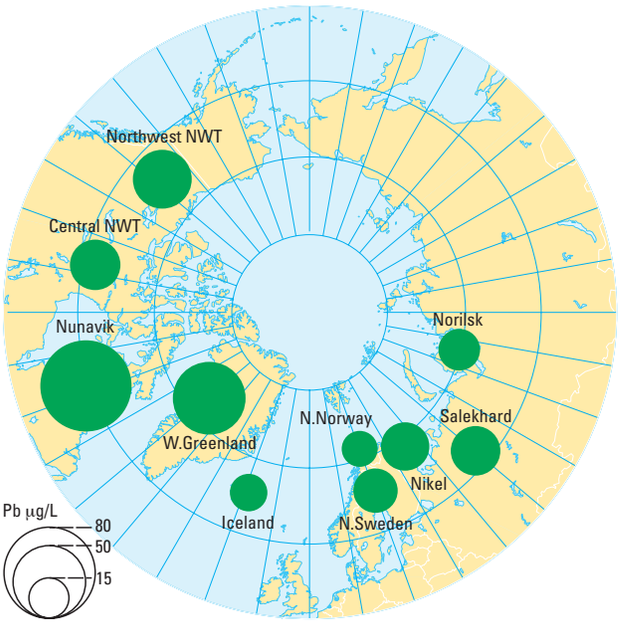


Figure 12·18. Geometric mean concentrations of PCB, HCB and β-HCH in maternal blood plasma; AMAP circumpolar study 1995, see Annex Table 12·A12.





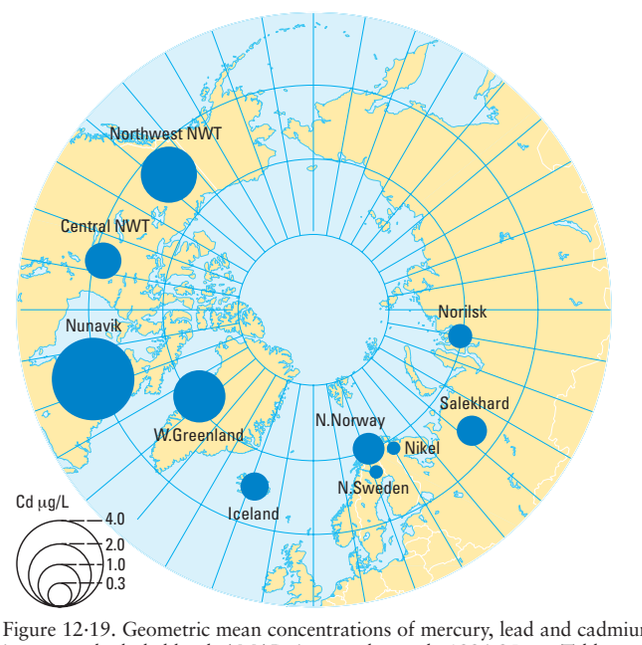


Figure 12·19. Geometric mean concentrations of mercury, lead and cadmium in maternal whole blood; AMAP circumpolar study 1994-95, see Table 12·A13.

graphic/ethnic pattern is seen as in Table 12·32. The concentrations of specific PCB congeners are only slightly higher in the maternal blood samples from Greenland than the Greenland cord blood samples. The predominant maternal and fetal PCB congeners are 153 > 138 > 180 > 187 ≈ 118 ≈ 99.

Metals and essential elements

Mercury levels in maternal blood from the six circumpolar areas sampled are relatively similar, except in Greenland where they are 5-12 times higher than the levels in the other countries. Even though blood mercury levels have declined (see Table 12·11) there is still a significant percentage of samples with values above the 20-50 μg/L risk range used by the WHO for adverse effects on fetal development.

Lead levels are again highest in Greenland, with intermediate levels in Canada, but all are well below the 100 μg/L no-observed-effect-level for protection of the fetus.

Cadmium levels are highest in Canadian and Greenlandic samples. Considering the lower levels in the other countries, and the relatively similar prevalence of smoking, it is likely that considerable amounts of cadmium are consumed in traditional food, especially organ meats, in Canada and Greenland. Levels in Canadian samples may also be affected by use of Canadian tobacco, which is known to have high cadmium content.

Selenium is included in Table 12·33 because it is beneficial for health and found in traditional food. Blood levels are related to traditional food consumption. Adequate levels of selenium are found in maternal samples from all the participating countries. Levels in Greenland are probably higher than shown because the values for Greenland were based on plasma, not whole blood.

Metal concentrations in the circumpolar region are shown in Figure 12·19.

Further assessment of the POPs and metals and essential elements data must await completion of analyses of the demographic data that applies to the samples.

12.6. Risk assessment, management and communication

General considerations

Environmental contaminants regardless of their origin (anthropogenic or natural), pose a potential risk to health. Evaluation of actual risks, and what needs to be done to reduce these risks, is complicated because there are both scientific and social components that must be taken into account (Figure 12·20).

Environmental risk assessment has been defined as the evaluation of 'potential adverse heath effects of human exposures to environmental hazards' (NAS 1983). The process of risk assessment is commonly divided into four major steps: hazard identification, exposure assessment, dose-response assessment, and risk characterization.

Risk management, which is distinct from risk assessment, is the process of weighing policy alternatives and selecting the most appropriate regulatory action that integrates the results of risk assessment with scientific data, and with social, economic, and political concerns (NAS 1983).

Risk communication is the process of informing the population of the risks, benefits and recommendations involved. A more detailed discussion is given by Scala (1991).

Identification of sources of individual contaminants, and the levels of contaminants in air, food and water can be accomplished through environmental monitoring programs. Estimates of human exposure can be derived from human tissue levels or calculated from individual intake of contaminants in food, air, water and consumer products. Estimates of health outcomes are more problematic. Hypotheses can be generated about expected human reactions using laboratory animal studies. These hypotheses have to be verified, when possible, by epidemiological studies of effects and exposure. Occupational exposure studies can be instructive, but occupational exposure levels tend to be far greater than those experienced through the environment, and frequently they concern only one or a very few principle compounds.

Limitations to the risk assessment process include: the uncertainty regarding the similarity of responses between experimental animals and humans; the fact that the epidemiological study approach needs to take into account other supporting information to establish causal relationships; the fact that experimental animal/human studies are often based upon single compound models, while human populations are exposed to a multitude of environmental chemicals and other stressors with possible mutual interactions; and the difficulty of estimating with precision human exposure from all sources. Human populations are generally exposed over several decades (i.e., a typical lifetime) to small doses of many different contaminants, and it is often the case that no single contaminant exposure is sufficient (or even necessary) to produce an adverse health outcome. Specific conditions, such as cancer and cardiovascular diseases, are often discussed in relation to environmental contaminant exposures, but a clear causal relationship to exposure to a single contaminant is seldom identified because the conditions are a result of inter-

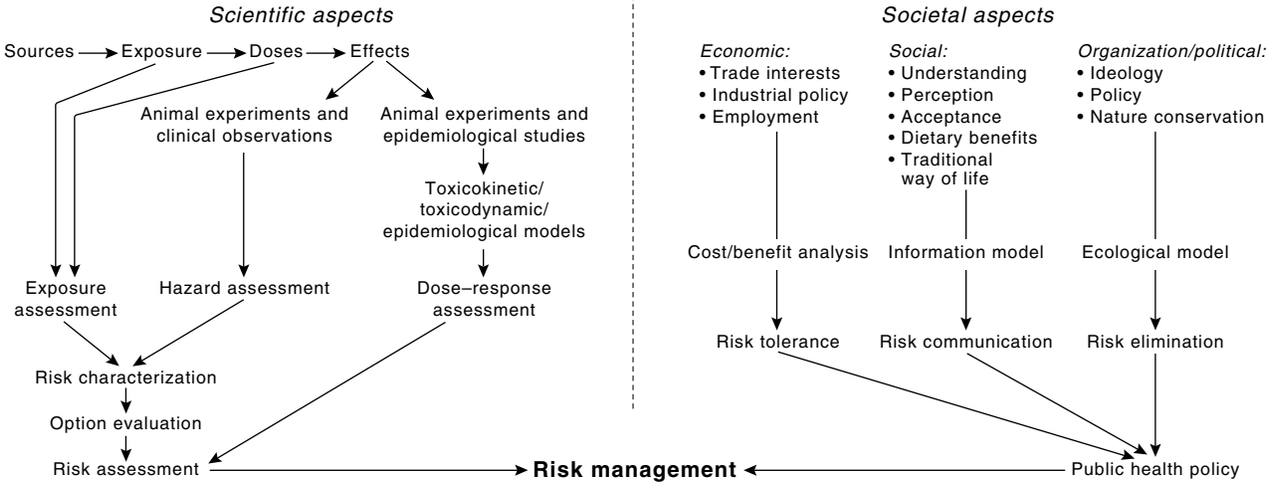


Figure 12·20. Elements involved in the evaluation and handling of risks from environmental contaminants.

action between genetic predispositions and overall environmental influences, i.e., contaminants and modifying factors such as diet, physical condition, age, etc. For these reasons, risk characterization may not represent the true risk, but rather the best estimate of risk based upon the current state of scientific knowledge and the assumptions used by the assessor. Where there is uncertainty in the adequacy of the toxicological or epidemiological data, risk characterization may utilize larger safety margins to develop the tolerable daily intake values. This approach may exaggerate risk relative to benefits. Methodologies for improved risk assessment and characterization following exposure to hazardous substances in the environment have recently been the subject of extensive discussion (Wilson *et al.* 1995).

Environmental risks cannot be expressed in absolute terms and, as a result, they are often vigorously debated. Various societal interests and conditions will influence the way the risks are managed. In many cases, environmental contaminants originate from industrial processes of economic importance to society. Economic consideration (cost/benefit analysis) will, therefore, be an important component in planning reductions in emissions. Imposing regulations on production as a consequence of concentrations of contaminants in foodstuffs may also need to address considerations related to the possible negative effects of loss of earning power on the producers. Risk managers will tend to take a pragmatic approach to the question of acceptability of risk, based upon a threshold of 'tolerable' or 'acceptable' effects which usually are greater than zero. These effects thresholds are compared to exposure concentrations in order to incorporate into legislation 'action levels' of some type for food, air, water and consumer products.

In the general population, workers and consumers are often at the receiving end of risks accepted as tolerable by the decision makers, and it is very important to inform these exposed groups of the risks, benefits and basis for actions taken. This is of special importance in the Arctic, where indigenous peoples often have limited influence on technological development and regulation. Contaminants in the local environments may change the conditions for herding, hunting, and fishing, and while their presence in traditional food items may result in direct health impacts, risk aversive behavior may harm health more than the exposure to the contaminants themselves. These 'risks' to health must be balanced with the health-promoting nutrients in traditional foods, such as n-3 fatty acids, vitamins and trace elements, as well as direct social and cultural benefits. A balance between the positive and negative influences must be achieved.

Risk communication should be carefully developed to convey an accurate and understandable message. People's reaction will depend a great deal on their perceptions of the credibility of the persons providing the information. Informing local populations must, therefore, be done by trained and informed people with a mandate to convey reliable information. In many cases, the news media are the only sources of information available, and they should be assisted in order to present information in its correct context. The success of any communication effort will depend on people's ability to understand the information provided, as well as their perception of the information, which is influenced by traditional, cultural, and religious concepts, and psychological factors. The complexity of providing information to people implies that risk communicators should have local knowledge, be prepared to listen, and accept dialogue as part of the communication process.

Acceptability of individual risk is largely influenced by an individual's perception of the risk. Table 12·34 shows some major factors that influence the level of acceptance. In the case of environmental pollution and exposure to contaminants through food, public acceptance of risks to their health will in general be low.

Table 12·34. Some factors influencing individual risk acceptance.

Acceptability	
High	Low
Risk known and understood	Risk unknown and not understood
Risk undertaken voluntarily	Risk imposed involuntarily
Personal benefit obvious	No personal benefit
Occupational setting	General environment
No alternatives	Alternatives available

A variety of organizations (e.g., those with interests in public health or environmental conservation) may have an impact on decision making. Some lobby groups may call for immediate and complete risk elimination, without adequate regard for hunting and herding traditions of indigenous peoples. Though well intentioned, this unidimensional approach may have a negative impact on cultural patterns of indigenous peoples, i.e., it would favor changes of lifestyle away from the traditional and towards a westernized way of life. A tendency toward westernization may impact not only on culture, but also on public health. Avoidance of traditional foods may actually increase health risks because these foods may protect against cardiovascular diseases, and supply essential, affordable nutrients.

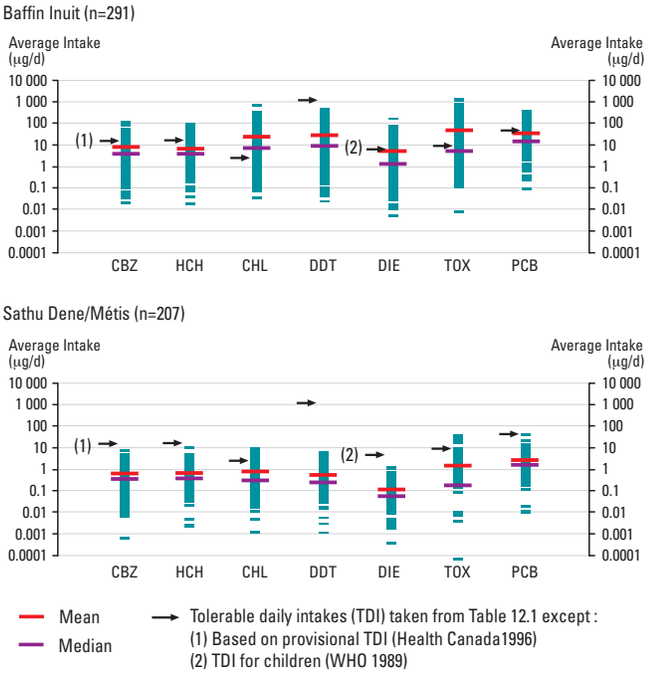
Risk management approaches are by nature complex, and in the Arctic they must strike a balance between well-documented benefits from traditional food and the potential risks from contaminants. In addition, risk management decisions should allow for special consideration of the needs of the indigenous peoples. The AMAP human health program recognizes these issues and aims to produce a set of basic recommendations, which can then be used to develop local or regional recommendations according to specific needs.

Risk assessment of individual contaminants

Persistent organic pollutants

Food consumption survey data can be combined with food contaminant concentrations to develop estimates of human exposure in the Arctic. This task is complicated by the varying levels of contaminants found in the many food items consumed (geographically and seasonally), the different amounts consumed by different ages and genders, and different methods of food preparation. Nevertheless, when these estimated one-day intakes are compared with currently held tolerable daily intakes (TDIs), it is possible to gauge the relative risk, but not the absolute risk, to which people are exposed. Exceedance of TDIs by an individual is of particular concern where that individual continues to exceed the TDIs over a significant period during their life. Much of the data on intakes in the Arctic is only for several one-day exposures to contaminants. This needs to be borne in mind when comparing daily intake estimates with TDIs, otherwise there exists potential for drawing conclusions which may not be justified on the basis of the available information.

Kuhnlein *et al.* (1995a) plotted the range of individual one-day contaminant intakes of POPs for Baffin Island Inuit and Mackenzie Valley Sahtu Dene/Métis (Figure 12·21). Among Inuit, the mean one-day contaminant intake values are higher by an order of magnitude or more for every contaminant shown than among the Sahtu Dene/Métis. This is expected from the different amounts and types of traditional food consumed by these two peoples, i.e., marine mammals and fish for the former and caribou and fish for the latter. These mean contaminant intakes can be compared to current



CBZ : sum of tetra-, penta- and hexachlorobenzene

HCH : sum of α-, β- and γ-hexachlorocyclohexane

CHL : chlordane ; sum of oxychlordane, *cis*- and *trans*-chlordane and heptachlor epoxide

DDT : sum of *p,p'*DDE, *p,p'*DDD, *p,p'*DDT, *o,p'*DDT

DIE : dieldrin

TOX : toxaphene

PCB : sum of congeners.

Figure 12·21. Organochlorine intake from traditional food consumed by indigenous women in the Canadian Arctic (adapted from Kuhnlein *et al.* 1995a).

TDI values (Figure 12·21). For the Inuit group, two of the mean one-day intake values (HCH and PCB) are within a factor of 10 of the TDI. For chlordane and toxaphene, these data indicate that mean one-day intakes exceed the TDI nearly ten-fold. For the Sahtu Dene/Métis, the estimated mean one-day contaminant intakes are well below the TDI values, except for toxaphene and chlordane which are only approximately a factor of 10 below the TDI (Figure 12·21).

Using different methodologies, Dewailly *et al.* (1996c) also estimated contaminant intakes among Nunavik Inuit women. Ayotte (1996) and Receveur (1996) have taken the data presented in Dewailly *et al.* (1996c) and Kuhnlein *et al.* (1995a), respectively, and calculated the proportion of women in the studies whose dietary contaminant intakes exceeded the TDI (Table 12·35). The Inuit in these two studies exceed the TDI much more frequently and to a greater extent than the Dene/Métis. Although the data sets are incomplete, chlordane, toxaphene, mercury and to a lesser extent cadmium and PCB, show the most frequent exceedances of the TDI. These exceedances of some contaminant TDIs by indigenous groups in Arctic Canada are in contrast to more southern Canadian populations where the market diet is considered to have negligible amounts of POPs (Connacher and Mes 1993). In the continental United States, the daily intakes of POPs are usually less than 10% of the acceptable daily intakes (Gunderson 1988).

Table 12·35. Proportion (%) of women whose daily intake exceeds the Tolerable Daily Intake.

	Inuit Baffin Island[a]	Inuit Nunavik[b]	Dene/Métis Mackenzie Valley[a]
CBz[c]	9	6[g]	0
HCH[d]	7	n.a.	0
Chlordane[e]	48	75	6
DDT[f]	0	0[h]	0
Dieldrin	16	n.a.[i]	0
Toxaphene	40	n.a.	2
PCBs (sum of congeners)	16	4	0
Mercury	29	37	n.a.
Cadmium	21	n.a.	n.a.

a. Source: Receveur 1996 (dietary interviews 1987-88). b. Source: Ayotte 1996 (dietary interviews 1992). c. Sum of tetra-, penta-, and hexachlorobenzene. d. Sum of α-, β-, and γ-hexachlorocyclohexane. e. Sum of oxychlordane, *cis*- and *trans*-chlordane, and heptachlor epoxide. f. Sum of *p,p'* DDE, *p,p'* DDD, *p,p'* DDT, and *o,p'* DDT. g. Hexachlorobenzene only. h. DDE only. i. n.a.: not available.

The surveys of the two Inuit populations show quite similar results despite the methodological difficulties in food contaminant intake studies. The possible differences in the proportion of TDI exceedances for chlordane and mercury between the Baffin and the Nunavik Inuit may be due to dietary differences, with one group consuming more beluga whale and ringed seal and the other consuming more walrus, narwhal and ringed seal (Dewailly *et al.* 1996c, Kinloch *et al.* 1992).

Support for these data can be found in the cord blood data on POPs (see section 12.4.1 and Figure 12·3). Since all of the contaminants listed in these studies are persistent, bioaccumulative, and cross the placenta, they can be expected to be present in the blood lipid of newborn babies. All of these contaminants occur at higher concentrations in cord blood of Inuit babies from both Nunavik and the western NWT than from Métis/Dene newborn. They also suggest that PCB and DDE levels in Inuit from the eastern Canadian Arctic are higher than those in Inuit from the western Canadian Arctic. In addition, back-extrapolation from maternal blood collected in Nunavik confirm that for those contaminants with well known biological half-lives and absorption factors, estimated maternal exposures were in the same order of magnitude as those calculated from the dietary intake data shown here (Dewailly, pers. comm.).

Cord blood data are of potential concern in the Arctic. Knowledge of effects of contaminants in animals and humans during fetal development is accumulating and makes a compelling case for reducing population exposures as quickly as possible, and with appropriate sensitivity to the various issues involved. The fetus is vulnerable to the effects of several contaminants, and there is little the mother or fetus can do to reduce exposure during this critical period of cell differentiation and organ development.

Breast milk data comparisons clearly demonstrate that increasing concentrations of all of the persistent organic pollutants reviewed are associated with increasing consumption of traditional food (in particular fish and marine mammals). The concentrations of POPs in some Canadian Inuit breast milk are 2-10 times greater than the levels in breast milk of southern Canadians (Table 12·36). It is worth noting that even though breast milk levels are considerably higher among some indigenous groups in the Arctic than among non-indi-

Table 12·36. Relative increase in breast milk contaminant concentration in Inuit women from Nunavik compared to non-indigenous women from southern Quebec[a].

Contaminant	Increase factor (north/south)
DDE	5.5
Toxaphene	n.a.[c]
Dioxins/furans (dioxin TEQs)	2.0
Dioxins/furans/PCB (dioxin TEQs)	2.4
Mirex	10.0
Chlordane[b]	10.0
Hexachlorocyclohexane	n.a.
PCBs (as Aroclor 1260)	5.6
HCB	4.9

a. See section 12.4.1; data collected in 1989/90. b. comparison between Nunavik (1989/90) and southern Canada (1992). c. n.a.: not available.

genous groups in the south, the levels of at least one contaminant for which there are historical data, DDT, indicate the levels in southern Canadian women in 1967 were three times higher than the levels now being reported in Inuit mothers (Mes 1994). Comparative data for levels of POPs in breast milk of indigenous groups in other Arctic regions are unavailable.

The effects on infants of contaminants taken in during breast feeding are not yet clear (Jacobson *et al.* 1990, 1992, Dewailly *et al.* 1993c, Koopman-Esseboom *et al.* 1994a, 1994b). In some cases in the Canadian Arctic, infants have been exposed to contaminants at levels which are above the TDIs for certain POPs. However, TDIs are calculated for lifetime exposures, whereas exposures through breast milk are limited to the first few months of life. These exposures, averaged over the lifetime of the individual, contribute very little to the total body burden. Given the well-known benefits of breast feeding, it is generally recommended that mothers continue breast feeding unless advised otherwise by their health care provider.

Mercury

Methylmercury poses a significant risk to the health of some peoples in the Arctic, especially in the regions where sea mammals are included in the diet. Large percentages (45-75%) of the Inuit sampled in the eastern Canadian Arctic have levels above 20 μg/L (Table 12·10); this was also found for Cree in the same area. Extensive research by Wheatley and his co-workers (Wheatley and Paradis 1995) indicate the extent and range of the problem. Blood mercury levels range from 1-660 μg/L in the 38 571 indigenous samples examined between 1970 and 1995. Some of the measurements among eastern Canadian Arctic Inuit and Cree were above the 200 μg/L suggested by the WHO as a guidance level (i.e., 5% of the population with levels above 200 μg/L may be at risk of developing neurological paraesthesia). Similar results have been reported by Hansen (1990) and co-workers in Greenland, in particular northern Greenland (Figure 12·8). While neurological signs have been observed clinically in some highly exposed native peoples, it has as yet been impossible to irrefutably diagnose methylmercury poisoning. Some recent data indicates that mercury levels may be decreasing in some Arctic populations.

The WHO has also proposed that there is a 5% risk of neonatal neurological damage associated with peak methylmercury exposures measured at 10-20 μg/g in maternal hair. Using the conversion factors proposed by Clarkson (1992) and Phelps *et al.* (1980), these hair levels can be roughly converted to 30-70 μg/L blood. While all mean values for Dene, Yukon First Nations and non-indigenous reported in Table 12·11 are below this range, 12 out of 15 mean values reported in the same table for Cree and for Inuit from Canada and Greenland are within this range. One recent food intake study of Inuit in the eastern Canadian Arctic supports these findings. Receveur (1996) reports that 29% of the women in the study had daily intakes of mercury that exceeded the WHO TDI (Table 12·35).

Lead

In general, current levels of lead in the Arctic do not pose a significant threat to health and, based on the declining global emissions of lead, are not likely to pose a threat to the health of northerners in the future. It is clear that average adult blood lead levels have declined, that they are only slightly higher in Arctic peoples than in populations in the south, and that these elevations are associated with consumption of large amounts of meat from marine mammals and fish, and with smoking. Mean values for various groups tested in Canada, Russia, Norway, Greenland and Sweden are below 100 μg/L blood and below a level of concern based on WHO blood guidelines for communities (Figure 12·9, Annex Tables 12·12 and 12·A13).

For children, who are most at risk from lead exposure during fetal development and early infant development (quantifiable IQ point reductions at or above lead levels of 200 μg/L blood and reduced gestation periods at or above 50 μg/L blood), there is a necessity to monitor cord blood and infant blood to ensure lead levels do not increase. As the majority of newborns are well below the 100 μg/L action level, they are not considered to be at risk from *in utero* lead exposure. However, lead is a cumulative toxin with exposure continuing during lactation, suggesting that, in the Arctic, biomonitoring should continue during early childhood development.

Cadmium

While traditional food (especially organ meats) is a source of cadmium, the greatest source of human exposure is from tobacco smoking. For smokers, cadmium levels are 20-30 times higher than for non-smokers. These exposures are similar both within and outside of the Arctic, and blood levels suggest that exposure to cadmium through smoking leads to large exceedances of the TDI.

Non-smoking indigenous peoples in the Arctic tend to have similar blood cadmium levels to people living further south (<1 μg/L). Using data from Canada and based on a conversion factor proposed by Louekari *et al.* (1991, 1992), non-smoker intakes in Nunavik Inuit would be in the range of 0.4-0.5 μg/kg bw/d which is half of the PTDI based on the WHO PTWI (Table 12·2). A recent dietary intake study of eastern Arctic Inuit women indicated that 21% consumed amounts of cadmium in diet greater than the current WHO PTDI. These exceedances are not likely to be far greater than the PTDI, but indicate further blood monitoring is needed in the Baffin region (Receveur 1996).

In an evaluation of cadmium exposure of northern Quebec Cree, it was estimated that regular consumption of moose and caribou kidney and liver would result in a cadmium intake near the WHO PTDI of 1 μg/kg bw/d. Cadmium exposure from smoking one pack of cigarettes per day results in a cadmium intake of a similar order of magnitude (Archibald and Kosatsky 1991). It was recommended to the Cree Regional Authority that no consumption restriction be applied but urinary cadmium biomonitoring be implemented. Traditional food use, where cadmium is the contaminant of concern, need not be altered if smoking habits can be modified.

Cadmium levels in caribou kidney tend to be highest in the Canadian Arctic in the Yukon herds and those between Great Slave Lake and Hudson Bay. It is also in these areas that caribou consumption is greatest. Risk assessments have been completed for various age ranges of caribou and endorsed by community leaders. Caribou kidney is a healthy food and significant amounts can be consumed without any concern for cadmium-related effects. More important for the protection of health from the point of view of exposure, is a reduction in tobacco smoking which leads to exceedances of the TDI without any contribution of cadmium from traditional foods whatsoever.

Cadmium levels reported in recent blood samples from the entire circumpolar region are generally similar, suggesting that exposures are relatively similar and from common sources, i.e., food and tobacco smoke. The levels reported in smokers could lead to some mild kidney damage.

The fetus is generally protected from exposure to cadmium because the placenta acts as a partial barrier.

Arsenic
Exposure to arsenic compounds in the Arctic comes from consumption of food of marine origin (organic arsenicals) and drinking water (inorganic arsenic). The only health risk seems to be associated with drinking water which is locally contaminated with inorganic arsenic.

Nickel
In communities where a significant proportion of the population is employed in nickel refining and comparable industries, environmental health assessments must take account of preexisting occupational disease conditions (such as reproductive or developmental impairments and cancer). Nickel contact dermatitis is a disease which may be exacerbated by intake of nickel in the diet and in drinking water.

Radionuclides
Amounts of radionuclides in the Arctic environment are generally about the same as, or even lower than levels found in the temperate zone. The two radiocaesium isotopes (^{137}Cs and ^{134}Cs) are considered to be the artificial radionuclides of greatest concern in the Arctic environment. In Canada, levels of radiocaesium have decreased from about 450 Bq/kg in 1965 to roughly 10 Bq/kg in 1990 due to the cessation of atmospheric nuclear testing. Of all radionuclides, natural and artificial, the natural radionuclides ^{210}Pb and ^{210}Po make the greatest contribution to human radiation doses in the Arctic. A recent study of caribou hunters in the Canadian Arctic indicated that they may be receiving up to 10 mSv per year from ingested ^{210}Po compared to a normal background of about 2 mSv.

If we assume (as an upper limit) that about one-half million Arctic residents depend on hunting, fishing and reindeer herding and that the highest average exposure is 11 mSv, then the collective radiation dose is 5500 person-Sieverts. With a cancer risk of 0.05 cases per Sievert of exposure, one would expect 275 radiation-induced cancers. This would be indistinguishable against a background of over 100 000 fatal cancers that would normally be expected to occur in a population of this size.

12.7. Conclusions and recommendations

12.7.1. Conclusions

General conclusions

1. Contaminants of anthropogenic origin, such as POPs, metals and radionuclides, are spread globally, and contaminate the Arctic. The combination of environmental conditions and biomagnification in the marine and freshwater aquatic food webs results in accumulation of certain persistent contaminants in local food at levels which are often in excess of contaminant concentrations in the mid-latitudes where these contaminants originate. Consumed fish, marine mammals, terrestrial mammals, and birds are the major sources of human exposure to environmental contaminants in the Arctic and, as a consequence, several Arctic population groups are much more highly exposed through the diet than most populations in the developed world.

2. The social, cultural, spiritual and physical health of Arctic indigenous peoples depends on the collection and consumption of country foods. A diet based on traditional foods is of high nutritional benefit. Consumption of n-3 fatty acids found in marine mammals and fish has been suggested as the component responsible for the lower incidence of cardiovascular disease in Alaska, Greenland, Canada and Japan. However, other nutrients may also influence the rate of cardiovascular disease. The consumption of local fish, meat, wild greens and berries provide the necessary dietary intake of most vitamins, essential elements and minerals. When market foods are purchased to supplement current country food diets, they should be selected for their nutrient quality. It is unlikely that market foods currently available to most Arctic indigenous populations can provide the nutritional equivalent of traditional food.

3. The influence of contaminants on fetal and neonatal development is of special concern. Preliminary results indicate that POP and methylmercury concentrations are two- to ten-fold higher in breast milk and cord blood in some Arctic areas than in breast milk and cord blood from regions south of the Arctic. The fetus and the neonate are very vulnerable to the effects of many of these contaminants during this critical period of development.

POPs

4. There is both scientific and public concern about the possible adverse effects of POPs on pregnancy outcome, fetal development, child development, reproduction, male and female fertility, and the immune system. Several of these effects may be mediated through endocrine disrupting properties of some POPs. DDT and its metabolites and some dioxin and PCB congeners have been implicated.

5. Despite the number of controls on several POPs imposed during the 1970s and 1980s, there is no evidence that levels in Arctic peoples have decreased. The persistence of POPs, their presence throughout the ecosystem, and the continued use of some POPs for disease vector control, all contribute to the constant influx of POPs into the arctic environment and sustained levels of human exposure. There is very little information on temporal trends of POPs in Arctic populations. Monitoring of POPs in blood over the next decade is essential to establish whether or not risk management strategies for POPs are effective.

6. Elevated levels of toxaphene and chlordane, coupled with current intake scenarios, suggest some indigenous groups are exposed to levels of these contaminants significantly above the Tolerable Daily Intake (TDI). Information on the levels of toxaphene in human tissues is limited.

7. There is insufficient information to conclude whether the TDI for dioxins and furans and dioxin-like PCBs is being exceeded in Arctic populations. Also, there is as yet little conclusive scientific information directly linking harmful human effects to low levels of exposure to these contaminants.

8. While current levels of exposure to POPs in the Arctic are unacceptable, it is not always clear what public health measures should be taken to reduce the exposure of Arctic populations who rely on traditional foods for spiritual, cultural, physical, and nutritional benefits. Decision-making would be greatly aided by studies of the interactive effects of current levels of mixtures of POPs found in the traditional food supply. In the interim, the risks associated with a shift in dietary preference need to be considered along with the risks associated with the presence of contaminants in Arctic wildlife consumed as traditional food. Weighing the uncertainty in some of the TDI values (e.g., toxaphene) against the benefits of traditional food gathering and consumption, it has been recommended in most Arctic jurisdictions that consumption continue.

9. The concentrations of some POPs in breast milk have raised justifiable concern among mothers in the Arctic. The health benefits to newborns of breast feeding are substantial,

e.g., mother-child bonding, immunological benefits transferred from mother to child, nutritional value, and reduced risk of bacterial contamination from poorly prepared formulas. Breast feeding should continue since the benefits of breast feeding outweigh the currently known risks attributed to infant exposure to contaminants through breast milk.

10. Existing epidemiological evidence on the adverse effects of POPs in humans is inconclusive and needs to be replicated because of the specific context in the Arctic in which there are differences in genetics, climate, food consumption patterns, and lifestyle among population groups.

Heavy metals

12. Existing data from the literature do not allow a valid estimate of spatial and temporal trends of exposure of Arctic peoples to mercury and cadmium, while for lead, a declining trend is observed. There is some evidence that the general decline in lead exposure parallels the decline in lead levels in industrialized areas.

13. The high exposure of indigenous peoples to methylmercury in some Arctic areas is a matter of concern because of its neurotoxic effects on the fetus. Further investigation of both the levels and the influence of mercury on fetal development is warranted.

14. Lead levels in Arctic indigenous peoples have declined since the implementation of controls on lead emissions. Concentrations of lead in blood currently reported are below a level of concern, however, continued monitoring is warranted because of the potent effects of lead on neurological development in the fetus and children.

15. As elsewhere in the world, cadmium intake in the Arctic is mostly through smoking. However, as with mercury, the dietary exposure level will vary according to choice of food. Recent research indicates that kidney tubular dysfunction may occur at lower levels than previously considered.

16. Arsenic and nickel exposures are mainly related to local industrial activities. The impacts on health of organo-arsenicals in marine food is at present not well understood. Studies of speciation of the naturally-occurring arsenicals in various animals and organs and their potential interactions with essential trace elements are needed.

Essential elements

17. In general, ample supplies of selenium are provided through the diet in populations depending on marine food. Muktuk (whale skin) is the richest source. Populations predominately depending on food of terrestrial origin may have a marginal deficit in selenium supply. While animal experiments indicate selenium protects against the effects of some heavy metals (i.e., mercury, lead, and cadmium), its role as an antidote for metal toxicity in humans is at present hypothetical. The role of selenium as a protective factor against lipoprotein peroxidation and its purported beneficial effects in protecting against cancer and cardiovascular disease remain to be elucidated.

Radiation

18. The level of human exposure to anthropogenic radionuclides such as radiocaesium in the Arctic has declined dramatically since the cessation of above-ground weapons testing. However, some Arctic populations still have higher exposures from anthropogenic radionuclides in the environment than those in the temperate zone because of unique features of Arctic terrestrial and freshwater ecosystems and the people's use of traditional foods. The cumulative dose to which a population is exposed must also include an estimate of the natural radionuclides. In some geographical areas, levels of natural radionuclides (derived from, e.g., radiolead and radiopolonium) have resulted in certain indigenous peoples being exposed to higher levels of radiation than the general population.

19. Increased UV radiation due to ozone depletion is not a major concern in terms of skin cancer because of the amount of clothing worn in Arctic environments. The primary health concern is the reflection of UV from snow and ice causing snow blindness. In addition, there is growing concern about the development of cataracts.

Estimates of exposure and effects

20. Food is the major exposure route for contaminants in the Arctic. The type and amount of human exposure to environmental contaminants varies throughout the circumpolar region according to the level of contaminants in the food, the amount and type of food consumed, and the method of food preparation. For these reasons, wildlife monitoring data provide a very uncertain basis for precise human exposure estimates. However, they are of great value for risk characterization as they identify the contaminants present in wildlife used as traditional food, and the most contaminated species, and can, therefore, contribute to the basis for dietary recommendations. Uniform methodology for dietary intake studies applied across the circumpolar region would greatly assist risk assessment.

21. Determining adverse health effects in human populations due to the presence of contaminants in traditional foods and human milk is extremely difficult for methodological and ethical reasons. Results are also difficult to interpret because of a wide range of confounding factors (socio-economic, lifestyle and gender/age related). Monitoring contaminant concentrations in human tissues and using these data to estimate exposures will continue to be necessary as will a general reliance on animal studies of the effects of mixtures of contaminants and nutrients found in Arctic foods. Tissue banks would greatly assist the requirements for retrospective comparative studies of contaminant levels and effects.

22. Risk assessments are, in general, conducted for individual contaminants and not mixtures of contaminants. They are based upon extrapolations from single compound animal studies combined, when possible, with data from occupational exposure or accidental intoxication events. These assessments do not adequately account for the metabolic transformations of contaminants in the food chain, the possible interactions between contaminants concomitantly present in the environment, or the modifying influences of nutrients, such as trace elements and antioxidants, naturally present in Arctic traditional food. Consequently, the accepted guidelines for exposure are not necessarily applicable to Arctic communities.

23. Very few studies of the effects of environmental contaminants on Arctic populations have been completed, therefore, existing literature does not provide convincing evidence of adverse health effects. On the other hand, the health-promoting effects of traditional diets have been well documented. This has lead to a reluctance to recommend changes to current patterns of traditional food consumption. Based on this review, there may be a need to consider providing food consumption advice to some people in some areas in order to protect the fetus from exposure to mercury and some POPs. It would be prudent for local health care providers to consider giving dietary advice to young women and pregnant

women in order to help them reduce exposure levels prior to and during pregnancy. The consumption of less contaminated traditional food items that provide the nutritional needs of women of child-bearing age should be promoted.

24. Contamination of the Arctic is part of a global process. While human exposures in the Arctic can be moderately reduced with some dietary modifications (provided these are culturally, socially and nutritionally suitable for the specific communities involved), it must be recognized that long-term exposure reductions can only be accomplished through international conventions resulting in bans and restrictions on production and use of the most toxic chemicals.

12.7.2. Recommendations

On the scientific assessment level, to:

1. Continue monitoring contaminants in human blood and tissues in order to reveal temporal and spatial trends.

2. Combine experiences from the rapidly expanding disciplines of biomarker research and molecular epidemiology with existing monitoring programs.

3. Develop uniform methods and initiate studies which will allow objective dietary assessments and exposure estimations.

4. Complete human population studies of contaminant-related effects on reproduction and fetal and child development.

5. Revise existing guidelines for tolerable intakes of contaminants based upon studies of interactions among individual contaminants present in Arctic foods, and between contaminants and nutrients.

6. Create and manage tissue/specimen banks in the circumpolar region.

On the public health policy level, to:

7. Apply communication and consultation approaches that enhance the development of local information and advice for indigenous peoples about contaminant exposures and effects.

8. Advise Arctic peoples to continue to eat traditional food and to breast feed their children, and develop dietary advice for girls, women of child-bearing age and pregnant women which promotes the use of less contaminated food items while maintaining nutritional benefits.

On the national and international policy level, to:

9. Initiate measures to reduce and control regional industrial emissions of contaminants.

10. Strengthen international efforts to control production and use of persistent organic pollutants.

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Annex

Contents