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Mercury Contamination in Arctic Canada: Possible Implications for Aboriginal Health

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Abstract

Methylmercury is a potent neurotoxin found at elevated concentrations in both the Arctic ecosystem and tissues of the local Aboriginal inhabitants. Combined studies of ecological contamination with the possible implications for human health, have made this one of the largest environmental research projects in Canadian history. Recent scientific advances have revolutionized the understanding of the global mercury cycle. The major source of mercury exposure is through the consumption of locally derived food sources. Mercury tissue concentrations are reaching alarming levels in some Aboriginal communities. Studies on both animals and humans have provided compelling evidence suggesting that methylmercury contamination induces neurological defects. Cognitive defects have been noted in children exposed congenitally in several other seafood-consuming communities around the world. Defects in motor function have been observed in both adults and children in Inuit Communities. Furthermore, environmental mercury has been linked to both autism and Alzheimer disease. Aboriginals are currently exposed to methylmercury in addition to several other environmental toxins. This may have serious repercussions for neurodevelopment and health in this population.

Mercury and the Environment of Arctic Canada

The Northern Contaminants Program (NCP)

Approximately 7.5% of Canada's aboriginal population inhabits the Arctic region in the northern part of the country, where they comprise just over half of the combined population (Statistics Canada, 2001). The lives of these

56,000 people are linked to the local environment, particularly through the consumption of traditional foods (Van Oostdam et al, 2005). Mercury contamination as a possible health concern was initially raised in the early 1970s, following contamination of fish due to effluents from chlor-alkali plants in northern Ontario. Similar concerns were later recapitulated in several other communities (CACAR, 2003). Moreover, early studies in the mid-80s indicated that the Arctic ecosystem harbored unusually high levels of contaminants such as persistent organic pollutants, radionucleotides, and heavy metals including mercury (Wong, 1986). In response to such concerns, the Department of Indian Affairs and Northern Development established the Northern Contaminants Program (NCP) in partnership with Federal and Territorial Departments, Aboriginal organizations, and University researchers (CACAR, 2003). Since its conception in 1991 the NCP has focused on determining the levels and sources of contaminants in the Arctic, and assessing the possible impacts and risks towards human health.

The Mercury Cycle in the Arctic Ecosystem

The initiatives of the NCP, in conjunction with the development of more sophisticated instrumentation, have lead to a scientific revolution in our understanding of the global mercury cycle (CACAR, 2003). Mercury exists in three states: elemental mercury, inorganic mercury salts, and organic mercury. In aquatic environments, inorganic mercury is converted to the more toxic organic state, otherwise known as methylmercury (MeHg). Methylmercury is found at elevated concentrations in the tissues of aquatic animals in the Arctic (Lockhart & Evans, 2000; Wagemann et al., 1995). When a contaminant enters the food web, it is passed on from prey to predator, and in the process successively increases in concentration. Animals higher up the food web are therefore at a higher risk for exposure. This process is known as biomagnification or bioaccumulation. Once it enters the food chain, mercury is biomagnified as methylmercury (Atwell et al., 1995), and results in global human exposure primarily through the consumption of contaminated fish (WHO, 1990).

Although the process of biomagnification enhances exposure levels, it is the physiochemical reactions of mercury in the air and water that ultimately determine the amount that enters the food web. Natural sources of mercury from local rocks and soils have remained steady for decades, while human-made, or anthropogenic sources are on the rise (CACAR, 2003). Anthropogenic emissions from fossil fuel consumption, waste incineration, chlor-alkali plants and metal smelting and processing release elemental

mercury in a gaseous state into the atmosphere (Pacyna & Keeler, 1995). Once in the atmosphere, gaseous mercury is capable of long-range transport in air currents (Schroeder and Munthe, 1998), which can reach isolated environments such as the Arctic from industrial regions such as Europe, Asia, and North America.

Canadian researchers have recently characterized a staggering discovery known as atmospheric mercury depletion events (MDEs) at Alert, Nunavut and Kuujjuarapik, Quebec (Schroeder et al., 1998; 1999a). During the polar sunrise in the spring after approximately five months of darkness, atmospheric mercury levels drop drastically. During this sudden exposure to solar radiation, atmospheric mercury is converted into a more reactive, oxidized form (Schroeder et al., 1999b), which deposits more easily in the snow (Schroeder et al., 2000). The occurrence of MDEs in the springtime correlates with the preparation of plants and animals for peak summertime activity possibly enhancing exposure. Approximately 60% of the mercury that reaches lakes and rivers flows out and 25% falls to the bottom and, and current data suggests that, at least in some areas, the levels of mercury in lake sediments are increasing (CACAR, 2003).

Mercury Exposure Levels

Aboriginal Perspectives on Food

> *For us to be fully healthy, we must have our foods, recognizing the benefits they bring. Contaminants do not affect our souls. Avoiding our food from fear does.* (Egede, 1995)

Exposure to mercury in Aboriginal communities occurs primarily through the consumption of traditional country foods (Van Oostdam, 2005). Country food refers to mammals, waterfowl/ seabirds, fish, and vegetation harvested from the local flora and fauna. The attitudes towards the collection, consumption, and trade of traditional food are different to those in Western life. Food is an integral part of the community, with social, cultural, economic and spiritual ramifications (Wheatly, 1996). Data from 1,721 interviews collected from five Inuit areas illustrated that traditional food is perceived to provide cultural and economic benefits in addition to basic nutrition (Kuhnlein et al., 2000). Cultural aspects aside, it would cost approximately 55 million dollars to purchase equivalent amounts of imported food, which is well above the 10,000 dollar aboriginal average household income (Usher & Wenzel, 1989). Despite the date of the previously

mentioned study, it illustrates that a major economic drawback would be associated with avoiding locally derived food sources. It is therefore apparent that exposure to contaminants such as mercury can be potentially confounded by socio-demographic, economic, and cultural factors.

Intake Levels and Guidelines

The major global source of MeHg exposure is through the consumption of contaminated fish (WHO, 1990). In aboriginal communities, exposure may also arise through the consumption of other local animals such as seals, polar bears, narwhal muktuk, and caribou (Kuhnlein et al., 2000). Metals such as mercury accumulate mostly in the internal organs of animals such as the liver and kidney (Chan et al., 1995). The WHO has specified a guideline for the provisional tolerable daily intake (pTDI) for total mercury (0.71μg/kg/day) and methylmercury (0.47μg/kg/day) (WHO, 1978). Health Canada has also issued a methylmercury pTDI of 0.2μg/kg/day for children and women of childbearing age (Health Canada, 1998). The U.S Environmental Protection Agency has established a MeHg dose of 0.1μg/kg/day for pregnant mothers (U.S. Environmental Protection Agency, 1997).

Mercury intake levels for various populations have been compiled based on a comparison of dietary surveys with the known mercury content of various foods. Care should be taken in interpreting these values since much of the available data is based on total mercury levels, not the more toxic organic form. This has important implications as certain species such as fish contain mostly MeHg while sea mammals contain mostly inorganic mercury (Wagemann, 1997). Furthermore, recent reconstructions of MeHg intakes using mathematical models based on biomarkers have illustrated that dietary surveys may have overestimated intake values (Gosselin et al., 2005). Finally, it should be understood that the data represent an average for the population. As in any statistical distribution, a few individuals may be exceeding the mean value by a large magnitude. Data from dietary surveys indicate that the Inuit have the highest intakes of mercury with levels close to the pTDI (Kuhnlein, 2001), while other groups have intake levels well below the pTDI. The data suggests that Inuit children and women of childbearing age may be exceeding the pTDI.

Tissue Levels and Guidelines

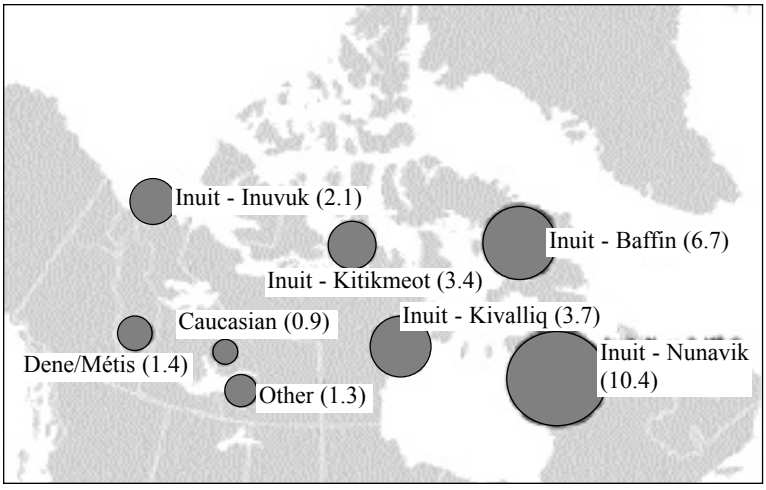
The Medical Services Branch of Health Canada, the First Nations and Inuit Health Branch (FNIHB), the Cree Board of Health and Social Services, and the Government of the Northwest Territories (GNWT) have accumulated a

wealth of data on tissue mercury levels. The combined efforts of these and other agencies have made this one the largest contamination research projects in Canadian history.

There are two commonly used biomarkers to assess mercury tissue levels: mercury levels in hair and mercury levels in maternal/umbilical cord blood. Health Canada has issued ranged guidelines for methylmercury blood levels (Health Canada, 1979). Levels below 20µg/L are acceptable, while those between 20 and 100µg/L are at increasing risk, and above 100µg/L considered at risk. More recently, the USA issued a benchmark level of 58µg/L and a recommended maternal level of 5.8µg/L (NRC, 2000).

The blood levels mirror intake levels, in that Inuit mothers are exceeding both Canadian and US guidelines, while no Caucasian, Dene, or Metis mothers exceed the lower guideline of 5.8µg/L (Butler & Walker, 2005). Within Inuit regions, Nunavik appears to have the highest proportion of mothers exceeding recommended guidelines (Figure.1). It should be noted that mercury concentrates on the fetal side of the placental circulation so umbilical cord levels would be 1.5 to 1.8 times higher than in maternal blood (Van Oostdam et al., 2005). The general historical trend is that the percentage of Inuit mothers exceeding blood guidelines is on the decline (Van Oostdam, 2005). However, issues regarding the statistical sampling of historical accounts have been raised (Van Oostdam, 1999).

Figure 1. Maternal contaminant levels in Arctic Canada: Total mercury (µg/L plasma)



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Mercury and the Nervous System

Toxicity of Mercury

Once ingested, 90% of MeHg is absorbed across the gastrointestinal tract. Once in the blood stream it can easily cross the normally protective blood brain barrier, due to its lipophilic nature (Mendola et al., 2002). It can be transferred from mother to fetus via the placenta (Kajiwara et al., 1996), and to infants through lactation (Sakamoto et al., 2002). Methylmercury is a neurotoxin that can induce severe, irreversible damage to the central nervous system (Philbert et al., 2000). Although the mechanisms remain to be fully elucidated, it appears that a major neurotoxic effect involves oxidative stress through the increased production of reactive oxygen species (ROS). Methylmercury also alters cell proliferation, differentiation, and migration (Mendola et al., 2002). These processes are crucial to the well-orchestrated and highly organized process of brain development.

A number of factors can potentially modulate the effect produced a neurotoxic agent such as MeHg. One obvious factor is dose, or the concentration that the nervous system is subjected to. Another would be the extent of exposure time. When dealing with low-level exposures, however, there may be more subtle factors involved. One such factor is the time point of exposure during development. Brain development proceeds in a very tightly regulated, highly organized pattern. Slight perturbations in this process may have profound consequences for the immature brain. On the other hand, a fully mature brain may be less vulnerable. Consequently, low-level exposure may induce different defects in prenatal, neonatal, adolescent, or adult nervous systems. Furthermore, subtle defects early in CNS development may not become apparent until relatively late stages of life. This is known as the Barker Hypothesis, which postulates that, certain parameters in early life, such as low birth weight or small head circumference induced by malnutrition, are indicators for disease development in later life (Osmond & Barker, 2000). In 2003, this hypothesis was expanded to include environmental toxins and brain development (Landrigan et al., 2005). This hypothesis is similar to that of "Silent Damage" (Weis & Reuhl, 1994). It has been postulated that the early exposures to neurotoxic chemicals reduces the number of neurons in critical brain areas, which becomes magnified later in life due to the aging process. This for example, includes a well-documented correlation between early life exposures to pesticides and Parkinson disease (Landrigan et al., 2005).

Acute Poisoning

The horrific neurotoxic effects of high level MeHg exposure are well characterized through catastrophic events of mass poisoning. On two separate occasions in Japan, fish become contaminated with MeHg from local industrial discharge (Tsubaki, 1977). The first event occurred in the 1950s at Minamata Bay, and resulted in severe developmental defects including cerebral palsy, microcephaly, blindness, and seizures in children exposed during pregnancy (Goto, 2000). This has been dubbed Congenital Minamata Disease. A similar episode occurred in Niigata, Japan in the 1960s. Based on lessons learned in Minamata, abortion was recommended for pregnant mothers exhibiting high hair mercury levels (Tsubaki, 1977). In exposed adults, the primary effect seems to be a targeted loss of neurons in areas of the brain involved in vision, motor function, as well as the disruption of sensory nerves (Reviewed in Castoldi et al., 2001). Consequently, acute exposure during adulthood induced defects such as hearing loss, muscle weakness, mental deterioration, and visual abnormalities. In 1971, accidental consumption of seed grains treated with a mercury containing fungicide resulted in hundreds of deaths in Iraq, with thousands becoming clinically ill. Children exposed during pregnancy exhibited higher frequencies of mental retardation, blindness, seizures, and other neurological defects (Marsh et al., 1987). Three major conclusions can be drawn from these unfortunate events. Firstly, high levels of MeHg can have a devastating effect on both the developing and mature nervous systems. Secondly, acute poisoning induces damage to several brain areas, resulting in a broad spectrum of clinical manifestations. Thirdly, exposures of the developing fetus to MeHg results in more severe neurological defects than exposures in later life.

Low Level Exposure

Animal Studies

As mentioned above, the acute neurotoxic effects of MeHg have been well documented. The effects of chronic low-level exposure are somewhat more controversial. Animal models have several advantages over human studies, the most important of which being that it permits testing in a controlled environment. This facilitates data interpretation by reducing the potential confounding effects of environmental factors such as diet or genetic variations. The results from animal models are mixed (Reviewed in Rice, 1996). The major drawback being difficulty in distinguishing between

defects in sensory/motor functions from defects in cognition, or "intelligence". Rodents and monkeys exposed developmentally to MeHg seem to display difficulty in performing simple tasks. This difficulty appears to be more related to sensory or motor deficits rather than a direct defect in cognitive aptitude. In one series of experiments, Macaque monkeys exposed to MeHg during pregnancy displayed infantile alterations in visual recognition tasks that are believed to assess cognitive function. However, this same group of monkeys was not impaired on similar tests later in life. In a separate series experiments no cognitive deficits were found in monkeys exposed, but visual and somatosensory defects were recorded. In one study, five monkeys were dosed from birth to seven years of age with 50µg/kg/day of MeHg (Rice, 1998). These monkeys displayed defects in auditory, visual, and somatosensory function at age 20 years. The monkeys were then tested for defects in speed of information processing, which is highly correlated with IQ in humans. A button pushing test in response to a visual stimulus experiment was set up to distinguish between reaction time (information processing) and motor time (speed of movement). Since no significant difference was observed between the reaction times in the experimental and control groups, and the authors concluded that cognitive defects were not impaired. Care should be exercised in interpreting these results since which is that five monkeys is hardly a large enough group to develop a statistical population. Despite the inconsistency in animal models a few conclusions may drawn with respect to low-level exposure. First, it is likely that developmental exposure can induce sensory and motor defects; however, evidence for direct cognitive defects is more controversial. Second, although a controlled scientific environment is a necessity, it is nonetheless a simplistic model of a much larger, genetically heterogeneous human population. Third, these experiments do not take into account the possible additive effect of exposure to multiple environmental neurotoxins, as is the case in Arctic Canada (Van Oostdam, 2005), which may have additive effects.

Congenital Exposure in Humans

A number of human studies have been carried out to address the issue of development defects following congenital exposure to low levels of MeHg through maternal consumption of contaminated seafood (Castoldi et al., 2001; Myers & Davidson, 2000). None of these studies have identified mental retardation or other severe development defects. However, it is ambitious to expect that such small sample sizes would have the sensitivity to detect increases in rare outcomes such as mental retardation.

Consequently, researchers have focused on more identifying subtle signals of neurological damage and developmental delay, which are anticipated to be more likely prevalent as a consequence of low-level developmental exposure. Table 1 compares known MeHg biomarkers in various populations including those in Canada. Note the wide variation in levels, illustrated in the range column. In general, the results of human studies mirror those seen in animals, in that there are inconsistencies. However, the data supporting cognitive defects are relatively more conclusive. Cognition is usually assessed by subjecting patients to standard tests that assess aptitude in areas such as language, memory, and attention. The National Research Council of the National Academy of Sciences (NAS) recently reviewed some of these studies and concluded that the evidence supporting neurodevelopmetal defects associated with methylmercury through contaminated seafood is compelling (NRC, 2000).

Table 1. Comparison of mercury (total) concentrations in Nunavik with those observed in other cohorts.

Cohort (reference)	*Medium*	*Years*	*N*	*Geometric mean*	*Range*	*Interquartile range*
Canada						
Nunavik Inuit	Cord blood (µg/L)	1996-2000	95	18.5	2.8-97.0	12.0-27.2
	Maternal blood (µg/L)	1993-1995	130	10.4	2.6-44.2	6.6-17.0
	Maternal hair (µg/g)	1992	123	3.7	0.3-14.0	2.5-6.2
Southern Quebec	Cord blood (µg/L)[a]	1977-1978	1108	1.0	0.9-1.0[b]	
James Bay Cree	Women hair, not pregnant (µg/g)[c]	1981	70	2.5	max=19.0	
Northern Quebec Cree	Maternal hair (µg/g)		215	6.0[d]	5.2[e]	
USA	Women hair, not pregnant (µg/g)	1981	1274	0.36[f]	0.14-0.90	
			1546	0.24[g]	0.09-0.62	

(continued)

Table 1. (cont'd)

Cohort (reference)	*Medium*	*Years*	*N*	*Geometric mean*	*Range*	*Interquartile range*
Faroe Islands						
First Cohort	Cord blood (µg/L)	1986-1987	894	22.9	13.4-41.3	
	Maternal hair (µg/g)	1994-1995	914	4.3	2.6-7.7	
Second cohort	Cord blood (µg/L)		163	20.4	1.9-102.0	11.8-40.0
	Maternal hair (µg/g)		144	4.1	0.4-16.3	2.5-7.4
Seychelles Island						
Main study	Maternal hair (µg/g)	1989-1990	740	5.9	0-25	6.0
Pilot study	Maternal hair (µg/g)		789	6.6	0.6-36.4	6.1
New Zealand	Maternal hair (µg/g)	1978-1984	935	8.3[d]	6.0-86.0	
Greenland, Disko Bay	Cord blood (µg/L)	1994-1996	178	25.3	2.4-181.0	
	Maternal blood (µg/L)	1994-1996	180	12.8	1.9-75.6	

Table reprinted from (Van Oostdam et al, 2005) with permission from Elsevier. Original source: (Muckle et al, 2005b)
[a] The average Hg concentration was reported in nmol/L, this concentration was divided by 5 to transform to µg/L.
[b] 95% confidence interval.
[c] Women aged between 15 and 39 years old.
[d] Arithmetic mean.
[e] Standard deviation.
[f] Among seafood consumers.
[g] Among non-seafood consumers.

In New Zealand, maternal exposure to MeHg, primarily through the consumption of contaminated fish, resulted in a lower performance on neurobehavioral tests in children at age 4 (Kjellstrom et al., 1986) and age 6 (Kjellstrom et al., 1989). At age 4, children exhibited abnormal test scores on the Denver Developmental Screening Test. Higher incidences of premature birth and low birth weight was also reported. At age 6, children

displayed poorer scores on the Wechsler Intelligence Scale for Children-Revised and in the Test of Language Development. However, the experimenters also found that social class and ethnic group affected scores. In both incidences, defects were correlated to higher levels of mercury in maternal hair.

A larger study in the Faroe Islands assessed the effects of maternal exposure through the consumption of contaminated fish and pilot whale (Grandjean et al., 1997). At seven years of age, the children were subjected to several neurobehavioral and sensory-motor tests. Defects were observed in verbal memory, language, attention, motor function, and visual-spatial abilities. Clinical testing revealed that the children had no apparent physiological defects, and were otherwise healthy. The same experimenters later found that these defects were also associated with umbilical cord mercury levels. Curiously, no evidence for a threshold hair level was found in these studies, with mothers exhibiting a broad range of levels. This population is also exposed to relatively high levels of PCBs, particularly through the consumption of whale meat. However, corrections for PCB cord blood levels suggested that concomitant exposure could not explain the mercury related defects, and that there was no additive effect between these two environmental contaminants (Budtz-Jorgensen, 1999). Another study conducted in the Faroe Islands correlated a decreased neurologic optimality score in 182 neonates to cord blood mercury levels (Steuerwald et al., 2000). Maternal hair mercury levels were also found to be associated with neurobehavioral defects in 351 children in Amazonian communities in Brazil (Grandjean et al., 1999a). Gold mining in the Amazon Basin has released mercury into rivers, and subsequently contaminates fish in downstream areas.

The results of two other major epidemiological studies did not report any effects of prenatal MeHg on neurobehavioral function. In the Republic of the Seychelles over 700 mother-children pairs were examined (Davidson et al., 1998). Deep sea and reef fish consumption is the source of MeHg for this population. Six age appropriate neurobehavioral tests were implemented on children age 66 months. Adverse effects were controversially noted in the pilot study; however, the main study, in which the covariates were better controlled, revealed no apparent neurobehavioral defects associated with maternal MeHg. The average maternal total mercury hair levels were intermediate between those recorded in the Faroe Islands and New Zealand. The authors of the Seychelles study recently reported that two of the 21 neurobehavioral endpoints examined at 9 years of age were correlated to MeHg exposure, but that this was probably due to chance as a consequence

of multiple analyses (Myers, 2003). The mercury levels in local fish were comparable to those found in the United States (Mahaffey & Rice, 1997), suggesting that the higher hair mercury levels were due to a greater consumption of fish, rather than higher contamination in the environment. Another smaller study on 131 infant-mother pairs in Mancora, Peru similarly found no neurodevelopmental anomalies.

The Seychelles, New Zealand, and Faroe Islands studies were all reviewed by the NAS. Despite the negative results in the Seychelles, the NAS still concluded that the evidence was compelling. Several factors were suggested to account for the apparent inconsistencies in findings between these studies (NRC, 2000). Differences in age at testing, the end points assessed, the source of mercury, and the pattern of exposure could all account for the differences between the results of the Faroe Island and the Seychelles studies. The exposure and experimental designs, however, were similar in the New Zealand and Seychelles study. Although it is curious that the pilot study found neurobehavioral effects in the Seychelles, differences in environmental and genetic factors may have also played a role.

Referring to Table 1, the Inuit of Disko Bay, Greenland have the highest maternal and cord blood levels. Similar, but lower levels are found in the Seychelles, Faroe Islands, New Zealand, and Nunavik regions. Lower levels are found across Caucasian Southern Quebec and U.S. populations. Given the compelling evidence that mercury contamination during pregnancy can cause neurodevelopmental defects, and that those aboriginal populations such as the Inuit harbor comparable maternal and cord levels, it seems reasonable to assume that such populations are at risk. Prospective longitudinal studies on the neurobehavioral effects of MeHg and other contaminants on Aboriginal populations have been ongoing since 1997 (Muckle et al., 2001a & 2001b). The exposure data have already been published and statistical analysis of the possible neurodevelopment defects are currently being undertaken and will be available soon.

Postnatal Exposure in Humans

The previous section addresses defects in cognitive functions. A recent study addressed the possible effects of postnatal MeHg exposure on neuromoter function in Inuit preschool children (Muckle et al., 2005). Blood mercury levels are an indicator for very recent exposure, within 1 or 2 months (WHO, 1976). The authors found that blood mercury levels at the time of testing were associated with tremor amplitude in pointing tasks. Additionally, 234

Quebec Cree children aged 12 to 30 months revealed abnormal tendon reflexes (McKeown-Eyssen et al., 1983). However, the abnormality was only correlated with blood mercury levels in males, and no evidence was observed relating increasing amounts of maternal hair mercury and abnormal tendon reflexes. Beuter and colleagues found a similar correlation between methylmercury exposure and static/ kinetic tremors in Quebec Cree adults (Beuter & Edwards, 1998; Beuter et al 1999a, Beuter et al., 1999). At mercury hair levels higher than 24ug/g, eye-hand coordination was also impaired in this group. Furthermore, adult Brazilian Amazon dwellers were found to be at increased risk for defects in arm movement and manual dexterity (Lebel et al., 1998).

In 1977, 306 Quebec Cree adults were analyzed for the possible neurological defects associated with chronic low-level exposure in response to litigation against 15 mining and industrial companies (Koffman et al., 1979). The authors found no correlation between MeHg levels and neurological problems. Recently, these data were reanalyzed using different statistical methods (Auger et al., 2005). Instead of using an overall neurological score, which may exclude subtle defects, the authors examined several possible neurological outcomes independently. The authors performed several tests for cognitive impairment, reflexes, and sensory-motor functions, and found only a correlation with tremor. There are several problems characteristically associated with studies of this type – for example, bias of subjects who have something to gain, such as a winning a lawsuit or public recognition. Other possible confounding factors include alcohol usage, which is known to induce neurological defects including tremor. The most difficult complication is attempting to assess whether the tremors are associated with chronic low-level postnatal exposure, or delayed neurotoxicity from prenatal exposure. Delayed neurotoxicity is well established in response to acute poisoning in Minamata patients, in which patients over the age of 40 years exhibit difficulties in performing daily activities (Kinjo et al., 1993). Auger and coworkers found the strongest association of tremors with average mercury levels along the hair shaft (Auger et al., 2005). These levels are believed to be a good indicator of long-term exposure, as opposed to peak levels along the shaft or scalp hair, which are believed to represent fluctuations of past exposure. However, similar to the Brazilian study, tremor development was associated with age in that younger adults exhibited the response exclusively. Thus, it is possible that induction of tremors in adults may be a delayed neurotoxic consequence of low level congenital exposure correlated to historical time points such as industrialization of these rural areas.

It has been suggested that elemental mercury exposure, particularly through dental amalgams, may be a risk factor for Alzheimer Disease (AD) (reviewed in Mutter et al., 2004). Patients with sporadic AD have a gene known as apolipoprotein E4 that is expressed in the brain. This compound may have a reduced ability to bind metals such as mercury, which may in turn increase the risk of AD development. As discussed previously, fish eating populations are exposed primarily to organic rather than elemental mercury. A study on 474 Baltimore residents aged 50 to 70 years exposed to mercury primarily through contaminated fish, however, revealed no association between blood mercury levels and neurobehavioral performance (Weil et al., 2005). This study has some limitations (Mutter and Naumann, 2005), the most serious of which is that blood methylmercury levels represent only recent exposures, and do not take into consideration exposure throughout life or during development.

In summary, there is currently no compelling evidence to suggest a correlation for cognitive defects with respect to chronic or acute low-level exposures in adult fish eating populations. This is supported by the fact that in the Faroe Islands the blood mercury levels at seven years of age were generally uncorrelated with neurobehavioural deficits, except in the area of performance on memory for visuospatial information (Grandjean, 1999b). This indicates that cognitive impairment is likely a function of congenital but not recent exposure. There may be neuromotor deficits associated with chronic low-level exposure, as indicated by previously mentioned studies; however, it is not absolutely clear whether theses effects are due to some form of delayed neurotoxicity associated with prenatal exposure.

Mercury and Autism

Autism is the most severe of the Autism Spectrum Disorders (ASD). It is characterized by impairments in social interaction, difficulties in communication, and repetitive or stereotyped behavioral patterns. Autism was first described in 1943, and by 1995 the National Institutes of Health had reported a prevalence of 1 in 500 (Bristol et al., 1996). The exact etiology of ASD is unknown, however, it is clear that development is subject to both genetic and environmental factors. For example, genetically identical twins have only a 60 % concordance rate in disease expression (Le Couteur, et al., 1996).

From the 1930s to 2001, a preservative known as thimerosal was commonly added to childhood vaccines. Thimerosal contains a slightly different form of organic mercury known as ethylmercury. It was proposed that exposure to

ethylmercury may contribute to the pathogenesis of ASD (Bernard et al., 2001; Bernard et al., 2002). Support for this hypothesis was based on temporal associations between onsets and prevalence of ASD with the introduction of ethylmercury into vaccines. Furthermore, autistic patients exhibited elevated levels of mercury in biological samples such as urine. Finally, the effects of low-level exposure to organic mercury in fish eating populations induces neurobehavioral defects characteristic of ASD. However, despite these correlations, the current viewpoint of the scientific community does not support this hypothesis. The conclusions of both the Institute of Medicine and the World Health Organization are that thimerosal does not cause neurobehavioural defects (IOM, 2004). However, the subject remains controversial with advocates of the thimerosal hypothesis vocally accusing the scientific community of covering up evidence (Kennedy, 2005).

Although the link between ASD and thimerosal is tenuous, a recent study has implicated environmentally released mercury as a risk factor (Palmer et al., 2006). The state of Texas has the fourth highest release rates of environmental mercury in the US. Investigators have found that, on average, a 61 % increase in the rate of autism among school children is associated with each 1000 lbs. of environmentally released mercury. The results of this study do not prove a causal relationship between environmental mercury and ASD. It is nonetheless a first step, with more detailed studies at the individual rather than ecological level required. None of the previously mentioned studies on fish eating populations has identified severe developmental disorders such as autism. It remains to be seen whether exposure to environmental mercury is a risk factor for ASD in aboriginal populations in Canada.

Outlook and Conclusions

Nutritional Benefits of Traditional Foods

Traditional country foods are of high nutritional benefit, providing a good source of vitamins, minerals, lipids, and proteins (Van Oostdam, 1999). Consumption of these foods has been associated with lower levels of saturated fat and carbohydrates (Kuhnlein et al., 2004). Health benefits from consuming 1-2 servings of fish a day have been well documented (Kromhout et al., 1995). Consumption of fish more than 4 times per week during pregnancy may actually improve cognitive functions in children (Daniels et al., 2004). Thus, the risks associated with mercury exposure should be carefully weighed against the benefits (Kuhnlein et al., 2000).

This risk benefit assessment should be assessed for different populations, which may have different eating habits. For example, caribou are currently the main source of mercury exposure for Inuit communities, which are the most highly exposed population (Kuhnlein et al., 2000). It would therefore seem unreasonable to advise against fish consumption in this population, given the benefits. However, a reduction in caribou consumption may be warranted, although the health benefits of this animal are currently unknown (Van Oostdam, 2005).

Possible Implications of Climate Change

It is become abundantly clear that the activities of the human race are inducing environmental changes on a global scale. Greenhouse gases and aerosols are being released into the atmosphere causing global warming, with the 1990s likely the warmest decade of the millennium (IPCC, 2001). The marine food webs are being disrupted (Pauly et al., 1998) and the hydrological cycle is being altered by excessive damming (Dynesius and Nilsson, 1994).

Current understanding of the possible implications of climate change are not well understood; however, speculation is rampant in the scientific communities. Global mercury levels in the atmosphere have more than doubled since the dawn of industry (Lamborg et al., 2002). The process of biomagnification makes aquatic environments the most vulnerable, especially in the Arctic due to mercury depletion events. Although global mercury emissions are decreasing, levels are on the rise in the Arctic, with climate change being a possible cause (CACAR, 2003). Several possible factors may contribute to alterations of environmental mercury levels (CACAR, 2003). For example, the loss of permafrost is expected to occur with global warming. This would increase the amount of wetlands, enhancing the influx of soils and organic materials into lakes and rivers, which may concurrently increase mercury levels. Animals reliant on the permafrost are already being found in different areas. The shift in animal species may alter exposure patterns of local communities, and possibly lengthen food chains enhancing biomagnification. Arctic oscillations are a natural phenomenon that result in the reversal of ocean and air currents into the north. Climate change is expected to increase the frequency and strength of these oscillations, which may increase the amounts of mercury brought into the Canada Basin from industrial regions such as Russia. Changes in the amount of sea ice and general salinity are also predicted to have effects.

Careful analysis of the neurological effects of mercury contamination can potentially be confounded by alterations of the mercury cycle in the environment, which itself may be influenced by global climate change. All longitudinal studies of human health implications represent static images of mercury contamination through an environment which may have very well changed since the time of initial measurements. As mentioned earlier, it is the physiochemical and biological properties of mercury that ultimately define exposure, not just the consumption of traditional foods. With the environment constantly evolving due to human activities, mercury exposure levels may fluctuate in an unpredictable fashion.

Conclusions

The evidence from cellular, animal and human studies all indicate that methylmercury can induce irreparable damage to the nervous system. Mercury and several other environmental contaminants are currently found in the arctic biota, and are contaminating traditional aboriginal foods sources. These contaminants are found in the tissues of the Aboriginal inhabitants, in some cases at alarming levels. Evidence from studies of seafood consuming communities around the globe strongly suggests that methylmercury can induce neurodevelopmental and motor defects in exposed populations. Studies are currently underway to completely assess the health impacts of mercury contamination in the Arctic. However, with the possibilities of confounding factors such as delayed neurotoxicity, climate change, and exposures to multiple contaminants, an in depth understanding of the effects on health is highly optimistic. The seemingly inexorable link between the Aboriginal people and their local environment may have social, cultural, spiritual, and nutritional ramifications; yet it may paradoxically have serious repercussions for their survival and future in Arctic Canada.

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