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Review

Mercury in fishes of Alaska, with emphasis on subsistence species

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Abstract

In the north, the presence of mercury (Hg) in food leading to chronic exposure is a scientific, economic and political issue. Guidelines have been established for the safe consumption of fish containing Hg, however, adherence to these guidelines must be weighed against the health benefits of consuming fish, such as from the omega-3 polyunsaturated fatty acids, vitamins and minerals. Alaskan Natives generally consume much more fish than the national average. This review summarizes and synthesizes the significant amount of data that has been generated on Hg in Alaska fish, particularly those consumed by Alaskans. Also included are a review of the benefits of eating fish, human health concerns relating to Hg toxicity and various risk assessment guidelines for food consumption. Emphasis was placed on methylmercury (MeHg), the most toxic form to humans. Hg concentrations were examined in 17 freshwater fish species and 24 anadromous and marine fish species, for a total of 2692 specimens. For freshwater fish the greatest database was on northern pike (*Esox lucius*). For anadromous and marine fish the greatest database was on Pacific halibut (*Hippoglossus stenolepis*) and the five species of Pacific salmon (*Oncorhynchus* spp.). Overall, most fish had muscle Hg concentrations of ≤ 1 mg kg^{-1} (wet wt.), within the USFDA's Action Level and Alaska's guideline for safe concentrations of MeHg in edible fish. Pacific salmon, the most commonly consumed fish group, had exceptionally low (≤ 0.1 mg kg^{-1}) Hg concentrations. Pacific halibut muscle Hg content was less than 0.3 mg kg^{-1}. Northern pike, a piscivorous (fish-eating) and long-lived fish, contained the highest muscle Hg values, often exceeding the state's guidelines for food consumption. A discussion of the safe consumption level for pike is included.

Keywords: Alaska; Subsistence; Mercury; Fish; Salmon; Pike; Halibut

Contents

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1. Introduction

Mercury (Hg) is a useful element that conducts electricity and combines easily with many metals forming alloys, called amalgams. Hg-containing compounds have many uses including acting as effective pesticides, fungicides and preservatives (ATSDR, 1999). Globally, deterioration of the environment has increased the vulnerability of various populations to Hg from contaminants such as Hg in the food supply. Metals appear in all Arctic ecosystems and can impact the local freshwater and marine food supply. With natural sources of metals, plants and animals, including humans, have time to adapt over many generations. As natural sources change with time, populations can continue to change because, on a relative time scale, these physical processes are slow enough to allow biochemical and genetic adaptation. With anthropogenic sources of metals such as erosion from mining activities, negative impacts can occur over a much shorter time scale, and biological adaptation of subsistence species may not be able to keep up with these rapid environmental changes. Thus, a rapid increase in contaminants can threaten the physical health of both fish species and the human populations which depend on them for their subsistence (AMAP, 1997, 2002; Nriagu, 1988; Egeland and Middaugh, 1997; Egeland et al., 1998; Nobmann et al., 1992; WHO, 1990, 1991; Arnold and Middaugh, 2004; Suk et al., 2004).

Bioaccumulation through the food chain increases the human risk of methylmercury (MeHg) chronic exposure mainly in those populations with high intake of fish or fish products (WHO, 1990; Sexton et al., 1993; Goyer and Clarkson, 2001; Macdonald et al., 2002; Hansen and Gilman, 2005). This is significant for Alaska because recent studies have begun to report that low concentration exposure to Hg in food is associated with an increased risk of neurochemical (Aschner, 2002; Weil et al., 2005) or cardiovascular damage (Bogler and Schwetz, 2002; Meyers et al., 2000; Sorensen et al., 1999). A major proportion of what is known about the disease processes that are associated with environmental contaminants, such as Hg, comes from epidemiologic research in the occupational health field. A recent report by Mahaffey et al. (2004) suggests that greater than 300,000 newborns may have been exposed in utero to MeHg concentrations higher than those considered to be without increased risk.

For Hg, it is clear that symptoms overlap with other sulfhydryl-reactive metals such as cadmium, lead, and arsenic. However, in Arctic wildlife and fish these metals do vary with respect to their site of deposition and action in the organism. Hg can build up in biological systems in its organic form; MeHg crosses the blood-brain barrier (Weil et al., 2005). There have been many reviews of Hg and its toxicity in recent years, including Sorensen (1991), Boening (2000), Braune et al. (1999), Wolfe et al. (1998), Morel et al. (1998), Grigal (2002), Goyer and Clarkson (2001) and National Academy of Sciences (NAS, 2000).

The objective of this paper is to review advances in research on current concentrations of Hg and MeHg in Alaska subsistence fish and note the human health concern relating to neurological, cardiovascular, and immune system development. Its focus is on the use of fish as a subsistence resource for Alaska and its indigenous peoples. All Hg and MeHg concentrations are presented in wet weight values as mg kg^{-1} (ppm; μg g^{-1}).

1.1. Absorption of mercury

Liquid metallic Hg, Hg^0, is only slowly absorbed by the gastrointestinal tract (approximately 0.01%), and is considered to be of no toxicologic consequence (Goyer and Clarkson, 2001). Although Hg^0 is poorly absorbed if indigested, Hg^0 vapor is efficiently absorbed through the lungs and quickly passes the blood–brain barrier (Pamphlett and Cotte, 1998). Due to its lipophilic nature, Hg^0 has a high affinity for myelin and lipid membranes. Once inside a cell, Hg^0 is oxidized by

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catalase to the highly reactive Hg^{2+}. MeHg, derived from fish (Bloom, 1992), and dimethylmercury are readily absorbed in the gastrointestinal tract. MeHg can be demethylated and oxidized to Hg^{2+}, but demethylation can also more commonly produce Hg^0. Once assimilated in the cell, Hg^{2+} and $MeHg^+$ form covalent bonds with glutathione and the cysteine residues of proteins as well as other sulfur-containing molecules. Gastrointestinal absorption of Hg^{2+} is about 15% while MeHg is about 90%. MeHg has a greater affinity for the brain, especially the posterior cortex.

Excretion of Hg from a mammal is by way of urine and feces. Some Hg is lost via hair and milk. About 90% of MeHg is excreted in feces after chronic exposure (Goyer and Clarkson, 2001), with the half-life of MeHg elimination being about 70days. The overt neurotoxic and nephrotoxic effects of high concentration Hg exposure are well established, but the more subtle effects of chronic, lower-concentration Hg accumulation to the fetus is a major concern. Concentrations of MeHg in fetal red blood cells are 30% higher than those in maternal red cells and maternal milk can contain up to 5% of the concentration of maternal blood (Grandjean et al., 1994).

Oxidation of Hg^0 to Hg^{2+} is mediated by catalases while MeHg is transformed to Hg^{2+} via the cleavage of the carbon–Hg bond. Within cells, Hg has an affinity for ligands containing sulfur groups such as glutathione and cysteine (Harris et al., 2003). MeHg cysteine has been recently identified in fish tissue (Harris et al., 2003). The cysteine complex of MeHg can enter endothelial and brain cells via the neutral amino acid transporter (Aschner et al., 1990).

1.2. Molecular mechanisms of effect from mercury

Although the underlying biochemical mechanisms that lead to impaired cell function are not completely understood, there is good evidence supporting damage from oxidative stress (Yee and Choi, 1996). Hg is a transition metal that can promote the formation of hydrogen peroxide and enhance the subsequent iron- and copper-induced production of lipid peroxides and the highly reactive hydroxyl radical (Miller et al., 1991). Lipid peroxides can alter membrane structure and are highly disruptive of mitochondrial function (Pratico et al., 2002). The pro-oxidant property of Hg is exacerbated by its inhibitory affect on antioxidant processes. Hg has high affinities for glutathione (GSH), which is the primary intracellular antioxidant and conjugating agent, and it can deplete the cell of GSH, decreasing antioxidant capacity. The Hg–GSH conjugation process results in the excretion of the toxic metal into the bile. It has also been demonstrated that Hg not only directly removes GSH from the cell, but also inhibits the activities of two key enzymes involved in GSH metabolism: GSH synthetase and GSH reductase (Zalups and Lash, 1996). Hg also inhibits the activities of the free-radical-quenching enzymes: catalase, superoxide dismutase, and GSH peroxidase (Quig, 1998).

Hg can disrupt the structure and function of proteins including Na/K ATPase through direct binding to free sulfhydryl groups. Metal-induced inhibition of Na/H^+ antiporter can result in astrocytic swelling and destruction; astrocytes are the primary cells responsible for the homeostatic regulation of synaptic 1) pH, 2) Na/K ions, 3) glutamate, and 4) metal sequestration in the central nervous system (Aschner et al., 1998, Aschner, 1996). Also, Hg inhibits the polymerization of tubulin, causes depolymerization of existing microtubules, and in animal studies results in brain lesions (Pendergrass et al., 1997). Cell studies have indicated that Hg exposure directly affects uptake and release of neurotransmitters such as dopamine and serotonin (Komulainen and Tuomisto, 1982; Newland, 2002; Ram and Sathyanesam, 1985). Hg burden is associated with depletion or poor assimilation of specific amino acids which are precursors of neurotransmitters. For example, available pools of sulfhydryl amino acids can be depleted by the metal-induced high turnover of GSH (Quig, 1998; Peroza et al., 1998).

Hg also acts as an endocrine disruptor by inhibiting the conversion of thyroxine (T4) to active T3 (Barregard et al., 1994). Hg may also interfere with steroid metabolism by binding to free sulfhydryl groups on receptors. These Hg-induced disruptions in hormone metabolism could certainly contribute to reproductive problems in wildlife and man (Aschner and Clarkson, 1988; Lundholm, 1991; Dansereau et al., 1999; Halbrook et al., 1994).

MeHg is the more important form of Hg in terms of toxic human health effects from food, because of the neurotoxic effects on fetuses exposed to MeHg from maternal diets during pregnancy (Meyers et al., 2000; NAS, 2000). Exposure of the fetus in utero can result in abnormal neuronal migration and unusual organization of neurons in the cortex (Goyer and Clarkson, 2001). Most of the Hg consumed from fish is in the absorbable MeHg form. The relative level of accumulation of the various forms of Hg in the nervous system is MeHg, $>Hg^{2+}$ (Schiønning and Møller-Madsen, 1991; Tiffany-Castiglioni and Qian, 2001; Shander et al., 2001). MeHg acts on diverse targets and neuronal death is caused by more than one mechanism (Atchison and Hare, 1994;

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Sarafian and Verity, 1991). Localization of MeHg has been studied in rats and monkeys (Møller-Madsen and Danscher, 1991). In rats treated with MeHg, Hg is predominantly accumulated in neurons. In contrast, in monkeys treated with MeHg, localization is predominantly in astroglial and microglial cells. Hg accumulation and storage in astroglia and neurons can occur by Hg^0 or MeHg diffusion across the plasma membrane. Once inside the cell, MeHg can be demethylated to Hg^{2+} and be stored in lysosomes. MeHg can also enter astrocytes via the neutral amino acid transporter (Aschner et al., 1990), where it can alter glutamate transport into astrocytes as well as cysteine uptake (Shander et al., 2001; Aschner et al., 1990). Linke et al. (2003) reported MeHg induced intracellular calcium dysregulation in granule neurons.

There is evidence that MeHg triggers reactive oxygen species (ROS) formation (Quig, 1998; Sanfeliu et al., 1999). The MeHg induced ROS can be partially mediated by *N*-methyl-D-aspartate (NMDA) receptor antagonists or antioxidants (Park et al., 1996). ROS is important in MeHg neurotoxicity since glutathione (GSH), as an antioxidant, will be less effective in modulating the toxic effects of MeHg-generated free radicals. Additionally, GSH concentrations are usually lower in neurons than in astrocytes (Shander et al., 2001). The neurotoxic effects of MeHg may be attenuated by protective effects of Se and omega-3 polyunsaturated fatty acids found in fish (Hansen and Gilman, 2005). Also, MeHg toxicity can be inhibited by other antioxidants such as *N*-acetyl-L-cysteine (Park et al., 1996; Ornagi et al., 1993).

1.3. Neurodevelopmental studies

Some studies have looked at neurological and neurobehavioral effects of elevated environmental MeHg exposure (Gilbert and Grant-Webster, 1995; Grandjean et al., 1997). Grandjean et al. (1997) correlated Hg concentrations in cord blood with neurobehavioral deficits in 7-year-old Faroese children. Studies in New Zealand looked at subtle neurobehavioral deficits in children born to mothers with MeHg hair concentrations in excess of 6 mg kg^{-1} (Kjellstrom et al., 1986). MeHg exposure in New Zealand is primarily due to consumption of shark meat. Neurodevelopmental status of 4-year-old children was assessed by means of the Denver Developmental Screening Test. Matching criteria was based on the mother's ethnic group (Pacific Islanders, Maori, and Europeans), age, as well as place and date of birth. About 50% of the high MeHg children had abnormal or borderline results, whereas this was true for only 17% of the reference children. No influence of confounding by socio-economic factors, maternal health, or smoking habits was found. Data on the fate and effects of combined alcohol and MeHg exposure is scarce and new data on possible synergistic mechanisms would be helpful in the development of public health policies related to Fetal Alcohol Syndrome. A significant dose–response association was found between mean hair Hg concentrations during pregnancy and developmental status on the Denver test. The Faroe Island and New Zealand studies suggest that doubling of the Hg exposure leads to a short delay in development for most test scores (AMAP, 2002; Murata et al., 2004).

The results from a follow-up study of 61 children with high Hg concentrations in maternal hair (6–86 mg kg^{-1}) were compared with a low concentration group (3–5.99 mg kg^{-1}), as well as two control groups with high and low fish consumption, but low Hg hair concentrations (0–3 mg kg^{-1}) (Clarkson, 1997). These groups were fully matched for ethnic group of the mother, sex of child, maternal age, smoking habits, place of residence, and duration of residence in New Zealand before the child's birth (Kjellstrom et al., 1989). Testing was done by means of a battery of scholastic and neuropsychological tests, including the Test of Language Development (TOLD), the Wechsler Intelligence Scale for Children (WISC-R), and the McCarthy Scales of Children's Abilities (MSCA). Significant inverse associations were found between hair Hg concentrations exceeding 6 mg kg^{-1} and performance on TOLD, MSCA, and WISC-R. The largest deficit was seen for children with maternal Hg hair concentrations exceeding 10 mg kg^{-1}. Once in the brain, MeHg can be demethylated to inorganic Hg which has a half-life in the brain that may be measured in years (Clarkson, 1997).

Intake of fish or fish oils (long-chain *n*-3 polyunsaturated fatty acids) has long been hypothesized to prevent cardiovascular disease (Slaonen et al., 1995; Krauss et al., 2000). Large, randomized clinical trials have shown reduced mortality after myocardial infarction among patients assigned to a diet rich in fatty fish or fish-oil supplements, but the generalizability of these finding to subjects without coronary heart disease is unclear. The results of an epidemiologic study relating fish intake or fish-oil concentrations to coronary events has suggested that Hg may counteract the beneficial cardiovascular effects of *n*-3 fatty acids in fish (Grandjean et al., 2004). Guallar et al. (2002) reported that toenail Hg concentration was directly associated with the risk of myocardial infarction. They concluded that high Hg content may diminish the cardioprotective effect of fish intake. On the other hand, Yoshizawa et al. (2002) reported that the Hg

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concentration was significantly correlated with fish consumption; but after age, smoking, and other risk factors for coronary heart disease had been controlled for, the Hg concentration was not significantly associated with the risk of coronary heart disease. Blood pressure in children is an important determinant of hypertension risk later in life and MeHg exposure is a potential environmental risk factor (AMAP, 2002). A birth cohort of 1000 children from the Faroe Islands was examined for prenatal exposure to MeHg, and blood pressure, heart rate, and heart rate variability were determined at seven years of age (Sorensen et al., 1999). Blood pressure increased when cord blood Hg concentrations increased from 1 to 10 $\mu g\ L^{-1}$. Birth weight acted as a modifier, with the Hg effect being stronger in children with lower birth weights. These findings suggest that prenatal exposure to MeHg might affect the development of cardiovascular homeostasis (AMAP, 2002).

MeHg can affect the cellular components of the immune system such as B cells and reduce the humoral-mediated response (Daum et al., 1993). Chronic exposure to low concentrations of Hg can result in immune activation leading to allergy and autoimmune disease (Stejskal, 1996). Hg is known to induce autoimmune disease in susceptible rodent strains. These effects include induction of specific autoantibodies, activation of T and B cells and increased serum immunoglobulin concentrations as well as cytokine dysregulation (Polland and Hultman, 1997). In a mouse model of acquired autoimmunity, Via et al. (2003) have reported that a brief, low-level exposure to Hg exacerbated disease manifestations. These results support the hypothesis that low-level environmental exposure to Hg is one potential factor in the development of autoimmune disease. Hg exposure may lower the threshold for disease development in susceptible individuals. The type of mutation is dependent upon metal concentration (Ariza and Williams, 1999). Later, an encounter with an appropriate toxic or infectious agent may trigger the autoimmune disease in these susceptible individuals. Since metal interactions can be synergistic, low concentrations of Hg and MeHg may affect several systems (Preston et al., 2000; Bemis and Seegal, 1999).

Past studies demonstrated the negative influence of Hg on survival of adults and juveniles in both mammals and fishes (Aulerich et al., 1974; Dansereau et al., 1999; Sorensen, 1991; Weis and Weis, 1991). High concentrations of Hg in the diet have adverse effects on reproduction in both males and females (Friedman et al., 1998). The general relationship between health effects and diet-related Hg exposure to humans has been debated (Laskowski, 1991; Zillioux et al., 1993; Egeland and Middaugh, 1997; Huggett et al., 2001). MeHg concentrations in fish tissues are of special concern because of the potential of MeHg to biomagnify through the food web in aquatic ecosystems (Hanisch, 1998; Wolfe et al., 1998; Ben-David et al., 2001). This MeHg biomagnification is especially evident in predatory, piscivorous (fish-eating) fishes because MeHg is accumulated more efficiently from food than Hg in an inorganic form such as Hg^{2+} (Pentreath, 1976; Frery et al., 2001). Also, fish excrete inorganic Hg increasing the biological half-life of MeHg in fish (Ribeiro et al., 1999). In addition, non-lethal concentrations of Hg were reported to damage the central nervous system in humans as well as in wildlife (Lebel et al., 1998; Wolfe et al., 1998). Such damage may influence performance of wild, free-ranging animals and result in additional indirect mortality (Halbrook et al., 1997). Reduced reproduction and high juvenile mortality resulted in low recruitment, and together with high adult mortality led to a decline in populations (Halbrook et al., 1994). Studies have demonstrated that the effects of Hg contamination could be exacerbated because of synergistic effects of other environmental pollutants or stressors such as organochorines (Newland, 2002; Bemis and Seegal, 1999, 2000).

1.4. Atmospheric deposition and biogeochemical exposure to Alaska

Hg has become a global pollutant due to its atmospheric transport (Mason and Fitzgerald, 1996; Van Oostdam et al., 1999). In a recent study of Hg concentrations in fish from streams and rivers throughout the western United States, Hg was detected in every fish tested (2707) and atmospheric transport is suggested as a key factor responsible for Hg in these fish (Peterson et al., 2007). Even remote areas such as Alaska are experiencing elevated concentrations of Hg (Fitzgerald et al., 1998). Anthropogenic sources of Hg to the atmosphere (about 2000t) are about twice that of natural geologic sources (about 1000t) (Lamborg et al., 2002; Porcella, 1994). Hg^0, as a vapor, resides in the atmosphere for a period of a year or more. Hg, as an element, can exist in three oxidation states: the vapor Hg^0 and two higher states, mercurous (Hg^{+1}) and mercuric (Hg^{+2}). Hg^0 is eventually converted to water-soluble Hg^{2+} and deposited to the surface in rainwater. Hg^{+2} can form several stable organic compounds by attaching to one or two carbon atoms. Hg^{2+} is methylated by methanogenic and sulfate-reducing bacteria present in sediments, streams and ocean waters (Compeau and Bartha, 1985). MeHg is the most important species from a human health and biomagnification point of view (Frery et al., 2001).

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Microbial processes play a key role in the geochemical cycling of Hg by methylating ionic Hg (Hg[II]) (Baldi, 1997; Barkay, 2000; Marvin-DiPasquale et al., 2000). Many microbes methylate ionic Hg in culture, but in the environment this activity has been attributed primarily to sulfate (SO_4^{2-}) reducing bacteria (SRB) (Compeau and Bartha, 1985). Syntrophic methylation by SRB metabolizing with methanogens has been demonstrated as well (Pak and Bartha, 1998).

Atmospherically-deposited Hg has increased since the industrial revolution with the bulk of this Hg being derived from coal-fired plants and waste incinerators (AMAP, 1997). As the global background of Hg and MeHg increases (AMAP, 2002), the MeHg enters the aquatic food chain in the north involving plankton, fish, and marine mammals. Anthropogenic sources for Hg pollution such as inputs from past mining (Frery et al., 2001; Gray et al., 2000; Lebel et al., 1996), and increased atmospheric deposition due to burning of fossil fuels rapidly increase concentrations of Hg in the environment. These sources of Hg pollution are particularly true in freshwater systems. The Hg deposition situation in Alaska may be exacerbated by the fact that major Alaskan rivers such as the Yukon–Kuskokwim (Y–K) river system drain mineralized geologic areas that are enriched in HgS, cinnabar. This form of Hg is especially prevalent near natural Hg deposits found in southwest Alaska (Nelson et al., 1978; Gray et al., 2000). Hg was also used in early Alaska gold mining activities, similar to those used in South America now (Frery et al., 2001, Malm et al., 1995; Yokoo et al., 2003). Naturally-occurring Alaskan Hg deposits have been known for decades such as the abandoned Red Devil Hg mine in the village of Sleetmute along the Kuskokwim River. It has been found that fish located downstream of Hg deposits in Alaska have Hg concentrations that are elevated relative to those upstream (Gray et al., 2000). Remobilization is not always readily apparent from measurements of Hg in water samples near the mine since much of the transport is as small as cinnabar particles that can be carried for hundreds of kilometers before being deposited in the sediment. Additionally, Hg contamination of the aquatic ecosystems can be enhanced from forest fires through erosion of soils following deforestation (http://www.nytimes.com/2006/12/05/science/05observ.html) and increased nutrient concentrations in watersheds (Hennig et al., 2005; Kelly et al., 2006). In 2004 and 2005 forest fire activity in interior Alaska increased 5-fold over the previous decade (http://www.dnr.state.ak.us/forestry/firestats/index.htm). This increase in fire activity may be linked to global climate change.

In addition to the microbial processes, biological transport of Hg and MeHg can influence biogeochemical systems. Salmon spend a major part of their life cycle in the ocean and contaminants incorporated into salmon while feeding in the pelagic environment can be re-deposited into spawning grounds (Ewald et al., 1998; Krummel et al., 2003). Based on escapement data (i.e. number of salmon allowed to escape from commercial and subsistence fisheries in order to spawn), sockeye salmon contributed approximately 5×10^6kg of organic matter to the Kvichak River in the Bristol Bay region of Alaska (ADFG, 1999a). Most of this salmon biomass is derived during the marine growth period and represents a substantial new source of both nutrients and contaminants to the Bering Sea region's aquatic and terrestrial food webs (Kline et al., 1993; Bilby et al., 1996; Ben-David et al., 1998; Watkinson, 2000). If a salmon contained about 0.035 mg kg^{-1} MeHg, a return of 2.25×10^6 sockeye salmon in 1980 to the Kvichak River would represent an estimated input of 0.1kg of MeHg into surface water (Zhang et al., 2001). Sockeye mean escapement data over 20 years for 8 rivers in the Bristol Bay region (ADFG, 1999a) was integrated with a MeHg mean value for sockeye (0.035 mg kg^{-1}) showing a total mass loading for MeHg to Bristol Bay river ecosystems of about 21kg MeHg from the ocean. This amount of MeHg is about 1/5000 of the Hg reported released in the US (USEPA, 1997a). This result supports the hypothesis of Ewald et al. (1998) regarding contaminant transport and the concept that salmon biomass is an additional transport pathway for MeHg in addition to atmospheric and local biogeochemical sources of Hg, moving MeHg to Alaska's interior waters. Similar biological transport to Arctic lakes by migrating birds has also been reported (Rothschild and Duffy, 2005).

Besides atmospheric deposition and biogenic transport, industrial contamination can lead to increases in Hg and its methylation. Previous evaluations of worldwide Hg contamination indicated that industrial areas in Japan, northern Europe, South America, and the Great Lakes are considered "hot spots" (Sorensen, 1991). For example, Hg can be released from 1) chlor-alkali plants; 2) pulp and paper mills; 3) coal-powered electrical power stations, and 4) large municipal operations such as near New York and Boston harbors (Hanisch, 1998; Nriagu, 1988). In Alaska, local industrial sources such as the above are rare.

1.5. Traditional fish-based foods and trophic levels

The fisheries of the North Pacific and the Bering Sea are among the most productive in the world. Over one quarter of the world total landings of fish, mollusks, and

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crustaceans were harvested from the North Pacific (Bechtel and Crapo, 2002). Pollock landings alone typically account for approximately 5% of the combined world landings of fish. However, economic models for predictions of changes in catches related to the effect of Hg on a fishery do not exist, and very little is known about the potential economic impacts of Hg on Alaska's marine and fresh water fisheries. The effect of "Hg scares" by advocacy groups may have similar economic effects on the fishery as the dolphin bycatch ban had on the tuna industry.

The subsistence division of the Alaska Department of Fish and Game (ADFG) periodically completes quantitative surveys of household employment and subsistence harvests (by species) for many coastal and interior communities (e.g., ADFG, 2001; Brown et al., 2005; Krieg et al., 2005; Fall et al., 2006). These studies provide recent, systematic "snapshots" of village wage and subsistence economies that are valuable as baseline information for predicting and tracking change. Among the many traditional foods consumed in Alaska (Fig. 1), fish are the primary stable food throughout the state. The ADFG estimated that finfish harvests represented 60% of all rural subsistence harvests in the state, based on a 1999 wild resource harvest survey (ADFG, 2001). Subsistence fishes include marine fishes, anadromous fishes (mainly salmon), and freshwater non-salmon species. Approximately 45% of the Alaskan statewide subsistence salmon harvest in 1999 occurred from the Yukon and Kuskokwim rivers of western Alaska. Dramatic declines in returning adult salmon and subsequent subsistence harvests occasionally occur (e.g., 1998–2002) in these rivers, and resident freshwater fishes may be targeted in greater proportions than previously taken by subsistence users. Subsistence fish harvests differ greatly between regions and communities. For example, in the four communities along the lower-middle Yukon River, whitefishes (mainly *Coregonus* spp.), Arctic lamprey (*Lampetra japonica*), inconnu (sheefish) (*Stenedus leucichthys*), and northern pike (*Esox lucius*) were the dominant non-salmon fishes harvested in 2002 (Brown et al., 2005). In the eight communities along the Kvichak River watershed of Bristol Bay, rainbow trout (*Oncorhynchus mykiss*), Dolly Varden/char (*Salvelinus* spp.) and pike dominated the fishes harvested (aside from commercially-important salmon) in 2002–2003 (Krieg et al., 2005). Pike made up approximately 10% of the fish diet in the Yukon (Brown et al., 2005) and Kvichak River (Krieg et al., 2005) studies.

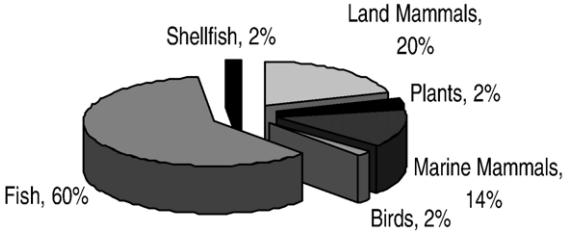

Fig. 1. Average composition of subsistence harvest by rural residents (source: ADFG 2001).

A survey showed that a western Alaskan villager ingested an average of 4.8kg of traditional subsistence foods per week. In that study the least amount of subsistence food consumed was 1.4kg per week and the most was 7.4kg (Rothschild and Duffy, 2002a). This variation is supported by recent salmon consumption data (ADFG, 2001). Among the Canadian Inuit and Deme, the average traditional food consumption varied from about 300g d^{-1} for a 13–19year age group to 600–700g d^{-1} for the 41–60 year age group (Kuhnlein et al., 1996).

Although industrial pollution can rapidly change concentrations of Hg, in Alaska natural geologic and hydrologic processes may create conditions of high Hg exposure from fish consumption similar to those reported for human-caused pollution (Jewett et al., 2003). Risk from Hg pollution is highest for human and animal populations that rely heavily on aquatic food webs that have the greatest number of trophic steps (levels). Subsistence food provides people with nutrients, and unfortunately, exposure to Hg. MeHg builds up "through the food chain" and this is called biomagnification (Macdonald et al., 2002). Not all contaminants biomagnify, but MeHg does (Zhang et al., 2001). Hg build-up in Alaskan animals depends on the number of "steps" before the trophic level where feeding occurs (Ben-David et al., 1998; Ben-David et al., 2001).

Bacteria, plankton, and algae are the lowest level on the Alaskan food chain. Plankton-eating animals generally have low concentrations of Hg, i.e. small fish and salmon, while polar bears tend to have the highest concentrations of contaminants. The amount of Hg body burden is not related to the size of the animal. For instance, a whale that eats mostly zooplankton, like a bowhead whale, may have lower contaminant concentrations than a killer whale that eats seals, even though the bowhead is larger (Endo et al., 2003). Seals that eat clams have lower concentrations of Hg than seals than eat fish (Dehn et al., 2005, 2006). People and animals that eat large amounts of marine mammals and fish are the most at risk for high Hg concentrations (Mahaffey et al., 2004; Wheatley and Paradis, 1996; Yardley et al., 1998).

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1.6. Risk assessment for food consumption guidelines

Public health scientists and regulators are generally in worldwide agreement regarding safe concentrations of MeHg dietary exposure guidelines for adults. However, agreement is lacking regarding the best guidelines for dietary intake of MeHg for the most sensitive population segments, namely pregnant women, nursing mothers, and young children. World Health Organization (WHO), U.S. Food and Drug Administration (USFDA), U.S. Environmental Protection Agency (USEPA), U.S. Agency for Toxic Substances and Disease Registry (ATSDR) and Health Canada use various epidemiological studies to derive their particular guidelines.

WHO recommends the Provisional Tolerable Daily Intake (PTDI) for MeHg not exceed 2.3×10^{-4} mg/kg body weight per day (or 1.6×10^{-3} mg kg^{-1} wk^{-1}) in order to sufficiently protect those at greatest risk, young children and women of childbearing age (http://www.who.int/mediacentre/news/notes/2003/np20/en.html).

Health Canada also recommends a PTDI for women of reproductive age and infants of 2×10^{-4} mg kg^{-1} body weight d^{-1}, and they use 5×10^{-4} mg kg^{-1} body weight d^{-1} for adults (NRC, 2000). Canada also uses the Frequent Consumers of Fish Guideline of 0.2 mg kg^{-1} and the Commercial Sale of Fish Guideline of 0.5 mg kg^{-1} (Evans et al., 2005).

USFDA recommends an action level of 1 mg kg^{-1} for MeHg in fish muscle tissue for commercial sale (http://www.cfsan.fda.gov/~lrd/fdaact.html#merc). This action level is an administrative guideline that defines the extent of contamination at which USFDA may regard food as compromised and represents the limit at or above which USFDA may take legal action to remove products from commercial sale. It was not intended for food caught locally by recreational or subsistence fishers. The action level was calculated assuming an acceptable MeHg daily intake of 5×10^{-4} mg/kg body weight per day, 226g (0.5lb) of fish consumed per week, and a 70-kg adult (1 mg kg^{-1} = [5×10^{-4} mg $kg^{-1}\times$ 7days × 70kg] / 226g of fish consumed).

USEPA established a more conservative reference dose (RfD) of 1×10^{-4} mg/kg body weight per day for MeHg in edible fish (USEPA, 2001a; Rice et al., 2003). The RfD is the safe dose that can be consumed daily without a risk of adverse effects when experienced without ill effects over a lifetime of 70years. USEPA also established a MeHg tissue-based water quality criterion or tissue residue criterion of 0.3 mg kg^{-1} fish for human consumption (USEPA, 2001a). This screening value is the MeHg concentration in fish tissue that should not be exceeded based on a total fish consumption-weighted rate of 0.0175kg fish d^{-1}. The fish tissue residue criterion is consistent with how fish advisories are issued. Fish advisories for Hg are based on the amount of MeHg in fish tissue that is considered acceptable, although they are usually issued for a certain fish or shellfish species in terms of a meal size. The USEPA strongly encouraged States and authorized Tribes to develop a tissue-based water quality criterion for MeHg using local or regional data rather than the default value if they believe that such a water quality criterion would be more appropriate for their target population (USEPA, 2001a).

While USFDA has legal jurisdiction over safe amount of Hg in commercial fish, each of the 50 United States has primary responsibility for protecting their residents from the health risks of consuming contaminated non-commercially-caught fish. States may select other scientifically defensible values for developing fish advisories. These advisories inform the public that concentrations of Hg have been found in local fish at levels of public health concern, especially for the most sensitive groups, i.e., pregnant women, nursing mothers and young children (USEPA, 2001b). Furthermore, state advisories recommend either limiting or avoiding consumption of certain fish from specific waterbodies.

ATSDR set an oral minimal risk level (MRL) of MeHg in fish as 3×10^{-4} mg/kg body weight per day (ATSDR, 1999).

The State of Alaska currently uses USFDA guideline of 1 mg MeHg kg^{-1} of fish, rather than the USEPA fish tissue residue criterion of 0.3 mg MeHg kg^{-1} of fish, as the safe level for human consumption, regardless of whether the fish is for commercial, recreational, or subsistence purposes (Arnold and Middaugh, 2004). This is because MeHg concentrations in the most frequently consumed fish (e.g., salmon, cod, halibut, pollock, sole, and herring) are very low, consistently below 0.2 mg kg^{-1}. Thus, the Alaska Division of Public Health (ADPH) maintains the general national fish advisories issued by USEPA and USFDA are inappropriate for Alaska and are not consistent with the ADPH recommendations. Based upon input from public health officials, research scientists, and Native health leaders, ADPH strongly recommended that all Alaskans, including the sensitive segments of the population, continue unrestricted consumption of fish from Alaskan waters as part of a balanced diet (Arnold and Middaugh, 2004). With this in mind, ADPH further recommended continuous monitoring to be conducted to ensure that Alaskans whom consume fish remain safe from Hg exposure.

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2. Mercury in Alaska fish

2.1. Mercury in freshwater fishes of Alaska

Fish accumulate Hg in their tissues, where it becomes bound to proteins. Concentrations of Hg in 17 freshwater fish species ($n=775$) from Alaska, including juvenile salmon, are summarized in Table 1. Over the past 22years, studies have focused on the Hg content of water, sediment, and fishes in western Alaska, particularly from National Wildlife Refuges (e.g. Snyder-Conn et al., 1992; Mueller et al., 1993, 1996; Gray et al., 1996; Mueller and Matz, 2002) (Fig. 2). Among the various fishes analyzed for Hg, the greatest database exists on the freshwater species northern pike (*E. lucius*) and Arctic grayling (*Thymallus arcticus*). Tissues of the piscivorous northern pike had total Hg (THg) concentrations that exceeded USEPA tissue-based water quality criterion relative to consumption of fish by humans (0.3 mg kg^{-1}) and USFDA action level for human consumption (1.0 mg kg^{-1}) (Fig. 3). For example, 44% of the pike examined from the Nowitna National Wildlife Refuge in 1987 had concentrations in tissues between 1.0 and 2.9 mg kg^{-1} (Snyder-Conn et al., 1992). A study on subsistence fishes in the Yukon–Kuskokwim Delta area reported 36% of the pike examined had THg in muscle tissue that exceeded the USFDA action level (Duffy et al., 1999). Most THg in pike is MeHg, the most neurotoxic form to humans (Dandborgy-Englund et al., 2001; Egeland et al., 1998; Duffy et al., 1999; Jewett et al., 2003). In general, Hg present in fish is greater than 85% MeHg for muscle (Storelli, 2000), but in pike muscle MeHg is nearly 100% of the THg (Jewett et al., 2003).

Only two of the 17 freshwater fish species listed in Table 1, northern pike and inconnu, exceeded the USFDA action level. As noted above, both of these fishes are subsistence foods in western Alaska. The highest mean THg concentration in pike, 1.5 mg kg^{-1}, came from a tributary on the lower Yukon River (Andreafsky River) where the Hg source apparently is naturally occurring. Arctic grayling from the same tributary had a mean value of less than 0.3 mg kg^{-1}. Both pike and grayling had substantially higher THg (2–3 times) than concentrations in the same species from the Kuskokwim River, indicative of the regional differences. As for inconnu, the mean THg in large (mean 865mm TL) specimens from the Innoko National Wildlife Refuge was 0.7 mg kg^{-1}, with only one fish exceeding 1.0 mg kg^{-1}.

MeHg concentrations in pike muscle were significantly higher than in the liver in Yukon and Kuskokwim river systems (Jewett et al., 2003). There were strong correlations between MeHg and THg in muscles of all pike, grayling, and whitefish. The mean percentage of MeHg in THg in pike and grayling muscle was 94 and 95%, respectively, while whitefish muscle had a lower percentage of MeHg at 81%.

An extensive examination of contaminants, including Hg, recently occurred in whole specimens of three fish species in the Yukon River Basin (Hinck et al., 2006). The fishes were the northern pike, longnose sucker (*Catostomus catostomus*), and burbot (*Lota lota*). Concentrations of Hg exceeded toxicity thresholds in one or more samples of these fishes and were included in a risk analysis for piscivorous wildlife. Hg concentrations in these fishes exceeded the 'no effect hazard concentrations' for all bird and small mammal models, which indicated that Hg concentrations in these fishes may represent a risk to piscivorous wildlife throughout the Yukon River Basin. Of the three fish species pike had the highest concentrations of Hg. In summary, the concentrations of Hg in northern pike from Alaska freshwater systems are generally higher than other fishes, but are highly variable in different systems, fluctuating nearly an order of magnitude. Also, the data available do not support any increase or decrease in Hg concentrations in pike over the past two decades (Jewett et al., 2003).

Because the accumulation of MeHg in freshwater fishes is related to the time of exposure and accumulation kinetics, Hg concentration in fish tend to rise with an increase in age, and therefore with the fish sizes as well (Johnels et al., 1967; Jewett et al., 2003). Age is the preferred parameter, but fish length or body weight can be used for approximation of age (Jewett et al., 2003; Zhang et al., 2001). In a study of fish in northern Manitoba, Derksen and Green (1987) showed that the Hg concentrations in walleye (*Stizostedion vitreum*) and northern pike correlated with the length more significantly than with the body weight. The Jewett et al. (2003) study came to similar conclusions and the data suggest eating smaller fish to retain benefits like omega-3 fatty acid while minimizing Hg exposure.

In general, there was no difference in Hg concentration between sexes of similar sized pike; however, female grayling had higher Hg content than male grayling (Jewett et al., 2003). Presumably the higher Hg value in females was because females were larger than males. Sufficient data on Hg content by size and sex of grayling is not available from other studies so interpretations must be careful. However, it appears that higher concentration of Hg in fish tissue is mainly a function of fish size rather than sex, and sex does not

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Table 1
Concentrations of total mercury in freshwater fishes from Alaska

Species and location	Year sampled	# of fish	Mean (SD) fish size (TL, mm) or weight (g)	Mean (SD) THg concentration (mg kg^{-1} wet weight)	Reference
Northern pike — *Esox lucius* (muscle)					
Yukon R Basin (composited whole body)	2002	157	498–725 mm	0.12–0.51	Hinck et al. (2006)
Yukon R. (Andreafsky R.)	2000	6	610 (33) mm	1.51 (0.296)	Jewett et al. (2003)
Yukon R. (Andreafsky R.)	1997	3	531 (33) mm	1.07 (0.803)	Duffy et al. (1999)
Yukon R. (Paimiut and Emmonak)	1997	6	796 (220) mm	0.73 (0.456)	Duffy et al. (1999)
Kuskokwim R. (Aniak, George and Takotna R.)	2000	15	524 (121) mm	0.63 (0.359)	Jewett et al. (2003)
Kuskokwim R. (Gweek and George R.)	1997	2	502 (153) mm	0.74 (0.359)	Duffy et al. (1999)
Innoko National Wildlife Refuge	1997	11	735 (192) mm	0.63 (0.439) [a]	Mueller and Matz (2002)
Innoko National Wildlife Refuge	1996	9	627 (179) mm	0.46 (0.283) [a]	Mueller and Matz (2002)
Innoko National Wildlife Refuge	1993	48	661 (163) mm	0.44 (0.184)	Headlee (1996)
Innoko National Wildlife Refuge	1991	13	678 (106) mm	0.40 (0.115) [a]	Mueller et al. (1996)
Koyukuk National Wildlife Refuge	1991	6	706 (139) mm	1.00 (0.448) [a]	Mueller et al. (1996)
Nowitna National Wildlife Refuge	1991	9	699 (176) mm	0.49 (0.363) [a]	Mueller et al. (1996)
Nowitna National Wildlife Refuge	1987	9	726 (152) mm	1.05 (0.827)	Snyder-Conn et al. (1992)
Selawik National Wildlife Refuge	1988	5	524 (52) mm	0.54 (0.257) [a]	Mueller et al. (1993)
Selawik National Wildlife Refuge	1987	15	521 (57) mm	0.19 (0.109) [a]	Mueller et al. (1993)
Kanuti National Wildlife Refuge	1989	23	526 (149) mm	0.31 (0.229) [a]	Mueller et al. (1995)
Kanuti National Wildlife Refuge	1988	7	472 (45) mm	0.11 (0.028) [a]	Mueller et al. (1995)
Kanuti National Wildlife Refuge	1987	6	551 (89) mm	0.34 (0.163) [a]	Mueller et al. (1995)
Kanuti National Wildlife Refuge	1986	1	601 mm	0.37	Mueller et al. (1995)
Kanuti National Wildlife Refuge	1985	3	541 (184) mm	0.37 (0.384) [a]	Mueller et al. (1995)
Arctic grayling — *Thymallus arcticus* (muscle)					
Yukon R. (Andreafsky R.)	2000	4	379 (19) mm	0.26 (0.030)	Jewett et al. (2003)
Kuskokwim R. (George R.)	2000	6	278 (23) mm	0.08 (0.015)	Jewett et al. (2003)
Kuskokwim R. (Tuluksak R.)	1997	4	N/A	0.10 (0.040)	Duffy et al. (1999)
Kuskokwim R. (3 rivers and tributaries)	?	13	N/A	0.19 (0.133)	Gray et al. (1994)
Southwestern Alaska (8 rivers and tributaries)	?	32	312 (118) mm	0.13 (0.068)	Gray et al. (1996)
Innoko National Wildlife Refuge	1997	7	371 (34) mm	0.15 (0.032) [b]	Mueller and Matz (2002)
Innoko National Wildlife Refuge	1996	5	379 (48) mm	0.16 (0.043) [b]	Mueller and Matz (2002)
Selawik National Wildlife Refuge	1988	5	366 (33) mm	0.33 (0.319) [b]	Mueller et al. (1993)
Selawik National Wildlife Refuge	1987	5	425 (126) mm	0.11 (0.370) [b]	Mueller et al. (1993)
Nowitna National Wildlife Refuge	1987	4	247 (7) mm	0.03 (0.004)	Snyder-Conn et al. (1992)
Kanuti National Wildlife Refuge	1988	3	362 (8) mm	0.21 (0.081) [b]	Mueller et al. (1995)
Kanuti National Wildlife Refuge	1987	37	326 (40) mm	0.13 (0.152) [b, c]	Mueller et al. (1995)
Kanuti National Wildlife Refuge	1986	2	481 (107) mm	0.33 (0.028) [b]	Mueller et al. (1995)
Kanuti National Wildlife Refuge	1985	2	N/A	0.14 (0.008) [b]	Mueller et al. (1995)
Coho salmon — *Oncorhynchus kisutch* (juvenile whole body)					
Innoko National Wildlife Refuge	1996	5	N/A	0.04 (0.006) [a]	Mueller and Matz (2002)
Kuskokwim R. Region	?	10	10 (4.4) g	0.07 (0.032)	Gray et al. (1996)
Chinook salmon — *Oncorhynchus tshawytsha* (juvenile whole body)					
Innoko National Wildlife Refuge	1996	19	N/A	0.04 (0.004) [a]	Mueller and Matz (2002)
Dolly varden — *Salvelinus malma* (muscle)					
Prince William Sound	1996–97	9	N/A	0.16 (0.046)	Duffy (unpublished)
Kuskokwim R. Region	?	6	451 (259) g	0.25 (0.230)	Gray et al. (1996)
Kuskokwim R. (Aniak R.)	2000	3	457 (26) mm	0.01 (<0.001)	Jewett (unpublished)
Innoko National Wildlife Refuge	1991	35	N/A	0.04 (0.004) [a]	Mueller et al. (1996)
Inconnu (sheefish) — *Stenodus leucichthys* (muscle)					
Kuskokwim R. (Johnson and George R.)	1997	46	N/A	0.16 (0.022)	Duffy et al. (1999)
Innoko National Wildlife Refuge	1991	5	865 (86) mm	0.70 (0.318) [a]	Mueller et al. (1996)
Nowitna National Wildlife Refuge	1987	2	628 mm	0.15 (0.056) [a]	Snyder-Conn et al. (1992)
Least cisco — *Coregonus sardinela* (muscle)					
Seward Peninsula (Snake R.)	1986	10	N/A	0.02 (0.015)	Jewett (unpublished)
Kanuti National Wildlife Refuge	1985	1	N/A	0.09 [a]	Mueller et al. (1995)

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Table 1 (*continued*)

Species and location	Year sampled	# of fish	Mean (SD) fish size (TL, mm) or weight (g)	Mean (SD) THg concentration (mg kg^{-1} wet weight)	Reference
Humpback whitefish — *Coregonus pidschian* (muscle)					
Kanuti National Wildlife Refuge	1985	1	N/A	0.04 [a]	Mueller et al. (1995)
Whitefish — *Coregonus* sp. (muscle)					
Kuskokwim R. (Aniak R.)	2000	6	389 (22) mm	0.03 (0.013)	Jewett, et al. (2003)
Lower Kuskokwim R. Region	1997	9	N/A	0.16 (0.076)	Duffy et al. (1999)
Burbot — *Lota lota*					
Yukon R. Basin (composited whole body)	2002	13	565–700 mm	0.13–0.26	Hinck et al. (2006)
Bethel (muscle)	1997	3	N/A	0.1 (0.005)	Duffy et al. (1999)
Longnose sucker — *Catastomus catastomus*					
Yukon R. Basin (composited whole body)	2002	45	394–474 mm	0.10–0.19	Hinck et al. (2006)
Kanuti National Wildlife Refuge (muscle)	1988	7	374 (46) mm	0.11 (0.179) [a]	Mueller et al. (1995)
Kanuti National Wildlife Refuge (muscle)	1987	10	430 (43) mm	0.16 (0.080) [a]	Mueller et al. (1995)
Slimy sculpin — *Cottus cognatus* (whole body)					
Innoko National Wildlife Refuge	1996	3	N/A	0.07 (0.036) [a]	Mueller and Matz (2002)
Kanuti National Wildlife Refuge	1990	10 [d]	N/A	0.07 (0.029) [a]	Mueller et al. (1995)
Kanuti National Wildlife Refuge	1989	2 [e]	N/A	0.27 (0.329) [a]	Mueller et al. (1995)
Coastrange sculpin — *Cottus aleuticus* (muscle)					
Prince William Sound	1996–97	8	N/A	0.16 (0.021)	Duffy (unpublished)
Sculpin — *Cottidae* (whole body)					
Kuskokwim R. Region	?	20	6 (2) g	0.05 (0.030)	Gray et al. (1996)
Three-spine stickleback — *Gastrosteus aculeatus* (muscle)					
Prince William Sound	1996–97	2	N/A	0.14 (0.056)	Duffy (unpublished)
Alaska blackfish — *Dallia pectoralis* (whole body)					
Innoko National Wildlife Refuge	1991	1	N/A	0.05	Mueller et al. (1996)
Lake chub — *Coueseius plumbeus* (whole body)					
Kanuti National Wildlife Refuge	1989	1	N/A	0.08 [a]	Mueller et al. (1995)

N/A = data not available.
[a] Converted from dry weight; dry weight × 25% solids (source Mueller, USFWS, Personal Communication).
[b] Converted from dry weight; dry weight × 39% solids (source Snyder-Conn et al., 1992).
[c] Matrix was whole body.
[d] Composed whole bodies at 10 sites.
[e] Composed whole bodies at 2 sites.

seem to influence their accumulation or elimination of Hg in tissues (Sorensen, 1991). Generally, the larger fish contained higher concentrations of Hg (Jackson, 1990; Grieb et al., 1990; Peterson et al., 2007). Results from a study recently conducted by the Alaska Department of Environmental Conservation (ADEC) (2003) seafood and food safety laboratory documented that mean MeHg concentrations were low in the most frequently consumed freshwater fishes from Alaska (http://www.state.ak.us/dec/deh/animal/fm-heavymetals.htm). The two freshwater species examined, northern pike and inconnu, had low mean (SD) values of MeHg in muscle, 0.15 (0.17) and 0.08 (0.26) mg kg^{-1}, respectively. However, fish size and sample locations were not available for that study, thus the ADEC data are not included in Table 1.

2.2. Mercury in anadromous and marine fishes of Alaska

Concentrations of Hg in anadromous and marine fishes (24 species and 1917 specimens) from Alaska are summarized in (Table 2). All of these fishes had mean concentrations of THg in muscle tissues well below the USFDA action level of 1.0 mg kg^{-1}. Since salmon spend a major part of their life cycle in the ocean, Hg incorporated into salmon, while feeding in the pelagic environment, will be deposited into spawning ground surface waters (Zhang et al., 2001). The mean concentrations of THg (Table 2) in the tissues of four adult Pacific salmon species at the four major rivers in Alaska for 1999 and 2000 were reported in Zhang et al. (2001). THg in these salmon had mean concentrations between

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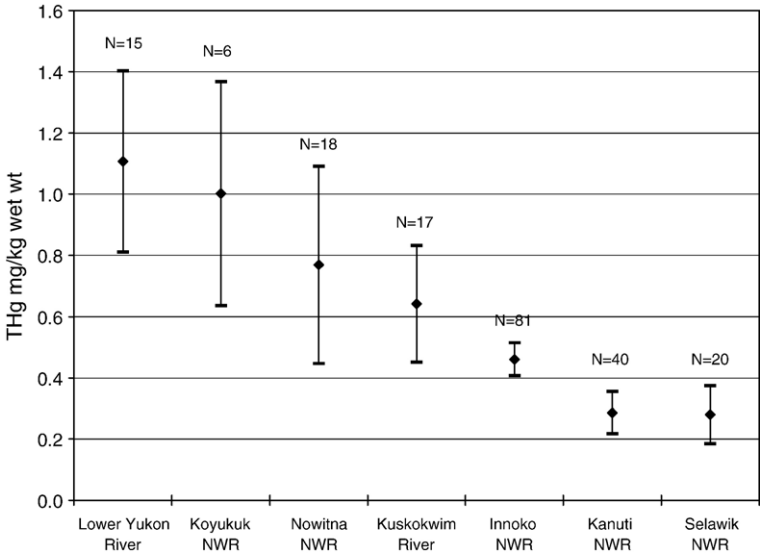

Fig. 2. Comparison (mean±95% CI) of total mercury concentrations in northern pike muscle from various regions of Alaska, 1985–2000.

0.03 and 0.10 mg kg^{-1}. In 1999, THg in individual salmon muscles ranged from 0.02 to 0.14 mg kg^{-1}, while in 2000, THg ranged from 0.03 to 0.11 mg kg^{-1}. THg concentrations in livers tended to be higher than those in muscles with exception of chum salmon. Mean THg in the salmon liver were from 0.05 to 0.11 mg kg^{-1}. Differences in the THg concentrations as well as MeHg differences between species were statistically significant ($p < 0.05$) reflecting trophic level differences. Differences between species in the Alaska river systems were not significant ($p < 0.05$) except for Chinook salmon (*Oncorhynchus tshawytscha*). For example, the mean THg in Chinook muscles from the Kuskokwim was 0.10 mg kg^{-1} in 1999 (Table 2), which is higher than the mean of the Yukon (0.05 mg kg^{-1}). In contrast, THg in Chinook livers from the Kuskokwim in 2000 (mean 0.08 mg kg^{-1}) was lower than in the mean in the Yukon (mean 0.10 mg kg^{-1}); (Zhang et al., 2001). Similar but non-significant variations can be seen for chum (*Oncorhynchus keta*), coho (*Oncorhynchus kisutch*), and sockeye (*Oncorhynchus nerka*) salmon for the mean THg in muscle and liver. Dehn et al. (2005) report similar concentrations of THg in total body homogenates of chum and Chinook salmon obtaining from Bering Sea subsistence fishers.

THg and MeHg concentrations in salmon muscle are correlated ($r=0.93$), with the mean MeHg concentrations lower ($p=0.001$) than the THg means. For salmon,

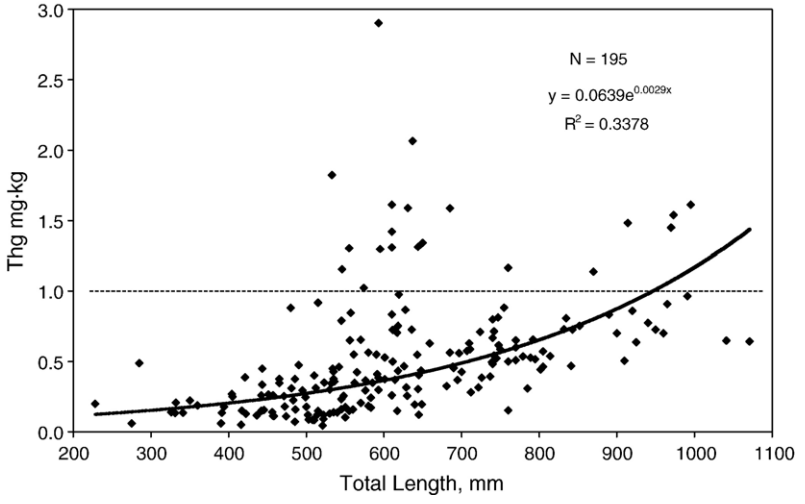

Fig. 3. Relationship between total mercury in northern pike muscle and size from various regions of Alaska, 1985–2000.

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Table 2
Concentrations of total mercury in anadromous and marine fishes from Alaska

Species and locations	Year sampled	# of fish [a]	Mean (SD) fish size (mm) [b] weight (g)	Mean (SD) THg concentration (mm kg^{-1} wet weight)	Reference
Chum salmon — *Oncorhynchus keta* (muscle of adults)					
Kotzebue Sound	1971–74	10 (6)	633 (47) mm	0.02 (0.006)	Hall et al. (1978)
Yukon R. (The Rapids)	2001	20	659 (23) mm	0.05 (0.021)	USFWS (unpublished)
Yukon R. (Beaver)	2001	20	636 (34) mm	0.04 (0.017)	USFWS (unpublished)
Yukon R. (Andreafsky R.)	2000	6	600 (31) mm	0.08 (0.011)	Zhang et al. (2001)
Yukon R. (Andreafsky R.)	1999	6	596 (22) mm	0.07 (0.023)	Zhang et al. (2001)
Yukon R. (Canada)	1971–74	10 (4)	596 (19) mm	0.09 (0.040)	Hall et al. (1978)
Kuskokwim R. (Bethel)	2001	20	645 (36) mm	0.05 (0.017)	USFWS (unpublished)
Kuskokwim R. (Bethel)	2000	6	567 (32) mm	0.07 (0.015)	Zhang et al. (2001)
Kuskokwim R. (Bethel)	1999	6	599 (43) mm	0.06 (0.014)	Zhang et al. (2001)
Nushagak R. (Portage Ck.)	2000	6	611 (11) mm	0.07 (0.018)	Zhang et al. (2001)
Nushagak R. (Portage Ck.)	1999	6	597 (40) mm	0.07 (0.018)	Zhang et al. (2001)
Interior Alaska	1971–74	9 (4)	582 (32) mm	0.06 (0.042)	Hall et al. (1978)
N. Gulf of Alaska	1971–74	9 (2)	615 (38) mm	0.04 (0.032)	Hall et al. (1978)
SE. Gulf of Alaska	1971–74	10 (4)	637 (40) mm	0.03 (0.022)	Hall et al. (1978)
Coho salmon — *Oncorhynchus kisutch* (muscle of adults)					
Yukon R. (Andreafsky R.)	2000	6	568 (19) mm	0.06 (0.012)	Zhang et al. (2001)
Yukon R. (Andreafsky R.)	1999	6	591 (26) mm	0.04 (0.016	Zhang et al. (2001)
Kuskokwim R. (Bethel)	2000	6	523 (33) mm	0.06 (0.014)	Zhang et al. (2001)
Kuskokwim R. (Bethel)	1999	6	564 (27)mm	0.05 (0.006)	Zhang et al. (2001)
Nushagak R. (Portage Ck.)	2000	6	554 (26)mm	0.06 (0.011)	Zhang et al. (2001)
Kvichak R. (Levelock)	1999	5	563 (22)mm	0.05 (0.004)	Zhang et al. (2001)
Innoko Natl. Wildlife Ref.	1991	1	670 mm	0.05 [3]	Mueller et al. (1996)
N. Gulf of Alaska	1971–74	10 (7)	670 (135) mm	0.11 (0.072)	Hall et al. (1978)
SE. Gulf of Alaska	1971–74	9 (2)	642 (34) mm	0.05 (0.061)	Hall et al. (1978)
SE. Gulf of Alaska	1971–74	10 (4)	557 (65) mm	0.03 (0.015)	Hall et al. (1978)
Chinook salmon — *Oncorhynchus tshawytscha* (muscle of adults)					
Yukon R. (The Rapids)	2001	20	763 (109) mm	0.05 (0.016)	USFWS (unpublished)
Yukon R. (Beaver)	2001	8	780 (100) mm	0.05 (0.007)	USFWS (unpublished)
Yukon R. (Andreafsky R.)	2000	6	734 (72)mm	0.07 (0.027)	Zhang et al. (2001)
Yukon R. (Andreafsky R.)	1999	6	711 (154) mm	0.05 (0.035)	Zhang et al. (2001)
Kuskokwim R. (Bethel)	2001	20	860 (104) mm	0.05 (0.016)	USFWS (unpublished)
Kuskokwim R. (Bethel)	2000	6	730 (158) mm	0.08 (0.026)	Zhang et al. (2001)
Kuskokwim R. (Bethel)	1999	6	875 (39) mm	0.10 (0.030)	Zhang et al. (2001)
Nushagak R. (Portage Ck.)	2000	6	695 (158) mm	0.06 (0.020)	Zhang et al. (2001)
Nushagak R. (Portage Ck.)	1999	6	860 (73) mm	0.09 (0.029)	Zhang et al. (2001)
Bristol Bay	1971–74	5	942 (62) mm	0.07 (0.021)	Hall et al. (1978)
Interior Alaska	1971–74	9 (3)	821 (72) mm	0.04 (0.021)	Hall et al. (1978)
N. Gulf of Alaska	1971–74	10 (8)	827 (105) mm	0.04 (0.025)	Hall et al. (1978)
SE. Gulf of Alaska	1971–74	10 (8)	843 (74) mm	0.02 (0.000)	Hall et al. (1978)
Sockeye salmon — *Oncorhynchus nerka* (muscle of adults)					
Kuskokwim R. (Bethel)	2000	6	578 (38) mm	0.05 (0.005)	Zhang et al. (2001)
Kuskokwim R. (Bethel)	1999	6	557 (39) mm	0.03 (0.006)	Zhang et al. (2001)
Nushagak R. (Portage Ck.)	2000	6	556 (23) mm	0.06 (0.006)	Zhang et al. (2001)
Nushagak R. (Portage Ck.)	1999	6	569 (84) mm	0.04 (0.007)	Zhang et al. (2001)
Kvichak R. (Levelock)	2000	6	516 (33) mm	0.06 (0.011)	Zhang et al. (2001)
Bristol Bay	1971–74	10 (4)	634 (36) mm	0.03 (0.021)	Hall et al. (1978)
NE. Gulf of Alaska	1971–74	10 (9)	657 (36) mm	0.02	Hall et al. (1978)
SE. Gulf of Alaska	1971–74	19 (9)	590 (25) mm	0.04 (0.016)	Hall et al. (1978)
SE. Gulf of Alaska	1971–74	9 (4)	516 (37) mm	0.03 (0.016)	Hall et al. (1978)

(continued on next page)

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Table 2 (*continued*)

Species and locations	Year sampled	# of fish[a]	Mean (SD) fish size (mm)[b] weight (g)	Mean (SD) THg concentration (mm kg^{-1} wet weight)	Reference
Pink salmon — *Oncorhynchus gorbuscha* (muscle of adults)					
N. Gulf of Alaska	1971–74	10 (6)	458 (24) mm	0.02 (0.013)	Hall et al. (1978)
SE. Gulf of Alaska	1971–74	9 (4)	501 (26) mm	0.04 (0.025)	Hall et al. (1978)
SE. Gulf of Alaska	1971–74	10 (8)	457 (17) mm	0.02 (0.007)	Hall et al. (1978)
Searcher — *Bathymaster signatus* (muscle)					
Prince William Sound	1996–97	8	N/A	0.10 (0.037)	Duffy (unpublished)
Arctic shanny — *Stichaeus punctatus* (muscle)					
Prince William Sound	1996–97	9	N/A	0.07 (0.019)	Duffy (unpublished)
Black prickleback — *Xiphister atropurpureus* (muscle)					
Prince William Sound	1996–97	4	N/A	0.16 (0.149)	Duffy (unpublished)
High cockscomb — *Anoplarchus purpurescens* (muscle)					
Prince William Sound	1996–97	3	N/A	0.10 (0.056)	Duffy (unpublished)
Crescent gunnel — *Pholis laeta* (muscle)					
Prince William Sound	1996–97	10	N/A	0.05 (0.023)	Duffy (unpublished)
Pacific sandlance — *Ammodytes hexapterus* (muscle)					
Prince William Sound	1996–97	4	N/A	0.02 (0.010)	Duffy (unpublished)
Pacific herring — *Clupea pallasi* (whole body)					
N. Gulf of Alaska	1971–74	20 (19)	N/A	0.06	Hall et al. (1978)
SE. Gulf of Alaska	1971–74	10 (10)	N/A	ND	Hall et al. (1978)
Sablefish — *Anoplopoma fimbria* (muscle)					
N. Gulf of Alaska (Kodiak)	N/A	30	920 g	0.04	Hall et al. (1976b)
N. Gulf of Alaska (Kodiak)	1971–74	10 (1)	635 (20) mm	0.12 (0.137)	Hall et al. (1978)
SE. Gulf of Alaska	N/A	120	2370 g	0.28	Hall et al. (1976b)
SE. Gulf of Alaska	1971–74	10	620 (88) mm	0.14 (0.056)	Hall et al. (1978)
Walleye pollock — *Theragra chalcogramma* (muscle)					
SE Bering Sea	1976	14	N/A	0.04 (0.038)[c]	Robertson and Able (1990)
N. Gulf of Alaska (Kodiak)	1971–74	5	475 (00) mm	0.12 (0.095)	Hall et al. (1978)
N. Gulf of Alaska (Kodiak)	1971–74	5	440 (00) mm	0.36 (0.220)	Hall et al. (1978)
SE. Gulf of Alaska	1971–74	28	498 (68) mm	0.14 (0.225)	Hall et al. (1978)
Pacific cod — *Gadus marcrocephalus* (muscle)					
N. Gulf of Alaska (Shelikof)	1971–74	9	666 (65) mm	0.13 (0.086)	Hall et al. (1978)
SE. Gulf of Alaska	1971–74	9	666 (58) mm	0.15 (0.095)	Hall et al. (1978)
Saffron cod — *Eleginus gracilis* (muscle)					
N. Norton Sound	1986	24	N/A	0.01 (0.008)	Jewett (unpublished)
Kelp greenling — *Hexagrammos decagrammus* (muscle)					
Prince William Sound	1996–97	6	N/A	0.04 (0.021)	Duffy (unpublished)
Fourhorn sculpin — *Myoxocephalus quadricornis* (whole body)					
Arctic National Wildlife Refuge	1989	5	35 (26) g	0.03 (0.004)	Snyder-Conn and Lubinski (1993)
Arctic National Wildlife Refuge	1988	39	46 (41) g	0.02 (0.007)	Snyder-Conn and Lubinski (1993)
Great sculpin — *Myoxocephalus polyacanthocephalus* (muscle)					
Aleutian Islands (Adak)	2004	18	45 (1) mm	0.32 (0.246)	Burger et al. (2007)

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Table 2 (*continued*)

Species and locations	Year sampled	# of fish [a]	Mean (SD) fish size (mm) [b] weight (g)	Mean (SD) THg concentration (mm kg^{-1} wet weight)	Reference
Tidepool sculpin — *Oligocottus maculosus* (muscle)					
Prince William Sound	1996–97	7	N/A	0.05 (0.160)	Duffy (unpublished)
Arctic flounder — *Liopsetta glacialis* (whole body)					
Arctic National Wildlife Refuge	1989	5	71 (44) g	0.03 (0.003)	Snyder-Conn and Lubinski (1993)
Arctic National Wildlife Refuge	1988	30	66 (51) g	0.03 (0.009)	Snyder-Conn and Lubinski (1993)
Rock sole — *Pleuronectes bilineatus* (muscle)					
SE. Bering Sea	1976	9	N/A	0.07 (0.026) [c]	Robertson and Able (1990)
Flathead sole — *Hippoglossoides elassodon* (muscle)					
Aleutian Islands (Adak)	2004	39	39 (0.27) mm	0.28 (0.100)	Burger et al. (2007)
Pacific halibut — *Hippoglossus stenolepis* (muscle)					
Bering Sea	N/A	152	2000–68,000 g	0.15	Hall et al. (1976a)
Gulf of Alaska	N/A	761	2000–>68,000 g	0.20	Hall et al. (1976a)
SE. Gulf of Alaska	N/A	70	2000–>68,000 g	0.26	Hall et al., 1976a
SE. Gulf of Alaska	1971–74	10 (3)	900 (122) mm	0.06 (0.370)	Hall et al. (1978)

N/A = data not available.
ND = not detected.
[a] Number in () is fish in which mercury was not detected and not included in mean THg.
[b] Length for salmon is mid-eye to fork of tail; length for other fishes is unspecified.
[c] Converted from dry weight; dry weight × 25% solids (source: Muller, K., USFWS, Personal Communication).

MeHg concentrations were 78% THg in muscle and salmon liver showed a lower relative abundance of MeHg (68%). Chinook salmon eggs have higher THg concentrations than those of other salmon species (Zhang et al., 2001). The high mean concentrations of both forms of Hg in Chinook salmon muscle are related to factors such as: (1) their larger size and thus longer ocean period, and (2) their piscivorous diet as adults. Recent results from a study conducted by the ADEC (2007) also documented that mean MeHg concentrations were well below the USFDA action level of 1.0 mg kg^{-1} in the most frequently consumed marine fishes (11 species) from Alaska, including the five Pacific salmon (http://www.state.ak.us/dec/deh/animal/fm-heavymetals.htm). No information was available concerning fish size and sampling location, thus the ADEC data are not included in Table 2.

In two subsistence and commercial marine fishes, Pacific halibut (*Hippoglossus stenolepis*) and sablefish (*Anaplopoma fimbria*), from the northeast Pacific Ocean, Hg concentrations in muscle approach the USEPA critical value for human consumption (0.3 mg kg^{-1}), but do not approach the USFDA action level for human consumption (1.0 mg kg^{-1}). In the early 1970s Hg concentrations in halibut and sablefish increased with decreasing latitude, with mean values from southeast Alaska of 0.26 (range 0.04–1.30) and 0.28 (range 0.06–0.77) mg kg^{-1}, respectively (Hall et al., 1976a, b) (Table 1). More recent testing of halibut and sablefish in Alaska revealed MeHg concentrations are similar in halibut (mean 0.22 [range 0.04–0.88] mg kg^{-1}), but lower in sablefish (mean 0.08 [range 0.01–0.21] mg kg^{-1}), to Hg concentrations of 30years earlier (ADEC, 2007).

2.3. Mercury exposure in Alaska

Hg was detected in food samples prepared from Alaska fishes (Rothschild and Duffy, 2002b). The range of Hg in prepared fish, mainly salmon, in which Hg was detected was greater than in raw fish. For example, half-dried adult sockeye salmon (*O. nerka*) (a Yup'ik food) from the lower Kuskokwim River had mean (SD) THg concentrations of 0.12 (0.072) mg kg^{-1} (Rothschild and Duffy, 2002b), while raw adult sockeye salmon muscle from the same location had considerably lower THg,

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i.e., mean 0.05 (0.005) mg kg^{-1} (Zhang et al., 2001). Chicourel et al. (2001) reported that MeHg is not removed from fish tissue by common cooking methods. Hg, including MeHg, remains bound to fish muscle, most likely as MeHg cysteine (Harris et al., 2003) during cooking (boiled) and drying. In one published study of Arctic char (*Salvelinus alpinus*), an Inuit food, Hg concentrations increased four- to six-fold during cooking and drying (Chan et al., 1995). Thus, cooking and drying subsistence foods like fish tends to remove the water without removing the Hg, resulting in elevated Hg concentrations. Nevertheless, dried or half-dried sockeye salmon (0.12 mg kg^{-1}), Chinook salmon (*O. tshawytscha*) (0.06 mg kg^{-1}), Alaska blackfish (*Dallia pectoralis*) (0.15 mg kg^{-1}), and Alaska whitefish (*Coregonus nelsoni*) (0.05 mg kg^{-1}) are still lower than the USEPA critical value for human consumption, 0.3 mg kg^{-1} (Rothschild and Duffy, 2002b).

On the commercial side, Endo et al., (2003) reported that some whale products sold in Japan for human consumption had high THg concentrations. Many of these whales where caught in the North Pacific. The THg concentrations in odontocete red meats ranged from 0.5 to 81 mg kg^{-1}. The false killer whale (27.3–81 mg kg^{-1}) and striped dolphin (1–63.4 mg kg^{-1}) had the highest Hg concentrations. The mystecete whales, in contrast, had meats with low concentrations of THg (0.01–0.54 mg kg^{-1}) reflecting their feeding at lower trophic levels (Endo et al., 2003).

Hg exposure has been documented in Arctic and sub-Arctic populations since the 1970's. Hair has been used as a surrogate indicator for monitoring human exposure to Hg because Hg concentrations in human hair reflect Hg concentrations in blood and internal organs, including the brain. Hair accumulates MeHg after its ingestion into the body and hair concentrations have correlated with concentrations in the blood at the time hair is being formed in the scalp. Cerneichiari et al., (1995) suggested using maternal hair for biomonitoring in prenatal studies. Mean hair Hg concentrations in Alaskan salmon-eating human populations are usually 3 mg kg^{-1} or less (Table 3). In 1976, Galster (1976) reported a range of 3.6 mg kg^{-1} (Yukon–Kuskokwim interior) to 4.3 mg kg^{-1} (Y–K coastal) for mean hair concentrations. Recent data (Rothschild and Duffy, 2002a; AKDHSS, 2003) indicate that riverside populations of subsistence users in western Alaska tend to have Hg concentrations less than 3 mg kg^{-1} in hair, probably because salmon is their main source of proteins. However, the average value of MeHg, while low, was still higher than the value of MeHg in urban areas like Fairbanks, Alaska. The availability of market food is believed to be part of the decline in the use of traditional plant foods such as berries and the use of fish and wildlife (Hansen and Gilman, 2005).

In non-fish consumers, blood values are around 0.002 μg L^{-1}, while in people who consume large amounts of fish, means around 0.02 to 0.04 μg L^{-1} are observed. The average half-life in human blood for MeHg in adults is 70days, in children 90days, and in lactating women 46days (Hightower and Moore, 2003). Dallaire et al. (2003) studied time trends of Hg in umbilical cord blood of Inuit infants born in Nunavik, Canada. Both cord blood and maternal hair Hg concentrations are associated with maternal blood concentrations (Stern and Smith, 2003; Bjornberg et al., 2003). In the Inuit cord bloods a significant reduction in Hg concentrations were found, but there was no clear linear or exponential trend. Since the high trophic level of the traditional Inuit diet is responsible for their Hg exposure, Dallaire et al. (2003) proposed that the observed decrease in Hg concentrations could be explained by a combination of decrease in Hg food contamination and by changes in dietary habits. Market food with increased carbohydrates, chicken and pork has a lower trophic level then does traditional food and will be less contaminated by Hg (Hansen and Gilman, 2005).

In Alaska, AMAP reported that mothers from the Barrow region had blood Hg concentrations of 1.3 μg L^{-1} compared to the Bethel region of 5.5 μg L^{-1} which agrees with hair concentrations (AMAP, 2002). A common factor of 1:250 has been proposed to convert

Table 3
Studies of human hair mercury concentrations in Alaska

Author agency and year	Location	Mean (mg kg^{-1})	SD or range	*N*
CDC 1972 (in Egeland et al., 1998)	Pribilof Islands natives	4.6	1.0	28
	Pribilof Islands whites	3.4	2.2	6
AKDHSS 1972 (in Egeland et al., 1998)	Pribilof Islands	5.8	0.3–13.2	13
	Bethel mothers	5.1	1.5–9.1	14
	Juneau	1.5	0.7–2.4	8
	Y–K river villages	1.2	0.0–0.3	56
Galster (1976)	Y–K coastal	4.3	0.6	12
	Y–K interior	3.6	0.7	6
	Urban	4.0	0.8	4
Lasorsa (1994)	Nome women	1.4	1.0	80
Rothschild and Duffy (2002a)	Napakiak	1.4 [a]	1.0	16
Rothschild and Duffy (2002a)	Fairbanks	0.2 [a]	0.1	20
AKDHSS (2003)	Pregnant women in 19 communities	0.65	ND-3.48	125

[a] Methylmercury; source: Egeland et al. (1998).

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human hair to whole blood (Mahaffey, 1999; Hightower and Moore, 2003), e.g. 4.0 μg $L^{-1} \times L$ / 1000g × 250 = 1.0 μg g^{-1} (mg kg^{-1}). For brain, when the initial distribution is complete the brain:blood concentration ratio is between 10:1 and 5:1.

To evaluate the possible human health impacts of Hg exposure from ingestion, various factors must be considered (Chapman and Chan, 2000; Chan et al., 1995). Exposure varies with percentage of different fish species in an individual's diet, as well as the amount consumed (Abe et al., 1995; Hightower and Moore, 2003). Exposure assessments were conducted on seven mature Alaska fishes from western Alaska — northern pike, Arctic grayling, whitefish, and Chinook, chum, coho, and sockeye salmon (Zhang et al., 2001; Jewett et al., 2003). Only pike exceeded the USFDA action level of 1.0 mg kg^{-1} and the USEPA critical value for human consumption of 0.3 mg kg^{-1}. The USEPA methods (USEPA, 1997b; Huggett et al., 2001) for analysis of human exposure to Hg through the ingestion of fish were followed by using the following equation to calculate exposure to adults and children:

$$\text{Intake}\left(\text{mg kg}^{-1}\text{d}^{-1}\right) = \frac{\text{CF} \times \text{IR} \times \text{EF} \times \text{ED}}{\text{BW} \times \text{AT}}$$

where CF is the mean Hg concentration in fish (mg kg^{-1}), IR is the ingestion rate (kg meal), EF is the exposure frequency (meals y^{-1}), ED is the exposure duration (y), BW is the body weight (kg), and AT is the averaging time (ED × 365d y^{-1}). The mean MeHg muscle value of each species was used for the concentration (CF); ingestion rate for adults and children was 0.227kg meal; exposure frequency for adults and children was 48meals y^{-1} (Huggett et al., 2001); average body weight for adults was 70kg, for children 14.5kg; it was assumed that 100% of the fish meals were from each species and that 100% of MeHg ingested is absorbed (Huggett et al., 2001).

The hazard index (HI) was determined for the seven fishes listed above. HI was calculated by dividing the ingestion by reference dose (RfD) for MeHg of 1.0×10^{-4} mg kg^{-1} d^{-1} (USEPA, 1997b). RfD is the dose that can be consumed daily over a lifetime without ill effects. A HI less than one indicates that no toxicity is expected to occur in individuals who consume the fish (USEPA, 1998). A HI greater than one generally suggests that a toxic stressor is present at a concentration that may pose risk to receptors. However, the relative risks associated with HIs of 1, 10 or greater are uncertain. For example, an HI of 10 may imply a greater risk but whether it is 10 times greater than 1 depends on many factors such as exposure scenario and the dose–response relationships and species differences. HI is only an estimate of risk and useful only as a screening tool (Huggett et al., 2001). Monthly consumption rates (CR) were calculated using the equation (USEPA, 1997b)

$$\text{CR}\left(\text{meals mo}^{-1}\right) = \frac{\text{RfD} \times \text{BW}}{\text{CF}} \times \frac{\left(30.44 \text{ d mo}^{-1}\right)}{\text{IR}}$$

where RfD is the reference dose (1.0×10^{-4} mg kg^{-1} d^{-1}), BW the body weight, CF the MeHg concentration in fish (mg kg^{-1}) and IR is the ingestion rate (0.227kg meal).

Based on the USEPA guidelines, there is little exposure from ingestion of grayling, whitefish or the four salmon species in western Alaska. The ingestion concentration from a 100% pike diet (48meals y^{-1}) would be 18×10^{-4} mg kg^{-1} d^{-1} MeHg (Fig. 4A) or a Hazard Index (HI) of 18 for an average child (Fig. 4B), while grayling have a HI of 3 for children. Salmon and whitefish have a HI around 1 and below for both children and adults (Fig. 4B). The HI for pike and grayling may indicate that pike and grayling consumption should be kept at a minimum for children (Fig. 5). Consuming 100% grayling is only hazardous for children while it is hazardous for all groups to consume large amounts of northern pike. Because of children's body size, it is more hazardous for them to consume pike and grayling than for the adults. Consumption limits for adults show that 16 Chinook salmon meals or 31 sockeye salmon meals may be eaten per month, while 1 pike or 6 grayling meals may be eaten per month (Fig. 5). Children have a consumption limit of 3 Chinook salmon or 6 sockeye salmon meals a month, 0.2 pike meals a month (2meals per year), or 1 grayling meal a month. Subsistence harvests of resident freshwater fishes, like pike and grayling, may increase if Pacific salmon catches continue to decline in the Yukon and Kuskokwim rivers.

The National Academy of Sciences (NAS, 2000) recommended keeping the whole blood Hg concentration at less than 5.0 μg L^{-1} or the hair concentration at less than 1.0 mg kg^{-1}. The USEPA (2001a) current reference dose per day (RfD) is an uncertain estimate of the daily exposure to the human population that should not cause deleterious effects during a person's lifetime (Mahaffey, 1999). With an RfD of 1×10^{-4} mg kg^{-1} d^{-1} of body weight (NAS, 2000), a 70kg person could ingest 0.07 mg Hg a day without risk, but there are different risk indicators used by other government agencies. ATSDR, (1999) uses a minimum risk level (MRL) of 3×10^{-4} mg kg^{-1} d^{-1}; however, ATSDR has stated that MRL's are not intended to be used in developing fish

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advisories. The USFDA level of 1.0 mg kg^{-1} is an action level calculated from an RfD of 5×10^{-4} mg kg^{-1} d^{-1}. The NAS has found the USEPA levels to be justifiable based on the latest evidence but also notes the need for further research to understand potential risk to a fetus (NAS, 2000). The uncertainties in the USEPA equations become apparent when applying this methodology to Alaska, especially to subsistence food users. Further, the actual amount of each species consumed is needed to develop a more complete exposure assessment (Hightower and Moore, 2003; Burger et al., 2001). At this time, such data are not available in Alaska.

As salmon is a common food for subsistence users in western Alaska (Nobmann et al., 1992), the bioconcentration factor (THg in fish/THg in water) for THg in these Bering Sea region salmon ranged around 2.5×10^{4} and varied 2-fold between salmon species. Despite this bioconcentration, the Hg concentrations in the Alaskan salmon do not exceed the USEPA critical value for humans. It is always important to note the location of fish, i.e. Alaskan salmon versus Great Lakes salmon because local conditions will influence exposure of salmon to Hg during the salmon's lifecycle. This low exposure to Hg by Alaskan subsistence users is supported by the Hg in hair data (Table 3).

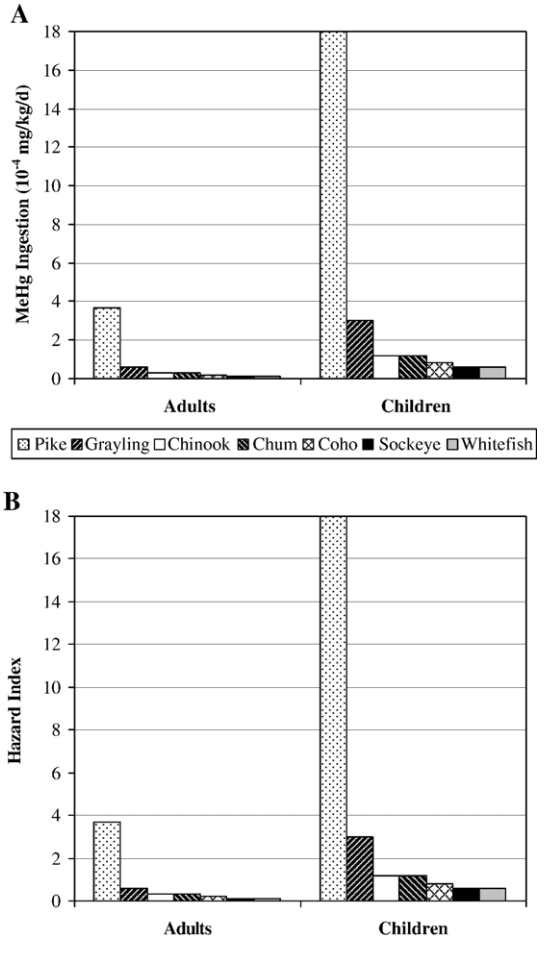

Fig. 4. MeHg ingestion (A) and Hazard Index, HI (B) associated with human consumption on 100 percent of the individual fish species from western Alaska (MeHg HI = MeHg ingestion/RfD; RfD = 1.0×10^{-4} mg kg^{-1} d^{-1}) (source: Jewett *et al* 2003).

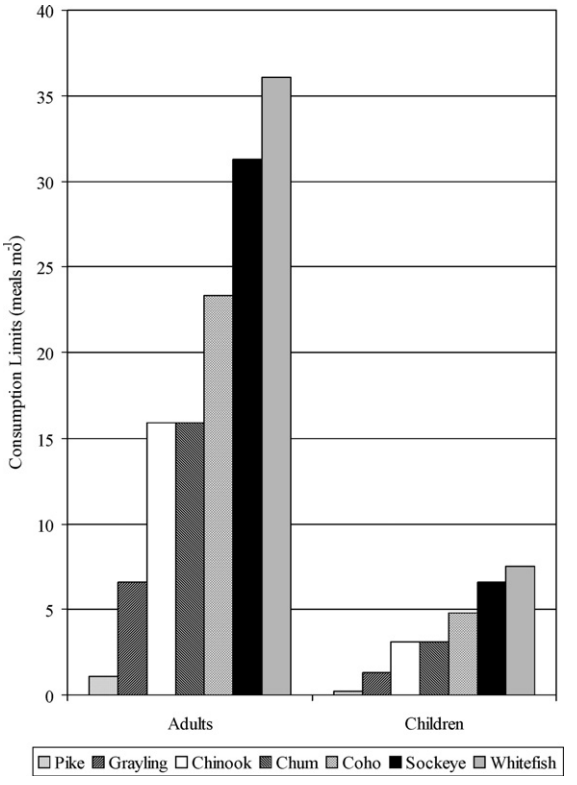

Fig. 5. Estimated consumption limits associated with human consumption of 100 percent individual fish species from western Alaska (source: Jewett *et al* 2003).

3. Discussion

With the increase in knowledge of the health hazards of MeHg, monitoring for spatial and temporal changes in MeHg in the environment, human foods, and humans is essential. Recent studies have reported that even low concentration exposure to Hg in food is associated with an increased risk of neurochemical (Aschner, 2002; Weil et al., 2005) or cardiovascular damage (Bogler and Schwetz, 2002; Meyers et al., 2000; Sorensen et al., 1999) (also see Arnold and Middaugh, 2004 for review). Currently there is insufficient historical data to address whether Hg concentrations in Alaska salmon have increased, decreased or not changed in recent years.

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However, regarding northern pike in western Alaska, there does not appear to be any change in Hg concentrations over the past two decades (Jewett et al., 2003). The temporal trends of Hg over the past 20–30years in the Canadian Arctic are inconsistent, with some animal populations exhibiting significant increases in Hg (e.g., eggs of some seabirds, beluga whales), whereas others are not (e.g., ringed seals, polar bears) (Braune et al., 2005). Temporal trend monitoring in the Canadian sub-Arctic and Arctic has revealed little evidence of declining Hg concentrations in fish that can be attributed to declining atmospheric inputs (Evans et al., 2005). Thus, currently it is not possible to determine if anthropogenic Hg is generally increasing in Canadian biota.

Although no consumption advisories have been issued for Hg in Alaska fish, such advisories have been issued for various lakes in the Mackenzie River Basin in the Canadian western Arctic (Evans et al., 2005). Fish mostly targeted to have exceeded Canada's Frequent Consumers of Fish Guideline of 0.2 mg kg^{-1} and the Commercial Sale of Fish Guideline of 0.5 mg kg^{-1} included northern pike, lake trout (*Salvelinus namaycush*) and walleye. Northern pike were investigated in 17 Yukon and 71 Northwest Territories (NT) lakes; 53% of Yukon and 92% of NT lakes had mean Hg concentrations >0.2 mg kg^{-1}, while 12% of Yukon and 77% of NT lakes had mean Hg concentrations >0.5 mg kg^{-1} (Evans et al., 2005). Small lake size, fish age, and predaceous feeding behavior were determined to be the primary variables affecting higher Hg concentrations in fish in the Mackenzie River Basin lakes. Hg concentrations in pike from these lakes were similar to values noted in pike from western Alaska.

The rationale for the State of Alaska to use the USFDA guideline of 1 mg MeHg kg^{-1} of fish, rather than smaller values such as the USEPA fish tissue residue criterion of 0.3 mg kg^{-1}, as the safe level for human consumption was provided by Arnold and Middaugh (2004).

The rationale is based on several factors, chiefly:

- MeHg concentrations in the most frequently consumed fish (e.g., salmon, cod, halibut, pollock, sole, and herring) are very low;
- Extensive scientific research has documented the numerous health, social and cultural, and economic benefits of eating fish;
- The subsistence lifestyle and diet are of great importance to the self-determination, cultural, spiritual, social, and overall health and well being of Alaska Natives; and
- The preponderance of data indicates the known benefits of fish consumption far outweigh the theoretical and controversial potential adverse health effects from MeHg found in Alaska fish.

Alaska's guideline for safe concentrations of MeHg in fish of ≤1 mg kg^{-1} seems appropriate for most fishes consumed by humans, since only one out of 41 species (n=2692) reviewed had any mean MeHg concentrations that exceeded 1 mg kg^{-1} (Tables 1 and 2). However, the exception may be the piscivorous and long-lived species like northern pike. Pike tested in various studies in western Alaska had mean MeHg concentrations in muscle tissue exceeding 0.3 mg kg^{-1} and some with mean values exceeding 1 mg kg^{-1} (Table 1; Jewett et al., 2003). Alaska pike/Hg data summarized in Arnold and Middaugh (2004) revealed the mean concentration of MeHg exceeded 0.5 mg kg^{-1} in 8 of 15 data sets; MeHg exceeded 0.3 mg kg^{-1} in 11 of 15 data sets. This may be a cause for alarm, especially since many Native households or communities in western Alaska rely considerably on pike as subsistence food, especially during winter months when fresh salmon are not available. Calculations, using the recent MeHg concentrations in pike muscle from western Alaska, revealed that adults should only consume one pike meal per month and children should only consume two pike meals per year (Fig. 5). Perhaps the State of Alaska should reconsider issuing local fish advisories for pike in select communities or drainages, especially targeting the Hg-sensitive groups. Applying the more restrictive USEPA screening value of 0.3 mg kg^{-1} would seem prudent for pike. Other states, albeit more industrialized than Alaska, use the more restrictive USEPA screening value of 0.3 mg kg^{-1} rather than the USFDA action level of 1 mg kg^{-1} (e.g., Utah: Ball, 2007). Also, as noted above, Canada's fish/Hg guidelines are more restrictive than Alaska. Whatever guidelines are used in Alaska for human consumption of fish, expanded biomonitoring for MeHg exposure is crucial, particularly for the Hg-sensitive populace in certain regions of the state.

4. Conclusion

The marine waters of Alaska contain one of the world's most important fisheries, with over half the fish harvested for consumption in the United States. Alaska provides 0.5% of fish to the global market and 15% to the domestic market for human consumption (Bechtel and Crapo, 2002). Fish taken from Alaska are valued at local, state, national, and global concentrations, and are inextricably linked to economic prosperity and cultural

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identity. These links are direct and indirect, and may be expressed at one scale through the use and preservation of the marine environment, and at another through local and regional responses to larger scale ecosystem drivers.

Recent dramatic global decreases in commercially valuable and culturally important marine species press for the need to understand the coupled dynamics of human and natural systems in the marine setting over long and short periods (Cannon et al., 1999). Human systems have intersected with and disturbed marine resources worldwide since people first began to live on the coast and to fish, but the nature and magnitude of food contamination varies spatially and temporally in conjunction with fishing practices, with the nature of human settlement, and with perturbations in the natural system (Fleming et al., 1995). Contamination from human activity has intensified with the occurrence of industrial enterprise, including mining and oil production (ADFG, 1999b; Picou, 1990). The issue of Hg in feed from farmed fishes and fish-feed has also attracted attention (Burger et al., 2001; Easton et al., 2002).

Contemporary communities throughout Alaska depend in various ways on fishing and/or on the use of coastal resources. Fish are nutritionally satisfying, but can contain low concentrations of Hg. Alaskan Natives generally consume much more fish than the national average. Whether for regional economic stability, subsistence, or for some combination of both, making a living offshore continues to be important for individual, group and community income, for individual and group identity, and for real and perceived individual and community health issues.

This review summarized the muscle Hg concentrations in 41 species of fish (17 freshwater and 24 anadromous/marine species), consisting of 2692 specimens from numerous studies. Most of these fish are subsistence food of Alaskans. Analyses of Hg concentrations in these fish generally showed most fish a safe to eat, usually below the USFDA's Action Level and Alaska's guideline for safe concentrations of MeHg in edible fish, i.e., 1 mg kg^{-1} (wet wt.). The notable exception was the northern pike (*E. lucius*). The deposition of Hg at high latitudes and new studies showing effects at low concentrations on neurological and cardiovascular function suggest fish and fish-derived foods should be monitored on a routine basis.

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References

Abe T, Ohtsuka R, Hongo T. High hair and urinary mercury levels of fish eaters in the nonpolluted environment of Papau New Guinea. Arch Environ Health 1995;50:367–73.

AKDHSS. Alaska mercury biomonitoring: statewide maternal hair mercury biomonitoring program. State of Alaska, Alaska Epidemiology Bull; 2003. No. 20.

Alaska Department of Environmental Conservation (ADEC). Publishing on the Internet, Anchorage, Alaska. Online. Available HTTP: http://www.dec.state.ak.us/eh/vet/heavy_metals.htm (accessed 1 June, 2007).

Alaska Department of Fish and Game (ADFG). Annual management report—Bristol Bay area. Anchorage, Alaska: Division of Commercial Fisheries, ADFG; 1999a.

Alaska Department of Fish and Game (ADFG). Subsistence harvests and uses in eight communities ten years after the Exxon Valdez oil spill. Technical Paper No. 252; 1999b.

Alaska Department of Fish and Game (ADFG). Alaska substance fisheries—1999 annual report. Division of Subsistence, ADFG; 2001. Juneau, Alaska, 154 pp.

Arctic Monitoring and Assessment Programme (AMAP). Arctic Pollution issues: a state of the arctic environment report; 1997. Oslo, Norway.

Arctic Monitoring and Assessment Programme (AMAP). Arctic pollution 2002; 2002. Oslo, Norway, 112 pp.

Ariza ME, Williams MV. Lead and mercury mutagenesis: type of mutation dependent upon metal concentration. J Biochem Mol Toxicol 1999;13:107–12.

Arnold SM, Middaugh JP. Use of traditional foods in a healthy diet in Alaska: risks in perspective. Mercury, State of Alaska, 8(11). Epidemiol Bull, Second Edition, vol. 2; 2004.

Aschner M. Astrocytes as modulators of mercury-induced neurotoxicity. Neurotoxicology 1996;17:663–9.

Aschner M. Neurotoxic mechanisms of fish-borne methylmercury. Environ Toxicol Pharmacol 2002;12:101–4.

Aschner M, Clarkson TW. Distribution of mercury 203 in pregnant rats and their fetuses following systemic infusions with thio-containing amino acids and glutathione during late gestation. Teratol 1988;38:145–55.

Aschner M, Eberle NB, Goderie S, Kimelberg HK. Methylmercury uptake in rat primary astrocyte cultures: the role of the neutral amino acid transport system. Brain Res 1990;521:221–8.

Please cite this article as: Jewett SC, Duffy LK. Mercury in fishes of Alaska, with emphasis on subsistence species. Sci Total Environ (2007), doi:10.1016/j.scitotenv.2007.07.034

Aschner M, Viterella D, Allen JW, Conklin DR, Cowan KS. Methylmercury-induced astrocytic swelling is associated with activation of the Na^+/H^+ antiporter, and is fully reversed by amiloride. Brain Res 1998;799:207–14.

Atchison WD, Hare MF. Mechanisms of methylmercury induced neurotoxicity. Fed Am Soc Exp Biol J 1994;8:622–9.

Agency for Toxic Substances and Disease Registry (ATSDR). Toxicology profile for mercury. Washington, D.C.: U.S. Department of Health and Human Services; 1999.

Aulerich RJ, Ringer RK, Iwamoto S. Effects of dietary mercury on mink. Arch Environ Contam Toxicol 1974;2:43–51.

Baldi F. Microbial transformation of mercury species and their importance in the biogeochemical cycle of mercury. In: Sigel A, Sigel H, editors. Metal ions in biological systems, vol 34. Marcel Dekker; 1997. p. 213–57.

Ball RW. An evaluation of mercury concentrations in fish sampled from streams, lakes and reservoirs in Utah for years 2004–2006. Final report to the Utah Department of Health; 2007. 51 pp.

Barkay T. The mercury cycle, in encyclopedia of microbiology, vol. 3. Academic Press; 2000. p. 117–81.

Barregard L, Lindstedt G, Schultz A, Sallsten G. Endocrine function in mercury exposed chloralkali workers. Occup Environ Med 1994;52: 536–40.

Bechtel PJ, Crapo CA. Alaska Fish Processing Byproducts. in Proceedings of the 53rd Arctic Science Conference, American Association for the Advancement of Science. Connectivity in northern waters; 2002. p. 18–21. September, Fairbanks.

Bemis JC, Seegal RF. Polychlorinated biphenyls and methylmercury act synergistically to reduce rat brain dopamine content in vitro. Environ Health Perspect 1999;107:879–85.

Bemis JC, Seegal RF. Polychlorinated biphenyls and methylmercury alter intracellular calcium concentrations in rat cerebellar granule cells. Neurotoxicology 2000;21:1123–34.

Ben-David M, Bowyer RT, Duffy LK, Roby DD, Schell DM. Social behavior and ecosystem processes: effects of river otter latrine sites on nutrient dynamics of terrestrial vegetation. Ecology 1998;79:2567–71.

Ben-David M, Duffy LK, Blundell GM, Bowyer RT. Natural exposure of coastal river otters to mercury: relation to age, diet and survival. Environ Toxicol Chem 2001;20:1986–92.

Bilby RE, Fransen BR, Bisson PA. Incorporation of nitrogen and carbon from spawning coho salmon into the trophic system of small streams: evidence from stable isotopes. Can J Fish Aquat Sci 1996;53:164–73.

Bjornberg KA, Vahter M, Petersson-Grawe K, Glynn A, Cnattingius S, Darnerud PO, Atuma S, Aune M, Becker W, Berglund M. Methyl mercury and inorganic mercury in Swedish pregnant women and in cord blood: influence of fish consumption. Environ Health Perspect 2003;111:637–41.

Bloom NS. On the chemical form of mercury in edible fish and marine invertebrate tissue. Can J Fish Aquatic Sci 1992;49:1010–7.

Boening DW. Ecological effects, transport, and fate of mercury; a general review. Chemosphere 2000;40:1335–51.

Bolger PM, Schwetz BA. Mercury and health. New Engl J Med 2002;347:1735–6.

Braune B, Muir D, Demarch B, Gamberg M, Poole K, Currie, R, et al. Spatial and temporal trends of contaminants in Canadian Arctic freshwater and terrestrial ecosystems: a review. Sci Total Environ 1999;230:145–208.

Braune BM, Outridge PM, Fisk AT, Muir DCG, Helm PA, Hobbs, K, et al. Persistent organic pollutants and mercury in marine biota of the Canadian Arctic: an overview of spatial and temporal trends. Sci Total Environ 2005;351–352:4-56.

Brown CL, Burr J, Elkin K, Walker RJ. Contemporary subsistence uses and population distribution of non-salmon fish in Grayling, Anvik, Shageluk, and Holy Cross. Federal Subsistence Fishery Monitoring Program. Final project report no. 02-037-2. Anchorage, Alaska: USFWS Office of Subsistence Management, Fisheries Resource Monitoring Program, Fishery Information Service; 2005.

Burger J, Gaines KF, Gochfeld M. Ethnic differences in risk from mercury among Savannah river fishermen. Risk Anal 2001;21: 533–44.

Burger J, Gochfeld M, Jeitner C, Burke S, Stamm T. Metal levels in flathead sole (*Hippoglossoides elassodon*) and great sculpin (*Myoxocephalus polyacanthocephalus*) from Adak Island, Alaska: potential risk to predators and fishermen. Environ Res 2007;103(1): 62–9.

Cannon A, Schwartz HP, Knyf M. Marine-based subsistence trends and the stable isotope analysis of dog bones from Namu, British Colombia. J Archaeol Sci 1999;26:399–407.

Cerneichiari E, Brewer R, Meyers JG. Monitoring methylmercury during pregnancy: maternal hair predicts fetal brain exposure. Neurotoxicology 1995;16:705–10.

Chan HM, Kim C, Khoday K, Receuevr O, Kuhnlein HV. Assessment of dietary exposure to trace metals in Baffin Inuit Food. Environ Health Perspect 1995;103:740–6.

Chapman L, Chan HM. The influence of nutrition on methylmercury intoxication. Environ Health Perspect 2000;108:29–51.

Chicourel EL, Sakuma AM, Zenebon O, Tenuta-Filho A. Inefficacy of cooking methods on mercury reduction from shark. Arch Latinoam Nutr 2001;51(3):288–92.

Clarkson TW. The toxicology of mercury. Crit Rev Clin Lab Sci 1997;34: 369–403.

Compeau GC, Bartha R. Sulfate-reducing bacteria: principal methylators of mercury in anoxic estuarine sediment. Appl Environ Microbiol 1985;50:498–502.

Dallaire F, Dewailly E, Muckle G, Ayotte P. Time trends of persistent organic pollutants and heavy metals in umbilical cord blood of Inuit infants born in Nunavik (Canada) between 1994 and 2001. Environ Health Perspect 2003;111:1660–4.

Danborgy-Englund G, Ask K, Belfrager E, Ekstrand J. Mercury exposure in utero and during infancy. J Toxicol Environ Health 2001;A63: 317–20.

Dansereau M, Larviviere N, Du Tremblay D, Belanger D. Reproductive performance of two generations of female semi-domesticated mink fed diets containing organic mercury contaminated freshwater fish. Arch Environ Contam Toxicol 1999;36:221–6.

Daum JR, Shepherd DM, Noelle RJ. Immunotoxicology of cadmium and mercury on B-lymphocytes. Int J Immunopharmacol 1993;15: 383–94.

Dehn LA, Sheffield GG, Follmann EH, Duffy LK, Thomas DL, Bratton, GR, et al. Trace elements in tissues of phocid seals harvested in the Alaskan and Canadian Arctic — influence of age and feeding ecology. Can J Zool 2005;83:726–46.

Dehn LA, Follmann EH, Thomas DL, Sheffield GG, Rosa C, Duffy, LK, et al. Trophic relationships in an Arctic food web and implications for trace metal transfer. Sci Total Environ 2006;362: 103–23.

Derksen AJ, Green DJ. Total mercury concentrations in large fishes from lakes on the Churchhill River diversion and Nelson river. Technical appendices to the summary report, Canada–Manitoba agreement on the study and monitoring of mercury in the Churchhill River diversion, vol. 4. Published by the Governments of Canada and Manitoba; 1987. Chap. 17.

Duffy LK, Scofield E, Rodgers T, Patton M, Bowyer RT. Comparative baseline levels of mercury, Hsp 70 and Hsp 60 in subsistence fish

Please cite this article as: Jewett SC, Duffy LK. Mercury in fishes of Alaska, with emphasis on subsistence species. Sci Total Environ (2007), doi:10.1016/j.scitotenv.2007.07.034

from the Yukon–Kuskokwim delta region of Alaska. Comp Biochem Physiol, Part C 1999;124:181–6.

Easton MDL, Luszniak D, Von der Geest E. Preliminary examination of contaminant loading in farmed salmon, wild salmon and commercial salmon feed. Chemosphere 2002;46:1053–74.

Egeland GM, Middaugh JP. Balancing fish consumption and benefits with mercury exposure. Science 1997;278:1904–5.

Egeland GM, Feyk LA, Middaugh JP. Use of traditional foods in a healthy diet in Alaska: risks in perspective. State of Alaska. Epidemiol Bull 1998;2(1).

Endo T, Hotta Y, Haragughi K, Sakata M. Mercury contamination in the red meat of whales and dolphins marketed for human consumption in Japan. Environ Sci Technol 2003;37:2681–5.

Evans MS, Muir D, Lockhart WL, Stern G, Ryan M, Roach P. Persistent organic pollutants and metals in the freshwater biota of the Canadian subarctic and Arctic: an overview. Sci Total Environ 2005;351–352:94-147.

Ewald G, Larsson P, Linge H, Okla L, Szarzi N. Biotransport of organic pollutants to an inland Alaska lake by migrating sockeye salmon (*Oncorhynchus nerka*). Arctic 1998;51:40–7.

Fall JA, Koster D, Davis B. Subsistence harvests of Pacific halibut in Alaska, 2005. Alaska Department of Fish and Game, Division of Subsistence, Juneau, AK; 2006. Technical Paper No. 320.

Fitzgerald WF, Engstrom DR, Mason RP, Nater EA. The case of atmospheric mercury contamination in remote areas. Environ Sci Technol 1998;32:1-10.

Fleming LE, Watkins S, Kaderman R, Levin B, Ayyar DR, Bizzio, M, et al. Mercury exposure in humans through food consumption from the Everglades of Florida. Water, Air Soil Pollut 1995;80: 41–8.

Frery N, Mavry-Brachet R, Maillot E, Deheeger M, deMerona B, Boudou A. Gold mining activities and mercury contamination of native Amerindian communities in French Guinea: key role of fish in dietary uptake. Environ Health Perspect 2001;109:449–56.

Friedmann AS, Chen H, Rabuck LD, Zirkin R. Accumulation of dietary methylmercury in the testes of the adult brown Norway rat: Impaired testicular and epididymal function. Environ Toxicol Chem 1998;17:867–71.

Galster WA. Mercury in Alaskan Eskimo mothers and infants. Environ Health Perspect 1976;15:135–40.

Gilbert SG, Grant-Webster S. Neurobehavioral effects of developmental methylmercury exposure. Environ Health Perspect 1995;103: 71–87.

Goyer RA, Clarkson TW. Toxic Effects of Metals. In: Klaussen CD, editor. Casarett and Dull's toxicology: the basic science of poisons. New York: McGraw-Hill; 2001. p. 811–67.

Grandjean P, Jorgensen PJ, Weihe P. Human milk as a source of methylmercury exposure in infants. Environ Health Perspect 1994;102: 74–77.

Grandjean NT, Weihe P, White RP, Debes F, Aiaki S, Yokoyama, K, et al. Cognitive deficit in 7-year old children with prenatal exposure to methylmercury. Neurotoxicol Teratol 1997;19:417–28.

Grandjean P, Murata K, Budtz-Jorgensen E, Weihe P. Cardiac autonomic activity in methylmercury neurotoxicity: 14-year follow-up or FAROESE birth cohort. J Pediatr 2004;144:169–76.

Gray JE, Theodorakos PM, Budahn J, O'Leary RM. Mercury in the environment and its implications, Kuskokwim River region, southwestern Alaska. U.S. Geol Surv Bull 1994:2107: 3–13.

Gray JE, Meier AL, O'Leary RM, Outwater C, Theodorakos, PM. Environmental geochemistry of mercury deposits in southwestern Alaska: mercury contents in fish, stream-sediment, and stream-water samples. U.S. Geol Surv Bull 1996;2152:17–29.

Gray JE, Theoorados PM, Bailey EA, Turner RR. Distribution, speciation and transport of mercury in steam-sediment, stream-water and fish collected near abandoned mercury mines in southwestern Alaska, USA. Sci Total Environ 2000;260:21–33.

Grieb TM, Driscoll CT, Gloss SP, Schofield CL, Bowie GL, Procella DB. Factors affecting mercury accumulation in fish in the upper Michigan peninsula. Environ Toxicol Chem 1990;9:919–30.

Grigal DF. Inputs and outputs of mercury from terrestrial watersheds: a review. Environ Res 2002;10:1-39.

Guallar E, Sanz-Gallardo MI, Van't Veer P, Bode P, Aro A, Gomez-Aracena J, et al. Mercury, fish oils, and the risk of myocardial infarction. New England J Med 2002;347:1747–54.

Halbrook RS, Lewis LA, Aulerich RI, Bursian SJ. Mercury accumulation in mink fed fish collected from streams on the Oak Ridge Reservation. Arch Environ Contam Toxicol 1997;33: 312–6.

Halbrook RS, Jenkins JH, Bush PB, Seabolt ND. Sublethal concentrations of mercury in river otters: Monitoring environmental contamination. Arch Environ Contam Toxicol 1994;27:306–10.

Hall AS, Teeny FM, Lewis LG, Hardman WH, Gauglitz Jr EJ. Mercury in fish and shellfish of the northeast Pacific. I. Pacific halibut, *Hippoglossus stenolepis*. Fish Bull 1976a;74(4):783–9.

Hall AS, Teeny FM, Gauglitz Jr EJ. Mercury in fish and shellfish of the northeast Pacific. II. Sablefish, *Analopoma fimbria*. Fish Bull 1976b;74(4):791–7.

Hall RA, Zook EG, Meaburn GM. National Marine Fisheries Service survey of trace elements in the fishery resource, U.S. Department of Commerce, National Oceanic Atmospheric Administration. Technical report NMFS SSRF-721; 1978. 313 pp.

Hanisch C. Where is mercury deposition coming from? Environ Sci Technol 1998;32:176A–9A.

Hansen JC, Gilman AP. Exposure of Arctic populations to methylmercury from consumption of marine food: an updated risk-benefit assessment. Int J Circumpolar Health 2005;64:121–36.

Harris H, Pickering IJ, George GN. The chemical form of mercury in fish. Science 2003;301:1203.

Headlee PG. Mercury and selenium concentrations in fish tissue and surface waters of the northern unit of the Innoko National Wildlife Refuge (Kaiyuh Flats), west central Alaska, 1993. Tanana Chiefs Conference, Inc. Fairbanks, Alaska. Water resources report 96-3; 1996. 3 pp. + appendix.

Hennig B, Reiterer G, Majkova Z, Oesterling E, Meerarani P, Toborek M. Modification of environmental toxicity by nutrients. Cardiovasc Toxicol 2005;5:153–60.

Hightower JM, Moore D. Mercury levels in high-end consumers of fish. Environ Health Perspect 2003;111:604–8.

Hinck JE, Schmitt CJ, Echols KR, May TW, Orazio CE, Tillitt DE. Environmental contaminants in fish and their associated risk to piscivorous wildlife in the Yukon River Basin, Alaska. Arch Environ Contam Toxicol 2006;51:661–72.

Huggett DB, Steevens JA, Allgood JC, Lutken CB, Grace CA, Benson WH. Mercury in sediment and fish from North Mississippi Lakes. Chemosphere 2001;42:923–9.

Jackson TA. Biological and environmental control of mercury accumulation by fish in lakes and reservoirs of Northern Manitoba. Can J Fish Aquat Sci 1990;4:2449–70.

Jewett SC, Zhang X, Naidu SA, Kelly JK, Dasher D, Duffy LK. Comparison of mercury and methylmercury in northern pike and Arctic grayling from western. Alaska rivers. Chemospere 2003;50: 383–92.

Johnels AG, Westermark T, Berg W, Person PI, Sjostrand B. Pike and some other aquatic organisms in Sweden as indicators of mercury contamination in the environment. Oikos 1967;18:323–33.

Please cite this article as: Jewett SC, Duffy LK. Mercury in fishes of Alaska, with emphasis on subsistence species. Sci Total Environ (2007), doi:10.1016/j.scitotenv.2007.07.034

Kelly EN, Schindler DW, St. Louis VL, Donald DB, Vladicka KE. Forest fire increases mercury accumulation by fishes via food web restructuring and increased mercury inputs. Proc Natl Acad Sci U S A 2006;103(51):19380–5.

Kjellstrom T, Kennedy P, Wallis S, Mantell C. Physical and mental development of children with prenatal exposure to mercury from fish. Stage I: preliminary tests at age 4. Solna, Sweden, National Swedish Environmental Protection Board Report 3080; 1986.

Kjellstrom T, Kennedy P, Wallis S, Steward A, Friberg L, Lind B, et al. Physical and mental development of children with prenatal exposure to mercury from fish. Stage II: interviews and psychological tests at age 6. Solna, Sweden, National Swedish Environmental Protection Board Report 3642; 1989.

Kline TC, Goering JJ, Mathisen OA, Poe PH, Parker PL, Scalan RS. Recycling of elements transported upstream by runs of Pacific salmon: δ^{15}N and δ^{13}C evidence in the Kvichak river watershed, Bristol Bay, southwestern Alaska. Can J Fish Aquat Sci 1993;50: 2350–65.

Komulainen H, Tuomisto J. Effects of heavy metals on monoamine uptake and release in brain synaptosomes and blood platelets. Neurobehav Toxicol Teratol 1982;4:647–9.

Krauss RM, Eckel RH, Howard B, Appel LJ, Daniels SR, Deckelbaum RJ. AHA dietary guidelines—revision 2000: a statement for healthcare professionals from the Nutrition Committee of the American Heart Association. Circulation 2000;102:2284–99.

Krieg T, Chythlook M, Coiley-Kenner P, Holen D, Kamletz K, Nicholson H. Subsistence fisheries assessment: Kvichak River watershed resident species. Federal Subsistence Fishery Monitoring Program. Final project report no. FIS 02-034. Anchorage, AK,: U.S. Fish and Wildlife Service, Office of Subsistence Management, Fisheries Resource Monitoring Program, Fishery Information Service; 2005.

Krummel EM, Macdonald RW, Kimpe LE, Gregory-Eaves I, Demers MJ, Smol, JP, et al. Delivery of pollutants by spawning salmon. Nature 2003;425:255.

Kuhnlein H, Soveida R, Receueur O. Dietary nutrient profiles of Canadian Baffin Island Inuit differ from food source, season and age. J Am Diet Assoc 1996;2:155–62.

Lamborg CH, Fitzgerald WF, O'Donnell J, Torgersen T. A non-steady-state compartmental model of global-scale mercury biogeochemistry with interhemispheric atmospheric gradients. Geochim Cosmochim Acta 2002;66:1105–18.

Laskowski R. Are the top carnivores endangered by heavy metal biomagnification? Oikos 1991;70:387–90.

Lasorsa B. Trends in mercury concentrations in the hair of women in Nome, Alaska: Evidence of seafood consumption or a biotic absorption. In: Wantras CJ, Huckabe JW, editors. Mercury Pollution. Boca Raton, Florida: Lewis Publications; 1994. p. 665–76.

Lebel J, Mergler D, Lucotte M. Evidence of early nervous system dysfunction in Amazonian populations exposed to low-levels of methylmercury. Neurotoxicology 1996;17:157–67.

Lebel J, Mergler D, Branches F, Lucotte M, Amorim M, Larribe, F, et al. Neurotoxic effects of low-level methylmercury contamination in the Amazonian Basin. Environ Res 1998;79:20–32 Section A.

Linke TL, Otero-Montanez JK, Atchinson WD. Evidence for interactions between intracellular calcium stores during methylmercury-induced intracellular calcium dysregulation in rat cerebellar granule neurons. J Pharmacol Exp Ther 2003;304:949–58.

Lundholm CE. Influence of chlorinated hydrocarbons, Hg^{2+} and methylmercury on steroid hormone receptors from eggshell gland mucosa of domestic fowls and ducks. Arch Toxicol 1991;65: 220–7.

MacDonald R, Mackay D, Hickie B. Contaminant amplification in the environment. Environ Sc Technol 2002;34:457a–62a.

Mahaffey KR, Methylmercury: September–October 1999. A look at the risks. Public Health Report, 1999, 114(5): 396-399, 402–413.

Mahaffey KR, Clickner RP, Bodurow CC. Blood organic mercury and dietary mercury intake. National Health and Nutrition Examination Survey, 1999 and 2000. Environ Health Perspect 2004;112: 562–70.

Malm O, Fernando JP, Branchesf ET. Mercury and methylmercury in fish and human hair from the Tapajos river basin, Brazil. Sci Total Environ 1995;175:141–50.

Marvin-DiPasquale M, Agee J, McGowan C, Oremland RS, Thomas M, Krabbenhoft, D, et al. Methyl-mercury degradation pathways: a comparison among three mercury-impacted ecosystems. Environ Sci Technol 2000;34:4908–16.

Mason RP, Fitzgerald WF. Sources, sinks and biogeochemical cycling of mercury in the ocean. In: Bayens W, editor. Global and regional mercury cycles: sources, fluxes and mass balances. Netherlands: Kluwer Academic Publishers; 1996. p. 249–85.

Meyers GJ, Davidson PW, Cox C, Shamlaye C, Cernichiari E, Clarkson TW. Twenty-seven years studying the human neurotoxicity of methylmercury exposure. Environ Res 2000;83:275–85.

Miller OM, Lund BO, Woods JS. Reactivity of Hg(II) with superoxide: evidence for the catalytic dismutation of superoxide by Hg(II). J Biochem Toxicol 1991;6:293–8.

Møller-Madsen B, Danscher G. Localization of mercury in CNS of the rat. IV. The effect of selenium on orally administered organic and inorganic mercury. Toxicol Appl Pharmacol 1991;108:457–73.

Morel FMM, Kraepiel AML, Amoyt M. The chemical cycle and bioaccumulation of mercury. Ann Rev Ecolog Syst 1998;29:543–66.

Mueller KA, Matz AC. Water quality, and metal and metalloid concentrations in water, sediment, and fish tissues from Innoko National Wildlife Refuge, Alaska, 1995–1997, Ecological Services, Fairbanks, Alaska, U.S. Fish and Wildlife Service. Technical Report NAES-TR-02-01; 2002. 155 pp.

Mueller KA, Snyder-Conn E, Doyle T. Contaminant baseline data from water, sediments, and fish of Selawik National Wildlife Refuge, Alaska, 1987–1988, Ecological Services, Fairbanks, Alaska, U.S. Fish and Wildlife Service. Technical report NAES-TR-93-02; 1993. 84 pp.

Mueller KA, Snyder-Conn E, Scannell PO. Metal and metalloid contaminants in water, sediments, and fish of Kanuti National Wildlife Refuge, Alaska, 1985–1990, Ecological Services, Fairbanks, Alaska, U.S. Fish and Wildlife Service. Technical report NAES-TR-95-02; 1995. 125 pp.

Mueller KA, Snyder-Conn E, Bertram M. Water quality, and metal and metalloid concentrations in sediments and fish of Koyukuk, Nowitna, and the northern unit of Innoko National Wildlife Refuges, Alaska, 1991, Ecological Services, Fairbanks, Alaska, U.S. Fish and Wildlife Service. Technical report NAES-TR-96-01; 1996. 79 pp.

Murata K, Weihe P, Budtz-Jorgensen E, Jorgensen PF, Grandjean NT. Delayed brainstem auditory evoked potential latencies in 14 year old children exposed to methylmercury. J Pediatr 2004;144: 177–83.

National Academy of Sciences (NAS) Toxicological Effects of Methylmercury. Washington D.C., 2000. Online. Available HTTP: <http://nap.edu/books/0309071402/html> (accessed 1 June 2007).

National Research Council (NRC). Toxicological effects of methylmercury. Washington, D.C.: National Academy Press; 2000. 344 pp.

Nelson H, Larsen BR, Jenne EA, Sorg DH. Mercury dispersal from lode sources in the Kuskokwim River drainage, Alaska. Science 1978;140:820–4.

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Newland MC. Neurobehavioral toxicity of methylmercury and PCB's: effects-profiles and sensitive populations. Environ Toxicol Pharmacol 2002;12:119–28.

Nobmann ED, Boyers T, Lanier AP, Hankin JH, Jackson ML. The diet of Alaskan native adults: 1987–1988. Am J Clin Nutrition 1992;55: 1024–32.

Nriagu JO. A silent epidemic of environmental metal poisoning. J Environ Pollut 1988;50:139–61.

Ornagi F, Ferrini S, Prati M, Giavini G. The protective effects of *N*-acetyl-l-cysteine methylmercury embryotoxicity in mice. Fundam Appl Toxicol 1993;20:437–47.

Pak KR, Bartha R. Mercury methylation by interspecies hydrogen and acetate transfer between sulfidogens and methanogens. Appl Environ Microbiol 1998;64:1987–90.

Pamphlett R, Cotte P. Entry of low doses of mercury vapor into the nervous system. Neurotoxicology 1998;19(1):39–48.

Park ST, Lim KT, Chung YT, Kim SU. Methylmercury induced neurotoxicity in cerebral neuron culture is blocked by antioxidants and an NMDA receptor antagonists. Neurotoxicology 1996;17: 37–46.

Pendergrass JC, Haley BE, Vimy MJ, et al. Mercury vapor inhalation inhibits binding of GTP to tubulin in rat brain: similarity to a molecular lesion in Alzheimer diseased brain. Neurotoxicology 1997;18(no. 2):315–24.

Pentreath RJ. The accumulation of mercury from food by the plaice *Pleuronectes platessa* L. J Exp Mar Biol Ecol 1976;25:51–65.

Peroza MA, Ayala-Fierro F, Barber DS, Casarez E, Rael LT. Effects of micronutrients on metal toxicity. Environ Health Perspect 1998;106:5203–16.

Peterson SA, Sickle JV, Herlihy AT, Hughes RM. Mercury concentrations in fish from streams and rivers throughout the western United States. Environ. Sci. Technol 2007;41:58–65.

Picou S. Social disruption and psychological stress in an Alaskan fishing community: the impact of the Exxon Valdez oil spill. Boulder Co. University of Colorado Natural Hazards Center; 1990.

Polland KM, Hultman P. Effects of mercury on the immune system. Met Ions Biol Syst 1997;34:421–40.

Porcella DB. Mercury in the environment — biochemistry. In: Watras CJ, Huckabee JW, editors. Mercury pollution, integration and synthesis. Boca Raton, FL: CRC Press; 1994. p. 3-19.

Preston S, Coad N, Towend J, Killham K, Paton G. Biosensing the acute toxicity of metal interactions: are they addictive, synergistic, or antagonistic? Environ Toxicol Chem 2000;19:775–80.

Pratico D, Reiss P, Tang L, Sung S, Rokach J, McIntosk T. Local and systemic increase in lipid peroxidation after moderate experimental traumatic brain injury. J Neurochem 2002;80:894–8.

Quig D. The prevention and treatment of metal toxicity. Altern Med Rev 1998;3:262–70.

Ram RN, Sathyanesam AG. Mercurial induced brain monamine oxidase inhibition in the teleost *Channa punctatus*. (bloch). Bull Environ Contam Toxicol 1985;35:620–6.

Ribeiro CAO, Rouleau C, Pelletier E, Audet C, Tjalve H. Distribution kinetics of dietary methylmercury in the Arctic charr (*Salvelinus alpinus*). Environ Sci Technol 1999;33:902–7.

Rice DC, Schoeny R, Mahaffey K. Methods and rationale for derivation of a reference dose for methylmercury by the USEPA. Risk Anal 2003;23:107–15.

Robertson DE, Abel KH. Natural distribution and environmental background of trace heavy metals in Alaskan shelf and estuarine areas'. U.S. Department of Commerce, National Oceanic Atmospheric Administration, Outer Continental Shelf Environmental Assessment Program, Final Report, vol. 69. 1990. p. 227–419.

Rothschild RFN, Duffy LK. Methylmercury in the hair of subsistence food users in a rural Alaskan village. Alsk Med 2002a;44:2–7.

Rothschild RFN, Duffy LK. Preliminary study on total mercury in the common prepared subsistence food of a rural Alaskan Village. Alsk Med 2002b;44:89–93.

Rothschild RFN, Duffy LK. Mercury concentrations in muscle, brain and bone of western Alaskan waterfowl. Sci Total Environ 2005;349:277–83.

Sanfeliu C, Cristofol R, Toran N, Rodriguez-Farre E, Kim SU. Use of human central nervous system cell cultures in neurotoxicity testing. Toxicol In Vitro 1999;13:753–9.

Sarafian TA, Verity MA. Oxidative mechanisms underlying methylmercury neurotoxicity. Int J Dev Neurosci 1991;9:147–53.

Schiønning JD, Møller-Madsen B. Autometallographic mapping of mercury in the spinal cord of rats treated with inorganic mercury. Acta Neuropathol 1991;81:434–42.

Sexton K, Olden K, Johnson BL. Environmental justice: the central role of research in establishing a credible scientific foundation for informed decision making. Toxicol Ind Health 1993;9:685–727.

Shander G, Allen JW, Mutleus LA, Aschner M. Mercury inhibits cysteine uptake in culture primary astrocytes but not in neurons. Brain Res 2001;914:159–65.

Slaonen JT, Seppanene K, Nyyssonen K. Intake of mercury from fish, lipid peroxidation, and the risk of myocardial infarction and coronary, cardio-vascular and any death in Eastern Finnish men. Circulation 1995;91:645–55.

Snyder-Conn E, Patton T, Bertram M, Scannell P, Anthony C. Contaminant baseline data from water, sediments, and fish of Nowitna National Wildlife Refuge, Alaska, 1985–1988. Ecological Services, Fairbanks, Alaska, U.S. Fish and Wildlife Service, Technical Report NAES-TR-92-02; 1992. 69 pp. + appendices.

Snyder-Conn E, Lubinski M. Contaminant and water quality baseline data for the Arctic National Wildlife Refuge, Alaska, 1988–1989. Raw Data, Ecological Services, Fairbanks, AK, U.S. Fish and Wildlife Service, Technical Report NAES-TR-93-03, vol. 2. 1993. 305 pp.

Sorensen EMB. Metal poisoning in fish. Boca Raton, Florida: CRC Press; 1991.

Sorensen N, Murata K, Budtz-Jorgensen E, Weihe P, Grandjean P. Prenatal methylmercury exposure as a cardiovascular risk factor at 7 year s of age. Epidemiology 1999;10:370–5.

Stern AH, Smith AE. An assessment of the cord blood: maternal blood methylmercury ratio: implications for risk assessment. Environ Health Perspect 2003;111:1465–70.

Stejskal VD. Mercury-specific lymphocytes: an indication of mercury allergy in man. J Clin Immunol 1996;16:31–40.

Storelli MM, Marcotrigano GO. Fish for human consumption: risk of contamination. Food Addit. Contam 2000;17:1007–11.

Suk WA, Auakian MD, Carpenter D, Groopman JD, Scammell M, Wild CP. Human exposure monitoring and evaluation in the Arctic: the importance of understanding exposure to the development of public health policy. Environ Health Perspect 2004;112:113–20.

Tiffany-Castiglioni E, Qian Y. Astroglia as metal depots: molecular mechanisms for metal accumulation, storage and release. Neurotoxicology 2001;22:577–92.

U.S. Environmental Protection Agency (USEPA) 1997–1996. Toxic release inventory: public data release EPA 745-R-97-005; 1997a. Washington D.C.

U.S. Environmental Protection Agency (USEPA). Guidance for assessing chemical contaminant data for use in fish advisories. Risk assessment and fish and fish consumption limits. EPA 823-B-97-009, vol 2. 1997b. Washington D.C.

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U.S. Environmental Protection Agency (USEPA). Guidelines for ecological risk assessment. EPA 630-R-95-002 F; 1998. Washington, D.C.

U.S. Environmental Protection Agency (USEPA). Water quality criterion for protection of human health: methylmercury. Technical report EPA/823/R-01/001; U.S. EPA; 2001a. Washington, D.C.

U.S. Environmental Protection Agency (USEPA). Mercury update: impact on fish advisories. EPA 823-F-01-011; 2001b. Washington, D.C.

Van Oostdam J, Gillamn A, Dewailly E, Usher P, Wheatley B, Kuhnlein H, Neve S, Walker J, Tracy B, Feeley M, Jerome V, Kwaunick B. Human health complications of environmental contaminants in Arctic Canada; a review. Sci Total Environ 1999;230:1-82.

Via CS, Nguyen P, Niculescu F, Papadimitriou J, Hoover D, Silbergeld EK. Low-dose exposure to inorganic mercury accelerators diseases and mortality in acquired murine lupus. Environ Health Perspect 2003;111:1273–7.

Watkinson S. Life after death: the importance of salmon carcasses to British Columbia's watersheds. Arctic 2000;53:92–9.

Weil M, Bressler J, Parsons P, Bolla K, Glass T, Schwartz B. Blood mercury levels and neurobehavior function. JAMA 2005;293: 1875–82.

Weis P, Weis JS. The developmental toxicity of metals and metalloids in fish. In: Newman MC, McIntosh AW, editors. Metal ecotoxicology. Boca Raton, Florida: Lewis Publications; 1991. p. 145–69.

Wheatley B, Paradis S. Balancing human exposure, risk and reality: questions raised by the Canadian Aboriginal methylmercury program. Neurotoxicology 1996;17:251–6.

World Health Organization (WHO). Methylmercury. Environmental health criteria, vol. 101. Geneva; 1990.

World Health Organization (WHO). Inorganic mercury. IPCS environmental health criteria, vol. 118. Geneva; 1991.

Wolfe MF, Schwarzbach S, Sulaiman RA. Effects of mercury on wildlife: a comprehensive review. Environ Toxicol Chem 1998;17: 146–60.

Yardley RB, Lazorchak JM, Paulsen SO. Elemental fish tissue contamination in northeastern U.S. lakes: evaluation of an approach to regional assessment. Environ Toxicol Chem 1998;17:1874–84.

Yee S, Choi BH. Oxidative stress in neurotoxic effects of methylmercury poisoning. Neurotoxicology 1996;17:17–26.

Yokoo EM, Valente JG, Grattan L, Schmidt SL, Platt I, Silbergeld EK, Low level methylmercury exposure affects neurophychological functions in adults. Environmental health: a global access science source 2003; 2: 8. http://www.ehjournal.net//content/2/1/8.

Yoshizawa K, Rimm EB, Morris JB, Spate VL, Chung-Chang H, Spiegelman D, Stampfer MJ, Willett WC. Mercury and the risk of coronary heart disease in men. New Engl J Med 2002;347: 1755–60.

Zalups RK, Lash LH. Interactions between glutathione and mercury in the kidney, liver, and blood. In: Chang LW, editor. Toxicol metals. Boca Raton, Florida: CRC Press; 1996. p. 145–63.

Zhang X, Naidu AS, Kelley JJ, Jewett SC, Dasher D, Duffy LK. Baseline concentrations of total mercury and methylmercury in salmon returning via the Bering Sea (1999–2000). Mar Pollut Bull 2001;42:993–7.

Zillioux EJ, Porcella DB, Benoit JM. Mercury cycling and effects in freshwater wetland ecosystems. Environ Toxicol Chem 1993;12: 2245–64.

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