

Peer Review

Methylmercury exposure through seafood diet and health in New Zealand: Are seafood eating communities at a greater risk?

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Abstract

Methylmercury (MeHg) is a developmental neurotoxin that presents a potential public health hazard to humans. MeHg exposure has been linked to developmental, cognitive and neurological disorders both pre and postnatally. Even very low environmental MeHg concentrations are thought to cause subclinical disorders especially in children. MeHg is highly absorbable with an important route of exposure through contaminated fish and seafood consumption. Many studies have shown dose-response MeHg effects in children whose staple diet is fish/seafood. It is also known that certain factors such as genetic susceptibility, socioeconomic, nutritional and cultural factors exacerbate the effects of MeHg exposure. This review examines the current knowledge on MeHg exposure through fish diet, our understanding of its effects on children and adults, the efforts at both national and international levels to tackle this pollutant. In particular, it raises concern that New Zealand (NZ) fish and seafood eating communities, such as Māori and Pacific population, may be at high-risk for MeHg exposure in addition to other Hg related factors

Introduction

There is substantial national and international interest in environmental contaminants, including the deleterious effects of mercury (Hg), a well known developmental neurotoxin.¹ Hg occurs in many different forms, including Hg vapour, inorganic Hg, ethylmercury, and methylmercury (MeHg). For the general population, primary exposure comes from a combination of fish/seafood consumption, dental amalgams and vaccines. There are also some occupational and local environmental (natural and man-made) exposures. Each of these forms of Hg have different toxicological profile and clinical symptoms.² This review focuses on MeHg, the most hazardous form of Hg. Most developed countries such as the United States (US), Canada and New Zealand (NZ) have strict guidelines and policies designed to protect the public from significant MeHg exposure.

In recent times there have been concerns about the health of children exposed to very low environmental MeHg concentrations, levels previously thought to be safe.³ Neurotoxins have been demonstrated to affect bioavailability

of various essential minerals, potentially leading to subclinical developmental disabilities, including behavioural problems.⁴ Individuals with elevated MeHg levels do not always show clinical symptoms and so symptoms can be difficult to detect and measure.⁵ Concerns are also growing about health impact on adults due to MeHg exposure, as studies are showing that MeHg exposure might interfere with vision, motor function, and memory.⁶

In NZ, an important route of MeHg exposure is through fish and seafood consumption.^{7,8} As children are more vulnerable to the effects of MeHg, there are growing concerns about fish/seafood consumption during childhood and its hazardous effects on the health and development of exposed children which is being raised by health authorities.^{1,9} However the benefits of fish consumption to children has also been well recognised.¹⁰ In the absence of widely publicised guidelines, this leaves parents and the general public to question what are appropriate and safe levels of fish consumption, particularly those in high seafood consuming sectors of the population. The purpose of this review is to provide an overview of MeHg exposure through seafood diet within the NZ context.

Hg exposure

There are a number of ways in which humans (both prenatally and postnatally) can be exposed to the different forms of Hg. Although Hg is present in water and air, the natural concentrations are extremely low and usually negligible to humans.² Traces of Hg are present in all food but uptake by plants from soil is low and therefore concentration of Hg in fruits and vegetables is also extremely low. The NZ Total Dietary (NZTD) survey 2003/04 of a large nationally representative sample found that vegetables and fruits all had Hg levels well below 0.0001mg/kg.¹¹ There are some Hg containing products, such as Hg thermometers and fluorescent lighting, which when damaged can be a cause of exposure to the individuals, however these instances are relatively rare. Also some occupational settings may expose people to Hg such as dentists.¹² However, apart from the workers directly involved, these exposures pose little risk to the general public. Instead, Hg exposure in the general population primarily occurs from fish/seafood consumption, which is in the form of MeHg,¹³ dental

amalgams absorbed through Hg vapours in the form of elemental Hg¹⁴, and from vaccines in the form of thiomersal.^{15, 16} In 2000, NZ phased out all thiomersal-containing vaccines in its childhood schedule vaccines, although some influenza vaccines still contain thiomersal.¹⁷ However, the estimated risk from thiomersal-containing vaccinations and dental amalgam is low.¹

MeHg exposure in humans

MeHg is the most toxic form of Hg and is highly absorbable relative to other Hg compounds.¹³ The consumption of contaminated fish (especially predatory fish) and seafood is the major source of exposure to MeHg in humans,¹³ and suckling infants can be exposed through breast milk.¹⁸ In NZ, dietary exposure to Hg may account for up to 54% of a person's total exposure to Hg and nearly all their exposure to MeHg compounds.¹⁹

In the environment, especially lakes, sea, and waterways, released elemental Hg is converted through methylation to MeHg by anaerobic bacteria in the aquatic ecosystem which then bioaccumulates in bigger fish and consequently larger predatory fish (e.g. mackerel, pike, shark, swordfish, barracuda, large tuna, marlin, whales and trout) have higher Hg concentrations.²⁰ Almost all Hg in fish muscles is MeHg.²¹ The level of MeHg varies in different fish species because each have different habitats, life-cycles and feeding patterns. In NZ, many fish contain levels of MeHg higher than the World Health Organization (WHO) recommended maximum MeHg levels (0.5 mg Hg/kg) due to volcanic and geothermal activities,²² or naturally occurring Hg deposits.²³ Freshwater fish in geothermal lakes and rivers in NZ may also accumulate high levels of MeHg.²⁴ For example, trout in NZ are known to contain MeHg levels that can reach 4.13 mg/kg.²² Since contaminants are high in some fish/seafood, people who consume large amounts of these kinds of fish/seafood, are vulnerable to adverse health effects.²⁵ However, currently there is no current information on the type of fish that is most eaten within Pacific, Māori or Asian people in the NZ population.

Transport of MeHg in humans

Approximately 90–100% MeHg is absorbed through the gastrointestinal tract where it enters into the blood stream and is distributed throughout the body.¹⁵ The pattern of this distribution is relatively uniform, except in red cells where the concentration is 10–20 times greater than the plasma concentration. The half-life of MeHg in people is dependent on the exposure length in addition to the dose of MeHg. For example, for people exposed to high doses over a short-term, the half-life of MeHg is approximately 44 days,^{26–28} whereas for people exposed to high doses over a long-term, the half-life is approximately 90 days.²⁸

MeHg readily crosses the blood–brain and placental barriers and thus is accumulated and concentrated in the fetus, especially in the brain.¹⁵ MeHg also accumulates in hair during its process of formation.²⁹ The vascular effects of MeHg include oxidative stress, inflammation, thrombosis, vascular smooth muscle dysfunction, endothelial dysfunction, dyslipidemia, immune dysfunction, and mitochondria dysfunction.³⁰

Seafood diet in NZ

Seafood is an important part of a healthy diet and is a major source of protein for many communities both nationally and internationally. A survey conducted in NZ by the Seafood Industry Council (2007) found that about 88% of NZ people eat fresh or frozen fish at least once a month and approximately 45% eat fish at least once a week.³¹ Many studies have shown that fish is an important source of the omega-3 fatty acids, eicosapentaenoic acid, and docosahexaenoic acid not found naturally in other food and could potentially contribute to a healthy heart, optimal brain function, cognition, improved eye and skin health and some protection against certain cancers,¹⁰ though not all studies have found such benefits.^{32, 33} Since seafood also contains environmental contaminants such as MeHg, balancing the risks and benefits of eating fish is an important public health issue.

Like MeHg, omega-3 fatty acid levels vary in different seafood.³³ Fatty fish tend to have higher omega-3 fatty acid levels than lean fish; while ocean fish are known to have higher levels of omega-3 fatty acid than fresh water fish.³³ No associations have been shown between MeHg levels and omega-3 fatty acids.³³ Large fish such as sharks and sword fish accumulate higher levels of MeHg but do not necessarily have high omega-3 fatty acids while fish such as anchovies, salmon, and herring have high omega-3 fatty acids and are known to have low MeHg concentrations.³³ The Ministry of Health (2009) in NZ has assessed various fish-types for omega-3.²⁴ In each fillet, fish such as eel contain an average 0.6g of omega-3, flounder contain 0.2g, and Hoki contain 0.1g.²⁴ However, some fish species, such as kahawai (also known as Australian salmon), were found to contain no measureable levels of omega-3,²⁴ and thus may be a source of MeHg without any omega-3 benefits. It is thus recommended that consumption of these fish-types should be limited, especially by pregnant women.³⁴ Historically, the consumption of fish in NZ was predominantly gained through eating fish and chips, especially in lower social economic status (SES) families.³⁵ Shark was routinely used as the meat source in fish and chips, yet shark is known to have high MeHg levels of up to 1.97 mg/kg.³⁵ A survey of fish in the early 1980s on Hg levels in 33 takeaway shops in South Auckland found that about 40% of the fish levels were above the WHO recommended maximum level of MeHg (0.5 mg/kg) and if eaten in excess by vulnerable people, could cause adverse health effects from MeHg.³⁶ The more recent NZTD survey 2003/04 found that battered fish total Hg concentrations reached up to 0.85 mg/kg,⁷ however the MeHg levels were not provided. While the routine consumption of shark may have changed in the intervening years, the risk of adverse health effects still remains and also led the NZTD survey to suggest that consumption of this fish-type should be limited, especially amongst vulnerable people.⁷

Many studies have shown that some ethnic groups are at a greater risk of MeHg exposure as they generally consume relatively more fish.^[25] Māori and Pacific populations traditionally have high fish consumption, and thus may be particularly vulnerable to adverse effects from MeHg. In a NZ study of 7331 mothers that was conducted in late 1970s, 1486 (20%) consumed fish more than three times per week while

pregnant.³⁵ Rates of fish consumption were greater for Māori (31%) and Pacific (53%) mothers. Amongst these mothers, 73 had MeHg levels (determined from hair samples) which were above 6 µg/kg; 8 (11%) of whom were Europeans, 20 (27%) were Māori, and 45 (62%) were of Pacific descent.³⁵ This study was conducted over three decades ago and the pattern of maternal fish consumption may have changed in the intervening years. No subsequent studies in NZ have explicitly examined these rates. However, a recent NZ National Children's Nutrition survey (2002/03) demonstrated that a higher proportion of Pacific children consumed fish (≈56%) than their Māori (≈37%) and European counterparts (≈35%). Furthermore, significantly more Pacific (≈27%) and Māori children (≈18%) children consumed fish cakes and fish fingers, which have high MeHg concentrations, compared to children of European origin (9%).³⁷ Unfortunately, little current information is available about MeHg exposure, levels and sequelae in these populations.

Health effects of MeHg

The deleterious health effects of MeHg have been known since the 1860s.³⁸ There have been several outbreaks since then due to dietary or occupational exposure with devastating health effects both pre/postnatally and in adults too. Two such major outbreaks occurred in Japan, Minamata Bay, through contaminated fish consumption.³⁹ Another outbreak occurred in Iraq from bread made from contaminated grains whereby thousands of victims suffered neurological symptoms.⁴⁰ These symptoms were found to be more pronounced in children born to mothers who were poisoned from MeHg.⁴⁰ More recently, there has been increased concern about the health of children exposed to environmental MeHg because it has been found to cause subclinical effects at even these low concentrations.³ It is demonstrated that toxic minerals can have antagonistic effect on various essential minerals leading to disturbances in metabolic utilisation which could lead to developmental disabilities including behavioural problems.

Many studies have been conducted to determine the effects of prenatal and postnatal low level MeHg exposure at subclinical and population levels and have reported poorer neurological status and slower development^{3,41} while some studies did not observe any adverse effects.⁴ Most attention has focused on three major longitudinal studies, namely: the Faroe Island (baseline n=1,022),^{42,43} Seychelles (baseline n=804),^{4,44} and NZ (baseline n=11,000) studies.^{35,45,46} While each of these populations consumed seafood in their diets, their findings differed. The NZ study showed a greater percentage of children in a high MeHg group (maternal hair MeHg levels >6 µg /g) who had lower developmental scores (a three-point decrement in intelligence quotient (IQ)) than children from the low MeHg groups (maternal hair MeHg <3 µg /g, 3-6 µg /g). The Faroe Island study also reported higher MeHg levels (average 22.9 µg/L) in cord blood and in maternal hair (geometric mean 4.3 µg/g) with lower developmental scores in children. The Seychelles study reported no association between maternal MeHg levels in hair (arithmetic mean maternal MeHg 6.8 µg/g) and developmental scores on neurological tests in children. There has been considerable debate over these mixed findings, however, it is likely due to the differences in study designs and sources of

exposure.⁴⁷ Nonetheless, using the information from these three major prospective studies, an attempt was made to develop a quantitative dose-response function for prenatal MeHg exposure and IQ.^{48,49} Axelrad and colleagues determined that an increment of 1ppm of MeHg in maternal hair during gestation can cause a decrease in IQ to range from 0.18 points⁴⁸ to 0.7 points.⁴⁹

Using the child behaviour check-list, a NZ cohort of 1,376 Pacific children aged 2 years found approximately 15.5% with behavioural problems within the clinical range.⁵⁰ Given these children's relatively high exposure to a sea-food/fish diet, MeHg may be one of the contributing factors. There have been many studies that have observed an association between neurodevelopmental problems and MeHg exposure^{51,52} however, studies need to be conducted within Pacific ethnic group in NZ to verify whether sea-food and fish diets are responsible or contribute to these high rates of behavioural problems.

Other studies have concluded that beneficial influence of nutrients (such as selenium and omega-3 fatty acids) from fish may counter any adverse effects of MeHg on the developing nervous system.⁵³ Again the results are equivocal. For example, a Faroe Island birth cohort study investigated omega-3 fatty acids and selenium as a potential modifier of the effects of MeHg exposure through fish/seafood diet but found no such associations.⁵⁴ Accordingly, Choi and Grandjean (2008) suggested in their review that to assess the full impact on the toxicity of MeHg and the beneficial effects of nutrients, both the good and bad effects should be assessed at the same time in order to separate opposite impacts on the outcomes.⁵⁵

Control measures and risk communications in NZ

The NZ Food and Safety Authority (NZFSA) manages a national monitoring program for heavy metals in fish while the Food Standards Australian New Zealand (FSANZ) prescribes a maximum level of MeHg in fish and seafood (0.5ppm for most fish and 1 ppm for certain fish).⁵⁶ The Joint Food and Agricultural Organization (FAO)/WHO Expert Committee on Food Additives (JECFA) established a provisional tolerable weekly intake (PTWI) for MeHg as 1.6 µg/kg body weight(bw)/week,⁵⁷ which is the equivalent of 0.23 µg/kg bw/day. This value is considered protective of developing foetuses, the most sensitive sub-group in the population. Therefore with the evidence, NZFSA adopted the 2003, JECFA revised PTWI for MeHg.¹¹ The NZFSA estimated the average daily intake of MeHg by adults, children, toddlers and infants from food sources which ranged from approximately 0.60 to 0.74 µg/kg bw/week.¹¹

The dietary exposures in the 2003/04 NZTDS were based on average energy diets for each of the age-sex groups.¹² According to Vanoort and Thomson (2005) some people might have significantly higher exposures, especially within the high exposure groups.^[11] There is specifically recommended dietary information provided by the NZFSA on its website. However, it is unclear whether this information reaches pregnant women or at-risk sub-populations (such as Māori and Pacific people).

Investigation is needed within NZ to identify the best method to inform and educate vulnerable sub-populations on the risks and benefits of fish consumption so these people can make well informed choices on consuming fish for good health whilst avoiding fish which may be harmful due to high MeHg levels. Also, with emerging evidence that even low MeHg levels may cause adverse health effects, further studies need to be conducted to confirm the patterns and extent of fish consumption in NZ. That is, the types of fish being consumed, the levels of MeHg in specific fish, and the extent of MeHg exposure within the NZ populations.

Conclusion

Accumulating evidence suggests that potentially damaging prenatal and postnatal exposure to MeHg may occur for some children born to vulnerable population sub-groups, primarily through fish/seafood consumption. Efforts to more fully understand and evaluate the health risks associated with this neurotoxin should be undertaken.

Any experimental or epidemiological data presents some uncertainty on whether the measured effects captures the most sensitive or critical effects. Neurodevelopmental effects are the most extensively studied sensitive end-point for MeHg exposure but there are other indications of adverse effects on humans such as immune functions, cardiovascular health and developmental disorders, and the relationships with broader societal factors. In addition to neurodevelopment effects, these other indicators need to be researched further in NZ particularly in view of the emerging literature of the subclinical effects of low level MeHg exposure.

The effects of MeHg exposure can be profound and debilitating. Yet, with appropriate monitoring and health promotion, excessive MeHg exposure is largely avoidable. NZFSA, FSANZ and NZ health authorities are making efforts to manage the risks involved with MeHg exposure but there is not enough

information about the current levels of MeHg in NZ people nor the types and amount of fish eaten to assess any causative relationship or effect. The NZFSA assessed MeHg exposure levels in NZ seafood/fish and adopted those recommended by the WHO guidelines (1.6 µg/kg body weight(bw)/week).¹¹ The NZFSA concluded, at that time, that the NZ population is highly unlikely to have any adverse health effects as a result of dietary exposure to MeHg. However, the emerging evidence suggests that NZ should perhaps reassess these levels.

The MeHg levels in high seafood consuming populations such as Māori and Pacific Island people, pregnant women, women of reproductive age, and children or adolescents who are most vulnerable is largely unknown in NZ. Unfortunately, Māori and Pacific Island communities, with a tradition of seafood eating also have risk factors which are disproportionately over-represented and could exacerbate the effects of MeHg exposure. The burden associated with the sequelae of MeHg exposure is likely to fall heaviest on these people. Māori and Pacific Island people also carry the highest oral health burden and lowest SES, which potentially exposes them to increased MeHg risk. No studies within NZ have researched the separate and synergetic effects of MeHg, elemental Hg and other neurotoxins in Māori and Pacific people. Such programs would be useful for health officials and communities to determine appropriate safety levels, and enable people to make informed dietary choices.

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