Mercury in Seafood:
Is Hawaii Seafood Safe?

Updated
July 15, 2009

Prepared by:

J. John Kaneko MS, DVM and Paul K. Bartram

PacMar, Inc.
3615 Harding Avenue, Suite 408-409
Honolulu, Hawaii 96816

Prepared for:

Hawaii Seafood Project-2 FY06
National Oceanographic and Atmospheric Administration
U.S. Department of Commerce

This document was prepared by PacMar, Inc. under NOAA Award No. NA06NMF4520222. The statements, findings, conclusions and recommendations are those of the authors and do not necessarily reflect the views of NOAA or the U.S. Department of Commerce.

Mercury in Seafood: *Is Hawaii Seafood Safe?*

John Kaneko and Paul Bartram
PacMar Inc., Honolulu, Hawaii
Hawaii Seafood Project-2 FY 06 (NOAA-funded)

Updated July 15, 2009

Mercury is toxic at high levels of exposure. However, its toxicity at the low levels found in ocean fish is uncertain. Public concerns have been raised about the potential health effects of mercury in open ocean (pelagic) fish including swordfish, marlin and tuna. These are extremely important food fish in Hawaii and elsewhere. The following is a discussion of the mercury issue in a question and answer format with references to the scientific literature intended to help clarify this complex public health and environmental management issue. This document is updated periodically to address new research findings pertinent to this important issue.

Q: Is mercury man-made, like pesticides or PCBs (polychlorinated biphenyls)?

A: NO. Mercury is a natural element that makes up a very small fraction of the earth's crust.[1]

Q: Is the amount of mercury increasing?

A: NO. There is a fixed amount of mercury on earth.

A: YES. Mercury in the atmosphere began its sharp increase with the start of the industrial revolution, 150 years ago. Industrial uses, especially burning coal[2], release mercury that was once stored in the earth's crust.

Q: Does atmospheric mercury enter seafood directly?

A: NO. Mercury in the atmosphere first has to be returned to earth, mostly by rainfall. In freshwater systems and nearshore waters, it may be converted by certain types of bacteria in sediments into a biologically active, organic form known as "*methylmercury.*" A very low-oxygen environment is required for *"methylation"* of mercury. The process by which inorganic mercury becomes "*methylated*" in the deep ocean is less certain.[3]

Q: Is "methylmercury" the form that could harm seafood consumers?

A: YES. Methylmercury is the major form of mercury in aquatic life. It enters at the bottom of the food chain and becomes more concentrated at each higher feeding level, reaching the greatest concentrations in long-lived aquatic species at the top of the food chain.[2,4,5] This process is known as *"bioaccumulation".* Everyone who eats seafood is exposed to small amounts of methylmercury. Accumulation of methylmercury occurs when it is ingested faster than the body is able to eliminate it. [6,7]

Q: Do we know why methylmercury is potentially harmful?

A: YES: Methylmercury is almost totally absorbed from the diet. The absorption of inorganic mercury is much less efficient. Methylmercury readily crosses the brain-blood and placental

barriers.[6] Methylmercury normally binds to sulfur within sulfur-containing amino acids which are components of protein. However, it binds more readily with selenium, an essential trace mineral.[8] When bound to mercury, selenium is no longer available for its essential functions. There is strong evidence that the toxicity of methylmercury is related to its interactions with selenium.[9]

Q: *Scientists have estimated that the amount of mercury in the atmosphere today is about 2-3 times what it was 150 years ago.[10] Has methylmercury in open ocean fish like tuna, increased by the same amount?*

A: NO. The relationship between mercury emissions in the atmosphere and methylmercury in open ocean fish is still being studied. Methylmercury is a very small fraction of the world's mercury supply.[6] Most of what is known about the conversion of atmospheric mercury into methylmercury is from studies of freshwater systems,[2] where the process is affected by water chemistry and bacterial activity in low oxygen environments at the bottom of lakes.[11]

Studies of mercury concentrations in open ocean fish and coastal seabirds over the past century using museum specimens as early controls have produced conflicting findings. There was no difference in the mercury concentration of Pacific tuna caught between 1878 and 1909 and in 1972.[12] In contrast, a significant increase in mercury was found in some studies[13,14] of fish-eating seabirds pre-1931 to post-1979 off the Atlantic coast of Europe. However, there remains some question about the accuracy of mercury testing of preserved museum specimens. [15,16]

If mercury in open ocean fish like tuna originated wholly or partly from atmospheric pollution, the increase in atmospheric mercury should be reflected to a measurable extent in tuna inhabiting the upper layers of the Pacific Ocean. Between 1971 and 1998, atmospheric mercury emissions increased by 26%. No change was found in mercury levels in Hawaii yellowfin tuna during this same period in surveys completed in 1971 (0.22 ppm) and repeated in 1998 (0.21 ppm).[3] On the basis of the constant mercury levels, scientists concluded that atmospheric mercury pollution was not the direct source of methylmercury found in tuna.

They hypothesize that methylmercury is formed in the deep ocean (> 900 m) or in deep bottom sediments. The specific source of mercury is not yet clear but researchers suggest it could be hydrothermal vents.[3] The mixing time between the deep ocean and the surface (where atmospheric mercury is deposited) is too long (hundreds of years) for the deep ocean to have been impacted by human mercury inputs over the past 150 years. A study in the Atlantic Ocean also found that uptake of methylmercury by fish is enhanced in low oxygen waters at ocean depths below the thermocline (> 300 m).[5] A study in the Pacific Ocean has documented a peak in methylmercury in water within the intermediate depths (500 to 700 m) of the water column characterized by low oxygen content and high levels of bacterial activity.[17] However, no mercury levels were determined in fish in this study. Another study has demonstrated that total mercury concentrations in predatory pelagic fishes and their prey increase with median depth of occurrence and mimic concentrations of dissolved organic mercury in seawater.[18] However, there remains no evidence that mercury levels of ocean fish near Hawaii have increased over time.

Q: Are people getting mercury poisoning from eating seafood?

A: NO. The risk of mercury poisoning from eating open ocean seafood remains hypothetical because it has not occurred. Not one person of any age in Hawaii or anywhere else has been reported with methylmercury poisoning from eating fish from the open ocean, including tuna, marlin and swordfish. There are no reports in the scientific literature confirming mercury poisoning from eating open ocean fish.

Q: Has mercury poisoning from eating any kind of seafood ever occurred?

A: YES. The only outbreaks of mercury poisoning from eating fish on record occurred in people dependent on seafood from Minamata Bay and the Agano River in Japan during the 1950s and 1960s. Severe neurological disorders and deaths occurred because of extreme mercury exposure from high consumption rates of highly contaminated fish and shellfish. These outbreaks were related to uncontrolled industrial pollution, not open ocean fish containing naturally low background levels of methylmercury.[19] Contaminated fish and shellfish contained extremely high levels of mercury, with averages of 5.6 to 35.7 ppm, with some fish testing up to 41 ppm in Minamata Bay.[20] In contrast, open ocean fish species from Hawaii on average contain less than 1 ppm mercury.[21]

Q: I hear health warnings about mercury in fish, but I also know that fish is good for your health. Is fish good for you?

A: YES. The American Heart Association's dietary guidelines for reducing the risk of cardiovascular disease recommend an increase in the consumption of foods rich in long chained omega-3 fatty acids found in ocean fish such as tuna. The guidelines target the general population and reference numerous studies showing cardiovascular health benefits.[22] A 2009 study by researchers at the Harvard School of Public Health found that low omega 3 fatty acid intake is the 8th highest risk factor among preventable causes of death in America.[23] This indicates that Americans could benefit from eating more fish, not less. Limiting the consumption of ocean fish that is associated with widely accepted health benefits to reduce low level mercury exposure must be carefully weighed. Alternative foods (ex. beef) may have other documented and potentially greater health risks.

Two studies have reported statistical associations between the risk of heart attack and mercury, mostly in the form of methylmercury associated with seafood consumption,[24,25] although one of these studies sampled a population where consumption of saturated animal fat was high.[25]

Many other, long-term studies sampling large populations have found no such association. A nested case-control study of more than 300,000 health professionals found no such association.[26] A 17-year study in Japan found that a fish diet has the effect of preventing strokes and heart attacks, cancer and other diseases. Cumulative mortality rates per 100,000 people were lower for those who ate fish daily than for those who did not.[27]

A study of 22,071 doctors, called the Physicians' Health Study,[28] suggests that consumption of fish rich in omega-3 fatty acids can reduce a man's risk of dying from a heart attack by 80%. A companion study, called the Nurses' Health Study,[29] found that omega-3 fatty acids can cut a woman's risk of heart attack death by 33%.

Q: If mercury poisoning has not happened from eating open ocean fish that contain mercury, is there something else in fish that makes it safe to eat?

A: YES. Yellowfin tuna was first reported in 1972 to protect against mercury toxicity in experimental diets laced with methylmercury.[30] Researchers concluded that the rich levels of selenium contained in tuna were responsible for the protective effect. Selenium is known to bind and sequester mercury on a 1:1 molecular basis, forming the biological inactive complex, mercuryselenide. The protective effect of selenium on mercury toxicity has been demonstrated in every animal model tested to date.[31,32,33]

Q: If there is such concern about mercury in ocean fish, why haven't there been any reported outbreaks of mercury poisoning from eating these fish?

A: There have been no reported outbreaks of mercury poisoning from eating open ocean fish in the US or in Japan where people eat nearly 10 times the amount of fish as the US consumer. A substantial and growing amount of scientific evidence indicates that mercury's toxic effects are countered by the protective interactions of selenium.

Q: What is selenium?

A: Selenium is an essential trace mineral required in the diet for the activity of 25-35 enzymes with critical functions.[34] These enzymes have powerful antioxidant functions especially important in the brain[35] and the endocrine system.[36] Anti-cancer effects of selenium have been demonstrated that may be the result of the anti-oxidant effects.[37] In addition, selenium has been known to detoxify mercury since 1967.[38] It should be noted that selenium can be toxic at extremely high doses.[39] Selenium toxicity in the US is extremely rare.

Q: Is there strong evidence that selenium detoxifies mercury?

A: YES. The National Research Council's Committee on the Toxicological Effects of Methylmercury[40] has concluded that "Although the effect of selenium on methylmercury (MeHg) toxicity has not been documented in humans, it has been known for over two decades that organic and inorganic selenium can influence the deposition of MeHg in the body and protect against its toxicity in animals." Protective effects have been demonstrated in every animal model tested.[31,32,33] Numerous papers presented by scientists at the international symposium dedicated to Selenium and Mercury Interactions held in La Jolla, California in 2007 were published in the journal *Biological Trace Element Research* Volume 119 No.3 in December 2007.

Q: How does selenium detoxify mercury?

A: It is known that selenium and mercury have an extremely strong binding affinity. Mercury normally binds with sulfur, but the binding attraction with selenium is 1 million times greater.[8] When mercury is bound to selenium, it forms a biologically inactive compound. The old theory was that selenium merely binds mercury and mercury is no longer available to cause toxic damage. The new and more logical mechanism is that excessive mercury binds up the available selenium so that the essential selenium functions are no longer active. In this sense, mercury poisoning is actually caused by profound selenium deficiency.[9] This makes sense because selenium has essential anti-oxidant functions (especially in the brain) and excessive mercury exposure is known to cause severe oxidative damage in the brain. For this reason, it is the ratio of selenium to mercury in fish that is important, not the mercury concentration alone.

Q: If selenium is health-promoting, where do we get selenium in the diet?

A: The USDA reports that of 1100 food evaluated, 17 of the top 25 food sources of selenium in the American diet are seafood.[41] Selenium has been shown to occur in rich amounts in Hawaii's open ocean fish.[21]

Q: If the ratio of selenium to mercury in foods determines which are health-promoting or potentially toxic, what are the ratios in Hawaii's ocean fish?

A: Hawaii's most popular open ocean fish provide a rich source of health-promoting selenium in excess of the naturally occurring levels of mercury they contain. A survey of selenium and mercury (total) was conducted for 15 different species of open ocean fish caught near Hawaii.[21] Four species of tuna (bigeye, yellowfin, albacore, skipjack), 4 species of billfish (swordfish, blue marlin, striped marlin, spearfish), 5 species of associated pelagic fish (mahimahi, wahoo, opah, sickle pomfret, escolar) and 2 species of sharks (mako, thresher) were evaluated. Thirteen of the 15 species contained a molar excess of Se over Hg, swordfish contained equal molar amounts of Se and Hg and only mako shark contained more mercury than selenium.

Q: Is there evidence that the ratio of selenium and mercury is the superior factor in determining mercury health risk or selenium benefits?

A: YES. A study in 2008 demonstrated that methylmercury toxicity in rats (slow growth, weight loss, neuromuscular signs) is not predictable by knowing the mercury concentration, but can be reliably predicted by knowing the molar ratio of mercury to selenium ($P < 0.001$)[42]. This study demonstrated that focusing on the mercury concentration of fish alone without considering the selenium content and protective interactions is incomplete and inadequate for assessing the risks or benefits from seafood consumption.

Q: I am a Hawaii resident who eats tuna steaks, seared tuna and tuna sashimi *regularly. Could my blood mercury level exceed the current guideline recommended by the Environmental Protection Agency (EPA) and Food and Drug Administration (FDA)?*

A: YES. In the case of seafood eaters, elevated blood mercury levels reflect higher seafood consumption rates. For most people, the primary route of exposure to mercury is from eating fish. But finding elevated mercury in hair or blood is not equivalent to mercury toxicity. In the case of people who do not eat ocean fish, finding elevated mercury in blood or hair has very different implications because they may be exposed to unnaturally high concentrations of mercury from pollution without protective amounts of selenium.

Q: The EPA and FDA currently recommend that women of child bearing age restrict fish consumption to no more than 2 meals per week.[43] I thought fish is good for you, what is the basis of this guidance?

A: The 2004 EPA and FDA guideline[43] provides a substantial margin of safety for seafood consumers. It was calculated from a "*benchmark dose level*" established by the National Research Council.[40] The benchmark dose is based primarily on the finding of a statistical correlation between pre-natal methylmercury exposure of children in the Faroe Islands (with umbilical cord blood concentration as a marker) and subtle changes in their neurological

development detected through a standard vocabulary test.[44] The Faroe Islands are located between Iceland and Norway and are part of Denmark.

The "*lower limit benchmark dose*"[40] was calculated by dividing the most sensitive endpoint (lowest value with significant statistical correlation) of the Faroe Islands study by an uncertainty factor of 10. This provides a large 10-fold margin of safety in the EPA and FDA recommendation for U.S. women of child-bearing age to limit consumption of certain species of fish, including tuna. In the Faroe Islands study, however, pre-natal exposure to methylmercury was due to the mothers' consumption of pilot whale – a marine mammal, and not from eating fish. The EPA and FDA guideline, therefore, is NOT the borderline between safe fish consumption and harm to mothers and children.

There are other concerns about the Faroe Islands study. The concentration of mercury in whole blood was used, but this is not a good indicator of possible long-term mercury exposure and may only indicate short-term exposure from recent meals. Methylmercury binds tightly to red blood cells and red blood cell values fluctuate.[45] Hair mercury levels reflect mercury exposure over the time it takes for the segment of hair to grow.[40] Hair mercury level is a much better indicator of long-term exposure.[40,46]

Only 10 percent of U.S. mainland women aged 15-49 years are estimated to have hair mercury levels higher than 1.4 ppm[47], or about 1/10th of the 12 ppm mercury in maternal hair that was a lower limit threshold of observed effects for subtle neurological changes in Faroe Island children[48] whose mothers consumed pilot whale meat high (3.37 ppm) in methylmercury.[49] Studies of U.S. women's exposure to methylmercury (concentration x consumption) and modeling of maternal and fetal blood and tissues and maternal dietary intake that occurs over the course of pregnancy and gestation indicate that the National Research Council's estimate that more than 60,000 newborns annually may be at risk for adverse neurodevelopmental effects due to pre-natal methylmercury exposure is an overestimation. It has been estimated (admittedly with substantial uncertainty) that in the Minamata mercury poisoning event, the average hair mercury concentrations of women who had babies and children with mercury poisoning effects was 41 ppm and may have range up to 133 ppm.[50] The EPA's reference dose should not be considered a "bright line" for evaluation of safety.[51]

Q: If the current 2004 EPA and FDA guidance[43] for fish consumption is based on studies of pilot whale meat in the diet, what did those studies find?

A: No one in the Faroe Islands thought there was a problem with their children. However, the Faroe Islands study found statistical correlations between pre-natal methylmercury exposure and subtle changes in neurological development in some tests of 7-year old children.[44,52] However, the results of development tests could not always be replicated when children were tested by different examiners. When results are adjusted for different examiners, the mercury effect is less evident.[51]

The main source of methylmercury was through their mothers' consumption of pilot whale meat. It should be noted that Faroe Islands pilot whale meat contains not only higher methylmercury concentrations than ocean fish, but also contains a 4:1 molar excess of mercury over selenium.[49] The meat of pilot whales caught off the Faroe Islands also contains elevated levels of cadmium, another toxic metal.[53] The women in the study also consumed whale blubber containing higher levels of PCBs, inorganic mercury and other toxic compounds than pilot whale meat.[52] Although other research has linked prenatal exposure to PCBs and other

organochlorine compounds to neurologic effects in children, PCBs had no discernible effect on neurologic function in the Faroe Islands study.[44] The mercury effect estimate remained almost unchanged after adjustment for concomitant exposure to PCBs.[54]

The lead Faroe Island scientist on the research team has stated that "*The Faroese children are not exposed to methylmercury by eating fish. They are exposed to mercury by the traditional consumption of pilot whale meat*."[55] The ability of small toothed whales to concentrate mercury and cadmium is well established.[56] Like methylmercury, cadmium can be transferred across the placenta to fetuses[57] and the hair cadmium level has been correlated with very significant learning disability or mental retardation in children in a large study in China.[58]

Q: Are unborn children and infants more sensitive to the harmful effects of methylmercury than older children and adults?

A: YES. The developing brain of an unborn child and infant is more sensitive to the harmful effects of methylmercury than the central nervous system of an older child or adult. Researchers investigated the tragic case of mercury poisoning that occurred in Iraq in the 1970's, when seed grain treated with a fungicide containing mercury was mistakenly used directly for food. These studies showed that extremely high levels of methylmercury crossing the placenta from the mother to the fetus could cause pre-natal brain damage, leading to mental retardation, delays in speech and motor development later in life. The level of methylmercury in the hair of the mothers was predictive of the adverse effects on infants in this case of accidental mercury poisoning.[59] It has been estimated that the mercury in the flour made from contaminated grain (9.1 ppm) far exceeded the amount of selenium (0.2 ppm), both by weight and molar basis.[60] The protective effects of selenium would have been overwhelmed by the great excess of methylmercury.

Q: Can exposure to methylmercury before birth through seafood consumption by pregnant mothers adversely affect children's neurodevelopment?

A: YES. If the seafood contains high mercury concentration and more mercury than selenium (ex. pilot whale meat, some types of sharks, or fish and shellfish from contaminated water).

A: NO. If the seafood contains rich levels of selenium and more selenium than mercury (ex. open ocean fish).

Q: Should shark meat be avoided during pregnancy?

A: It depends on the type of shark. Not all sharks contain a molar excess of mercury over selenium, stressing the importance of documenting selenium to mercury molar ratios as the criteria for determining potential mercury health risks (or selenium health benefits) in seafood. Results of a New Zealand study of prenatal exposure to methylmercury from consumption of a seafood diet high in shark meat showed adverse neuropsychological outcomes in school-aged children.[61] In New Zealand, the shark meat consumed as fish and chips, a popular take-out food, had a mean mercury concentration of 2.2 ppm, with some samples more than 4 ppm.[61] Mako shark caught near Hawaii contain a mean mercury concentration of 1.8 ppm and twice as much mercury as selenium on a molar basis making this fish a net dietary source of mercury.[21]

Q. Is there better evidence about the safety of ocean fish consumption than studies about pilot whales and sharks?

A. YES. The 9-year Seychelles Islands Child Development Study[46] assessed neurological development of children who were exposed to methylmercury before birth up to the age of 9. Their mothers consumed an average of 12 meals per week of ocean fish containing an average concentration of 0.3 ppm methylmercury and had an average maternal hair mercury level of 6.9 ppm (range 0.5 to 26.7 ppm). This hair mercury level is about 4 times higher than the highest level (90th percentile) found in U.S. mainland women of child-bearing age. The Seychelles Islands study found no detectable adverse effects in a population consuming large quantities of ocean fish.[46] The results of this study could not be used to calculate the risk of mercury toxicity simply because no harmful effects occurred. For this reason the EPA and FDA fish consumption guidance is based on studies of the effects of pilot whale meat in the diet.

The variety of fish consumed in the Seychelles Islands better approximates what is consumed in Hawaii, so the results of this study are more applicable than the Faroe Islands Study. Seychelles Islanders eat a variety of ocean fish but do not eat pilot whale meat, in contrast to the diet of Faroe Islanders. Ocean fish eaten in the Seychelles are rich in selenium and contain more selenium than mercury.[62] The results of this study are also more applicable because researchers used interscore reliability to eliminate variances between different examiners, a weakness in the Faroe Island study.[51]

Q: Is Hawaii ocean fish a safe choice?

A: YES. Eating ocean fish from uncontaminated environments, such as Hawaii in the mid-Pacific, exposes consumers to very low doses of methylmercury[6] and a rich source of health-promoting selenium.[21]

Of the three major studies of children with pre-natal exposure to methylmercury, the Seychelles Islands Child Development Study[46] is the most relevant to Hawaii seafood consumers and Hawaii fish. Seychelles Islanders have one of the highest fish consumption rates in the world, exceeding 175 lb per person annually. The Seychelles Island fish diet includes bonito, jacks, yellowfin tuna, skipjack tuna, wahoo and barracuda. The results of the Seychelles study do not support the hypothesis that there is a significant and detectable health risk to children from pre-natal methylmercury exposure resulting solely from ocean fish consumption (not whale or shark).[46]

In the Seychelles, fetal exposure is continuous through frequent consumption of ocean fish containing low methylmercury concentrations comparable to the average of the wide variety of species (0.3 ppm) consumed by the Hawaii and mainland U.S. populations. Hawaii consumers are estimated to eat nearly 3 times (41 lb/person/year) the U.S. per capita average amount of seafood with the majority of it being open ocean fish.[63] The most important open ocean fish species caught in the Hawaii fishery have been demonstrated to contain a molar excess of selenium over mercury, making them more likely to prevent mercury toxicity than contribute to it.[21]

Q: Do U.S. and international agencies agree on a safe level for methylmercury intake by mothers that will protect developing fetuses and infants?

A: NO. The EPA's guideline for a level methylmercury intake that will protect the developing fetus is the equivalent of 0.1 millionth of a gram per kilogram of their mothers' body weight per day (0.1 mcg/kg BW/day). The Joint Expert Committee on Food Additives, administered by the

United Nations' World Health Organization and the Food and Agriculture Organization, considers 0.23 mcg/kg BW/day by women of child-bearing age a sufficient limit to protect the developing fetus.[64] Based on the findings of the Seychelles Child Development Study, the U.S. Agency for Toxic Substances and Disease Registry (U.S. Department of Health and Human Services) currently recommends 0.3 mcg/kg BW/day as a minimal risk level for methylmercury intake, three times greater than the current EPA and FDA guidance.[65] It should be noted that the ATSDR provides scientific advice to the FDA on toxic substances.

Q: How does this translate to a recommended number of fish meals (e.g. yellowfin tuna) per week for pregnant women?

A: Agencies differ in their recommendations for safe fish consumption because they are based on different studies of mercury health effects and sources of mercury (e.g., pilot whale meat versus fish). The results of the Seychelles Island study are more likely to reflect safe fish consumption rates than guidance based on the consumption of pilot whale meat.

Agency recommendations for fish consumption such as yellowfin tuna during pregnancy and Seychelles Island study results without detectable adverse health effects are as follows.

- EPA-FDA Joint Advisory 2004 — 1 fish meal per week[43]
- World Health Organization — 2.3 fish meals per week[64]
- Agency of Toxic Substances and Disease Registry — 3 fish meals per week[65]
- Seychelles Islands Child Development Study — 12 fish meals per week[46]

Q: What marine life should women of child-bearing age avoid because of exposure to harmful levels of methylmercury and low levels of selenium?

A. DO NOT EAT TOOTHED WHALE OR DOLPHIN MEAT. Toothed whales and dolphins are eaten in some cultures. Mercury accumulates in these long lived animals and may greatly exceed selenium levels. Faroe Island pilot whale meat contains much more mercury than selenium.[49] The same has been shown in toothed whales and dolphins in Japan, leading to health warnings.[66] Mercury poisoning from eating marine mammals is suspected in Japan, but no outbreaks have occurred in Japan from eating open ocean fish. Tests on meat from toothed whales in Japan have revealed high levels of methylmercury, 5 ppm in small-toothed whales, with much higher concentrations in livers.[56] It is clear that consumption of toothed whale meat should be strongly discouraged.[67]

A: DO NOT EAT FISH OR SHELLFISH FROM MERCURY CONTAMINATED RIVERS, LAKES, ESTUARIES OR BAYS. Scientists have established a strong link between air pollution and elevated methylmercury levels in some freshwater species.[11] A study of northern Ontario, Canada lakes shows a strong correlation between lake size and methylmercury concentrations in fish, possibly because smaller lakes have warmer water that can increase the rate at which bacteria convert mercury deposits into methylmercury.[68] However, it should be stressed that selenium and mercury molar ratios need to be considered in determining the health risk of mercury.[9,21,42] Unlike ocean fish that tend to be rich in selenium, freshwater fish may not be due to the variability of the selenium content of soils and bodies of freshwater.

A. DO NOT EAT CERTAIN TYPES OF SHARK MEAT. Results of a New Zealand study of prenatal exposure to methylmercury from consumption of a seafood diet high in shark meat

showed adverse neuropsychological outcomes in school-aged children.[61] Some species of shark may contain a molar excess of methylmercury over selenium and should be avoided

Q: If I play it safe and don't eat tuna, could I be exposing my baby to other health risks?

A: YES. The risk posed by exposure to mercury from ocean fish consumption is currently speculative. Attempts to reduce exposure to mercury may pose greater health risks than those hypothesized to occur from low levels of mercury in open ocean fish.[67] Omega-3 fatty acids, of which tuna are a rich source, are vital for the proper development of an infant's brain and retina. Omega-3 fatty acids are necessary for the complete development of the human brain during pregnancy and the first two years of life.[69] The omega-3 fatty acids are so essential to a child's development that if a mother and infant have a deficient diet, the child's nervous and immune system may never fully develop. The developing fetus draws on supplies of omega-3 fatty acids stored in the mother's body. There is a growth spurt in the human brain during the last 3 months of pregnancy and the first months after birth with a large increase in the cerebral content of certain long-chain omega-3 fatty acids.[70]

There is strong evidence that the health benefits of fish consumption far outweigh the risks of low level mercury exposure for the developing fetus. Children born to mothers that ate 2 meals or less of fish during pregnancy in the ALSPAC Study in the U.K. fell into the lowest 25% in verbal IQ and other developmental tests.[71] The results of some developmental tests that were conducted on prenatally exposed children in the Seychelles Islands Child Development Study indicate beneficial outcomes in neurologic development that correlate with relatively high hair mercury levels during pregnancy. Higher fish consumption and the potential beneficial role of nutrients in fish is a plausible explanation for this finding.[62]

A diet that includes fish containing healthy fish oils has also been shown to have specific health benefits for pregnant mothers and their babies. One study has found that women whose daily intake of fish was less than 15 grams, corresponding to a fish oil intake of 150 mg/day, were significantly more likely to give birth to a premature or underweight baby than were women with higher fish intakes. Low consumption of fish, therefore, was a strong risk factor for preterm delivery and low birth weight.[72]

Q: Is there something positive that can be said about the safety of low level mercury exposure from a diet including ocean fish?

A: There are no reported cases of mercury poisoning associated with open ocean fish.

A: No adverse health effects were detected in children born to mothers who ate 12 meals per week of ocean fish during pregnancy in the Seychelles Islands.

A: The widely accepted health benefits of fish in the diet outweigh potential adverse health effects of low level mercury exposure from a diet of ocean fish.

REFERENCES

[1] Hammond, L. 1971. Mercury in the environment: natural and human factors. *Science* 171 (3973): 788-789.

[2] Annapolis Center for Science-Based Public Policy. 2003. Mercury in the environment: the problems, the risks, and the consequences. Annapolis, MD.

[3] Kraepiel, A.M.L., K. Keller, H.B. Chin, E.G. Malcolm and F.M.M. Morel. 2003. Sources and variations of mercury in tuna. 2003. Sources and variations of mercury in tuna. *Environmental Science and Technology*. 37(24): 5551-5558.

[4] Bolger, P.M. and B.A. Schwetz. 2002. Mercury and health. *New England Journal of Medicine*. 347(22): 1735-1736.

[5] Monteiro, L.R., V. Costa, R.W. Furness and R.S. Santos. 1996. Mercury concentrations in prey fish indicate enhanced bioaccumulation in mesopelagic environments. *Marine Ecology Progress Series* 1996. 141: 21-25.

[6] Dalton, L.W. 2004. Methylmercury toxicology probed. *Chemical and Engineering News*, January 19, 2004: 70-71.

[7] U.S. Environmental Protection Agency. 1997. Mercury white paper. Obtained from www.epa.gov/ttn/oarpg/t31/memoranda/whitepaper.html.

[8] Dyrssen, D. and M. Wedborg. 1991. The sulfur-mercury(II) system in natural waters. *Water Air Soil Pollut* 56:507-519).

[9] Ralston, N.V.C., J.L. Blackwell III, and L.J. Raymond. 2007. Importance of molar ratios in selenium-dependent protection against methylmercury toxicity. *Biol Trace Elem Res* 119:255-268.

[10] Pirrone, N., G. J. Keeler and J.O. Nriagu. 1996. Regional differences in worldwide emissions of mercury to the atmosphere. *Atmos Envir* 30(17): 2981-2987.

[11] Rudd, J.W.M. 1995. Sources of methyl mercury to freshwater aquatic ecosystems: a review. *Water Air Soil Pollut*. 80: 697-713.

[12] Miller, G.E., P.M. Grant, R. Kishore, E.J. Steinkruger, F.S. Rowland and V.P. Guinn. 1972. Mercury concentrations in museum specimens of tuna and swordfish. *Science* 175 (4026): 1121-1122.

[13] Monteiro, L.R. and R.W. Furness. 1997. Accelerated increase in mercury contamination in north Atlantic mesopelagic food chains as indicated by time series of seabird feathers. *Environmental Toxicology and Chemistry*. 16: 2489-2493.

[14] Thompson, D. R., R.W. Furness and L.R. Monteiro. 1998. Seabirds as biomonitors of mercury inputs to epipelagic and mesopelagic marine food chains. *Science Total Environ*. 213: 299-305.

[15] Gibbs, R.H., Jr., E. Jarosewich and H.L. Windom. 1974. Heavy metal concentrations in museum fish specimens: effects of preservatives and time. *Science* 184 (4135):475-477.

[16] Renaud, C.B., J.O. Nriagu and H.K.T. Wong. 1995. Trace metals in fluid-preserved museum fish specimens. *Science Total Environ*, 159: 1-7.

[17] Sunderland, E.M., D.P. Krabbenhoft, J. W. Moreau, S.A. Strode and W.M. Landing. 2009. Mercury sources, distribution and bioavailability in the North Pacific Ocean: Insights from data and models. *Glob Biogeochem Cycles* 23, GB2010, doi:10,1029/2008GB003425. p. 14

[18] Choy, C.A., B.N. Popp, J.J. Kaneko and J.C. Drazen 2009. Influence of depth on mercury levels in pelagic fish and their prey. *Proc National Academies of Science*. www.pnas.org/cgi/doi/10.1073/pnas.0900711106

[19] Myers, G. 1998. Statement by the University of Rochester Research Team studying the effects of methylmercury read before the Senate Subcommittee on Clean Air, Wetlands, Private Property and Nuclear Safety, Committee on Environment and Public Works, October 1, 1998. 4 p.

[20] Harada, Y. 1968. Congenital (or fetal) Minamata Disease. In *Study Group of Minamata Disease*. Kumamoto University, Kumamoto, Japan 93-117.

[21] Kaneko, J.J. and N.V.C. Ralston. 2007. Selenium and Mercury in Pelagic Fish in the Central North Pacific near Hawaii. *Biol Trace Elem Res* 119:242-254.

[22] Krauss, R.M., R.H. Eckel, B. Howard, L.J. Appel, S.R. Daniels, R.J. Deckelbaum, J.W. Erdman, P. Kris-Etherton, I.J. Goldberg, T.A. Kotchen, A.H. Lichtenstein, W.E. Mitch, R. Mullis, K.Robinson, J. Wylie-Rosett, S. St. Jeor, J. Suttie, D.L. Tribble and T.L. Bazarre 2000. AHA dietary guidelines, revision 2000. #71-0193, Circulation. 2000; 102: 2284-2299. *American Heart Association*.
(Also, AHA Scientific Statement: fish consumption, fish oil, omega-3 fatty acids and cardiovascular disease, #71-0241 Circulation. 2002; 106: 2747-2757.)

[23] Danaei, G. E.L. Ding, D. Mozaffarian, B. Taylor, J. Rehm, C.J.L. Murray and M. Ezzati. 2009. The preventable causes of death in the United States: Comparative risk assessment of dietary, lifestyle and metabolic risk factors. PLoS Med 6(4): e1000058. Doi:10.1371/journal.pmed.1000058.

[24] Guallar, E., M.I. Sanz-Gallardo, P. van't Veer, P. Bode, A. Aro, J. Gomez-Aracena, J.D. Kark, R.A. Riemersma, J.M. Martin-Moreno and F.J. Kok. 2002. Mercury, fish oils and the risk of myocardial infarction. *New England J Medicine*. 347: 1747-1754.

[25] Salonen, J.T., K. Seppanen, T.A. Lakka, R. Salonen and G.A. Kaplan. 2000. Mercury accumulation and accelerated progression of carotid atherosclerosis: a population-based perspective 4-year follow-up study of men in eastern Finland. *Atherosclerosis*, 148 (265-273).

[26] Yoshikawa, K., E.B. Rimm, J.S. Morris, V.L. Spate, C.C. Hsieh, D. Spiegelman, M.J. Stampfer and W.C. Willett 2002. Mercury and the risk of coronary heart disease in men. *New England J Medicine*. 347: 1755-1760.

[27] Hirayama, T. 1990. Life style and mortality: a large-scale census-based cohort study in Japan. *Contributions to Epidemiology and Biostatistics* 6. Basel, New York: Karger 138 p.

[28] Albert, C.M., C.H. Hennekens, C.J. O'Donnell, U.A. Ajani, V.J. Carey, W.C. Willett, J.N. Ruskin and J.E. Manson 1998. Fish consumption and risk of sudden cardiac death. *J American Medical Assoc*. 279: 23-28.

[29] Hu, F.B., L. Bronner, W.C. Willett, M.J. Stampfer,K.M. Rexrode, C.M. Albert, D. Hunter and J.E. Manson.. 2002. Fish and omega-3 fatty acid intake and risk of coronary heart disease in women. *J American Medical Assoc*. 287: 1815-1821.

[30] Ganther, H.E., C. Goudie, M.L. Sunde, M.J. Kopecky, P. Wagner, S.H. Oh and W.G. Hoekstra. 1972. Selenium: relation to decreased toxicity of methylmercury added to diets containing tuna. *Science*. 175:1122.

[31] Culvin-Aralar, L.A. and R.W. Furness. 1991. Mercury and selenium interaction: a review. *Ecotoxicol Environ Saf* 21:348-364.

[32] Ralston, C.R., J.L. Blackwell III and N.V.C. Ralston. 2006. Effects of dietary selenium and mercury on house crickets (*Acheta domesticus* L.): Implications of environmental co-exposures. *Environmental Bioindicators* 1(1):98-109.

[33] Chapman, L. and H.M. Chan. 2000. The influence of nutrition on methylmercury intoxication. *Environ Health Perspect* 108: 29-56.

[34] Rayman, M. 2000. The importance of selenium to human health. *Lancet* 356:233-241.

[35] Chen, J. and M.J. Berry. 2003. Selenium and selenoproteins in the brain and brain diseases. J *Neurochem* 86:1-12.

[36] Kohrle, J., F. Jacob, B. Contempre and J.E. Dumont. 2005. Selenium, the thyroid and the endocrine system. *Endocr Rev* 26(7):944-984)

[37] Schauzer, G.N. 2000. Anticarcingenic effects of selenium. *Cell Mol Life Sci* 57(13-14):1864-1873).

[38] Parizek, J. and I. Ostadalova. 1967. The Protective Effect of Small Amounts of Selenite in Sublimate Intoxication, *Experientia.* 23(2):142–143.

[39] Koller, L.D. and J.H. Exon. 1986. The two faces of selenium-deficiency and toxicity are similar in animals and man. Can J Vet Res 50: 297-306.

[40] National Research Council (NRC) 2000. Toxicological effects of Methylmercury. *National Academy Press*. Washington DC, p. 344.

[41] USDA National Nutrient Database for Standard Reference, Release 17. Selenium, Se (µg) Content of Selected Foods. http://www.nal.usda.gov/fnic/foodcomp/Data/SR21/nutrlist/sr21w317.pdf (accessed 7/15/09).USDA.

[42] Ralston, N.V.C., C.R. Ralston, J.L. Blackwell III, L J. Raymond. 2008. Dietary and tissue selenium in relation to methylmercury toxicity. *Neurotoxicology* 29:802-811.

[43] EPA and FDA. 2004. What you need to know about mercury in fish and shellfish. March 2004 www.cfsan.fda.gov/~dms/admehg3.html

[44] Steuerwald, U., P. Weihe, P.J. Jorgensen, K. Bjerve, J. Brock, B. Heinzow, E. Budtz-Jorgensen and P. Grandjean. 2000. Maternal seafood diet, methylmercury exposure and neonatal neurologic function. *J Pediatrics.* 136: 599-605.

[45] Myers, G. 2003. Statement by the University of Rochester research team studying the developmental effects of methylmercury. Handout, *U.S. Senate Committee on Environment and Public Works*, July 29, 2003.

[46] Myers, G.J., P.W. Davidson, C. Cox, C.F. Shamlaye, D. Palumbo, E. Cerichiari, J. Sloane-Reeves, G.E. Wilding, J. Kost, L.S. Huang and T.W. Clarkson. 2003. Prenatal methylmercury exposure from ocean fish consumption: 9-year evaluations in the Seychelles child development study. *Lancet*. 361: 1686-1692.

[47] Center for Disease Control and Prevention. 2001. Blood and hair mercury levels in young children and women of childbearing age – United States, 1999. 1999 National Health and Nutritional Examination Survey (NHANES). *Morbidity and Mortality Weekly Report*, March 2, 2001, 50(8): 140-143.

[48] Budtz-Jorgenson, E., P. Grandjean, N. Keiding, R.F. White and P. Weihe. 2000. Benchmark dose calculations of methylmercury-associated neurobehaviorial deficits. *Toxicology Letters*. 112-113: 193-199.

[49] Juhlshamn, K. A. Andersen, O. Ringal and J. Morkore. 1987. Trace element intakes in the Faroe Islands. I. Element levels in edible parts of pilot whales (*Globecephalus melaeanus*). *Science Total Environ* 65:53-62.

[50] Akagi, H., P. Grandjean, Y. Takizawa, and P. Weihe. 1998. Methylmercury dose estimation from umbilical cord blood concentrations in patients with Minamata Disease. *Environ. Res*. 77(2):98-103.

[51] Center for Food Safety and Applied Nutrition. 2002. Food advisory committee – methylmercury. Transcript of July 23, 2002, meeting at Sheraton College Park Hotel, Beltsville, MD. CFSAN, FDA, U.S. Dept. Health and Human Services. 227 p. (www.fda.gov/OHRMS/DOCKETS/ac/02/transcripts/3872t1.htm).

[52] Grandjean, P., P. Weihe, R.F. White, F. Debes, S. Araki, K. Yokoyama, K. Murata, N. Sorensen, R. Dahl and P.J. Jorgensen. 1997. Cognitive deficit in 7-year-old children with prenatal exposure to methylmercury. *Neurotoxicol. Teratol*. 19: 417-428.

[53] Caurant, F., M. Navarro and J. C. Amiard. 1996. Mercury in pilot whales: possible limits to the detoxification process. *Science Total Environ*.186:95-104.

[54] Budtz-Jorgensen, E., N. Keiding, P. Grandjean and P. Weihe. 2002. Estimation of effects of mercury exposure using structural equation models. *Environmental Health*.1: 2.

[55] Weihe, P. 2004. Letter dated 9 February 2004 To Whom It May Concern: Faroe Islands women do not eat mercury-tainted fish and fish consumption does not harm Faroese children.

[56] Endo, T., K. Haraguchi, F. Cipriano, M.P. Simmonds, Y. Hotta and M. Sakata. 2004. Contamination by mercury and cadmium in the cetacean products from Japanese market. *Chemosphere*. 54(11):1653-1662.

[57] Ahmed, F.E. ed. 1991. Seafood safety. Committee on evaluation of the safety of seafood products. *National Academy Press*. Washington, D.C.

[58] Jiang, H.M., G.A. Han and Z.L. He. 1990. Clinical significance of hair cadmium content in the diagnosis of mental retardation of children. *China Medical Journal (Engl).* April 1990; 103(4): 331-334.

[59] Riley, D.M. and V. M. Thomas. 2004. Mercury pollution: PU/CEES Working Paper No. 140, Center for Energy and Environmental Studies, Princeton University. 7 p.

[60] Ralston, N.V.C. 2009. Selenium Health Benefit Values as Seafood Safety Criteria. EcoHealth 5(4): 442-455.

[61] Crump, K.S., T. Kjellstrom, A.M. Shipp, A. Slivers and A. Stewart. 1998. Influence of Prenatal Mercury Exposure upon Scholastic and Psychological test performance: Benchmark Analysis of a New Zealand cohort. *Risk Analysis*. 18(6): 701-713.

[62] Clarkson, T.W. and J.J. Strain. 2003. Nutritional factors may modify the toxic action of methylmercury in fish-eating populations. *J Nutrition.* 133 (5 Supplement 1): 1539S-1543S.

[63] Pan, M. 1998. Multilevel and Multiobjective Programming Model for Hawaii Fisheries Management. PhD dissertation in Agricultural and Resource Economics, University of Hawaii.

[64] Joint FAO/WHO Expert Committee on Food Additives. 2003. Summary and conclusions of 61th meeting. June 10-19, 2003. Rome.

[65] Agency for Toxic Substances and Disease Registry. January 2004. Minimal risk levels for hazardous substances. U.S. Dept. of Health and Human Services. Atlanta, GA.

[66] Endo, T., K. Haraguchi and M Sakata. 2002. Mercury and selenium concentrations in the internal organs of toothed whales and dolphins marketed for human consumption in Japan. *Sci Total Environ* 300(1-3):15-22.

[67] Clarkson, T.W., L. Magos and G.J. Myers. 2003. The toxicology of mercury – current exposures and clinical manifestations. *New England J Medicine.* 349(18): 1731-1737.

[68] Rudd, J.W.M. 1995b. Mercury cycling in the boreal forest at the Experimental Lakes Area (ELA), Northwest Ontario. *Proceedings of 1995 Canadian Mercury Network Workshop.* Environmental Monitoring and Assessment Network, Environment Canada.

[69] Holman, R.T., S. Johnson and P. Ogburn. 1991. Deficiency of essential fatty acids and membrane fluidity during pregnancy and lactation. *Biochemistry, Proc. Natl. Acad. Sci*., June 1991. Vol. 88: 4835-4839.

[70] Helland, I.B., L. Smith, et al. 2003. Maternal supplementation with very-long-chain n-3 fatty acids during pregnancy and lactation augments children's IQ at 4 years of age. *Pediatrics*. 111(1): 39-44.

[71] Hibbeln, J.R., J.M Davis, C. Steer, P. Emmett, I. Rogers, C. Williams and J. Golding. 2007. Maternal seafood consumption in pregnancy and neurodevelopmental outcomes in childhood (ALSPAC study): an observational cohort study. *Lancet* 369:578-585.

[72] Olsen, S.F. and H.J. Secher. 2002. Low consumption of seafood in early pregnancy as a risk factor for preterm delivery: prospective cohort study. *British Medical J*. 324: 1-5.