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The Fish Paradox: Are Maternal Omega-3 (n-3) DHA and Selenium (Se) Intake Protective Against Negative Effects of Methylmercury Exposure on Infant Cognitive Development?

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Scientific uncertainty surrounds the consumption of fish and seafood by pregnant and breastfeeding women. The developing fetal brain is especially sensitive to the effects of methylmercury. Methylmercury bound to cysteine is the predominant form found in fish tissues and this adduct is able to be transported across placenta [1]. Mercury concentrations in fetal brain have been reported to be about five to seven times those of maternal blood [2]. Methylmercury readily crosses the blood-brain barrier and causes oxidative damage to the developing central nervous system. Neurotoxic effects of severe mercury poisoning resulting from catastrophic accidents in Minamata, Japan and Iraq although unusual are well established [3]. Low level exposure (<10 ppm maternal hair levels) from maternal seafood consumption is more common and the effects are inconsistent. Although a number of epidemiologic studies have shown a subtle negative impact of methylmercury exposure from high seafood intake on certain domains of learning, [4] maternal seafood consumption at lower levels is most often associated with improved cognitive development [5-7] or to have no effect, or inconsistent effects, on scholastic achievement upon long term follow up of children [8]. Seemingly paradoxical is the observation that, at relatively low levels of methylmercury exposure, cognitive ability improves as both fish consumption and maternal methylmercury increase [6,9]. An explanation for this paradox is the confounding effects of other nutrients, selenium which plays in protective role against the neurotoxicity of methylmercury and n-3 DHA which is an essential nutrient for optimal brain development. Although ocean fish are a source of methylmercury, seafood is the richest dietary source of omega-3 DHA and also an excellent source of selenium.

Seleniuim is an essential component of the antioxidant enzymes, glutathione peroxidase and thioredoxin. Methylmercury in excess of dietary selenium intake irreversibly inhibits these selenium-dependent enzymes and compromises the antioxidant defense systems in the brain [1]. The effect is related to reduction in intracellular selenium bioavailability and/or to covalent binding of methylmercury to selenium in the active sites of selenium-dependant enzymes [1]. Neural tissue is especially susceptible to peroxidative damage due the high rate of production of reactive oxygen species and to the neuronal cell morphology with a high ratio of membrane polyunsaturated lipids to cytosolic repair systems. High Se:mercury (Hg) ratios may mitigate the negative effects of methylmercury [1]. The strength of the evidence from animal models is good. In mice, supplementation with selenomethionine was shown to reverse inhibition of seleno-enzymes and prevent the neurotoxic effects of mercury [10]. Mitigation of methylmercury induced cerebral degeneration also been demonstrated in rats receiving selenomethioinine in utero [11]. Evaluation of the Se:Hg ratio may be a better method for examining neurocognitive effects of methylmercury exposure from seafood in humans. A report from the Faroe Island study showed that the negative effects of maternal intake of marine foods disappeared when adjusted for intake of fish, eliminating intake of flesh from marine mammals which is high in Hg low in Se [12]. However, a recent regression analysis of Hg and Se failed to show consistent interactions in two birth cohorts in the Faroe Islands [13].

Similarly, negative effects of higher levels of methylmercury exposure, have been demonstrated in the China only after controlling for n-3 DHA [14], although positive benefits of n-3 DHA were diminished at high maternal levels of mercury in cohorts from the Seychelles and U.S [6,15]. Results from the large British ALSPAC Study showed that mothers who consumed the greatest amount of n-3 DHA from fish throughout pregnancy and breastfeeding gave birth to babies who performed better on a wide variety of neurocognitive tests compared to the least amount of DHA. Effects of fish intake on cognitive development from the longitudinal Seychelles Child Development Nutrition Study have largely been attributable to the maternal n-3 DHA status which parallels dietary seafood intake [9,15,16].

The potential of nutrients to modify mercury toxicity remains an area of great interest. Current evidence suggests that selenium may mitigate the negative effect of methyl mercury and that the benefits of n-3 DHA outweigh the risks of consumption of most seafood during pregnancy and lactation. The greatest cognitive benefits are derived from maternal consumption of seafood which is highest in n-3 DHA and low in methylmercury. The modulating effect of selenium, although clear in animal models, has not been well documented in humans. Further research is needed to address this question.

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