Balancing Fish Consumption Benefits with Mercury Exposure

Grace M. Egeland and John P. Middaugh

The element mercury (Hg) occurs naturally in Earth's crust, but human activities—for example, coal burning, trash incineration, and industrial emissions—increase its release into the environment. Hg is methylated by organisms in fresh and marine water and concentrated through the food chain in the tissues of fish and marine mammals. Me-

thylmercury (MeHg), in toxic doses, causes neurological and developmental disorders in humans.

In an effort to reduce industrial emissions of Hg, the U.S. Environmental Protection Agency (EPA) developed a new reference

dose (RfD) for MeHg in 1996 (1). This RfD (the daily dose of MeHg that can be safely consumed over a lifetime) is used by states to develop their fish consumption advisories. The EPA RfD of 0.1 µg per kilogram per day is only one-fifth of the intake guidelines set by the World Health Organization (WHO) (0.47 μ g per kilogram per day) (2, 3) and, if followed, would severely restrict fish and seafood consumption. For example, average total Hg concentrations in bass, crappie, dolphin, halibut, mackerel, pike, snapper, and tuna range from 0.2 to 0.3 parts per million (ppm) (1). For an adult woman of average weight (60 kg), routine consumption of 4 ounces per week of fish containing average Hg tissue concentrations of 0.25 ppm would provide the RfD for methylmercury exposure.

Although the process of developing an RfD is valuable in efforts to regulate industrial emissions or to establish target levels in site-specific clean-up efforts, food consumption advice should occur within a broader multidisciplinary public health context that incorporates and weighs information on both risks and benefits. Fish and shellfish are food sources that are high in protein and low in saturated fat, are direct dietary sources of beneficial omega-3 polyunsaturated fatty acids (PUFAs) [eicosapentaenoic (20:5) and docosahexaenoic acid (DHA) (22:6)], and contain antioxidants

The authors are in the Section of Epidemiology, Division of Public Health, Alaska Department of Health and Social Services, Anchorage, AK 99524–0249, USA. E-mail: johnm@health.state.ak.us such as selenium and vitamin E (4–6).

Omega-3 PUFAs are important for optimal brain and retinal development, maturation of the visual cortex, and motor development; they may also help regulate the duration of quiet sleep episodes in human infants (7). During the third trimester of pregnancy, large amounts of the long-

> chain omega-6 and omega-3 PUFAs—arachidonic acid (20:4) (omega-6) and DHA—are mobilized to meet the demands of increased neural and vascular growth. Because up to 50% of the total fatty acids in the phospholipids of the cere-

bral cortex and retina consists of DHA, findings of diminished DHA in cell membranes during lactation and pregnancy (8) have suggested a need for omega-3 PUFA supplementation during pregnancy (7).

Fish consumption has also been related to a reduced risk of coronary heart disease (9). Although not all studies show a protective effect of fish consumption on coronary heart disease, fish and fish oils lower very-low-density lipoprotein cholesterol and triglyceride levels, inhibit platelet aggregation, and may reduce blood pressure (10, 11). As reported in the book *Risk vs. Risk*, the estimated benefits of reduced cardiovascular disease mortality from fish consumption far outweigh the imperceptible estimated increase in cancer risk associated with the presence of certain low-level polychlorinated biphenyls and related compounds (12).

Severely limiting the consumption of fish and seafood may do more harm than good by reducing the consumption of foods with health benefits and by increasing the consumption of alternative foods that have potential health risks. For populations that rely heavily on subsistence fishing, restrictive fish consumption advisories could damage the social, economic, and personal well-being of entire villages. In many areas in remote Alaska, commercially available foods are prohibitively expensive, grocery stores are poorly stocked or inaccessible, and there is insufficient variety in products to provide healthy alternatives to traditional foods. In Canada, the general health status of subsistence populations worsened after the social, economic, lifestyle, and dietary changes associated with fish consumption advisories (13, 14). In some cases, consumption advisories resulted in a complete avoidance rather than reduced consumption of a particular food (15).

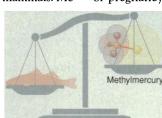
Other facts suggest caution in simple application of the EPA's RfD. Although MeHg exposure in the U.S. population occurs primarily through the consumption of fish and seafood, the RfD was generated by analysis of an acute poisoning episode in Iraq (1971–1972), when consumption of bread baked from grain treated with a Hg fungicide led to the hospitalization of over 6000 people and caused 400 deaths (16, 17).

Data from the Iraqi event may not be appropriate for calculating risks from lowlevel MeHg exposures through fish consumption. The Iraqi exposures were highdose and acute, and exposures may have included Hg compounds other than MeHg (17,



Preparing fish in Alaska. [Photo courtesy of the Alaska Division of Tourism]

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18). In addition, the delay of several years before data was collected could have limited the accuracy of recall of relevant developmental milestones by the mothers of affected children (19). A wide range in risk estimates was obtained by using different assumptions and modeling approaches (19).

Hg is ubiquitous in the environment, and some level of MeHg has always been present in the freshwater and marine food chain (20). Dietary factors present in fish and seafood, such as

naturally occurring selenium and vitamin E, may protect against the potential effects of lowlevel MeHg exposure.

The neurodevelopmental damage associated with industrial mercury pollution in Minimata and Nigata, Japan, and the effects of contaminated bread in Iraq are clear. However, published studies of populations consuming fish indicate no deleterious effects associated with MeHg exposures at levels comparable to or twice as high as WHO

intake guidelines (2, 21, 22). The WHO dietary intake guidelines correspond to hair Hg concentrations of 5 to 6 ppm. A comprehensive and rigorous study of over 700 infant-mother pairs in the Seychelles Islands found no deleterious

effects associated with MeHg exposures among the offspring of heavy fish consumers who were followed up to 29 months of age (21). Seventy-five percent of the women studied ate 10 to 14 fish meals per week (21). The median hair Hg level during pregnancy was 6.6 ppm (maximum 36 ppm; 80% of participants (659) \leq 12 ppm).

In a Peruvian study of 131 heavy fish consumers, peak maternal hair Hg levels ranged from 1.2 to 30 ppm, with a geometric mean of 8.3 ppm (22). No neurodevelopmental abnormalities were noted in the offspring. Among 234 Cree Indian children from northern Quebec, aged 12 to 20 months, the mean maternal hair Hg concentration during pregnancy was 6 ppm, and 6% had hair levels above 20 ppm (2). Abnormal muscle tone or reflexes were observed in 5 out of 13 boys in the highest exposure category of maternal hair Hg concentrations (13 to 24 ppm), but no consistent dose response was observed, and no association was noted among girls. In addition, no other associations were observed between the amount of Hg in mothers' hair during pregnancy and



Drying fish. [Photo courtesy of the Alaska Division of Tourism]

Methylmercury

their offspring's neurologic test results. In a study conducted in New Zealand, a slight decrease in the Weschler Intelligence Scale for Children was observed at 6 years of age for 15 offspring of women who had average hair Hg levels during pregnancy of 13 to 15 ppm and peak segmental hair Hg levels of 25 ppm. However, these smaller studies are of limited value (2).

In the Faroe Islands, where MeHg exposure occurs primarily through consumption

of pilot whale meat (23– 27), analyses of 917 children at 7 years of age found no clinical or neurophysiological Hg-related abnormalities (27). However, subtle decreases in neuropsychological test performance were associated with

prenatal Hg exposure at maternal hair levels below 10 ppm, "although test scores obtained by most of the highly exposed children were mainly within the range seen in the rest of the children . . ." (27). The longterm predictive value of these findings is not known, and the generalizability of these data to fish consumers is questionable. Interestingly, the Faroese children had excellent visual contrast sensitivity (27) that may be attributed to the ample supply of dietary omega-3 fatty acids.

Given all the available data, we question the scientific merits of restrictive fish consumption advisories. The EPA's new RfD could result in fish consumption advisories for sport and subsistence fish consumers when average fish tissue concentrations are as low as 0.2 ppm, although fish with Hg tissue concentrations up to 1 ppm are fit for commercial sale as regulated by the U.S. Food and Drug Administration, and the federal program for Women, Infants and Children provides food vouchers for tuna (which contains, on average, 0.2 ppm Hg) to participants because of its superior nutritional value. As state public health workers use the new RfD to develop fish consumption advisories, additional factors that should be considered include the uncertainties in risk, the potential health benefits of fish consumption, the competing risks associated with other available food sources, the potential medical impact of dietary and lifestyle changes on a population, and the social and economic ramifications of restrictive fish and seafood consumption advisories.

References and Notes

- EPA, "Mercury Study Report of Congress: Characterization of Human Health and Wildlife Risks from Anthropogenic Mercury Emissions in the United States," SAB Review Draft EPA-452/R-96-001f (1996).
- World Health Organization, "Environmental Health Criteria 101: Methylmercury" (WHO, Geneva, Switzerland, 1990).
- 3. L. Tollefson and F. Cordle, *Environ. Health. Perspect.* 68, 203 (1986).
- J. E. Kinsella, Seafoods and Fish Oils in Human Health and Disease (Marcel Dekker, New York, 1987).
- J. Bauernfeind, in *Vitamin E, A Comprehensive Treatise*, L. J. Machlin, Ed. (Marcel Dekker, New York, 1980), pp. 99–168.
- K. W. M. Siu and S. S. Berman, in *Occurrence and Distribution of Selenium*, M. Ihnat, Ed. (CRC, Boca Raton, FL, 1989), pp. 263–293.
- R. Uauy-Dagach and P. Mena, *Clin. Perinatol.* 22, 157 (1995).
- R. T. Holman, S. B. Johnson, P. L. Ogburn, *Proc. Natl. Acad. Sci. U.S.A.* 88, 4835 (1991).
- M. L. Daviglus *et al.*, *N. Engl. J. Med.* 336, 1046 (1997).
- H.O. Bang, J. Dyerberg, A. B. Nielson, *Lancet* 1, 1143 (1971).
- National Institutes of Health, Effects of Fish Oils and Polyunsaturated Omega-3 Fatty Acids in Health and Disease, Bibliography 1995-A (NIH, Bethesda, MD, 1995).
- P. D. Anderson and J. B. Wiener, in *Risk vs. Risk: Tradeoffs in Health and Environmental Protection*, J. D. Graham and J. B Wiener, Eds. (Harvard Univ. Press, Cambridge, MA, 1995), pp. 104–123.
- 13. B. Wheatley, Arctic Med. Res. 53, 386 (1994).
- A. M. Shkilnyk, A Poison Stronger Than Love: The Destruction of an Ojibwa Community (Yale Univ. Press, New York, 1985).
- P. J. Usher et al., Communicating About Contaminants in Country Food: The Experience in Aboriginal Communities (Research Department, Inuit Tapirisat of Canada, Ottawa, Ontario, 1995).
- 16. D. O. Marsh *et al.*, *Arch. Neurol.* **44**, 1017 (1987).
- F. Bakir *et al.*, *Science* **181**, 230 (1973).
 S. B. Skerfving and J. F. Copplestone, *Bull.*
- S. B. Skerfving and J. F. Copplestone, Bull. World Health Org. 54, 101 (1976).
- 19. K. Crump et al., Risk Anal. 15, 523 (1995).
- J. C. Hansen, T. Y. Toribara, A. G. Muhs, Meddeleiser om Gronland Man & Soc. 12, 161 (1989).
- 21. G. J. Myers *et al.*, *Neurotoxicology* **16**, 629 (1995).
- D. O. Marsh, M. D. Turner, J. C. Smith, P. Allen, N. Richdale, *ibid.*, p. 717.
 P. Grandjean *et al.*, *Arch. Environ. Health* 47,
- 23. P. Grandjean *et al.*, Arch. Environ. Health **47**, 185 (1992).
- P. Grandjean, P. Weihe, J. B. Nielsen, *Clin. Chem.* **40**, 1395 (1994).
 P. Grandjean, P. Weihe, R. F. White, *Neuro-*
- P. Grandjean, P. Weihe, R. F. White, Neurotoxicology 16, 27 (1995).
- R. Dahl *et al.*, *Neurotoxicol. Teratol.* **18**, 1 (1996).
 P. Grandjean, P. Weihe, R. F. White, *Neuro-toxicology* **20**, 1 (1997).
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