Conclusions and Recommendations

Methyl mercury is a compound worthy of scientific and societal concern. It is clear that MeHg is a widespread environmental contaminant and a potent neurotoxicant that adversely affects the developing nervous system. Mercury continues to be released into the environment by both natural and human-generated sources. It is readily converted to MeHg and accumulates in the food supply, primarily in fish and marine mammals. MeHg is readily absorbed and distributed throughout the body, including the brain and the fetus. Fetal exposure appears to be at a level that is greater than maternal blood levels. Studies of humans exposed to elevated levels of MeHg clearly demonstrate its neurotoxic potential. Animal studies using rodents and nonhuman primates have confirmed the neurotoxic potential of MeHg. However, research into cellular and molecular mechanisms has yet to produce an understanding of MeHg sufficient to allow accurate prediction of its neurotoxicity. Furthermore, human and animal studies on the neurobehavioral effects of developmental MeHg exposure have not determined a level of exposure that is convincingly harmless to the developing fetus.

In many ways, our understanding of the neurotoxic potential of MeHg is similar to that of lead 20 years ago; MeHg is a known neurotoxicant at high levels of exposure but there is little understanding of its effects at lower levels of exposure. The failure to adequately characterize the functional effects of low-level MeHg exposure has compromised the formulation of a sound policy regarding the safe levels of MeHg exposure, particularly for pregnant women or women of child-bearing age. Examination of the results of human studies on the effects of MeHg indicate that maternal hair levels of 10 to 20 ppm may result in adverse effects on fetal outcome. Making the appropriate assumptions and calculations, a level of exposure not expected to be hazardous (RfD) would be 0.06 μg/kg/day. Evaluation of results from animal studies on the developmental effects of MeHg provided an estimated RfD of 0.025 μg/kg/day. The human and animal RfDs are in very good agreement.

Given the current state of knowledge with regard to MeHg exposure, the following recommendations are offered:

- reduce environmental release of all forms of mercury;
- consider restricting the global production and sale of mercury;
- strongly advise pregnant women and women of child beating age to limit their exposure to sources of MeHg;
- establish an RfD (reference dose) for MeHg of 0.025 to 0.06 μg/kg/day;
- continue research to determine a level of MeHg exposure that would not harm the developing nervous system;
- continue research to understand the underlying molecular mechanisms of action of MeHg;
- assess the long-term neurodegenerative effects of developmental MeHg exposure.

REFERENCES

1. OTA. Neurotoxicity; Identifying and Controlling Poisons of the Nervous System. Washington:U.S. Congress, Office of Technology Assessment, 1990.
2. NRC. Environmental Neurotoxicology. National Research Council, 1991.
3. Landrigan PJ, Graham DG, Thomas RD. Strategies for the prevention of environmental neurotoxic illness. Environ Res 61:157-163 0 993).
4. WHO. Inorganic Mercury Environmental Health Criteria 118. Geneva:World Health Organization, 1991.
5. Burbacher TM, Rodier PM, Weiss B. Methylmercury developmental neurotoxicity: a comparison of effects in humans and animals. Neurotoxicol Teratol 12:191-202 (1990).
6. Chang L, Reuhl K. Mercury in human and animal health. In: Trace Elements in Health: A Review of Current Issues (Rose J. ed). Boston:Butterworths, 1983;132-149.
7. Chang LW. Neurotoxic effects of methylmercury intoxication—a review. Environ Res 14:329-373 (1987).
8. Clarkson TW, Hursh JB, Sager PR, Syversen TLM. Mercury. In: Biological Monitoring of Toxic Metals (Clarkson TW, Friberg L, Nordberg GF, Sager PR, eds). New York:Johns Hopkins Press, 1988,199-246.
9. Clarkson TW. Mercury: major issues in environmental health. Environ Health Perspect 100:31-38 (1992).
10. Eccles CV, Annau Z. The Toxicity of Methylmercury. Baltimore:Johns Hopkins University Press, 1987.
11. ATSDR. Toxicological Profile for Mercury. Atlanta:Agency for Toxic Substances and Disease Registry, 1994.
12. Fitzgerald WF, Clarkson TW. Mercury and monomethylmercury: present and future concerns. Environ Health Perspect 96:159-166(1991).
13. Reuhl KR, Chang LW. Effects of methylmercury on the development of the nervous system: a review. Ncurotoxicology 1:21-55 (1979).
14. Stern AH. Re-evaluation of the reference dose for methylmercury and assessment of current exposure levels. Risk Anal 13:355-364 (1993).
15. U.S. EPA. U Methylmercury-IRIS (Integrated Risk Informarion System). Washingotn:U.S. Environmental Protection Agency, 1992.
16. WHO. Methylmercury. Environmenral Health Criteria 101. Geneva:World Health Organization, 1990.
17. WHO. Mercury—Environmental Aspects. Environmenral Health Criteria 86. Geneva:World Health Organization. 1989.
18. Pfeiffer WC, de-Lacerda LD, Malm O, Souza CM, da-Silveira EG, Bastos WR. Mercury concentrations in inland waters of gold-mining areas in Rondonia, Brazil. Sci Total Environ 88:233-240(1989).
19. Brosset C. The mercury cycle. Water Air Soil Poll 16:253-255 (1981).
20. Pfeiffer WC, Drude de Lacerda L. Mercury inputs into the Amazon Region, Brazil. Environ Technol Lett 9:325-330 (1988).
21. Drude de Lacerda L, Pfeiffer WC. On AT. Gloria da Silveira E. Mercury contamination in the Madeira River, Amazon—Hg inputs to the environment. Biotropica 21:91-93 (1989).
22. Magos L. The absorption, distribution and excretion of methyl