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Methylmercury in the Environment: A Review of Current Understanding

by W. Stopford* and L. J. Goldwater*

The danger of methylmercury poisoning appears to be slight when the environment is not directly contaminated with methylmercury. Sediments rapidly bind mercury and decrease its availability to aquatic organisms. Sediments further have a greater propensity to demethylate than to methylate mercury. In noncontaminated aquatic ecosystems, the concentrations of methylmercury and inorganic mercury are many times lower than those that have been found to cause toxicity, even in the most sensitive organisms. Methylmercury bound to protein is comparatively less toxic than methylmercury salts, and selenium present in this protein appear to be one of the major detoxifying agents for methylmercury. This is particularly important in seafood, where there is an excess of selenium compared to methylmercury.

Interest in mercury in the environment has been greatly stimulated since the epidemics of methylmercury poisoning in Minimata and Niigata due to ingestion of contaminated seafood and because of concern over similar poisonings elsewhere. Substantial work has been done in the past five years to delineate the passage and effect of mercury in ecosystems and its eventual impact on man. We shall try to summarize some of the more important recent findings in this area.

Mercury in Sediments

When mercury is first deposited in a sediment, it is rapidly and strongly complexed to various components of the sediment. Mercury is most strongly bound to sulfur-containing organic and inorganic particles. In surface sediments, up to 62% of the mercury present is bound to these types of particles (*1*). To a lesser extent, mercury is also bound strongly to clays, mineral sediments containing iron and manganese oxides, and to fine sands (*2*). Only a small portion of mercury in sediments is released into the pore water. In this interstitial water, mercury appears to be associated primarily with organic acids such as fulvates and humates with little or none of the mercury in the unbound form (*3, 4*). Of mercury present in deeper sediments, 65 to 75% is also bound to organic acids (*1*). With or without agitation, the rate of release of mercury from sediments is slow and from sulfur-containing sediments is hardly measurable (*2*).

Methylation

Because of the greater toxicity of methylmercury as compared to nonalkylmercury compounds, great attention has been directed toward the formation and passage of methylmercury in aquatic sediments. Organisms present in many sediments are able to methylate inorganic mercury under ideal laboratory conditions (*5, 6*). Methylating organisms that have been isolated grow only under very strict conditions: they are microaerophilic, being killed if the sediment is agitated; they grow only in a narrow pH range; and, even under ideal conditions, they are slow growers (*7*). Methylation appears to occur only in the top 1–2 cm of sediment. Burrowing sediment organisms, however, can expose mercury present at deeper layers to the methylating process (*8*). When the pH of a sediment is raised, mercury is bound less tightly to organic acids and sulfide complexes and is more readily available for methylation (*9*). When mercury is bound to sulfides, there is little demonstra-

*Department of Community Health Sciences, P.O. Box 2914, Duke University Medical Center, Durham, N.C. 27710.

ble methylation under anaerobic conditions. Even under aerobic conditions the rate of methylation is only about 0.001 that for mercuric chloride under the same conditions (*10*). Methylation even under ideal conditions can at best convert less than 1.5% of the inorganic mercury present per month (*11, 12*).

Demethylation

Little or no methylmercury, however, is found in sediments (*13*). This might be explained by methylmercury's lesser tendency to be absorbed by sediment constituents and greater tendency to be desorbed than inorganic mercury. Methylmercury tends to be bound only to sulfur containing sediment particles, and, even in sulfur-containing sediments, the rate of absorption is one half to one third that for inorganic mercury salts. The rate of desorption of methylmercury from any type of sediment is from 10 to 1000 times that for inorganic mercury (*2*).

Another possible explanation for not finding appreciable amounts of methylmercury in sediments is that there is a greater tendency for sediments to demethylate than to methylate mercury compounds. As much as 15% of bacterial isolates from mercury-containing sediments have been found to demethylate mercury (*14*). These organisms are hardy, being able to demethylate both aerobically and anearobically. The demethylation process is rapid, with 100% of any methylmercury added to the cultures being demethylated within 4 days and, in some cases, within 1 day (*7, 15*). A majority of the organisms isolated that demethylate mercury have been identified as belonging to the *Pseudomonas* species (*7*).

When methylmercury is released into surface water, it can rapidly be photodegraded to inorganic mercury if bound to sulfide or thiol complexes (*16, 17*). In surface water, unbound methylmercury is rapidly and nearly completely bound to seston, that is, microorganisms such as plankton, algae, and bacteria, inorganic suspended material, and organic detritus (*18, 19*). Indeed, seston are the major reservoir for methylmercury in aquatic ecosystems, with the seston compartment containing 90–99% of the methylmercury in these systems (*19*). In eutrophic lakes where there is a large amount of organic matter, seston can decrease the amount of methylmercury reaching higher orders of aquatic organisms (*20*). This could be a dilutional effect, with less methylmercury per gram of organic matter available to organisms feeding within the seston compartment. There is a possibility, however, that algae decrease methylmercury's availability to higher biologic orders by converting methylmercury to inorganic mercury. In one experiment, algae completely absorbed all of the methylmercury added to the test system. After about 1 week, however, only 10% of the mercury present in the algae was in the form of methylmercury (*18*).

Aquatic Food Chains

Mercury is avidly taken up by lower biologic orders in aquatic ecosystems (*19–21*). Greater than 75% of methylmercury present in these lower orders is taken up directly from water. Even in higher orders, such as predatory fish, as much as 60% of methylmercury present is taken up from water (*22*). At each higher trophic level the concentration of methylmercury usually increases (*18, 22*). In fish this might be explained by methylmercury's prolonged half-life. Methylmercury is rapidly cleared from the aquatic environment and bound mostly to muscle tissue. When exposed to similar concentrations of methylmercury and inorganic mercury, fish are able to absorb methylmercury from water 100 times as fast as the inorganic mercury and are able to absorb five times as much methylmercury from food as compared to inorganic mercury (*23*). Once absorbed, methylmercury is retained two to five times as long as inorganic mercury. With increased fish size, both the uptake of methylmercury from the environment and the clearance of methylmercury from the fish is decreased. Because, however, methylmercury is strongly bound to muscle, methylmercury does accumulate appreciably with increased muscle mass and increased duration of exposure. With fish of the same size and with similar conditions of exposure, the rate of uptake and clearance of methylmercury is approximately the same in all species (*23*).

Direct methylation of inorganic mercury by members of higher biologic orders has been postulated to account for the higher methylmercury levels found in these orders. For instance, liver homogenates of certain species of tuna and trout have been found to methylate mercury (*24*). *In vivo* experiments, however, where fish and rats have been exposed to methylmercury, have suggested the occurrence of demethylation, with a larger fraction of the mercury in the liver and kidneys being in the form of inorganic mercury (*25, 26*).

Mercury Toxicity in Food Chain Organisms

In aquatic ecosystems, mercury is quite toxic to lower biologic orders and to juveniles of certain species. At concentrations of less than 0.1 ppb, methylmercury causes a decrease in the growth rate of phytoplankton and a decreased reproduction of daphnia (*21, 27*). At similar levels inorganic mercury causes a decreased long term survival of fiddler crab larvae (*28*). In fish toxicity has been noted at 3 ppb for both methylmercury and mercuric chloride (*29, 30*). These toxic levels of mercury compare with normal methylmercury levels in surface water of less than 0.001 ppb and inorganic mercury levels of less than 0.05 ppb (*13, 31*).

Biologic Versus Chemical Methylmercury

Methylmercury complexed to organic matter appears to be less toxic than methylmercury salts. Both fish and mice absorb only one third as much biologic bound methylmercury as compared to methylmercury chloride (*32*). Japanese quail fed methylmercury introduced in the form of tuna fish meal experienced decreased mortality and increased growth as compared to quail fed similar amounts of methylmercury added directly to corn–soya meal (*33*). An explanation for this difference might be the presence of selenium in certain biologic materials. Selenium readily complexes with methylmercury. When present in near equimolar amounts, it greatly detoxifies methylmercury. Indeed, in seawater and in seafood, selenium is present in concentrations in excess of methylmercury *(33)*. When introduced into the diet, selenium protects against the toxic effects of methylmercury in both rats and quail (*33, 34*). It is interesting that fish taken from Minimata Bay were found to be high in methylmercury but comparatively low in selenium, with a molar ratio of selenium to methylmercury of about 1:10 (*35*).

Toxicity of Methylmercury in Man

Recently, more has been elucidated about the toxicity of methylmercury in man. Although chromosomal breaks have been found in onion root tips exposed to concentrations of methylmercury that cause neurotoxicity in animals (*36*), no genetic defects or excessive chromosomal abnormalities have been found in children with congenital methylmercury poisoning (*37*). With severe long-term methylmercury poisoning, brain atrophy with associated presenile dementia and atrophy of the Islets of Langerhans of the pancreas with associated diabetes mellitus have resulted (*37*). Methylmercury poisoning has yet to be demonstrated in human populations not exposed directly to methylmercury or to food contaminated with methylmercury. Recent studies of populations who subsist mainly on seafood that is naturally high in methylmercury have failed to demonstrate any evidence of methylmercury poisoning, even with whole blood methylmercury levels that average three to eight times that a comparative non-fish-eating population *(38,39)*.

In summary, the danger of methylmercury poisoning appears to be slight when the environment is not directly contaminated with methylmercury. Sediments rapidly bind mercury and decrease its availability to aquatic organisms. Sediments further have a greater propensity to demethylate than to methylate mercury. In noncontaminated aquatic ecosystems, the concentrations of methylmercury and inorganic mercury are many times lower than those that have been found to cause toxicity even in the most sensitive organisms. Methylmercury bound to protein is comparatively less toxic than methylmercury salts, and selenium present in this protein appears to be one of the major detoxifying agents for methylmercury. This is particularly important in seafood where there is an excess of selenium compared to methylmercury.

REFERENCES

1. Walters, L. J., Jr., and Wolery, T. J. Transfer of heavy metal pollutants from Lake Erie bottom sediments to the overlying water. Ohio State University, Columbus, 1974.
2. Reimers, R. S., and Krenkel, P. A. Sediment sorption phenomena. CRC Crit. Revs. Environ. Control 4:265 (1974).
3. Fitzgerald, W. F., and Lyons, W. B. Organic mercury compounds in coastal waters. Nature 242: 452 (1973).
4. Lindberg, S. E., and Harriss, R. C. Mercury-organic matter associations in estuarine sediments and interstitial water. Environ. Sci. Technol. 8: 459 (1974).
5. Jernelov, A. Conversion of mercury compounds. In: Chemical Fallout. Miller, M. W., and Berg, G., Eds. C. C Thomas, Springfield, Ill., 1969.
6. Gillespie, D. C. Mobilization of mercury from sediments into guppies. J. Fisheries Res. Board Can. 29: 1035 (1972).
7. Spangler, W. J., Spigarelli, J. L., and Miller, H. M. Studies on the biomethylation of mercury. Midwest Research Institute, Kansas City, 1972.
8. Jernelov, A. Release of methyl mercury from sediments with layers containing inorganic mercury at different depths. Limnol. Oceanog. 15: 958 (1970).
9. Matsumura, F., Gotoh, Y., and Boush, G. M. Factors influencing the translocation and transformation of mercury in river sediment. University of Wisconsin, Madison, 1972.
10. Fagerstrom, T., and Jernelov, A. Formation of methyl mercury from pure mercuric sulfide in aerobic sediment. Water Res. 5: 121 (1971).
11. Jensen, S., and Jernelov, A. Biologic methylation of mercury in aquatic organisms. Nature 223: 753 (1969).

12. Jacobs, L. W., and Keeney, D. R. Methylmercury formation in mercury-treated river sediments during *in situ* equilibration. J. Environ. Qual., in press.
13. Andren, A., and Harriss, R. C. Methylmercury in estuarine sediments. Nature, 245: 256 (1973).
14. Spangler, W. J., et al. Degredation of methylmercury by bacteria isolated from environmental samples. Appl. Microbiol. 25: 448 (1973).
15. Spangler, W. J., et al. Methylmercury: bacterial degradation in lake sediments. Science 180: 192 (1973).
16. Zepp, R. G., et al. Methylmercuric complexes in aquatic systems. Environ. Letters, 6: 117 (1974).
17. Baughman, G. J., et al. Chemistry of organomercurials in aquatic systems. Environmental Protection Agency, Research Triangle Park, 1971.
18. Matida, Y., and Kumada, H. Distribution of mercury in water, bottom mud and aquatic organisms of Minamata Bay, the River Agano and other water bodies in Japan. Bull. Freshwater Fish. Res. Lab 19:73 (1969).
19. Huckabee, J. W., and Goldstein, R. A. Dynamic redistribution of methylmercury in a pond ecosystem. In: Proceedings of the First Annual NSF Trace Contaminants Conference. National Science Foundation, 1973.
20. Fagerstrom, T., and Jernelov, A. Biological methylation of mercury, food chain accumulations, ecological effects and routes of exposure to man. CRC Crit. Rev. Environ. Control 4: 296 (1974).
21. Biesinger, K. E. The chronic toxicity of mercury to *Daphnia magna*. National Water Quality Laboratory Report, U.S. Environmental Protection Agency, Duluth, 1974.
22. Jernelov, A., and Lann, H. Mercury accumulation in food chains. OIKOS 22: 403 (1971).
23. de Freitas, A. S. W., Qadri, S. U., and Case, B. E. Origins and fate of mercury compounds in fish. In: Proceedings of the International Conference on Transport of Persistent Chemicals in Aquatic Ecosystems, Ottawa, 1974.
24. Imura, N., Pan, S., and Ukita, T. Methylation of inorganic mercury with liver homogenate of tuna fish. Chemosphere 5: 197 (1972).
25. Burrows, W. D., Taimi, K. I., and Krenkel, P. A. The uptake and loss of methylmercury by freshwater fish. In: Congreso Internacional Del Mercurio, Barcelona, 1974.
26. Norseth, T., and Clarkson, T. W. Studies on the biotransformation of 203 Hg-labelled methyl mercury chloride in rats. Arch. Environ. Health 21: 717 (1970).
27. Harriss, R. C., White, D., and MacFarlane, R. Mercury compounds reduce photosynthesis in plankton. Science 170: 736 (1970).
28. DeCoursey, P. J., and Vernberg, W. B. Effect of mercury on survival, metabolism and behavior of larval *Uca pugilator* (Brachyura). Oikos 23: 241 (1972).
29. Matida, Y., et al. Toxicity of mercury compounds to aquatic organisms and accumulation of the compounds by the organisms. Bull. Freshwater Fish. Res. Lab. 21: 197 (1971).
30. Weir, P. A., and Hine, C. H. Effects of various metals on behavior of conditioned goldfish. Arch. Environ. Health 20: 45 (1970).
31. Hartung, R. A survey of heavy metals in the lower Mississippi River. University of Michigan, Ann Arbor, 1973.
32. Jernelov, A. Are there any differences between "biologically" and "chemically" synthesized methyl mercury? Vatten 3: 304 (1969).
33. Ganther, H. E., et al. Selenium: relation to decreased toxicity of methylmercury added to diets containing tuna. Science 175: 1122 (1972).
34. El-Begearmi, M. M., et al. Attempts to quantitate the protective effect of selenium against mercury toxicity using Japanese quail. Fed. Proc. 32: 886 (1973).
35. Uzioka, T. Analysis of mercury in food and animal organs. Kumamoto Igakkai Zasshi 34: 383 (1960).
36. Ramel, C. Genetic effects of organic mercury compounds. I. Cytological investigations on allium roots. Hereditas 61: 208 (1969).
37. Research Committee on Minamata Disease. Pathological, clinical and epidemiological research about Minamata Disease; 10 years after. (R. Ishizaki, translator). Interuniversity Consortium for Environmental Studies, Durham, N.C., 1974.
38. Turner, M. D., et al. Methyl mercury in a population eating large quantities of fish. I. Northern Peru. In: Proceedings Congreso Internacional Del Mercurio. Barcelona, 1974.
39. Marsh, D. O., et al. Methyl mercury (MeHg) in human populations eating large quantities of marine fish. II. American Samoa; cannery workers and fishermen. In: Proceedings Congreso Internacional Del Mercurio. Barcelona, 1974.