# Methyl mercury poisoning in Canada

By David A.E. Shephard, FRCP[C]

Is mercury poisoning Canadians? Might Canadians die of mercury poisoning?

These questions are of increasing concern because mercury can no longer be ignored as a lethal environmental pollutant. They demand our attention because some Canadians have blood mercury concentrations within the range associated with mercury poisoning in other countries, notably Japan and Iraq.

There is no doubt that mercury can kill and maim: during the last 2 centuries an estimated 8500 persons have been poisoned by mercury, of whom perhaps 700 died.[1] Mercury poisoning must therefore be considered a hazard to public health wherever mercury is used and wherever mercury is discharged into the environment. This is certainly true for Canada but, so that the entirety of the problem of mercury as an environmental pollutant be understood, wider aspects of mercury pollution — those relating to Canadian society — must be considered also.

## Consequences

Consider some of the effects of mercury pollution. In industry, vast quantities of mercury are used in a variety of technologic processes. In 1967 world consumption of mercury was 7364 metric tonnes, a large proportion being used by but a few industrial nations: the USA, for example, used 2127 tonnes.[2] (References cited give the figures in US tons of 2000 lb each; these have been translated into tonnes of 1000 kg or 2200 lb each). In Canada chloralkali plants, pulp and paper mills and the electrical industry consume much of the total mercury used; for example, in 1969, chloralkali plants used some 100 of a total of 140 tonnes used.[3]

With this industrial use, large amounts of mercury are discharged into the environment: of the total 7364 tonnes of mercury consumed by the world in 1967, 2573 tonnes was lost to the air and 2045 tonnes was lost to waterways.[2] Again, a Canadian example: until 1970, for every 100 tonnes of chlorine produced by chloralkali plants, 20 kg of mercury was lost,[3] and from 1961 to 1970 in one small area (the Wabigoon-English river system near the chloralkali plant in Dryden, Ont.) about 2 to 4.5 kg of inorganic mercury was dumped daily into local waterways.[4]

As well as effects on health, mercury pollution in Canada has had ecologic, sociocultural, political, racial and international implications. With other factors such as alcohol, it has disrupted the lives of Indians and has radically altered their traditional way of life, for the fish on which so many Indians depend for protein have become contaminated with mercury discharged into waterways from chloralkali plants and paper and pulp mills. This is particularly serious in northwest Ontario and northwest Quebec. Not surprisingly the ecologic and sociocultural effects of mercury pollution have had political impact, and federal and provincial authorities have been accused of making light of the dangers of mercury pollution to man and environment in Canada. Nor is it surprising that mercury pollution in Canada has had racial implications, for the Canadians who have the highest mercury concentrations are Canadian Indians.

Finally the international twist: first, Japanese victims of mercury poisoning have allied themselves with Canadian Indians of northwest Ontario, as have Japanese environmentalists, in an attempt to help Canadian Indians bring their plight to the attention of the federal and Ontario governments; second, Japanese physicians with much experience in mercury poisoning have examined Canadian Indians and, convinced of mercury poisoning in these Indians, have taken a stronger line than have Canadian physicians on mercury pollution in Canada. Unfortunately there is as yet no agreement on the clinical diagnosis of mercury poisoning.

Currently, the answers to the lead questions cannot be written, and speculation abounds. But the evidence must be examined to enable us to reach a rational conclusion, so that Canadian physicians may clarify the problem and decide what, if any, action need be taken.

## Quicksilver, slow killer

Mercury has long fascinated man: it has been dubbed "a mineral of a perfectly singular kind".[5]

Ramazzini, the father of industrial medicine, spoke in the 18th century of "the most cruel bane of all that deals death and destruction to miners",[6] and of course Lewis Carroll's Mad Hatter lives on as a symbol of the damage done to the nervous system by mercury.

The Mad Hatter conjures up an image of poisoning due to inorganic mercury, and this form of mercury poisoning was for long the most familiar. Today, however, because of the burgeoning of sophisticated technologic processes, it is poisoning from organic mercury that is of major concern — methyl mercury in particular, which damages the central nervous system irreversibly and sometimes fatally. The experience of several countries merits attention.

## Mercury poisoning in Japan

In 1965 a Japanese factory doctor in Minamata, a small town on the western coast of Kyushu in southern Japan, reported that "an unclarified disease" of the central nervous system had broken out.[7] Soon some 30 persons were found to have contracted the disease now known as Minamata disease during the previous 3 years. Diligent investigation finally disclosed that the disease was a disorder of the nervous system caused by poisoning with methyl mercury.[8] The disorder appeared to affect only fishermen and their families in and around Minamata; the patients evidently ate large amounts of fish regularly (the average consumption of fish in Minamata exceeds 300 g/d, in contrast to the average consumption of most Japanese fishermen of 200 g/d); the fish and shellfish of Minamata Bay were found to contain high concentrations of mercury — of the order 5 to 20 parts per million (ppm); the concentration of mercury in the mud of Minamata Bay was related directly to the proximity of the sample to a plastics factory in Minamata that produced acetaldelhyde and vinyl chloride (an inorganic mercury compound being used as the catalyst); and a similar disorder was produced in cats by feeding them fish contaminated with methyl mercury.

Kumamoto University physicians elucidated the clinical features of the disease. The main symptoms were numbness and paresthesias of the extremities, dysarthria, coordination disturbances, ataxia, constriction of the visual fields and impairment of hearing; its acute and subacute forms affected adults, children, infants and even the newborn. Of much interest was the mercury concentration in hair; in 25 patients the concentration ranged

Reprint requests to: Dr. D.A.E. Shephard, *Canadian Medical Association Journal*, PO Box 8650, Ottawa ON K1G 0G8

from 3.60 to 705 ppm,* in contrast to a range of 0.14 to 7.49 ppm among healthy persons outside the Minamata area.[8e]

Pathological studies revealed the sites of the damage in the nervous system: in the cerebral cortex, the neurons of the calcarine cortex, the precentral frontal cortex and the postcentral parietal cortex; in the cerebellum, the granular cells; and in the peripheral nervous system, the nerve fibres, with demyelination and destruction. The damage appeared to be severe and was often lethal; among survivors who had been incapacitated the damage was irreparable, so that many of the survivors have remained incapacitated.

Of epidemiologic interest was the observation that the number of cases reported between 1956 and 1960 was directly proportional to the amount of acetaldehyde and vinyl chloride produced by the plastics factory in Minamata.

Another outbreak of Minamata disease occurred in Niigata, Japan, in 1964 and 1965; it was virtually identical to the eponymous disease of Minamata itself.

To December 1974, 798 persons in Japan had been verified as having Minamata disease; of these, 107 died.

The Japanese experience is relevant to the Canadian situation because the Canadian Indians in whom high blood concentrations have been found are, like the Japanese of Minamata, heavy fish eaters — and fish in some Canadian waters have high concentrations of mercury. However, the Japanese data are less useful than those of studies of methyl mercury poisoning elsewhere because blood concentrations apparently were not documented at the time of intoxication. From analysis of hair content of mercury (hair and blood mercury concentrations are related in a ratio of either 300:1 or 200:1) Dinman and Hecker[9] have estimated that the concentration of mercury in the blood of persons with "fully-developed" intoxication would have ranged from 1230 to 1840 ppb (normal range, 5 to 10 ppb, with an upper limit of 50 ppb).[10] In this context, useful data are available from analysis of methyl mercury poisoning in Iraq.

### Iraq, New Mexico, Sweden

Iraq has had three outbreaks of organic mercury poisoning, two of which were major. In 1960 approximately 1000 persons were affected, and in 1971 and 1972 the huge total of 6430 cases was recorded, of which 459 were fatal.[11] The 1971-72 outbreak followed ingestion of bread made from grain treated with a methyl mercurial

*Ppb is the unit preferred for blood, ppm for hair.

fungicide; the symptoms and signs were similar to those of other outbreaks of methyl mercury poisoning, particularly those in Japan, though the Iraqi outbreak comprised acute cases. For some of the Iraqi patients, data for blood concentrations of mercury are available, and these provide valuable information, even though the first blood samples were taken at an average of 65 days after cessation of ingestion of mercury.

Bakir and his colleagues[11] were able to relate the blood concentrations of mercury to the incidence of symptoms (Table I). Four ranges of mercury concentration are of interest. Although the range of 0 to 100 ppb was associated with a 9.5% incidence of paresthesia and a 5% incidence of ataxia, Bakir and his group evidently believed that, at this concentration, the symptoms were probably caused by factors other than mercury. The next range of interest is 101 to 500 ppb, which was associated with incidences of paresthesia and dysarthria of 5%. Above this range the incidence of symptoms increased in direct proportion to the blood concentration, until concentrations exceeding 3000 ppb were associated with lethal damage.

In 1969 three members of a family in Alamogordo, New Mexico, were crippled by methyl mercury poisoning after the family had eaten hog meat over a period of several months; the meat had become contaminated when the hogs had eaten discarded waste seed grain that had been treated with an alkyl mercury fungicide. Three children were subsequently found to have severe brain damage of the Minamata type, and an infant born 8 weeks after the contaminated pork had been impounded was later found to be hypotonic, blind and retarded.[12] Dinman and Hecker[9] have calculated, from analysis of hair mercury concentrations, that the blood mercury concentrations of these children would have been 2900 to 4440 ppb.

Swedish workers have contributed much to our knowledge of mercury poisoning. Many Swedish waterways are polluted with mercury and the Swedes eat varying amounts of mercury-contaminated fish. Two papers are examples of Swedish work.

Birke and colleagues[13] studied two groups of persons — those who ate either no fish or small to moderate amounts of fish originating mainly from the ocean, and those who ate small, moderate or large amounts of fish contaminated by methyl mercury. Among 8 of 12 of the last group the whole-blood mercury concentration ranged from 7 to 650 ppb. Major neurologic symptoms and signs were recorded for

**Table I—Relation of clinical features of methyl mercury poisoning in Iraq to blood concentration**

| Whole-blood methyl mercury concentration (ppb) | Incidence of Symptoms (%) | | | | | |
|---|---|---|---|---|---|---|
| | Paresthesia | Ataxia | Visual changes | Dysarthria | Hearing defects | Death |
| 0 — 100 | 9.5 | 5 | 0 | 5 | 0 | 0 |
| 101 — 500 | 5 | 0 | 0 | 5 | 0 | 0 |
| 501 — 1000 | 42 | 11 | 21 | 5 | 5 | 0 |
| 1001 — 2000 | 60 | 47 | 53 | 24 | 0 | 0 |
| 2001 — 3000 | 79 | 60 | 56 | 25 | 12.5 | 0 |
| 3001 — 4000 | 82 | 100 | 58 | 75 | 36 | 17 |
| 4001 — 5000 | 100 | 100 | 83 | 85 | 66 | 28 |

**Data taken from Bakir et al.[11]**



only one of this group, a person whose whole-blood concentration was 125 ppb; he had a coarse tremor of the suggested intentional type and dysgraphia. Excepting one person, all the others, including the person with the highest mercury concentration, had minor neurologic symptoms or signs or both: occasional slight numbness of the legs or fingers, slight finger tremor and unspecifically abnormal electroencephalogram or learning deficiency or both; none, however, manifested visual field constriction. In other words, none of the eight persons who ate moderate or large amounts of contaminated fish showed clear-cut clinical evidence of methyl mercury poisoning — though the occurrence of nervous system abnormalities is certainly not without interest.

Skerfving[14] examined fish-eaters for concentrations of mercury in the blood cells and evidence of symptoms or signs of mercury poisoning. Except in one individual with a very high concentration of 1100 ng/g, the range of blood-cell mercury concentrations was from 8 to 390 ng/g. When these persons were divided into two groups according to the blood-cell concentration (12 to 75 ng/g and 82 to 1100 ng/g) and screened for signs and symptoms of mercury poisoning no clear-cut evidence of poisoning emerged. Even so, some nervous system signs and symptoms were apparent — notably, there was one person in the high-mercury group with constriction of visual fields — but there was no statistical difference in incidence of signs or symptoms between the two groups As with Birke's group the occurrence of nervous system signs is interesting, though the significance of the findings is difficult to interpret.

## Dose-response relationship

The study of Bakir and his group[11] documents a relationship between the amount of mercury ingested and the body's response. We know much more about the effects of *high* concentrations of mercury than we do about those of *low* concentrations, yet for public health we should be more concerned about the latter; therefore, no consideration of mercury poisoning can be complete without making use of all data as are available to clarify the problem of threshold concentrations.

Dinman and Hecker[9] analysed data for three groups of persons: those not known to be exposed to unusual mercury contamination, those known to have absorbed organic mercury compounds and manifesting signs and symptoms of organic mercury intoxication and those known to have absorbed organic mercury compounds and yet believed to be healthy.

The Niigata experience indicates that, in patients with "fully developed" intoxication, blood concentrations of mercury would have ranged from 1230 to 1840 ppb (depending on the hair: blood ratio used), so it is prudent to accept 1000 ppb as being the concentration above which poisoning may be evident — and this is consistent with the findings of the study by Bakir and colleagues.[11] The problematic range is that from 50 to 1000 ppb. For this range Dinman and Hecker[9] have accepted the range of 100 to 200 ppb as being insignificant and concentrations as high as 600 ppb similarly so. The range about which one cannot be sure, then, is 600 to 1000 ppb — but one must be concerned about this range in view of the uncertainty that surrounds the whole problem of the threshold for mercury poisoning.

The intake of methyl mercury is obviously important. Grant[15] has estimated that the average daily intake of methyl mercury in the USA is less than 0.06 mg — well below the intake of 1 mg/d that Berlin and colleagues[16] have stated is toxic to the brain. But the intake of methyl mercury increases sharply when all of the food eaten is fish, in which methyl mercury is concentrated to a greater extent than in many other foods. And if all the food eaten contained 0.5 ppm of mercury and the total amount eaten were 0.5 kg, the intake

there is no evidence that mercury poisoning does *not* exist (which, admittedly, would be hard to find). Moreover, the lifestyle of Canadians living near polluted waters is consistent with the likelihood of their being poisoned by mercury. Consider just one aspect of this — the eating habits of Canadian Indians. In northwest Quebec in the area of Quevillon (a polluted area) the average daily consumption of fish over a year is 0.3 kg, but the daily consumption varies according to the season, so that these naturally heavy fish-eaters may eat up to 0.7 kg/d during the summer; and the fish in this area of Quebec may contain up to 2 ppm of mercury. If one considers now the estimate by Grant[15] that if all food contained 0.5 ppm of methyl mercury and the amount of this food eaten daily were 0.5 kg, the daily intake of methyl mercury would amount to 0.25 mg (one quarter of the amount calculated by Berlin and colleagues[16] to be toxic to the brain), it is clear that ingestion of larger amounts of more seriously contaminated fish would lead to intake of methyl mercury approaching much more closely the toxic value. This seems to be the case with Canadian Indians.

The question whether some Canadians are being poisoned by mercury is not easy to answer, but there is sufficient evidence that methyl mercury is harmful and that some Canadians do have unacceptably high whole-blood concentrations of mercury. Because of the sinister possibility of mercury-induced irreversible brain damage the current situation demands our full attention. Action is required. What should be done?

First, the multidepartmental approach to mercury poisoning in Canadians should be replaced by one that is the responsibility of a single group. At present three governments, several government departments, at least one outside consultant, various local authorities, numerous neurologists and many other interested persons have separate responsibility.

In addition, various task forces and conferences on mercury contamination have added to the wealth of information now available on mercury poisoning. What is required is a cohesive effort to synthesize this information and the approaches of the past so that the Indians, the group mainly affected, can be given a clearer indication that a constructive approach will be followed.

Second, still more needs to be learned of the effects of methyl mercury, particularly its subclinical effects. Much of the information we have today is based on outbreaks of mercury poisoning elsewhere, the relevance of which to the situation in Canada is not entirely clear. Much more basic research is required into the problem of dose and response, and more epidemiologic research is needed concerning, for example, the degree to which mercury poisoning is irreversible, and autopsy findings in those known to have been exposed to high mercury whole-blood concentrations.

Third, it is desirable that differences between Canadian and Japanese neurologists, and between Canadian neurologists, be resolved. This will not be easy because these differences stem from differences in interpretation of clinical findings relating to Canadian Indians examined. In part the differences are attributable to a fragmented and quasipolitical approach to the problem of mercury poisoning.

Finally, we need a mature philosophical approach to the management of environmental problems, of which mercury contamination is but one. The current approach is a stopgap one, for, like so many environmental problems, mercury contamination is just one of many that require solution at any one time, and the solution to any one problem, then, is inadequate. Many of those who are trying to deal with an urgent problem must simultaneously be dealing with others; this is true, for example, of the medical services branch of the Department of National Health and Welfare, which is attempting to overcome the difficulties of mercury contamination in Indians while also dealing with many other unrelated matters, despite limitations in personnel and resources. It is in part a matter of priorities, but to a much greater extent our current problems have a more fundamental origin: Canada lacks the aggressive environmental philosophy of a country like Sweden, which puts teeth into its environmental legislation and muscle into the actions required.

Preventive medicine is always less glamorous than therapeutic medicine. Yet if the health of Canadians is to be preserved it is issues such as mercury pollution that are likely to be more important in the long run than many other apparently more pressing issues.


### Acknowledgements

I very much appreciate the generous help and ready cooperation of Drs. T.H. Patterson, T.F. McCarthy and B. Wheatley of the medical services branch, Department of National Health and Welfare, who made available to me valuable sources of information and constructively criticized this report. I also thank Drs. A. Eisen, A. Barbeau and W.H. Feindel for informal comments on mercury contamination in Canada.


### References and notes


1. D'ITRI FM: *The Environmental Mercury Problem.* Cleveland, OH, CRC Pr, 1972, p 73. Morbidity and mortality were estimated by this author at 1800 to 2000 and 120 to 150 respectively, but this did not take into account the additional figures for the 1971-72 outbreak in Iraq (6530 and 459 respectively) (see BAKIR F, et al, ref 11 below)
2. PARKER JS: Mercury, in *Minerals Yearbook,* Washington, US Dept of Interior, Bureau of Mines, 1967, p 721
3. FLEWELLING RJ: Loss of mercury to the environment from chloralkali plants. Paper read at symposium on mercury in man's environment, Ottawa, Feb 15-16, 1971
4. BERNSTEIN AD: Final report, Task Force on Organic Mercury in the Environment: Grassy Narrows and White Dog, Ontario. Ottawa, Health and Welfare Canada, 1973
5. HILL J, cited by PUTNAM JJ, in Quicksilver and slow death. *Natl Geogr Mag* 142: 507, 1972
6. RAMAZZINI B, cited by PUTNAM JJ, ibid
7. SMITH EG, SMITH AM: *Minamata,* New York, Holt, Rinehart and Winston, 1975
8. Of the many reports on Minamata disease, most are in Japanese. The most useful accounts in the English literature are: (a) MCALPINE D, ARAKI S: Minamata disease: an unusual neurological disorder caused by contaminated fish. *Lancet* 2: 629, 1958; (b) KURLAND LT, FARO SN, SIEDLER H: Minamata disease: the outbreak of a neurologic disorder in Minamata, Japan, and its relationship to the ingestion of seafood contaminated by mercurial compounds. *World Neurol* 1: 370, 1960; (c) KURLAND LT: The human health hazards of methylmercury, in *Mercury in the Western Environment,* BUHLER DR (ed), Corvallis, OR, Cont Ed Publs, 1973, p) 283; (d KURLAND LT: An appraisal of alkyl mercury compounds, in *Mercury, Mercurials and Mercaptans,* MILLER MW, CLARKSON TW (eds), Springfield, IL, CC Thomas, 1973, p 24; (e) TAKEUCHI T: Biological reactions and pathological changes in human beings and animals caused by organic mercury contamination, in *Environmental Mercury Contamination,* HARTUNG R, DINMAN BD (eds), Ann Arbor, MI, 1972, p 247
9. DINMAN BD, HECKER LH: The dose-response relationship resulting from exposure to alkyl mercury compounds, in *Environmental mercury contamination,* op cit, p 290
10. GOLDWATER LJ: "Normal" concentrations of metals in urine and blood. *WHO Chron* 2: 191, 1967
11. BAKIR F, DAMLUJI SF, AMIN-ZAKI L, et al: Methylmercury poisoning in Iraq; an interuniversity report. *Science* 181: 230, 1973
12. SNYDER RD: Congenital mercury poisoning. *N Engl J Med* 284: 1014, 1971
13. BIRKE G, JOHNELS AG, PLANTIN L-O, et al: Studies on humans exposed to methyl mercury through fish consumption. *Arch Environ Health* 25: 77, 1972
14. SKERFVING S: Methylmercury exposure, mercury levels in blood and hair, and health status in Swedes consuming contaminated fish. *Toxicol* 2: 3, 1974
15. GRANT N: Mercury in man. *Environment* 13 (4): 2, 1971
16. BERLIN MH, CLARKSON TW, FRIBERG LT, et al: Maximum allowable concentrations of mercury compounds. *Arch Environ Health* 19: 891, 1969
17. MCDUFFIE B, in discussion of KURLAND LT: An appraisal of alkyl mercury compounds, in *Mercury, Mercurials and Mercaptans,* op cit, p 50
18. Missing mercury mystifies govt. *Ottawa Citizen,* Oct 16, 1975, p 83
19. WOBESER G, NIELSEN NO, DUNLOP RH, et al: Mercury concentrations in tissues of fish from the Saskatchewan River. *J Fish Res Bd Can* 27: 830, 1970
20. KLEIN DH: Sources and present status of the mercury problem, in *Mercury in the Western Environment,* op cit
21. FEIT HA, cited by BERKES F, OTT B, BUTLER MJA, et al (eds): *Environmental Aspects of the Pulp and Paper Industry in Quebec,* 2nd ed, Montreal, 1972, p 172
22. BERNSTEIN AD: The significance of reports of mercury in various body tissues in the perspective studies in various Canadian populations. Paper read at Third International Symposium on Circumpolar Health, July 11, 1974, Yellowknife NT
23. One example is in TSUJI M: Minimizing Minamata. *MacLean's,* Dec 15, 1975, p 21
24. 57 Cree reach danger level in mercury. *Montreal Gazette,* Jan 21, 1976 p 1
25. CLARKSON TW: *Exposure to methyl mercury in Grassy Narrows and White Dog Reserves: an Interim Report.* Ottawa, Health and Welfare Canada, 1976
26. TAKEUCHI T, cited by KURLAND LT: in An appraisal of alkyl mercury compounds in *Mercury, Mercurials and Mercaptans,* op cit, p 29