Bull. Environ. Contam. Toxicol. (2005) 74:689–697

DOI: 10.1007/s00128-005-0638-7

Bulletin of Environmental Contamination and Toxicology

Followup Study of Mercury Pollution in Indigenous Tribe Reservations in the Province of Ontario, Canada, 1975–2002

M. Harada,[1] T. Fujino,[2] T. Oorui,[3] S. Nakachi,[4] T. Nou,[5] T. Kizaki,[6] Y. Hitomi,[6] N. Nakano,[6] H. Ohno[6]

[1] Department of Social Welfare Studies, Kumamoto Gakuen University, Kumamoto 862-8680, Japan
[2] Department of Neuropsychiatry, Kyoritsu Hospital, Minamata 867-0045, Japan
[3] Middle Gate Winnipeg, MB, R3C 2C5, Canada
[4] Environmental Monitoring Institute, Osaka 552-0007, Japan
[5] Kumanichi News Paper Office, Kumamoto 860-8506, Japan
[6] Department of Molecular Predictive Medicine and Sport Science, Kyorin University, School of Medicine, Mitaka 181-8611, Japan

Received: 15 August 2004/Accepted: 19 January 2005

In 1975, we went twice to the Province of Ontario, Canada, to make epidemiological and clinical investigations following mercury contamination. Minamata disease (mercury poisoning) was already evident, although the symptoms were slight (Harada et al. 1976; 1977). Later, although the provincial government did not officially recognize an outbreak of Minamata disease, they issued an advisory against fishing and eating fish, and made plans for financial compensation. However, even later they refused to recognize wide-scale mercury poisoning. We think that, in order to predict changes in the mercury level in the hair and the clinical symptoms over the long term, it is more important to know the long-term effects of methylmercury than to know the safe level (Harada 1995). Such a long-term follow-up study of mercury poisoning has not even been done in Minamata in Japan, to say nothing of other countries. Therefore, in September 2002, we went back to make a follow-up study.

MATERIALS AND METHODS

Between August 31 and September 3, 2002, we collected information by interviewing local citizens in Grassy Narrows where the poisoning took place (Figure 1). We also examined 57 people using the Japanese criteria for Minamata disease. The details are shown in Table 1. The clinical study consisted of routine neurological examinaton and examination of visual fields using Förster's perimeter in addition to setting out a questionnaire on subjective symptoms. Whenever permitted, we collected 47 hair samples and determined the amount of total mercury as described previously (Ikingura and Akagi 1996), with precision and accuracy (Harada et al. 1999). We analyzed only the total mercury level in hair, taking into consideration that over 90% of the mercury derived from the food chain is methylmercury (Harada et al. 2001).

Briefly, for total mercury analysis, 10 mg of finely cut hair sample was placed in a 50-ml volumetric flask with a long neck and digested with a mixture of 2 ml HNO_3-$HClO_3$ (1: 1), 5 ml H_2SO_4, and 1 ml distilled H_2O at 230-250°C for 20 min. After coolimg, each digested sample was made up to 50 ml by adding distilled water to the sample bottle and shaking to homogenize the sample. Total mercury

Correspondence to: H. Ohno

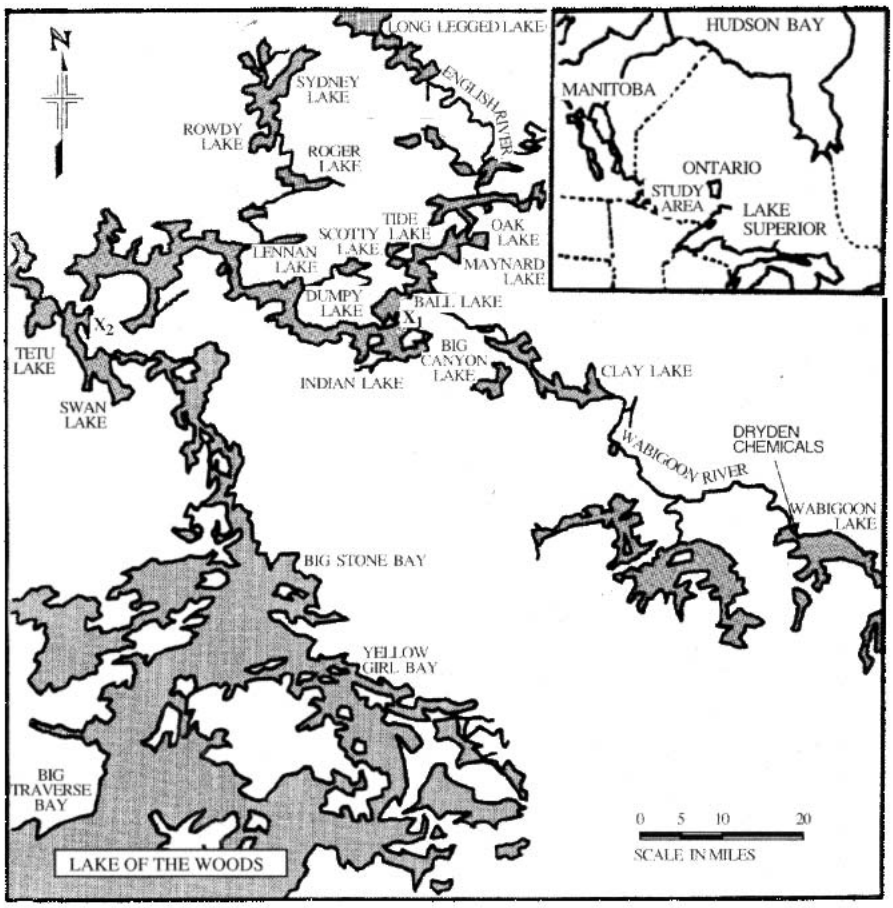

Figure 1. Map of the Wabigoon river system (Fimreite and Reynolds, 1973)
X_1: Grassy Narrows, X_2: White Dog

level in the sample solution was determined by cold vapor atomic absorption spectrometry using the RA-1 Mercury Analyzer (Nippon Instruments Corp., Tokyo). The Mercury Standard Solution for atomic absorption spectrometry (Kanto Chemical Co., Inc., Tokyo) was employed as standard samples. The detection limit is approximately 0.1 ppm. The precision and accuracy of the method used for total mercury analysis have been repeatedly verified by inter-laboratory calibration exercises (Department of Urban Engineering, Faculty of Engineering, University of Tokyo, Tokyo). There existed a high correlation between both results obtained (the correlation coefficient r = 0.983, n = 10), thereby indicating that the techniques used in the current study were reliable and accurate. All reagents used (Wako Pure Chemical Industries Ltd., Osaka) were of analytical grade.

The statistical significance of the data was assessed by ANOVA, followed by the Bonferroni post hoc comparison. When applicable, Student t test was used for

Table 1. Total mercury level in head hair from inhabitants in northwestern Ontario.

Group	n	Sex		Age		Total mercury (ppm)	
		M	F	Mean ± S.D.	Min-max	Mean ± S.D.	Min-max
Minamata disease	11	9	2	60.7 ± 14.5	38 – 90	2.24 ± 1.31 (n = 10)	0.84 – 4.50
Minamata disease with complications	12	9	3	49.4 ± 15.2	24 – 73	3.35 ± 4.84	0.24 – 18.1
Light Minamata disease	22	10	12	43.5 ± 14.8*	18 – 65	1.15 ± 1.37 (n = 21)	0.11 – 5.10
Others	12	6	6	20.5 ± 20.2	1 – 62	2.71 ± 3.06 (n = 4)	0.12 – 6.60

M, male; F, female. *P < 0.01 vs. Minamata disease group. The statistical significance of the data was assessed by exclusion of "others", because they included 8 children and the total mercury level in hair was determined only for 4 of them.

unpaired samples. Level of significance was set at $P < 0.05$.

RESULTS AND DISCUSSION

Of the 57 residents who underwent medical examinations, 34 were male and 23 female. Their ages ranged from 1 to 90 years old and their age distribution was: 7 aged 1 –10; 2 aged 11 –20; 6 in their 20's; 11 in their 30's; 7 in their 40's; 8 in their 50's; 13 in their 60's; 2 in their 70's; and 1 aged 90.

Seven children (1-11 years old) and one adult (49 years old) had no subjective symptoms at all. As for the other 49, apart from four who complained of pain in all limbs and two who complained of only numbness, they all had multiple subjective symptoms. The common subjective symptoms were: numbness in the hands and feet in 38; pain in the joints of all limbs and the hips in 26; cramps in the legs in 24; dizziness in 17; hearing difficulty in 15; a disturbed gait in 14; headache in 13; trembling in 12; forgetfulness in 12; and stiff fingers and difficulty in grasping in 10 (Table 2).

As for neurological symptoms, the most prominent were sensory symptoms. There were 31 cases of glove and stocking-type neuropathy, 21 of difficulty in balance and walking (standing on one leg, walking in a straight line, positive Mann's syndrome), 12 of tremors, 9 of perioral sensory impairment, 9 of mental retardation, 7 of ataxia, 7 of impaired speech, 7 of disturbed ocular movement, 6 of sensory impairment of the whole body, 6 of tunnel vision, 4 of convulsions, 4

Table 2. Clinical symptoms of inhabitants in northwestern Ontario (n = 57).

	n	%
Subjective symptoms		
Numbness	38	66.7
Pain in limb extremities and hips	26	45.6
Cramp in legs	24	42.1
Dizziness	17	29.8
Hearing difficulty	15	26.3
Disturbed gait	14	24.6
Headache	13	22.8
Trembling	12	21.1
Forgetfulness	12	21.1
Stiff fingers and difficulty in grasping	10	17.5
Objective symptoms		
Glove and stocking-type sensory disturbance	31	54.4
Difficulty in balance and walking	21	36.8
Tremor	12	21.1
Perioral sensory impairment	9	15.8
Mental Retardation	9	15.8
Ataxia	7	12.3
Impaired speech	7	12.3
Disturbed ocular movement	7	12.3
Sensory impairment of whole body	6	10.5
Tunnel vision	6	10.5
Convulsion	4	7.0
Fainting	4	7.0
Sensory impairment of either side of body	3	5.3

of fainting, and 3 of sensory impairment of either side of the body (Table 2).

Minamata disease was diagnosed based on the standard criteria; in addition to sensory impairment of extremities and around the mouth, there was one or more of the following: motor ataxia, tunnel vision, impaired hearing, difficulty in standing up and a disturbed gait, disturbed ocular movement, tremors, and impaired speech. The patients were categorized into three groups: the Minamata disease group; the Minamata disease with complications group; and the light Minamata disease group. The Minamata disease group had 11 cases. The Minamata disease with complications group had 12 cases. The light Minamata disease group had 22 cases, some of whom had sensory impairment, predominantly of the fingers and toes, while others had other symptoms, which changed easily. Counting all three groups, there were a total of 45 (78.9%) cases of Minamata disease, which is a higher rate than that in the Minamata incident.

There were 3 cases of cerebral palsy and 3 cases of mental retardation. However,

there is no evidence that these were caused by methylmercury when the children were in the womb. Excluding these children in addition to two children and four adults without any symptoms of Minamata disease, the mean age with S.D. (age range) of the Minamata disease group together with the Minamata disease with complications group was 54.8±15.6 years (24 – 90) as opposed to only 43.5± 14.8 years (18 - 65) for the light Minamata disease group (P<0.02) (Table 1). Therefore, the older people tended to have more severe symptoms, such as various neurological symptoms, particularly glove and stocking-type sensory disturbance.

The company that caused the pollution and the provincial and federal governments provided compensation for the victims through the Mercury Disability Board set up in 1986. Of the subjects examined in the current study, 21 victims who satisfied diagnostic criteria received Can $250 - 800 per month. Twenty claimants were denied and a decision is pending for 3 claimants. Thirteen of the 57 whom we examined did not file claims. Eight of the 10 claimants in the Minamata disease group, 7 of the 10 claimants in the Minamata disease with complications group, and only 3 of the 18 claimants in the light Minamata disease group were deemed eligible for compensation. Of the 6 claimants who had either cerebral palsy or mental retardation, only 2 were deemed eligible for compensation. Of another 4 claimants who were considered by us to have contracted other diseases, only 1 was deemed eligible for compensation. Excluding the children, the age range (mean±S.D.) of those deemed eligible was 39 – 90 years (56.7±18.6) and that of those deemed ineligible was 18 – 65 years (41.3 ± 13.8). Therefore, those deemed eligible were significantly older (P<0.01). Although the peak of the mercury pollution was in the 1970s, it seems that the symptoms increased or worsened with aging.

We determined the total mercury level in hair taken from 47 of the people. The level ranged from 0.11 to 18.1 ppm (2.07±2.87 ppm). With one exception, all the subjects showed a mercury level of less than 10 ppm. Today, very little mercury pollution remains in the area. Moreover, there was no significant difference in mercury level in hair among the Minamata disease group, the Minamata disease with complications group, and the light Minamata disease group, although that in the light Minamata disease group appeared to be lower (P<0.10) ((Table1).

Nineteen (43.2%) of the 44 people examined in our previous investigation in Grassy Narrows had died, although the cause of death remains unclear. In addition to the previous investigation in Grassy Narrows, we also made an investigation in 1975 of 45 residents in White Dog. Of the 89 people examined, 44.9% (40) had pain in all limbs, 31.4% (28) had numbness, and 17.9% (16) had leg cramps. As for neurological symptoms, 44.9% (40) had impaired hearing, 23.5% (21) had tremors, 16.9% (15) had glove and stocking-type neuropathy (considered important), 10.1% (9) had tunnel vision, 8.9% (8) had ataxia, and 5,6% (5) had perioral sensory impairment. At that time, almost all of these people had high levels of mercury in their hair; consequently, we think that they

had light symptoms of Minamata disease.

In the current investigation, as in the previous investigation, there was a high rate of subjective and neurological symptoms of Minamata disease. These symptoms would probably be due only to methylmercury (Harada 1995; Harada et al. 2001). However, because the current level of total mercury in the hair is low, the symptoms are thought to have been caused by mercury poisoning over a long time. Perhaps aging and the presence of medical complications might also be contributing to the worsening symptoms.

The previous analysis of the mercury level in the hair was performed in the Tokyo Metropolitan Research Laboratory of Public Health, Tokyo. The hair was sliced every 3 cm from the base. This enabled us to ascertain that the mercury uptake was the highest during summer, which is when the subjects typically eat a lot of fish. In nine subjects examined in both the current and previous studies, mercury is still detectable in their hair today, but at much lower levels than the recommended safe level of 50 ppm (Ohno et al. 1984). These cases are important as they demonstrate that chronic Minamata disease can occur due to long-term mercury poisoning even though the level is not above the recommended safe level.

In 1970, after a fish in the English-Wabigoon River system was found to have a mercury level as high as 27.8 ppm (Fimreite and Reynolds 1973), fish after fish with high levels of mercury were reported (Harada et al. 1976; 1997). In response, commercial and non-commercial fishing were prohibited. Otters and mink in the area, which feed on fish, disappeared (Fimreite and Reynolds 1973). Turkey vultures which could not fly properly were observed, and the mercury level in the liver from one of them was 96 ppm (Fimreite 1972). Likewise, high levels of mercury were found in the liver, muscle tissues and eggs of waterfowl (such as turkey vulture and common loon) in the area (Fimreite 1972). Furthermore, a cat caught in the area manifested symptoms of Minamata disease including pathological observations (Harada et al. 1976), and the levels of mercury in its brain, liver, and fur were very high, 16, 67, and 392 ppm, respectively, which is highly indicative of Minamata disease (Takeuchi et al. 1977). In an experiment, cats fed on the local fish developed symptoms of Minamata disease after about 90 days (Chabonneau et al. 1974). The mercury pollution in the environment and in the food chain was therefore very patent. The source of the pollution was a factory upstream that manufactured caustic soda (Harada et al. 1976; 1977). In the polluted area, there are also two reservations for indigenous tribes. Most of the people who live in the reservations are fishermen, hunters and tour guides. Not surprisingly, the mercury levels in their hair and blood became very high. In 1970, in Grassy Narrows, as much as 95.8 ppm of mercury was detected in a hair sample, and in the nearby settlement, White Dog, as much as 198 ppm was detected. After that, a branch of the Environmental Health Services reported that the mercury level was in decline (The Ontario Ministry of Health 1974). In 1975, however, we analyzed the hair

of 77 people and, in one hair sample, detected as much as 80.3 ppm. We also detected more than 30 ppm in the hair of 23 people, and more than 20 ppm in the hair of 44 people (Harada et al. 1976). At the same time, Clarkson (1976) detected as much as 105 ppm in a hair sample.

In 1975, we visited the contaminated area in March and again in August to carry out clinical and epidemiological investigations (Harada et al. 1976; 1977). The same doctor did this follow-up study and the studies 27 years earlier, and this is also unique. The serious and indicative symptoms of Hunter-Russell syndrome which were observed in the Minamata incident in 1956 were not observed in this incident. Later, however, research has shown that observation of such severe symptoms is exceptional, and that slight, chronic Minamata disease usually occurs (Harada 1995). In the 1970s, because it seemed that Minamata disease was chronic and atypical, questions arose in Japan as to "what are the minimum effects of methylmercury ?" and "what is Minamata disease ?" (Harada 1995). In 1959, in Minamata, a special committee decided the diagnostic criteria for Minamata disease. The set of criteria was that, in addition to sensory impairment of the extremities, there should be two or more of the following: tunnel vision, ataxia, impaired speech, and impaired hearing. On the other hand, we think that severe sensory impairment of the digits indicates damage to the central nervous system, and that there is a high probability that it is due to methylmercury poisoning (Ninomiya et al. 1995). Thus, we consider that, when this symptom is present, the diagnosis should be Minamata disease. In that case, an outbreak of Minamata disease was already apparent in Grassy Narrows and White Dog in 1975.

In the earlier studies, we could not confirm Minamata disease because the symptoms were light. In the current study, the characteristic symptoms were apparent. Because there was a high rate of occurrence of multiple-limb sensory disturbance and symptoms such as ataxia and tunnel vision, we confirmed the presence of the disease. Likewise, we concluded that the cause was mercury pollution, certainly due to the food chain. We also claim that the disease was already present in 1975. Furthermore, it is clear that chronic Minamata disease can occur even when the level of mercury in the hair is below the provisional 50 ppm recommended as the safe level over a long period of time. Thus, although it has been correctly reported that the fetus is at risk even when the mercury level in the hair of a pregnant woman is below 50 ppm (Grandjean et al. 1997), adults may also be at risk from mercury poisoning at such levels. In both the current and previous investigations, many cases of glove and stocking-type sensory disturbance were found, as was the case in Minimata and the Amazon basin where there was mercury contamination (Harada 1995; Harada et al. 1976; The Ontario Ministry of Health 1974). Collectively, we may emphasize that sensory impairment of the extremities is strongly characteristic of methylmercury poisoning (Harada 1995). In the current investigation, not only did we confirm Minamata disease based on the Japanese criteria, but we demonstrated that it can be diagnosed by sensory impairment of the extremities alone.

Although the Mercury Disability Board did not officially admit an outbreak of Minamata disease, their diagnosis was very similar to ours, with the exception of those involving light symptoms and strong psychogenic reactions. Therefore, their criteria were similar to those applied in Japan. The Mercury Disability Board also gave compensation for complications, but it only covered medical fees and loss of earnings. To further enrich each life , it will be necessary to confirm beyond doubt that the individuals have Minamata disease.

This time we obtained much valuable data. Because the current investigation was short, the findings are only superficial. A more detailed study should be done, involving not only the effects of methylmercury but also financial consequences. Such a study would benefit the entire world, because mercury contamination in all corners of the earth has recently been reported.

REFERENCES

Chabonneau SM, Munro LC, Nera EA, Willes RF, Kuiper-Goodman T, Iverson F, Moodie CA, Stoltz DR, Armstrong FAJ, Uthe JF, Grice HC (1974) Subacute toxicity of methylmercury in the adult cat. Toxicol Appl Pharmacol 27: 569-581

Clarkson TW (1976): Exposure to Methyl Mercury in Grassy Narrows and White Dog Reserves. Report, The Medical School, Univ of Rochester

Fimreite N (1972) Mercury Contamination of Aquatic Birds in Northwestern Ontario. Report of Univ of Tromsö, Norway

Fimreite N, Reynolds LM (1973) Mercury contamination in fish of northwestern Ontario. J Wildlife Manage 37: 62-68

Grandjean P, Weihe P, White RF, Debes F, Araki S, Yokoyama K, Murata K, Sorensen N, Dahl R, Jorgensen PJ (1997) Cognitive deficit in 7-year-old children with prenatal exposure to methylmercury. Neurotoxicol Teratol 19: 417-428

Harada M (1995) Minamata disease: Methylmercury poisoning in Japan caused by environmental pollution. Crit Rev Toxicol 25: 1-24

Harada M, Fujino T, Akagi T, Nishigaki S (1976) Epidemiological and clinical study and historical background of mercury pollution on Indian reservations in northwestern Ontario, Canada. Bull Inst Constit Med Kumamoto Univ 26: 169-184

Harada M, Fujino, T, Akagi T, Nishigaki S (1977) Mercury contamination in human hair at Indian reservation in Canada. Kumamoto Med J 30: 57-64

Harada M, Nakachi S, Cheu T, Hamada H, Ono Y, Tsuda T, Yanagida K, Kizaki T, Ohno H (1999) Monitoring of mercury pollution in Tanzania: relation between head hair mercury and health. Sci Total Environ 227: 249-256

Harada M, Nakanishi J, Yasoda E, Pinheiro MCN, Oikawa T, de Assis Guimaraes G, da Silva Cardoso B, Kizaki T, Ohno, H (2001) Mercury pollution in the Tapajos River basin, Amazon: mercury level of head hair and health effects. Environ Int 27: 285-290

Ikingura JR, Akagi H (1996): Monitoring of fish and human exposure to mercury due to gold mining in the Lake Victoria goldfields, Tanzania. Sci Total Environ 191: 59-68

Ninomiya T, Ohmori H, Hashimoto K, Tsuruta K, Ekino S (1995) Expansion of methylmercury poisoning outside of Minamata: an epidemiological study on chronic methylmercury outside of Minamata. Environ Res 70: 47-50

Ohno H, Doi R, Tani Y, Harada M (1984) Mercury content of head hair from residents on the coast of Jakarta Bay. Bull Environ Contam Toxicol 33: 382-385

Takeuchi T, D'Itri FM, Fischer PV, Annett CS, Okabe M (1977) The outbreak of Minamata disease (methyl mercury poisoning) in cats on Northwestern Ontario reserves. Environ Res 13: 215-228

The Ontario Ministry of Health (1974): Methyl Mercury in Northwestern Ontario, Prepared in Mid-November, The Ontario Ministry of Health, Toronto