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Long-term study on the effects of mercury contamination on two indigenous communities in Canada (1975-2004)

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1. Background of mercury pollution in Canada

In 1970, fish caught from the English-Wabigoon River system in northwestern Ontario indicated mercury contamination, the highest levels reaching 27.8 ppm (parts per million)*1.

Since then, many more mercury contaminated fish were reported*2*3. The government banned commercial fishing and issued a warning against taking fish for food. Otters and minks, which eat fish in the area, disappeared*1. People witnessed eagles flying abnormally*3*4. An eagle indicated a mercury level of 96 ppm in its liver. High levels of mercury were detected from the liver, the muscle, and the eggs of ducks in the same area. Cats in the area showed symptoms of Minamata Disease.

One of the cats was sent to Japan for autopsy by a neuro-pathologist, Tadao Takeuchi. Dr. Takeuchi found typical Minamata Disease in the cat. Very high levels of mercury were detected in the cat, such as 16 ppm from its brain, 67 ppm from its liver and 392 ppm from its hair. The cat's owner also indicated a high mercury level, 58.7 ppm. Experiments proved that cats, which were fed fish from the same river system, contracted Minamata Disease in about 90 days. Contamination of the environment and the food web was clear. The source of contamination was a caustic soda factory up stream, in Dryden, Ontario*3.

Two indigenous communities, Grassy Narrows First Nation and Wabaseemoong (Whitedog) Independent Nations, are located in the contaminated area. The main occupations for the residents were commercial fishing, guiding and hunting. Hair samples and blood samples from the residents indicated high mercury levels. In 1970, the highest hair mercury level in Grassy Narrows was 95.77 ppm, and 198 ppm in Whitedog. Since that year, mercury levels started decreasing according to the Medical Services Branch, Health Canada.

According to our research in 1975, the hair sample with the highest amount of mercury, collected from residents, indicated 80.3 ppm. 44 out of 71 residents indicated mercury

levels above 20 ppm and 23 indicated higher than 30 ppm*10. Dr. Clarkson, who was testing the residents of these communities at the same time, detected a 105 ppm sample, the highest number he found.

Mercury levels in the blood samples from the residents of Grassy Narrows were higher in 1970, averaging 46.37 ppb, the highest being 159 ppb. It was even higher in Whitedog, averaging 77.39 ppb (parts per billion) and the highest being 385 ppb *9. With this data, I was reasonably concerned with the impact of mercury on people. By the time cats had contracted Minamata Disease in Japan, we had already experienced people that were impacted by mercury in Minamata and Niigata*2.

Invited by these two communities, the Pollution Research Committee (Chairperson: Shigeto Tsuru) sent a research team (Leader: Kenichi Miyamoto) to Canada. I, as a member of the team, visited these two Indigenous communities in March and August 1975. I did epidemiological research and clinical examinations and collected hair sample to test mercury levels*3*10. Some hair samples exceeded the safety guideline of 50 ppm.

2. The result of the research in 1975

We examined a total of 89 residents from Grassy Narrows and Whitedog in 1975. The most frequently claimed subjective symptom was pain in the limbs (40 cases, 44.9%). Other symptoms claimed were numbness (28 cases, 31.4%) and cramps of the limbs (16 cases, 17.9%). Among the neurological symptoms that caught our attention were sensory disturbances [the loss of sensation in the extremities around the mouth] (glove and stocking type) (15 cases, 16.8%), (perioral) (5 cases, 5.6%), concentric constriction of the visual field (9 cases, 10.1%), ataxia (8 cases, 8.9%), tremor (21 cases, 23.5%). All of these symptoms are found among Minamata Disease patients. Those residents who had these symptoms also indicated higher mercury levels. Therefore, we considered them to have mild Minamata Disease.

Hair samples from the residents indicated higher mercury levels at the section of hair that grew during the summer. It is because they ate more fish in the summer and that mercury contamination came from locally caught fish*3*10.

Sensory disturbances (the loss of sensation in the extremities) are a very characteristic symptom of mercury poisoning, from my experience. In that sense, mild Minamata Disease had occurred in those two communities in 1975*3. However, because severe and typical cases, which were found in Minamata around 1956, had not been found, both federal and provincial governments did not acknowledge the occurrence of Minamata Disease*3*10. Despite that, the governments still banned commercial fishing, issued a warning against taking fish for food and compensated the communities, nominally, for the loss*3.

The Mercury Disability Board was established and funded with the compensation paid by the polluters and both federal and provincial governments. The board started examining

applicants and providing pensions to the acknowledged applicants, which began in 1986. The board pays monthly pensions, between 250 dollars to 800 dollars per person, for the roughly 140 persons who meet the neurological criteria.

The Mercury Disability Board consists of a chairperson, two physicians, a representative from each reserve, two members chosen by a committee made up of a representative from the federal and the provincial government and the chiefs from each reserve. A neurologist, designated by the board, examines applicants and his diagnosis is translated into a point system that is presented to the board. The neurologist does not participate in the process of awarding claims. The symptoms included in the assessment are tremor, ataxia, inco-ordination, dysarthria (speech impairment), the absence of tendon reflexes, sensory abnormality and concentric constriction of the visual field, all of which strongly reflect the Hunter-Russell syndrome*14 However, the awarding of compensation by the board does not mean that they believe the applicant is to be diagnosed with Minamata Disease.

3. Significance of our research in Canada

During the early stages of the Minamata Disease crisis in Minamata, patients, who were diagnosed with Minamata Disease, seemed to be typical and severe cases of the Hunter-Russell syndrome, including sensory disturbance, concentric constriction of the visual field, ataxia, speech impairment and impaired hearing*12*13*14. However, through more study, it became clear that such severe cases were rather exceptional and many more mild cases were progressing to chronic Minamata Disease *15.

I observed that the symptoms of Minamata Disease became more chronic and atypical in the 1970s. Also I have been proving that sensory disturbances, specifically stronger loss of sensation in the extremities, were seen among patients of methylmercury poisoning at a very high rate, through clinical examinations on family members of patients with severe and typical symptoms or mothers of congenital Minamata Disease patients*15*16. Tsuda demonstrated epidemiologically that this type of sensory disturbance rarely occurs in other diseases*17.

Ninomiya stated that Minamata Disease should be diagnosed when sensory disturbances caused by the damage to the central nervous system was proven. It is because sensory disturbances, in the case of Minamata Disease, is a deficiency of the central nervous system*18. However, in Minamata, the Judgment Committee (established in 1956) is in charge of acknowledging Minamata Disease patients. The Committee does not acknowledge Minamata Disease unless the applicant has two or more symptoms of concentric constriction of the visual field, inco-ordination, speech impairment and impaired hearing, in addition to sensory disturbances*12.

Therefore, many Minamata Disease patients were not acknowledged. The number of the denied reached over fifteen thousand people. I have been stating that Minamata Disease should be acknowledged if sensory disturbances and epidemiological conditions (proof of living in the polluted area) are confirmed. I have opposed the Judgment Committee's

decisions since the second Minamata Disease lawsuit in January 1973*16. In court, arguments over the symptoms of the disease such as "What is the minimum effect of methylmercury?" or "What is Minamata Disease?" continued*15*16*17. The Supreme Court of Japan reached their final conclusion on this matter on the 15th of October 2004. The Court defined Minamata Disease with broader criteria than the Committee's criteria. A long medical dispute ended with the judgment of the Judiciary*19.

In order to understand what are the minimum effects of Minamata Disease, examining residents living in the area where contamination is continuing, is necessary. That is why I have been conducting examinations in mercury-contaminated areas in Canada, Brazil and China. I test mercury levels and conduct clinical examinations for each examinee, as well*20. There are quite a few reports with the only mercury data collected being hair samples from the residents who live in the contaminated area. On the other hand, surprisingly, there are only a few reports about the state of the resident's health. There is hardly a report with data of both hair mercury levels and clinical examinations, together, on each examinee, even in Canada.

Clarifying how the mercury levels change after a long time, in hair samples and the clinical symptoms from 1975, is very meaningful in order to understand mercury's effect on human health and the safety issue of long-term low exposure of methylmercury. At the same time, it is a crucial contribution to conclude the long-term dispute over the definition of Minamata Disease in Japan. Long-term follow-up research like this case in Canada is significant and rarely see in the world.

I organized a research team consisting of researchers from Kumamoto Gakuen University and physicians. We conducted examinations in September in 2002 and September in 2004, 27 years after the first research in 1975.

4. Research method

The areas of research were two reserves, Grassy Narrows First Nation and Wabaseemoong (Whitedog) Independent Nations, the same two communities where we conducted research in 1975. The registered population is 1214 people in Grassy Narrows and 1649 people in Whitedog.

Research #1 in 2002

We stayed in Grassy Narrows from August 31 to September 3 2002 and conducted clinical examinations the same way as the Minamata Disease examinations are done in Japan. At the same time, we collected hair samples. Though it is clear that the mercury contents in hair acquired through the food web is more than 90% methylmercury, the total amount of mercury was reflected in the numbers*21. We examined applicants with the co-operation provided by the residents.

Research #2 in 2004

We stayed in Grassy Narrows from August 27 to 30 and in Whitedog from August 31 to

September 2, 2004. We conducted clinical examinations and collected hair samples in the same way as our visit in 2002. This time, we did tests on sensation of a third dimension, delicate fingertip movement and sociological research.

The total number of people tested during the 2002 research trip was 57 residents from Grassy Narrows.

The total number of people tested in 2004 was 87 residents from Grassy Narrows and 69 residents from Whitedog: 156 people in total. 26 residents from Grassy Narrows overlapped between Research #1 and #2 making the actual total number 187 people, as the 31 individuals that did not overlap from 2002 are added to the final total for the study.

There were 108 male examinees and 79 female examinees, aged from 1 to 90 years old. There were 12 examinees aged from 1 to 10 years old, 12 examinees from 11 to 20 years old, 12 examinees from 21 to 30 years old, 35 examinees from 31 to 40 years old, 27 examinees from 41 to 50 years old, 40 examinees from 51 to 60 years old, 30 examinees from 61 to 70 years old, 13 examinees from 71 to 80 years old, 6 examinees from 81 and over. The oldest was 90 years old. Some applicants did not remember their birthday clearly.

5. The results of the research

(1) Subjective symptoms

19 examinees did not have any subjective symptoms. Most of them were young, 12 of them were under 10 years old and 3 of them were from 11 to 20 years old. 18 examinees above 10 years old have only one subjective symptom. All the rest above 10 years old had more than one subjective symptom. The most frequently claimed subjective symptom was numbness (126 cases). This is 72.0% of the 175 examinees, excluding those under 10 years old. The next frequently claimed subjective symptoms were pain in the limbs, joints and back (107 cases, 61.1%), impaired vision or deteriorated sight (70 cases, 40.0%), impaired hearing (66 cases, 37.7%), cramps in the limbs (59 cases, 33.7%), dizziness (47 cases, 26.8%), tendency to fall, imbalance (39 cases, 22.2%), forgetfulness (39 cases, 22.2%), impaired finger movement, difficulty grasping, dropping things (27 cases, 15.4%), tremor (21 cases, 12.0%), dysarthria (speech impairment), (18 cases, 10.2%). These symptoms are commonly found among Minamata Disease patients.

(2) Neurological symptoms

Sensory disturbances were the most noticeable symptom and appear in different parts of the body. There were 114 cases (65.1%) with the stronger loss of sensation in the extremities (glove and stocking type), 35 cases (20%) around the mouth (perioral), 32 cases (18.2%) of sensory disturbances throughout the entire body, 10 cases (5.7%) with sensory disturbances on one side of the body or irregular sensory disturbances. There were 36 cases (20.5%) with difficulties balancing and walking (walking a straight line, standing on one leg), 47 cases (26.8%) of impaired hearing, 37 cases (21.1%) of tremor, 44 cases (25.1%) of ataxia, 12 cases (6.8%) of speech impairment, 17 cases (9.7%) of

disturbed ocular movement (Saccadic movement), 19 cases (10.8%) of concentric constriction of the visual field (tunnel vision), 9 cases (5.1%) with mental deficiency, 14 cases (8.0%) of mental retardation (including dementia), 6 cases (3.4%) of convulsion, 5 cases (2.8%) of fainting, 3 cases (1.7%) of muscular dystrophy.

(3) Complications

There were 49 cases (28.0%) of high blood pressure, 42 cases (24.0%) of diabetes, 19 cases (10.8%) of heart disease, 13 cases (7.4%) of stroke, 7 cases (4%) of thyroid problems, 5 cases (2.8%) of Kennedy-Alter-Sung Syndrome (genetic diagnosis). In addition to those, dystrophy, an impaired spinal column in the cervical vertebrae, wounds such as broken bones, cancer, tuberculosis, sniffing, liver disease, kidney disease, Buerger's disease and sarcoidosis were seen. In particular, many residents (24.0%) contracted diabetes; sever cases being 1 case of gangrene and 2 cases of loss of sight.

(4) Diagnosis

We diagnosed Minamata Disease if an examinee showed more than one symptom, such as sensory disturbances (specifically the loss of sensation in the extremities), ataxia, disturbed ocular movement, imbalance, concentric construction of the visual field (tunnel vision) and speech impairment. In cases where examinees were showing only sensory disturbances, we acknowledged Minamata Disease as long as there was no other disease causing the symptom. If an examinee had another disease and still showed symptoms of Minamata Disease that were not explained by that other disease, we defined the case as Minamata Disease with complications. If we saw some Minamata Disease symptoms but could not confirm due to complications or their mental state, or symptoms were inconsistent, we categorized it as possible Minamata Disease. If symptoms, caused by another disease, were seen and no symptoms caused by methylmercury poisoning were observed, we either categorized it as another disease or normal.

There were 60 cases of Minamata Disease (34.2% of total examinees, excluding people 10 years old and younger), 54 cases (30.8%) of Minamata disease with complications and 25 cases (14.2%) of possible Minamata Disease for a total of 139 cases (79.4%). The rest were other diseases or normal.

It is very high rate of neurological symptoms for a sample of a population, even though examinees came because of other health issues. It is as high as contaminated areas in Minamata. We cannot help but recognize the effects of methylmercury from the symptoms we have seen. Considering current low mercury levels from hair samples, long-term low exposure in the past must have caused the symptoms. In addition, the symptoms (the level of difficulty added to conducting their daily lives) became worse.

(5) Symptoms of toddlers and children

There were 7 cases of cerebral palsy, 7 cases of intellectual developmental delay. The effects of methylmercury during pregnancy might be considered, but there is no evidence in any control studies to prove as such so far.

(6) Acknowledgment by the Mercury Disability Board

Of the people examined, 54 out of 187 examinees were acknowledged. 77 were rejected, 7 were suspended and 47 had not applied.

(7) Comparison of diagnoses

Comparing the decisions by the Mercury Disability Board with our diagnosis, among the 60 examinees we diagnosed to have Minamata Disease, 21 examinees were acknowledged and 39 were not acknowledged by the MDB. Among the 54 people we diagnosed as Minamata disease with complications, 27 were acknowledged and 27 were not by the MDB. In total, the Mercury Disability Board acknowledged 38.1% of the people we diagnosed with Minamata Disease, Minamata Disease with complications and possible Minamata Disease.

Among the 75 examinees that were not acknowledged by the MDB, 28 examinees were diagnosed to have Minamata disease, 21 were diagnosed to have Minamata disease with complications and 11 were diagnosed to have possible Minamata disease, a total of 60 examinees (80.0%). Most of them had mild Minamata disease with sensory disturbances or sensory disturbances and impaired hearing, or mental symptoms.

Of seven cerebral palsy patients 2 children (10 years old and 13 years old) were acknowledged. 2 more children whom we diagnosed with other diseases were acknowledged as well.

The average age of people acknowledged was 61.1 years old. This is because the peak of contamination was in 1970s and also the effect of the symptoms as one ages. The youngest of the acknowledged was 18 years old, excepting examinees with possible congenital Minamata disease. The examinee had the highest mercury level in Canada when he was 3 years old. He had sensory disturbances (the loss of sensation in the extremities), tremor and imbalance.

The average age of the people who were not acknowledged was 42.8 years old. There seemed to be more younger Minamata Disease patients and mild cases in the communities and the surrounding area.

We noticed that examinees with Minamata Disease with complications were acknowledged by the MBD at a higher rate.

(8) Analysis of mercury levels in hair samples

128 people provided hair samples. The lowest was 0.06 ppm and the highest was 25 ppm, the average was 2.32 ppm. Currently, mercury pollution is decreasing in this area, however some people still indicate high mercury levels.

(9) Comparison with the examinations in 1975

19 examinees (43.18 %) out of 44 residents of Grassy Narrows, whom we examined in 1975, are deceased. The number of the deceased in Whitedog was unknown. It is significant that we were able to re-examine 27 of the same people that we examined in 1975.

The age of the 27 residents range from 49 to 90 years old, the average being 66.2 years old; naturally, many of them were elderly. The residents who indicated mercury levels higher than the safety guideline of 50 ppm in 1975 were not among the examinees this time. They are deceased. The highest mercury level at this time was 44.2 ppm and most of them indicated between 10 to 30 ppm. Comparing the symptoms of the 27 residents between 1975 and 2002-04, 2 cases of sensory disturbances (the loss of sensation in extremities and around the mouth) and 6 cases of only sensory disturbances (the loss of sensation in extremities) in 1975 increased to 6 cases and 13 cases respectively in 2002-04. Seven of them also indicated the loss of sensation in whole body (2 cases duplicated with the loss of sensation in extremities). Three of them did not have sensory disturbances. Those with ataxia increased from 2 to 16 cases, concentric constriction of the visual field increased from 3 to 4 cases, speech impairment increased from 3 to 8 cases, imbalance increased from 2 to 7 cases, tremor increased from 5 to 10 cases, walking difficulties increased from 0 to 6 cases and stroke (cerebral infraction) increased from 0 to 4 cases. There were 7 diabetic patients. Although, we acknowledged the effects of aging and complications, we also found that the symptoms of Minamata Disease had progressed. Therefore, the change cannot be explained by only aging and complications. According to our diagnosis, 13 residents had Minamata Disease, 11 residents had Minamata Disease with complications for a total was 24 residents (88.8%). The MDB acknowledged 21 applicants (77.7%) out of 27 residents and provides them with a pension. This means that the occurrence of Minamata Disease is acknowledged in a practical sense even though they do not recognize it officially. Those cases indicate that even being exposed under the safety guideline, if prolonged, it could cause Minamata Disease (chronic type).

(10) Case studies

#1

53 year old, male, commercial fisherman and hunter.

There were no subjective and neurological symptoms in August 1975. Mercury levels from his hair sample ranged between 2.0 ppm and 23.4 ppm. It was 3.0 ppm in August 2002. He had sensory disturbance (extremities) and imbalance in August 2002. He was acknowledged and receives a monthly pension from the MDB of 300 dollars. We examined him in August 2004 and found sensory disturbances (extremities and around the mouth), imbalance, disturbed ocular movement, tremor, impaired hearing, high blood pressure & diabetes. The diagnosis was Minamata Disease.

#2

68 year old, male, former fishing guide.

He already had sensory disturbances (extremities and around the mouth) and tremor in August 1975, however, he was not aware of these symptoms. His mercury levels ranged between 7.5 ppm and 35.3 ppm in 1975. He had severe sensory disturbances (the loss of sensation in the whole body), disturbed ocular movement, ataxia, tremor and impaired hearing in August 2002. By 2002, the mercury level in his hair was 2.1 ppm. In August 2004, it was mostly the same but the symptoms became more typical. He was acknowledged and receives a monthly pension from the MDB of 450 dollars.

#3

90 year old, male, former fishing guide.

He claimed numbness and pain in the limbs in August 1975. We found sensory disturbance (extremities.) His mercury levels ranged between 30.8 ppm and 44.2 ppm in 1975. In 2002, he had Parkinson's Disease symptoms, loss of sight, impaired hearing, difficulty walking, mild dementia, sensory disturbances (severe loss of sensation in the extremities and around the mouth) and numbness on one side of his body due to cerebral infarction. Our diagnosis was Minamata Disease with complications, cerebral infarction and aging. The MDB acknowledged him ten years ago and provided a monthly pension of 600 dollars. He passed away in 2003. He contracted Minamata Disease before the cerebral infarction.

#4

49 years old, male, hunter.

In 1975, he had mild tremor but no subjective symptoms. His mercury levels ranged between 2.5 ppm and 8.1 ppm in 1975. In 2004, we found sensory disturbance (extremities) and disturbed ocular movement. Our diagnosis was a mild case of Minamata Disease. The Board rejected him. Young applicants with milder symptoms were not acknowledged.

6. Conclusions

High-density mercury contamination had occurred in areas where methylmercury was discharged by acetaldehyde production facilities or where agricultural chemicals that contained an organic mercury fungicide were massively sprayed. Now the situation is changing into lower density contamination over broader areas. Sources of contamination are mostly inorganic mercury discharged into the natural environment, which methylate into methylmercury, as in the case of caustic soda plants or gold mines. In the late 1980s, mercury contamination was found in several areas in the world, like the Amazon, where analysis was conducted on the air, earth, organism, fish, shellfish and hair sample of local residents. However, very few clinical reports were done on the health of local residents in the contaminated area. Even after the high mercury levels of the residents were reported, it was not clear what kind of symptoms the residents had. For example, residents living along the Amazon River indicated mercury levels that are high enough to contract Minamata Disease, but there are few reports about clinical examinations of the residents.

*21*22*23

Our research method examined life conditions, medical history, family history, subjective symptoms, mental symptoms and complications as well as collecting hair samples. The same doctors conducted both studies, in 1975 and the follow-up studies. There is no such precedent. Therefore, our research from 1975 and the follow-up studies in 2002 and 2004 are significant contributions in considering mercury contamination and the health of the residents in these two communities.

As a result, we confirmed a very high rate of occurrence of the symptoms of Minamata Disease such as sensory disturbance (loss of sensation in the extremities), ataxia and concentric constriction of the visual field. Considering the background of mercury contamination for decades, we cannot help but diagnose Minamata Disease. The residents, who had mild symptoms or almost no symptoms in 1975, and whom we could not, with confidence, diagnose with Minamata Disease because of the symptoms seemed too mild, showed almost typical symptoms by the second visit 27 years later. Although it was mild, Minamata Disease, had occurred in Grassy Narrows and Whitedog in 1975, as we pointed out at that time.

Sensory disturbance (extremities), which was found at a very high rate among the residents in 1975 and during the follow-up studies, were also found at a high rate in Minamata, Niigata and amongst the people of the Amazon.*10*15*22 We would say this type of sensory disturbances is very characteristic of methylmercury poisoning (Minamata Disease).*15*16*17*23

Considering the current low levels of mercury in the Indigenous residents of Grassy Narrows and Whitedog, no new cases of Minamata Disease are expected. The Canadian government has declared the safety of eating fish from contaminated river systems and denied the possibility of Minamata Disease occurrence. (+After the Ontario government issued the warnings against taking fish for food in 1970, the government started publishing the “Guide to Eating Ontario Sport Fish” in 1977, which is based on the national safety guideline for mercury and other toxins. The latest version is 2007-8 edition) (+Health Canada concluded, in 1979 and again in 1999, that “While it can be stated categorically that severe methylmercury poisoning, “Minamata Disease” has not been found in Canada, milder forms of mercury poisoning, although difficult to prove conclusively, possibly have occurred. However, with the lower levels currently being found, even mild forms are unlikely to occur in adults at the present time.”) Surely, as mercury levels in the fish are low, there is no possibility of new cases of Minamata Disease. However, our research implies that there are many patients still suffering that have not been acknowledged yet. It is a very familiar response by the state , as witnessed in Minamata over the course of 50 years.*16

In 1975, most of the residents indicated mercury levels lower than the safety guideline of 50 ppm, except a few. We diagnosed many of them to have Minamata Disease after the follow-up studies, even though their mercury levels have been decreasing since 1975 and they were not subjected to a further heavy dose of mercury contamination. We concluded from these cases that it is possible to contract Minamata Disease (chronic type) even under the safety guideline if they keep eating contaminated fish for a long period of time. Currently, it is being reported that fetuses could be affected by mercury even though the mother's mercury level is lower than the safety guideline.*24*25 Our research implies that it is not only pregnant women and fetuses that are at risk.

The possibility of congenital Minamata Disease occurrence is very high in these two communities. We examined 7 cases of cerebral palsy and 7 cases of mental deficiency. Two of them were acknowledged by the MDB. We could not reach a conclusion quickly

because the symptoms of congenital Minamata Disease are not as characteristic as those of Minamata Disease for adults. Further epidemiological research on stillbirths and miscarriages needs to be done.

The Mercury Disability Board has stated that though they may acknowledge a patient, it does not mean that they agree with the diagnoses of Minamata Disease. But we claim, from their actual symptoms, that they have Minamata Disease. In fact, the board's acknowledgements correspond well with our diagnoses. At the same time, many mild cases and patients with severe psychogenic symptoms were not acknowledged. In those cases, the board's criteria are another version of the Japanese government's criteria. This is a typical problem; the globalization of criteria from Japan. (In 1978, the Japanese government tightened the criteria for acknowledgement of Minamata Disease based on the report by Dr. Tsubaki and Dr. Irukayama. This report is mentioned in "Methylmercury in Canada" by Health Canada and Canadian officials used the Japanese report to define Minamata Disease, denying the occurrence of Minamata Disease in Canada.) On the other hand, I appreciate that many cases with complications are acknowledged by the MDB. I also appreciate the procedure for acknowledgement and who was chosen to be on the board. (In Minamata, the board consisted only of physicians. Some people stated the board should include representatives from the community. The MDB has one representative from each reserve.) However, simply providing a pension is not true relief. Having observed the actual conditions the residents live in, I think, in addition to committing some money to improve the health services, support such as improving the living standards and employment development is needed. Official acknowledgement of Minamata Disease and corporate responsibility are important in order to implement this policy.

From now on, cooperation between patients, sharing findings among researchers and the exchange of information is necessary. We hope that medically unknown issues such as the safety guideline, long-term low exposure and the effects of minimal amounts of methylmercury will be clarified by further study.