INTRODUCTION
Tragedy of Minamata Disease and Environmental Chemical Problems Today

1. Outline of Minamata disease

(1) Outbreak of Minamata disease and its casualties

Minamata disease is a poisoning disease of central nervous system developed among the inhabitants who routinely had large quantities of fishes and shellfishes in which methylmercury compound had been absorbed directly through the gills or through the intestinal tracts or been accumulated at high concentrations by food chains after discharge from chemical plant to the sea and rivers.

In the beginning of the outbreak, however, the disease occurred as a peculiar nervous disease of unknown cause in the districts along the shore of the Shiranui Sea (Yatsushiro Sea) centering on the areas around Minamata Bay in Kumamoto Prefecture. The outbreak was also confirmed later in the reaches of the Agano River in Niigata Prefecture.

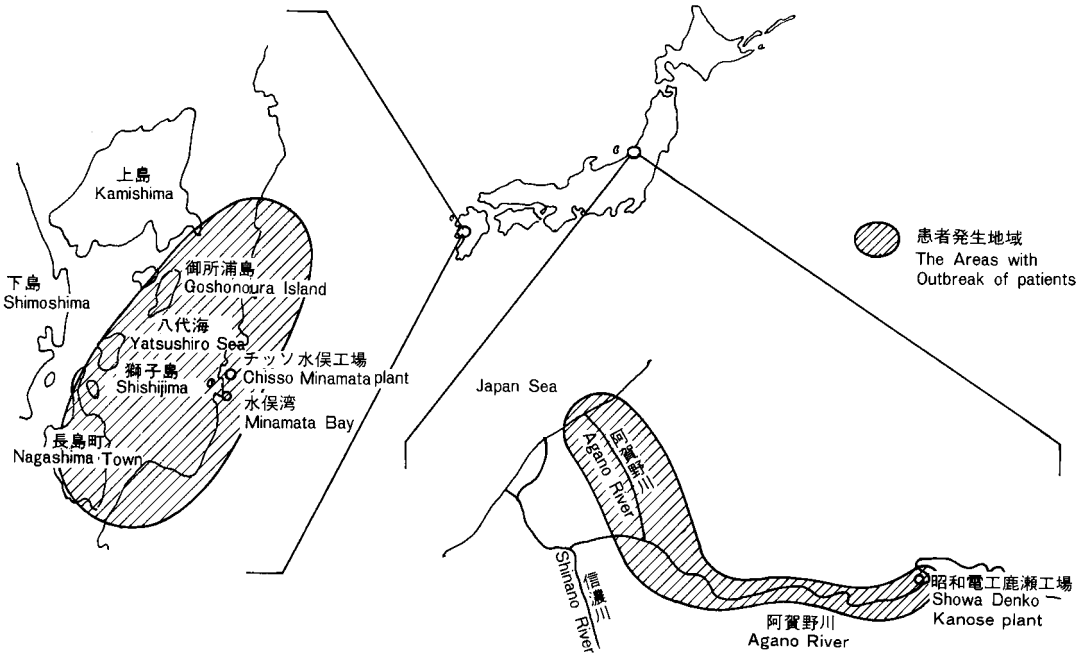


Figure 1. The areas with outbreak of Minamata disease (Cited from "Our Investigative Efforts to the Tragic History of Minamata Disease 1999" by Environmental Health Department, Environment Agency)

With regard to Minamata disease occurring in the areas around Minamata Bay in Kumamoto Prefecture, the patient was reported for the first time in May 1956, and it was confirmed at the end of the year that 54 patients had had the disease since December 1953 and that 17 of the patients already died. The disease became called "Minamata disease" from 1957 onward.

With regard to Minamata disease occurring in the reaches of the Agano River in Niigata Prefecture, the patient was reported for the first time in May 1965. In July 1965, it was confirmed that there were 26 patients and that 5 of the patients died.

Certification of a person to be a patient with Minamata disease in Kumamoto Prefecture is made by Governors of Kumamoto Prefecture and Kagoshima Prefecture on the basis of the law(*); 2,263 patients with Minamata disease have been certified by these Governors by the end of November 1999. Since the outbreak of Minamata disease was reported, at least 40 years have passed, and 895 patients were alive as of the end of November 1999.

(*)N.B.: (Old) Low Concerning the Relief of Pollution-Rellated Health Damage. At present Law Concerning Compensation and Prevention of Pollution-related Health Damage.

Certification of Minamata disease in Niigata Prefecture was made by Governor of Niigata Prefecture and the Mayor of Niigata. By the end of November 1999, 690 patients with Minamata disease have been certified by the Governor and the Mayor. As of the end of November 1999, 352 patients with Minamata disease are alive.

In addition to the people who have been certified to be Minamata disease patients on the basis of the law, the following people were decided to receive medical benefits from the Government and the prefecture, which would have expense to bear them, and to receive a single sum from Chisso Co., Ltd. or Showa Denko Co., Ltd.: the persons who have been exposed to the methylmercury compound at the concentrations higher than usual and certified to have sensory disturbance mainly in the peripheral portions of extremities by the Governor or the Mayor, among the people who were not certified to have Minamata disease by the law because of “the medically low probability of Minamata disease” as a measure of relief of the administrative structure by political measures to solve the problem of Minamata disease on the basis of mutual consent of the three Government parties (the Murayama Cabinet) [the Liberal Democratic Party, the Japanese Socialist Party (Social Democratic Party), and Shinto-Sakigake] in December 1995. In total, 11,152 people were the subjects (10,353 in Kumamoto and Kagoshima Prefectures and 799 in Niigata Prefecture).

Minamata disease caused not only serious casualties to lives and health of the patients but also serious damage, which are unable to be easily relieved, to natural environments, human relations, and economical activities in the districts, as well as the daily lives of the patients and family members.

(2) Tragedies of Minamata disease

Minamata City is located on the southern tip of Kumamoto Prefecture, and adjacent to Kagoshima Prefecture. Nature abounded on the city that was surrounded by the sea and mountains. The Shiranui Sea which Minamata City faces was a calm beautiful inland sea surrounded by the Kyushu mainland and the Amakusa Islands on the other side of the city. The sea area teemed with fish. Particularly, the areas around Minamata Bay were fishery spawning grounds, which were blessed with abundant natural rocky places under the water where fish tended to gather, and fishing grounds. The areas were dotted with small fishing villages, and the people led their self-sufficient lives with the blessed sea. On the days of large catches, anchovies, scabbard fishes, horse mackerels, etc. were netted in so large amounts that there were no spaces for drying grounds. The fishes and shellfishes caught in the Shiranui Sea were valuable protein sources for the inhabitants of the districts.

However, strange extraordinary events began to appear in the calm fishing villages in around 1953 at latest; cats went mad, ran around, and went into the sea, and crows and birds living along the shore of the sea fell to die. Thereafter, a disease of unknown cause stroke the inhabitants in sequence.

Among the inhabitants who had not ever entertained any apprehension about their health, some had numbness and trembling of extremities, narrowed visual field, and hearing disorder. Some people could not speak clearly, stumbled or staggered. Some could not walk in an ordinary way. Some people had convulsion or became bedridden. The persons with particularly serious ones of these symptoms lost consciousness, moved the extremities and the body terribly, cried by day and night, or scratched off the wall. Then, they died about 1 month after the onset.

Further tragedies occurred; some infants were bone with physical and mental retardation and severe

difficulty of moving, and died in the early stage after birth.

The grief and troubles of the patients who suffered from the disease, whose cause and treatment were unknown, and of family members who cared for their blood relatives were serious, and the lives of the families that lost the supporters because of the disease were worked to the limit.

Fifty-four patients were confirmed by the end of 1956, and 17 of them died. However, that is not all grief or trouble Minamata disease has induced. The patients and family members were treated discriminately because of some apprehensions about the possibility of the disease being infectious, and they could not make purchases in the neighborhood. Thus, human relations between them and the surroundings were broken. If a patient appeared in a fishing village, the fishes taken by fishermen in the village would not be sold. For this reason, some persons were stopped to announce themselves as the patients. The inhabitants who had treated the patients discriminately also became patients, and some of them died. In 1959, patients and the family members moved to ask for compensations to the Chisso Minamata plant that had been strongly suspected as the causative company. Therefore, the patients and family members met with resistance from the citizens of Minamata City governed by the company. This was one of the main reasons by which the victims were confined to an obscure part in the local community.

Minamata disease that caused such tragedies is one of the most serious environmental pollutions humans experienced worldwide as well as in Japan. It occurred during the time period when Japan was going to revive from destruction of the economic base due to World War II, which ended in defeat of Japan, and to take the new course of economic growth, and when the Government, local governments, politicians, the mass media, and most of the nation, as well as companies, thought that economic development centering on heavy chemical industries is most important. The activities of companies, in which only the productivity took priority over others and which lacked consideration to environments, took many persons' lives, damaged many people mentally and physically, and further gave serious irrecoverable casualties to natural environments, economic activities, and human relations in the districts.

Despite the outbreak of such serious tragedies in the districts along the shore of the Shiranui Sea, any efficient measure to arrest the spread of the casualties was not taken by the Government, leasing to the second outbreak of Minamata disease in the reaches of the Agano River in Niigata Prefecture in May 1965.

Drainage from the process of acetaldehyde production by Chisso, which was a causative factor for Minamata disease in Kumamoto Prefecture, completely stopped by the cessation of operation of production facilities in May 1968, i.e., 12 years after May 1956 when the outbreak of Minamata disease was reported for the first time to the administrative organs. Operation of the process of acetaldehyde production by Showa Denko, which was a causative factor for Minamata disease in Niigata Prefecture, was ceased in January 1965.

(3) Confirmation of the causes of Minamata disease by the Government

The methylmercury compound as a by-product of the process of production of acetaldehyde at the Minamata plant of Shin Nippon Chisso Fertilizer Co., Ltd. ("Nippon Nitrogen Fertilizer Co., Ltd." until January 1950; "Shin Nippon Chisso Fertilizer Co., Ltd." until January 1965; "Chisso Co., Ltd." at present; hereinafter referred to as "Chisso") and at the Kanose plant of Showa Denko Co., Ltd. (hereinafter referred to as "Showa Denko") as discharged with factory wastes. It contaminated Minamata Bay, the Shiranui Sea, and the Agano River, and accumulated in the fishes and shellfishes inhabiting there. The disease that occurred in the inhabitants of the districts, who continued to eat the fishes and shellfishes in large quantities, was Minamata disease.

With regard to the cause of Minamata disease occurring in the districts around Minamata Bay in Kumamoto Prefecture and in the reaches of the Agano River in Niigata Prefecture, the Government's unified views were announced on September 26, 1968; i.e., it was concluded that the causative agent for Minamata disease in Kumamoto Prefecture was the methylmercury compound formed as the by-product of the process

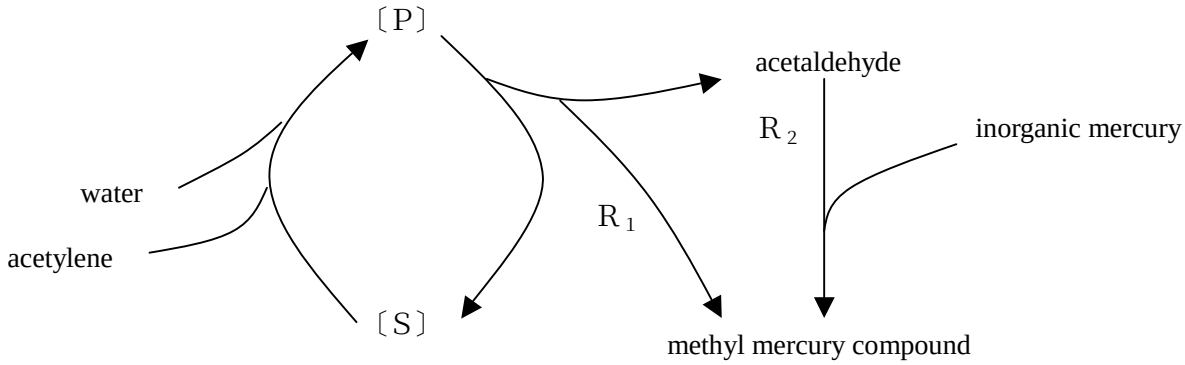
of acetaldehyde production in the Chisso Minamata plant", and that "the development of poisoning in Niigata Prefecture was based on the drainage containing the methylmercury compound as the by-product of the process in the Showa Denko Kanose plant". It was then concluded that the methylmercury compound discharged from these plants accumulated in fishes and shellfishes and Minamata disease was the disease of the central nervous system, which occurred in the inhabitants who continuously took these fishes and shellfishes in large quantities.

2. Mechanisms underlying the outbreak of Minamata disease and morbid conditions

(1) Mechanisms underlying the by-production of methylmercury and the outbreak of Minamata disease

A. The mechanism underlying the by-production of methylmercury

The methylmercury compound as the causative agent for Minamata disease is generated in the process of production of acetaldehyde by addition reaction with acetylene water, as follows: ① produced in a reactor as a side reaction of acetaldehyde generation, i.e., as one of the catalyst deterioration reactions; ② produced in response to reaction of generated acetaldehyde to inorganic mercury ion. The reaction shown as ② may occur not only in a reactor by addition reaction with acetylene water but also in other instruments in which acetaldehyde may come in contact with inorganic mercury. The whole relevant reactions are illustrated below.



With regard to the mechanisms underlying the generation of the methylmercury compound in the actual reactor, there is one hypothesis; i.e., the methylmercury compound is produced as an adverse reaction (R1) in the process of the degradation, while acetaldehyde is generated in the process of degradation of the intermediate product [P] developing through the reaction of acetylene to mercury.

Furthermore, *Hajime Nishimura, Prof. Emeritus of University of Tokyo (Chemical Industry) and his co-workers, who have recently investigated results of past experiments by various investigators by the analysis from the aspect of reaction kinetics, have indicated that it is important to generate the methylmercury compound via the R2 tract as the reaction in the process of the actual production of acetaldehyde and that the contribution of R1 to the generation is relatively slight.

B. Onset mechanisms of Minamata disease

The methylmercury compound may have been generated in acetaldehyde plants which had the process similar to that in the Chisso Minamata plant. The following possible reasons for the occurence of a large number of patients in Minamata are considered to be that: ① the amount of acetaldehyde produced in the

Chisso Minamata plant was large; ② the amount of the secondarily generated methylmercury compound per unit production amount was extraordinarily large; ③ in the case of the Chisso Minamata plant, since a large amount of chloride ion was contained in city water, the methylmercury compound generated changed to volatile methylmercury chloride, which may have been transferred to a rectifier in the process of evaporation and distillation of acetaldehyde in the mother liquor and excreted with the essential drain waste water; ④ the excreted methylmercury compound did not diffuse adequately and accumulated at high concentrations in fishes and shellfishes, because Minamata Bay is the inland sea of the Shiranui Sea; and ⑤ the fishing people who eat the fishes and shellfishes in large quantities existed there.

In the case of Minamata disease in Niigata Prefecture, the following possible reasons for the outbreak are considered to be that: ① The inhabitants who eat river fishes existed in the areas around the Agano River to which waste water from the Showa Denko Kanose plant was discharged; and ② a large quantity of factory wastes may have been thrown away as a consequence of the cessation of operation of the acetaldehyde process.

[*Comments*] According to the recent study by Hajime Nishimura ["Gendai Kagaku (Chemistry Today)", February, 1998], some possible reasons for the high frequency of the methylmercury compound generated in the Chisso Minamata plant are described below. From August 1951 onward, the procedural system was changed; manganese dioxide used conventionally as a promotor for maintaining the activity of mercury catalyst was changed to ferric sulfide, and ferrous ion reduced in the reaction mother liquor was oxidized with nitric acid. As a result, the amount of methylmercury generated in the process was rapidly increased, and since the factory water with which the sea water was mixed was used, the methylmercury compound was transmitted from the evaporator to the rectifier in the form of methylmercury chloride and discharged. Also, waste matter from a sulfuric acid plant, i.e., burned draff of ferrum sulfide ore, has been used as a promotor for 3 years since 1951, frequently leading to troubles; the reaction mother liquor containing the methylmercury compound has been thrown away.

(2) Morbid conditions of Minamata disease

When the fishes and shellfishes contaminated with the methylmercury compound are orally taken into the body, the compound is absorbed almost completely into the digestive tract, and distributed in organs of the whole body via blood circulation. The compound partly passes the blood-brain barrier to accumulate in the central nervous system and damages nerve cells to cause neuropsychic symptoms. The methylmercury compound is characterized by the fact that it passes the blood-brain barrier more easily than other mercury compounds. The compound is partly transmitted into hairs.

Minamata disease shows a variety of clinical symptoms as follows, and the patients have persistent subjective complaints: sensation of numbness, arthralgia and myalgia of extremities, disturbance of speech, disability to use fingers, disability to grasp objects, easy stumbling and unsteadiness, ageusia, anosmia, difficult hearing, cramp, headache, failure of memory, insomnia, etc. The major clinical pictures of nervous system in the typical patient include sensory disturbance mainly in the peripheral parts of extremities (,which may be expressed as glove and stocking anesthesia in some cases), cerebellar ataxia, dysarthria, concentric constriction of visual field, central hearing impairment, central disturbance of ocular moviement, central dysequilibrium, tremors, etc. Sensory disturbance, ataxia, constriction of visual field, and hearing impairment among these are called Hunter-Russell syndrome after the names of the physicians in England, who have reported for the first time details of the patients with methylmercury poisoning. These symptoms are regarded as typical clinical features of methylmercury poisoning.

Some severe cases of Minamata disease show restlessness, wild excitement, and consciousness disorder or the state called apallic syndrome, and occasionally show the fatal course. With regard to patients in the early stage of Minamata disease, 16, 4, and 1 patients died within 3 months, 6 months, and a year, respectively, after the onset. As of 1965, the fatality rate was 43.8% in the patients except fetal cases which

will be described later ("Minamata byo – Yuki-suigin chudoku ni kansuru kenkyu [Study on Minamata disease: an organic mercury poisoning]").

[*Comments*] Including fetal cases, all patients of Minamata disease who had been confirmed by 1968 at latest, were serious cases. Since the disease is a methylmercury poisoning that occurred through fishes and shellfishes via environmental pollution, it is a matter of course that there are patients with the diseases of many various patterns of the severity in addition to these severe cases. Thereafter, some patients were actually confirmed to have had the disease as a consequence of chronic exposure to the methylmercury compound, and a number of patients were confirmed to have had mild incomplete-type Minamata disease. As the mild type of fetal Minamata disease as well, some patients were confirmed to have had high mercury levels in the umbilical cord and shown intellectual disorder as the main symptom.

Minamata disease is pathologically characterized by cortical damages of the cerebrum and the cerebellum. In other words, cell deficits are selectively observed in the calcarine areas (visual center), precentral gyrus (motor center), postcentral gyrus (sensory center), and the transverse temporal gyrus (auditory center) of the cerebrum. In the cerebellar cortex, Purkinje's cells remain, and granular cells markedly fall off, showing an atrophy of granular cell type. The severe cases show spongiosis and the mild cases show slight nerve cell loss and glia cell proliferation. Thus, lesions of various degrees have been confirmed. It is characteristic that the lesions of the basal ganglion, brainstem, and the spinal cord are mild even in the severely affected cases. Damages mainly to sensory fibers including the sural nerve and the dorsal root of spinal nerves have been reported as peripheral neuropathy.

[*Comments*] With regard to the peripheral neuropathy, some reports have shown that biopsy and so on revealed the absence of damage mainly to the sensory fibers.

The methylmercury compound passes the blood-plancenta barrier in a way similar to that for the blood-brain barrier. It is therefore clear that the methylmercury compound widely damages the fetal brain via the placenta from the mother who eats the fishes and shellfishes in which the compound accumulates. Consequently, cerebral palsy-like symptoms, which include (congenital) intellectual disturbance, developmental disorder, speech disorder, difficulty of moving of extremities, gait disturbance, abnormality in ocular movement, other paroxysmal symptoms, and abnormal posture deformation, occur. The condition showing these symptoms is called fetal Minamata disease, mainly because the disease is considered to be due to damage in the fetal stage (in late pregnancy).

<Column> "*Hunter-Russell syndrome*"

In 1940, three investigators: Hunter, Bomford, and Russell in England, reported an accident of poisoning in the workers who were engaged in production of the methylmercury compound at a plant of seed disinfectants production.

In the plant, 16 workers were exposed to the methylmercury compound (the route of invasion of the compound was the respiratory system, unlike Minamata disease), only 4 of them showed toxic symptoms. Twelve other workers showed no symptoms. From the symptoms observed in the 4 patients with manifestations, three symptoms of methylmercury poisoning were regarded as ataxia, speech disturbance, and constriction of visual field, and they are called three signs of Hunter and Russell syndrome. At present, however, sensory disturbance and hearing loss were added to these three signs (speech disturbance was included in ataxia in some cases), and under these circumstances of elucidation of Minamata disease these symptoms are called Hunter-Russell syndrome, which shows symptoms of typical Minamata disease.

<Column> "*Clinical features of Minamata disease patients*"

Details of symptoms in Minamata disease patients from the records by the physician (N.B.) in those days are shown below. "S.T. A girl aged 5 years and 11 months. She has had pyrexia for only a day in late March, 1956. Whenever she had meals thereafter, she was awkward at holding chopsticks and the foods dropped. She could not wear her shoes well. Since around April 14, it has been noted that she was unsteady on her feet. On April 17, she had twists in her tongue and she was choked with something to eat. At night, she became displeased and could not sleep. She became to increasingly show wild excitement. On April 21, she was examined at Shin Nippon Chisso Hospital (Chisso Hospital). The findings on examination showed a middle stature, undernutrition, and dementia-like facial expression. She always let out sudden cries. She showed slight pupillary dilatation, dry tongue, and no other abnormalities from the aspect of internal medicine. She was admitted to the hospital on April 23. Difficulty of moving of extremities was being increased. On April 26, the tendon reflex of the upper and lower extremities was increased, a pathological reflex was observed, insomnia continued, tonic convulsions were occasionally observed in the whole body, and she bit her tongue that bled. On May 2, systemic tonic convulsions frequently occurred with remarkable sweating and muscle stiffness of extremities. On May 28, the patient became blind, the frequency of systemic convulsions gradually increased, she had no response to any stimuli, and she had flexion and remarkable deformity of the extremities." (Died on January 2, 1959)

"J.T. A girl aged 2 years and 11 months. On April 23, 1956, she was unsteady on her feet, she became unable to walk freely, and she had difficulty of moving of her hands. Simultaneously with these conditions, she was unable to speak distinctly, and she complained of pains in the right knee and hand fingers. On May 7, she could stand up, but was unable to walk. Her grasping power was also reduced. She did not chew any food. She had slight swallowing difficulty, enhanced dysphasia (she was unable to speak distinctly), and her neck became unsteady. On May 8, she was unable to eat any meals. She had insomnia. On May 10, she could not grasp any object. On May 14, difficulties of mastication and swallowing were considered to have been reduced, but alalia was present."

"K.E. A girl aged 5 years and 4 months. Since April 28, 1956, she has been unsteady on her feet and shown unnatural gait. She has been increasingly unable to speak distinctly and to grasp objects. On May 8, she received initial examination, and showed ataxic gait. On May 9, she frequently spilled water when she drank water, and she choked over her water. On May 10, she was unable to stand. On May 16, she was unable to grasp any object. On May 17, she had aphagia and stiffness of extremities. On May 21, she had pneumonia and frequent convulsions. She had severe systemic convulsions, deformation, and loss of consciousness. On May 23, she died."

Thus, symptoms of Minamata disease patients are philosophically and simply described, but the records written by the physicians in those days show how dreadful the disease is. It is surprising that these patients lived next door to each other. In the family of the patient reported last, a boy aged 11 years and 8 months had the disease on May 8. Their mother had the disease on May 16. Thereafter, his younger brother aged 8 years and 7 months had the disease on June 14.

Their father and other brothers, who had not been dealt with on that occasion have also shown a variety of complaints. In 1971, sensory disturbance, constriction of visual field, and incoordinaion were revealed by our survey, and all members of the family were revealed to have been influenced by the methylmercury compound. It is a matter of course that these results were observed, because they had eaten the same foods. However, this is fear of poisoning with environmental pollution.

[Cited from "Minamata disease" Masazumi Harada, Iwanami Shinsho (1972)]

(N.B.) "The physician in those days" indicates Hajime Hosokawa, Director of Chisso Hospital

3. Lessons of Minamata disease

The lives lost by Minamata disease and the mental and physical damages are irreparable.

Causative companies did not cooperate with other institutions' activities to clear up the cause of the disease and veiled even the achievements of internal research, leading to delays of elucidation of the cause and countermeasures and to the spread of the casualties.

Compensation for the casualties was decided to be paid by causative companies to the patients, who have been certified to have Minamata disease on trials on the basis of "Law of compensation for casualties of environmental pollution to health". The compensation paid accumulated, and at present, Chisso is obliged to pay the compensation (including the interest of borrowed money for the payment of compensation) to patients, which exceeds markedly the ordinary profit.

From the aspect of cost as well, there has been a trial balance showing that the expenses for countermeasures against the prevention of Minamata disease would have been much lower than the actual amount of compensation paid to the certified patients by the causative companies ("*Nippon no Kogai Keiken* [Experience of Environmental Pollution in Japan]" ed. by the Research Group for Global Environments and Economics, Godo Shuppan, 1991). This indicates that the prophylactic countermeasures eventually become profitable for the companies' management as well.

We Japanese, who experienced Minamata disease, should contribute actively not only in Japan but also to foreign countries on the basis of the experience as lessons, so that such a tragic environmental pollution will not be repeated on the earth.

For this purpose, the delay of the policy decision and the countermeasures by investigators, the inhabitants in the districts, and the causative companies on each occasion should be inspected with the investigation of the reasons for the outbreak of Minamata disease, the spread of tragic consequences, and for the fact that it took 12 years after the discovery of the disease for the official views to have been offered by the Government. By doing so, the lessons should be clarified from the experience of Minamata disease.

4. The object of the present compilation

The history of Minamata disease may be regarded as that of the following tragedy: Only the strict need for scientific elucidation of the cause has been done, and no responsible decision has been made by the Government; any necessary measure to counter the disease has not been considered for a long period; consequently, serious casualties were given to the inhabitants. In addition, the tragedy was repeated twice. Repetition of such a failure in the countermeasures against Minamata disease should not be allowed any more.

With regard to the problems about pollution with chemical substances, which all men currently face, there are two standpoints of the safety; i.e., from one standpoint, chemical substances, safety of which is not or will not be confirmed not only at present but also in the future, should not be discharged into environments, and from the other standpoint, a certain chemical substance may be discharged before the chemical substance is confirmed to be harmful.

Considering from viewpoints of preliminary prevention of environmental pollution and prevention of the spread, everybody should agree with the former principles of the priority of safety. However, when concrete measures to counter environmental pollution with chemical substances are considered, various objections against the measures are raised, as follows: "Any causative chemical substance has not been identified"; with regard to the chemical substances, harmfulness of which has not been demonstrated, "regulation of the chemical substances may damage considerably industrial activities." Thus, decision of important policy or social countermeasure is not necessarily made rapidly because of such dissenting voices.

Considering the seriousness at the time point when the current pollution with chemical substances, the

spread of the pollution, and the casualties were confirmed, it should be beyond medical aid to consider countermeasures after the scientific elucidation of harmfulness and the mechanisms. There are many things that can be learned from the failed experience of Minamata disease; e.g., the way how to rapidly decide the opinion as the Government under the state in which insecurity remains.

Minamata disease is a typical poisoning with chemical substances, which occurred as a result of the following process: the causative agent (methylmercury compound), a by-product of the process of production at chemical plants, was discharged with factory wastes to the natural world, and accumulated in fishes and shellfishes, and the people ate them in large quantities. This affair has the leading meaning in problems with chemical substances as the event as well, in which a chemical substance damaged seriously a fetus via the maternal placenta.

The subjects of the studies by the Study Group included the process of inspection of the cause, which indicated the most important period for considering countermeasures against Minamata disease, i.e., from the outbreak of the disease to September 26, 1968 when the Government's unified views were presented. Problems with Minamata disease have involved central ministries and offices, the Diet, Prefecture, prefectural assembries, city, municipal assemblies, causative companies, trade groups, research institutions, the mass media, victims in the districts, inhabitants, fishing people, and so on. How have these social groups or the individuals belonging to them behave regarding the disease during the period? How have they recognized the period and judged the situation? What kind of results have been induced by the behavior under the recognition and circumferential judgment? These problems mainly with Minamata disease in Kumamoto Prefecture were discussed from a socioscientific viewpoint at meetings of the Study Group. The present research papers were designed to extract social factors, which make the common quick countermeasures against the current problems with chemical substances to be hard, from the process of inspection of the cause of the disease and to dispatch the lessons obtained from the factors to various foreign countries over the world as well as all countries in Japan.

Many valuable lessons, which will become useful on considering environmental problems in the future, particularly relief measures, may be induced from the history from 1968 onward as well as the period included in the subjects of the present study, i.e., the process of inspection of the cause before 1968. Therefore, it should be added that similar research is required for these lessons.

[*Comments*] The present research papers do not show any conclusion about the presence or absence of legal responsibility of the nation or prefecture. With regard to the presence/absence and the extent of legal responsibilities of the Government's and prefectural countermeasures in those days, the opinions may vary with individuals' standpoints. When the actual situation of the casualties of Minamata disease is exposed, however, nobody may be satisfied with the countermeasures made in those days, regardless of the standpoints. There is the common critical moment in which the Minamata disease affair is inspected in spite of the different standpoints.

It is easy to criticize the behavior of each group made in those days after the results of the affair have already been obtained. However, on reading the papers, suppose the case in which the readers are placed in the situation as the person concerned in those days. Imagine how the readers should have or should not have behaved under the situation. Read the papers as if the readers face the troubles. On reading the final chapter particularly, review the readers' current standpoints and ask again whether or not the readers are going to repeat the same serious mistake at present.