

MINAMATA DISEASE IN JAPAN

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Summary

Mercury and its compounds have many unique properties, which have made them extremely useful in industrial and agricultural technology. In Japan, inorganic mercury was used as a catalyst in the industrial production of acetaldehyde. A highly toxic

methylmercury compound was produced as a by-product in this process. When discharged in wastewater into a sea-bay or a river, it was taken up by organisms of sediments and eventually this contaminant accumulated in fish and other marine creatures through mercury amplification in the food chain.

1. Introduction

The outbreak of Minamata Disease was first discovered in 1956 in certain villages around Minamata Bay, in Kumamoto Prefecture. In 1965, another similar epidemic occurred along the Agano River, in Niigata Prefecture (Figure 1). Two Minamata Diseases are a methylmercury poisoning disease of the central nervous system. There have been cases of fetal-type Minamata Disease which were caused when the mothers had been exposed to the methylmercury during pregnancy.

Through the diagnostic criteria based on the Compensation Law, 2,952 people have been certified as Minamata Disease patients in the Kumamoto and Niigata area, and a total of approximately 136 billion yen has been paid in compensation from the companies responsible for the pollution (as of March, 1997).

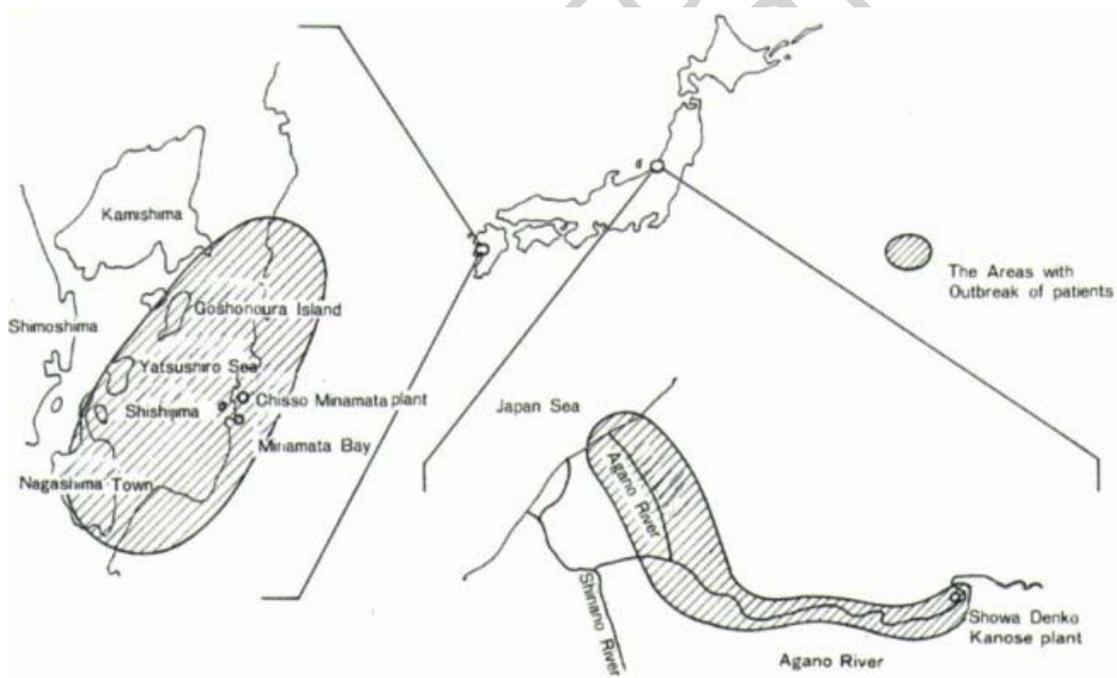


Figure 1. Map of two areas with outbreak of Minamata Disease.

In 1966, the Japanese government reached an agreement with non-certified victims and their supporters, and legal issues surrounding Minamata Disease were finally resolved, 40 years after the first outbreak.

The Minamata experience has left us with an invaluable understanding of the importance of taking thorough measures to protect public health from environmental pollution.

2. Outbreak of Minamata Disease

2.1. First outbreak of Minamata Disease in Kumamoto

A strange disease with peculiar neurological symptoms occurred in a fishing village in Minamata Bay at the end of 1953. The onset of this disease is very slow and sometimes a person manifests the disease suddenly after drinking liquor, without showing any precursory or general symptoms, such as fever. Researchers who were the first to examine the victims reported that they felt a tingling sensation at the ends of the extremities and complained of inability to grasp, difficulty in walking, inability to run, slurred speech, diminution of vision, hardness of hearing, and difficulty in swallowing. Investigations revealed that the basic pathological picture of this disease closely resembled that of the cases of methylmercury poisoning reported by from Great Britain.

At the beginning of the outbreak of this disease, ‘infantile cerebral palsy’ was mentioned as the cause of death on the death certificate of a female child who died crying with limbs quivering. It was about that time that rumors of mad cats began to spread in the areas around the victims’ homes. With the frequent outbreak of victims who died mad as in ‘cat-dancing disease’, a Committee for Countermeasures against the Strange Minamata Disease was organized with the cooperation of the local medical association and health center in 1956.

At the request of this committee, a ‘Study Group on Strange Minamata Disease’ was organized at the Kumamoto University School of Medicine in August of 1956 to conduct epidemiological research on the cause and to formulate plans to deal with the situation. The first recorded case of Minamata Disease was of a young child in 1953, and the number of Minamata Disease victims increased to seven adults and five children (a total of 12) the following year, to eight adults, two children and five congenital cases (a total of 15) in 1955, and to 22 adults, 21 children and seven fetal cases (a group of 50, the highest number recorded up to that time), in 1956.

A look at the incidence at the beginning of the outbreak (1953-1956) shows that there were 51.3 victims per 10 000 of the population in the area in which Minamata Disease occurred, and that a high incidence of 101.0 victims was recorded in the fishing villages alone. The fatality of 111 cases was very high at 36.9%. In 1958, as victims died one after another, a group of researchers, who had performed pathological autopsies of six cases, suggested that Minamata Disease could be due to alkylmercury poisoning, since the findings of the cases showing a chronic clinical course were similar to those reported in 1954.

In 1959, Minamata Disease started to be found in areas near the mouth of the Minamata River and also in remote areas north-east and south-west of Minamata City, the former including the Tsunagi district and the latter the Izumi district in Kagoshima Prefecture. One hundred and eleven patients were identified at that time. The great number of patients in areas near the mouth of the Minamata River contracting the disease apparently resulted from the fact that in 1957, plants and factories of Chisso Co., Ltd. in areas around the mouth of the river changed the outlets of their waste drainage channels from Minamata Hyakken Port to Hachiman pool, from which wastewater was then discharged into the mouth of the Minamata River.

It can be inferred from the transition in pollution levels within fish that continuous methylmercury exposure at a level that could possibly have caused Minamata Disease may have existed in the areas around Minamata Bay in and before 1968 (Figure 2). In 1970, the number of patients with acute or sub-acute symptoms reached 121. Of these patients, 61 people were fisherfolk living in the areas where the disease produced a great number of victims.

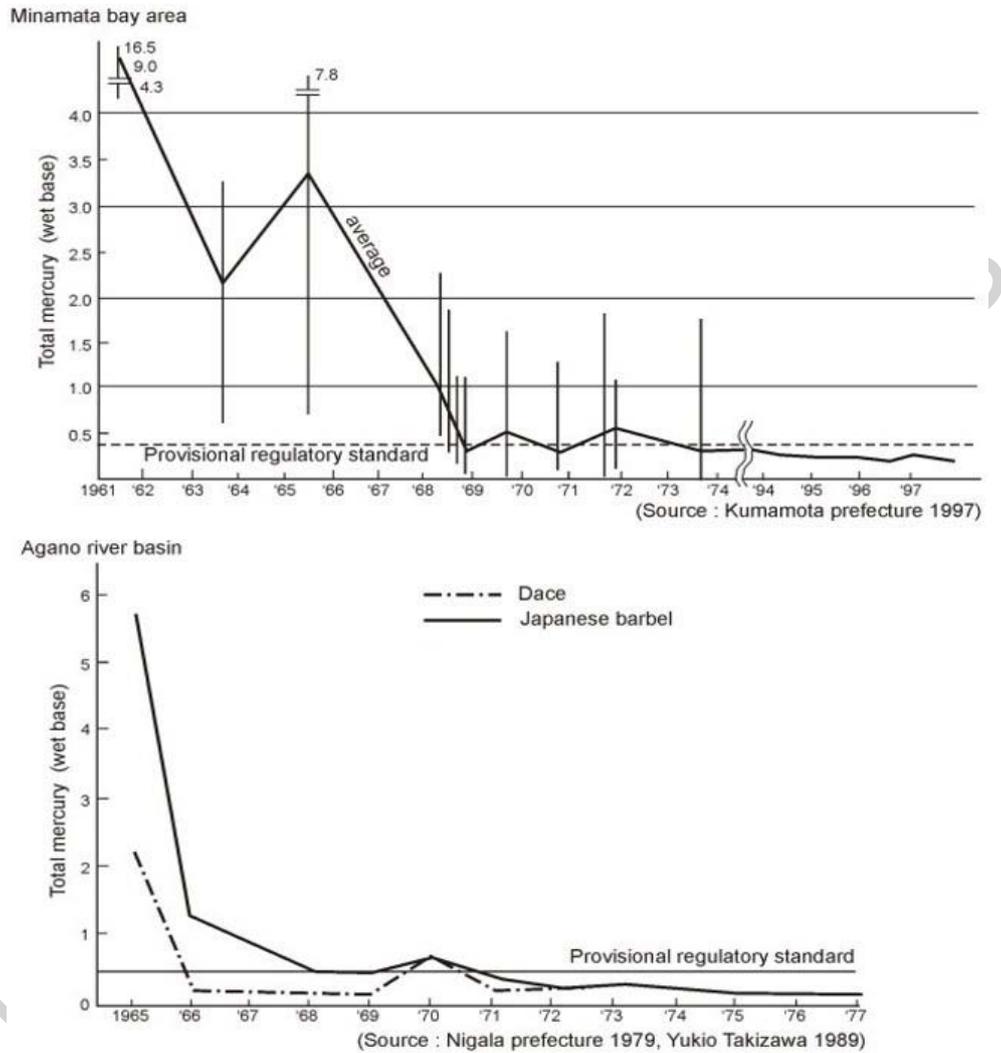


Figure 2. Transition in total mercury levels of fishes in Minamata Bay area and Agano River basin.

The possibility that a third outbreak of Minamata Disease might occur on the Sea of Ariake was presented in May 1973, by the 2nd Minamata Disease Research Group of Kumamoto University's Medical Department. They suggested that there might be chronic effects of very small amounts of mercury encountered regularly for an extended period of time, and they emphasized that the possibility that this might have already turned into a reality. This opinion was examined by a national committee of experts on mercury from various fields, but was finally contradicted, with the committee's conclusion that there was no evidence to support this opinion. In September 1974, the

Pollution-related Health Damage Compensation Law was enacted, substantially improving relief measures for the victims.

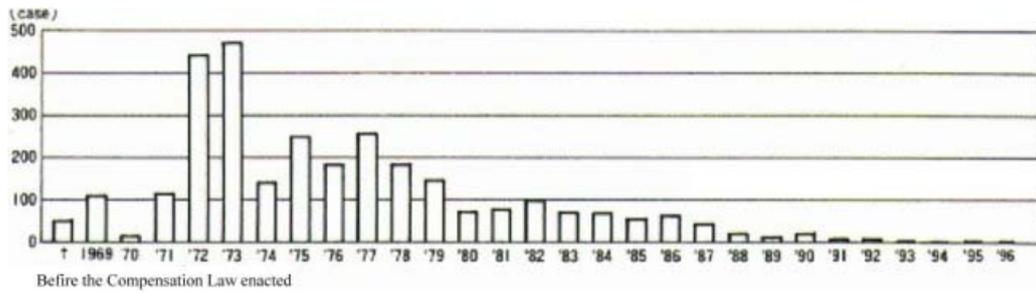


Figure 3. Transition in the number of certification as Minamata Disease patient.

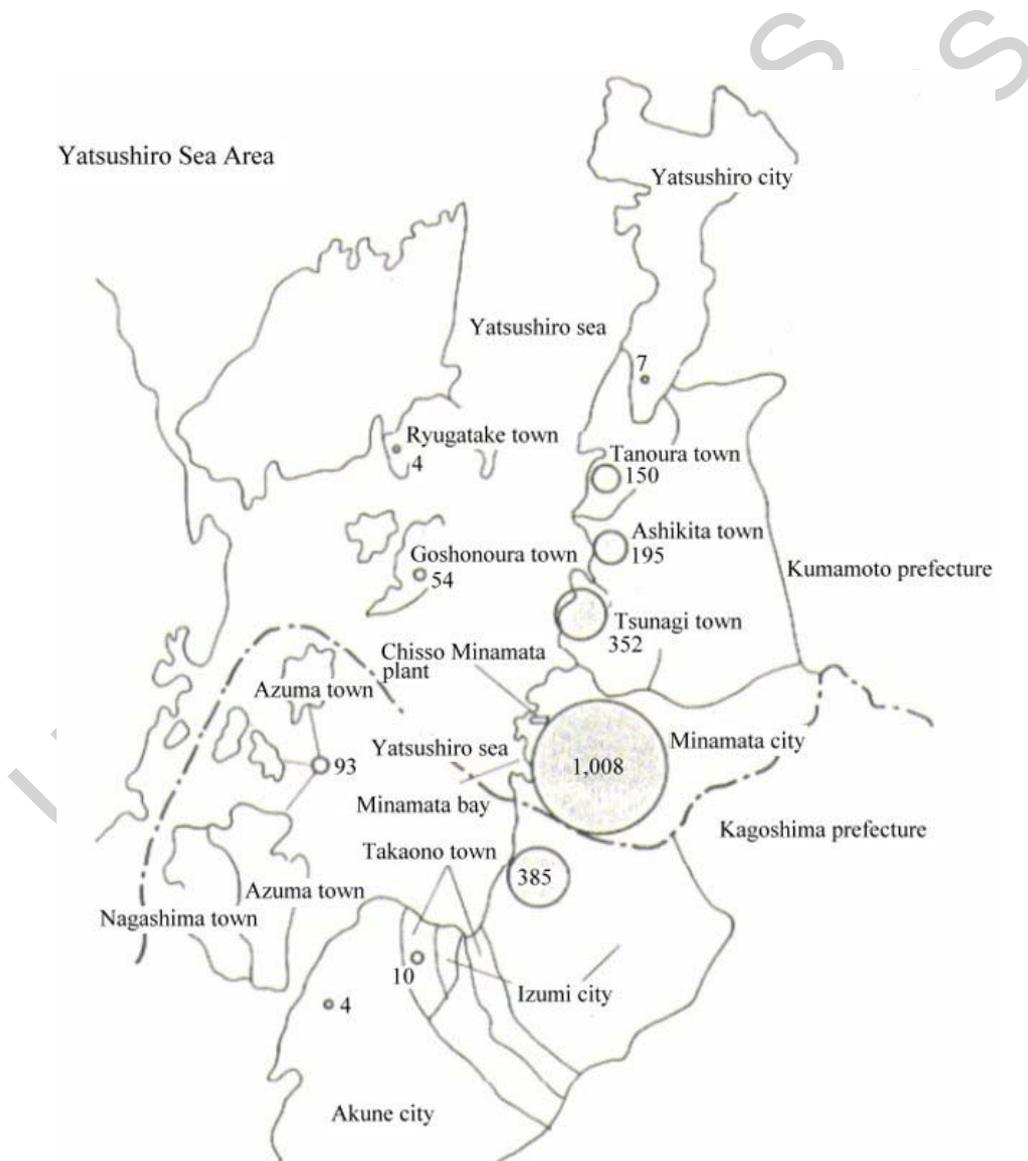


Figure 4. Distribution of certified patients with Minamata Disease in the Yatsushiro Sea Area.

Parallel with the enactment of this Law, the number of victims who were officially certified as Minamata Disease patients peaked during the 1972 through 1973 period. A great number of victims were observed who showed no clinical symptoms at the beginning of the outbreak of Minamata Disease, but who started to complain of superficial numbness of the extremities several years or decades after the first outbreak of the Disease. On the other hand, chronic Minamata Disease started to increase in 1975-1979.

Through the Compensation Law, 2,261 victims have been certified as Minamata Disease patients in the Yatsushiro area as of December 1996 (Figure 3). With regard to the distribution of the patients, in Kumamoto Prefecture, Minamata City had the greatest number of patients who contracted the disease, accounting for more than 50% of the patients in the entire prefecture, followed by Tsunagi-machi, Ashikita-machi and Tanoura-machi; in Kagoshima Prefecture, Izumi City had the greatest number of patients who contracted the Disease, accounting for 80% of the patients in the entire prefecture, followed by Higashi-machi (Figure 4).

2.2. Outbreak of Second Type of Minamata Disease in Niigata

In May 1965, an outbreak of organic mercury poisoning similar to Minamata Disease occurred sporadically in villages along the Agano River from its mouth to about 6 km upstream. The first victim diagnosed with organic mercury poisoning complained of numbness of limbs around the mouth in October 1964, and the symptoms became aggravated.

When symptoms, such as constriction of the visual field and ataxia in walking appeared, this victim was referred from a town hospital to Niigata University Hospital for medical examination and treatment.

Two similar cases were discovered in April and May 1965. An investigation was started immediately by a study group, with Niigata University School of Medicine at the center. A special study group was organized by the Ministry of Health and Welfare in September 1967, and the work involved clinical and epidemiological investigations, as well as determinations of mercury concentrations.

With the discovery of one patient in January, April and May 1965, an epidemiological investigation was initiated by a study group of Niigata University School of Medicine in June of the same year.

As a result, 26 victims, including five fatalities, were discovered. Though the Compensation Law, 690 victims have also been certified as Minamata Disease patients in the Agano River Basin, as of December 1996 (Figure 5).

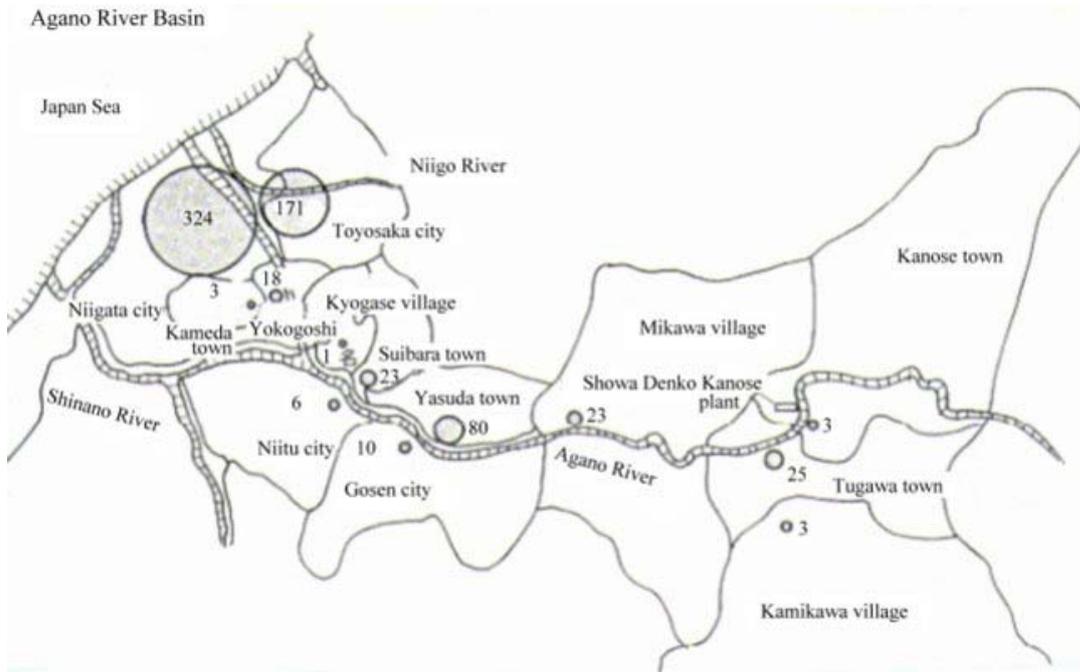


Figure 5. Distribution of certified patients with Minamata Disease in the Agano River Basin.

A clear correlation is observed between the amount of river fish ingested and mercury content in hair in areas where victims appeared. That is evident from the high mercury levels (total mercury levels) shown in $56.8\text{-}570 \mu\text{g g}^{-1}$ in hair, $6.4\text{-}90.8 \mu\text{g per dl}$ in blood, and $92\text{-}915 \mu\text{g per day}$ in urine from most cases found in the first general medical survey.

Measures to restrict the fish catch in the Agano River were taken immediately after the incident was made public, and further appearance of victims ended about one year from the time the fishing was restricted.

3. Investigation of Cause of Minamata Disease in Two Areas

3.1. Research on the Causative Agent of Minamata Disease in Kumamoto

The epidemiological features of Minamata Disease were found to be: (1) people who ate a great deal of fish and shellfish succumbed to this Disease; (2) both men and women, in each age bracket (except infants), contracted this Disease; and (3) cats kept by patients showed similar symptoms. Deducing from these facts, food poisoning caused by fish and shellfish, specifically bacterial cerebritis, was suspected. However, none of the patients showed such signs and symptoms as high fever, inflammation of the blood or lymph systems, or stomach or intestinal disorders; they showed only neurological symptoms, and results were negative when they were checked for bacteria or viruses. Therefore, the assumption arose that chemical poisoning caused by fish and shellfish was responsible. Considering the geographical and chronological distribution of the outbreak of the disease, the investigation focused on the chemical substances contained in the waste water discharged from the plant of Chisso Co. Ltd. It took three years to

identify the chemical substance as a mercury compound because of the peculiarity of the mechanism of the Disease outbreak. During this time, chemical analyses were repeated for countless numbers of poisonous substances, including Mn, Zn, Cu and Se, and many experiments were performed to test their effects on animals.

While these various investigations, from different perspectives, were going forward in the search for the possible cause of the disease, highly concentrated mercury contamination of the sludge in Minamata Bay attracted the attention of the investigators. At locations near the drainage outlet of the plant, the concentrations measured were as high as $2,010 \mu\text{g g}^{-1}$. The concentration gradually decreased toward the mouth of the bay, where it measured only $12.2 \mu\text{g g}^{-1}$. On the other hand, in the areas that were subject to the investigation, the concentration ranged from 0.4 to $3.1 \mu\text{g g}^{-1}$. This great difference suggested that contamination by mercury inside the bay resulted from effluent from the drainage outlet of the plant. This evidence immediately caused a great number of fishermen to rush and turn to the plant for relief and petition the civil authorities concerned. Thus, the Disease became a political as well as a health problem. The results of a comprehensive study of Minamata Disease, incorporating epidemiology, microbiology, pathology and clinical medicine, led to the decisive conclusion that organic mercury contained in fish and shellfish in Minamata Bay was the cause of Minamata Disease. The fish and shellfish in the bay accumulated from 10 to $35 \mu\text{g g}^{-1}$ of mercury, directly after the first outbreak after the disease.

The fish and shellfish in Minamata Bay became polluted by mercury contained in wastewater discharged from the Minamata plant. This plant engaged in vinyl chloride and acetic acid manufacturing processes, for which inorganic mercury was used as a catalyst. The great amount of mercury accumulated in the sludge in the drainage outlet was presumed to come from the acetic acid manufacturing process in the plant. The plant acknowledged this was likely, considering the amount of mercury lost from the plant. It was also discovered that in those days, wastewater was discharged into the bay without undergoing any treatment. The factory admitted this fact, and according to the announcement in 1959 made by the company that owned the factory, wastewater from the acetic acid equipment contained $10\text{-}20 \mu\text{g g}^{-1}$ of mercury, and the amount of wastewater was 6 m^3 per min. Moreover, the equipment was overhauled for cleaning four times a year, at which times the amount reached 30 m^3 per min. The wastewater had been discharged untreated into the bay. In July 1959, Kumamoto University made public its intention to concentrate investigations on organic mercury as the cause of Minamata Disease, for the following reasons (1) clinical and pathological findings from the victims closely resembled cases of methylmercury poisoning with Hunter-Russell's syndrome; (2) a great deal of mercury was found in urine of victims and in the tissues of autopsy cases; (3) clinical pathology of spontaneously affected cats closely resembled that of the victims; (4) a large amount of mercury was found in the organs of these cats; (5) symptoms of spontaneously affected cats were clinically and pathologically reproduced by the administration of fish and shellfish containing mercury to cats; and (6) methylmercury compounds were detected in fish and shellfish. The Investigation Group of Kumamoto University detected methylmercury in fish and shellfish and methylmercury crystals in the acetic acid equipment of the factory, providing evidence for the methylmercury theory.

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Biographical Sketch

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