5 Minamata disease: a challenge for democracy and justice

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Minamata disease, which can induce lethal or severely debilitating mental and physical effects, was caused by methylmercury-contaminated effluent released into Minamata Bay by Chisso, Japan's largest chemical manufacturer. It resulted in widespread suffering among those who unknowingly ate the contaminated fish. This chapter documents the story in three phases.

The disease first came to prominence in the 1950s. It was officially identified in 1956 and attributed to factory effluent but the government took no action to stop contamination or prohibit fish consumption. Chisso knew it was discharging methylmercury and could have known that it was the likely active factor but it chose not to collaborate and actively hindered research. The government concurred, prioritising industrial growth over public health. In 1968 Chisso stopped using the process that caused methylmercury pollution and the Japanese government then conceded that methylmercury was the etiologic agent of Minamata disease.

The second part of the story addresses the discovery that methylmercury is transferred across the placenta to affect the development of unborn children, resulting in serious mental and physical problems in later life. Experts missed this at first because of a medical consensus that such transfer across the placenta was impossible.

The third phase focuses on the battle for compensation. Initially, Chisso gave token 'sympathy money' under very limited criteria. In 1971 the Japanese government adopted a more generous approach but after claims and costs soared a more restrictive definition was introduced in 1977, justified by controversial 'expert opinions'. Legal victories for the victims subsequently made the government's position untenable and a political solution was reached in 1995–1996. In 2003, the 'expert opinions' were shown to be flawed and the Supreme Court declared the definition invalid in 2004.

In September 2011 there were 2 273 officially recognised patients. Still, the continuing failure to investigate which areas and communities were affected means that the financial settlement's geographic and temporal scope is still not properly determined. Alongside deep-seated issues with respect to transparency in decision-making and information sharing, this indicates that Japan still faces a fundamental democratic deficit in its handling of manmade disasters.

This chapter is followed by three short updates on the effects of mercury poisoning since Minamata; on attempts to contain it, including the 2009 global agreement to phase mercury out of economic activity; and on the need for better information about contaminant exposures to enable policymakers to make informed choices that balance the benefits of fish consumption against the assumed adverse effects of low-level methylmercury exposures.

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5.1 Introduction

The Minamata disease story is one of blinkered awareness by industry and government, of inaction, refusal to take evidence seriously, insistence on high levels of proof before addressing the problem, and delay, delay, delay.

In 1972 scientists, politicians and the public were shocked by the presence of two Minamata disease patients on the platform at the first global environmental conference, held in Stockholm — the United Nations Conference on the Human Environment (Harada, 2004). They were halting and unsteady, they struggled to speak. They had been poisoned by mercury in their environment.

It took a further thirty-eight years for the first session of an Intergovernmental Negotiating Committee, also held in Stockholm, in June 2010, to start developing a global legally binding instrument on mercury pollution prevention, following the elaboration of a legally binding instrument on mercury at the United Nations Environment Programme (UNEP) Governing Council in February 2009.

At the Intergovernmental Negotiating Committee's opening session, the representative of Japan reported that his government wished to host the conference at which the global mercury agreement would be agreed (UNEP, 2010). He proposed that it be called the 'Minamata Convention', indicating the international community's resolve to ensure that the human health and environmental disaster caused by methylmercury in the Bay of Minamata would never be repeated (UNEP, 2010). He also reported that the Japanese government would contribute all it had learnt about reducing the risk of mercury (METI, 2010).

Despite the Japanese government's apparent determination, many problems remain unresolved in Japan. Key aspects of the disaster are unknown, such as the number of Minamata disease sufferers and exposed residents, and the area and duration

Photo: In 1972 United Nations Conference on the Human Environment in Stockholm focused on the Minamata disease. (From the left) Dr Noboru Ogino, Mr Tsuginori Hamamoto, Dr Masazumi Harada, Ms Fujie Sakamoto (Shinobu's mother), Ms Shinobu Sakamoto, unknown person, and Dr Soubei Togo. Mr Hamamoto and Ms Shinobu Sakamoto are Minamata disease patients.

of exposure. There is not even a consensus on the definition of Minamata disease (Ekino et al., 2007), making it hard to count the number of Minamata disease patients and determine who qualifies for compensation. Indeed, the diagnostic criteria that the Government has consistently used to certify Minamata Disease were judged medically invalid by the Japanese Society of Psychiatry and Neurology (JSPN) in 1998 (JSPN, 1998) and declared invalid by the Supreme Court in 2004 (McCurry, 2006). Nevertheless, the government has not changed the criteria.

The criteria currently being used to diagnose Minamata disease are too strict, meaning that even patients with the related neurological symptoms lack government accreditation. And without government recognition of a Minamata disease diagnosis, a patient will not be properly compensated. Consequently, in September 2011 although 2 273 individuals were officially recognised as Minamata Disease patients (Minamata Disease Museum, 2010), several tens of thousands have neurological symptoms characteristic of methylmercury poisoning but remain formally unrecognised as Minamata disease patients (McCurry, 2006; Watts, 2001; Sankei Shinbun, 2011; Yorifuji et al., 2013).

Given this continuing conflict and suffering, Masazumi Harada ([2]), has observed that when the government of Japan expressed a desire to share its experience and expertise, and to name the new global convention on mercury phase-out after Minamata, it should not merely report its technical success in controlling mercury pollution. It should also 'report to the world that there still remain unsolved problems in Minamata. Not only cases of success but also cases of failure can be valuable lessons for the world' (Kumamoto Nichinichi Shinbun, 2010b).

In this chapter, the history of Minamata disease is presented chronologically, broadly separating the discussion into three parts: the period before 1968; specific issues associated with congenital Minamata disease; and the period after 1968. The chapter concludes with the lessons that can be drawn from the history of Minamata disease.

5.2 Minamata disease in the period up to 1968

5.2.1 Early warnings and signs of Minamata disease: from wildlife to children

> 'Don't think of labourers as humans; treat them as cattle and horses'

This quote is widely attributed to the Chisso factory founder Shitagau Noguchi, suggestive of past attitudes towards workers, residents and the environment in Minamata (Miyazawa, 1996).

Minamata is the south-western part of Kumamoto Prefecture in Japan, facing Shiranui Sea (Map 5.1), 1 000 km from Tokyo. In 1908, the Nihon Carbide factory was established in Minamata. Later that year it merged with Sogi Electric to form Nihon Chisso Hiryo Kabushiki Gaisha (Japan Nitrogenous Fertilisers). The firm initially used carbide to produce ammonia for fertilisers but, having purchased a German patent for producing ammonia without carbide in 1921, it began using carbide and acetylene (derived from carbide) to manufacture a wider range of organic synthetic compounds.

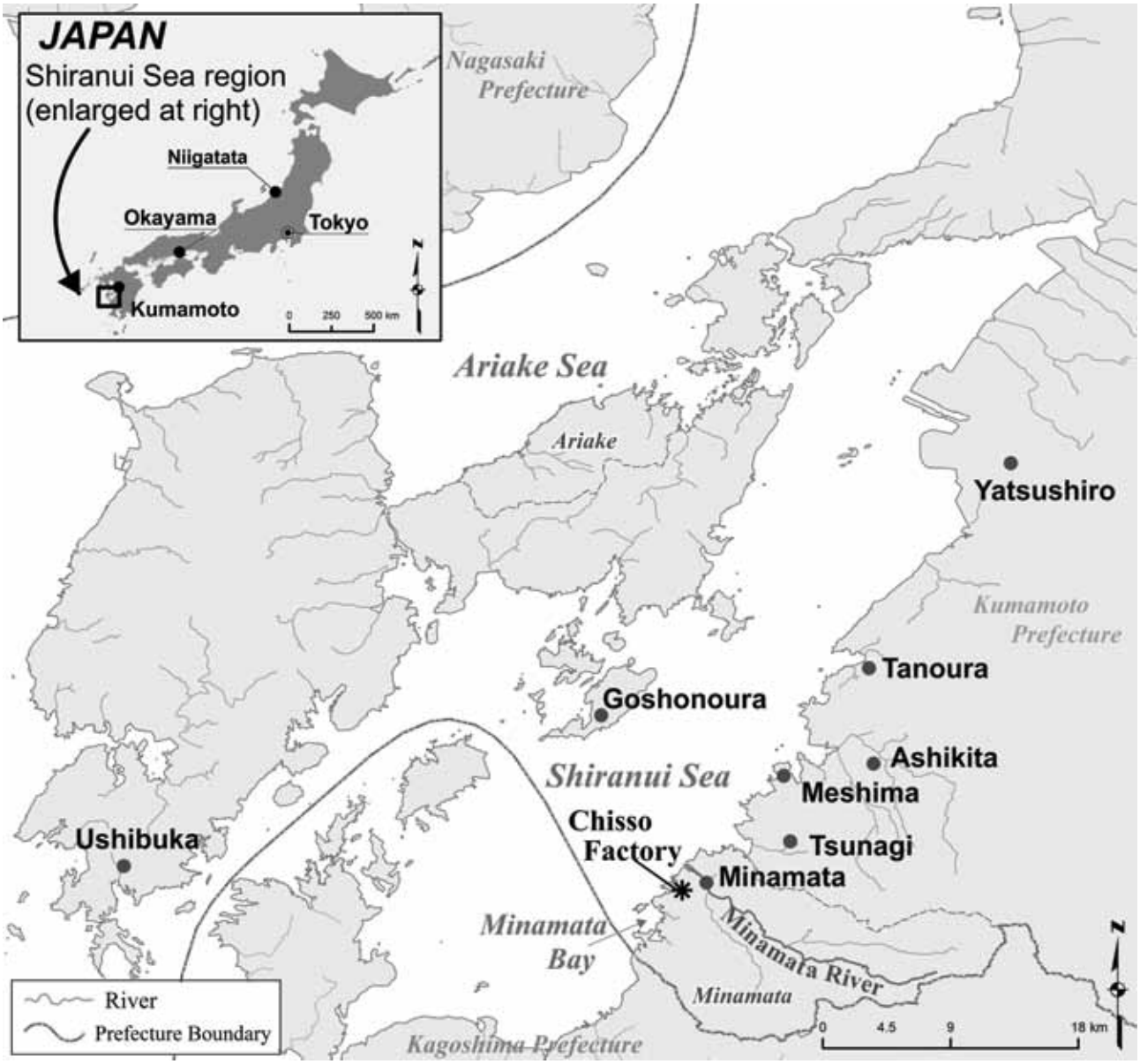
One such compound was acetaldehyde. The factory began producing it in 1932 from acetylene gas, using mercury as a catalyst. The process was developed by Hikoshichi Hashimoto, who later became factory manager and served as Minamata's mayor ([3]).

It is now understood that the effluent from the acetaldehyde production contained methylmercury and this caused Minamata disease. In fact, Vogt and Nieuwland had already shown in 1921 that organic mercury was synthesised in producing acetaldehyde (Ishihara, 2002). In the 1930s, Zangger (1930) and Koelsch (1937) reported on intoxication due to occupational exposure to organic mercury or methylmercury (short-chain organic mercury) (Ishihara, 2002). A researcher at Chisso factory demonstrated in 1951 that organic mercury is synthesised in the production of acetaldehyde (Arima, 1979) but it is not clear whether the Chisso factory was aware of the toxic effects at that time.

([2]) Masazumi Harada, co-author of the present chapter, started researching Minamata disease in 1961 and went to Stockholm with the Minamata patients in 1972.

([3]) He seems to have played a similar role as that of Peter Stockmann, the mayor in Ibsen's play 'An enemy of the people', where he opposed taking action on the public health doctor's report on pollution of the town's spa baths and in favour of suppressing the report. The Chisso doctor Hosokawa, whose report on Minamata disease in the Chisso factory cats was also suppressed, drew some comfort from reading 'An enemy of the people.'

Map 5.1 Map of Shiranui Sea region



Source: Dr. Saori Kashima, Department of Public Health and Health Policy, Hiroshima University Graduate School of Biomedical Sciences, Hiroshima, Japan.

The factory's knowledge of the Zangger study (1930) was considered at the Minamata Disease Trial in 1987 (Hashimoto, 2000) but the findings were inconclusive.

The Chisso factory's acetaldehyde production initially peaked at 9 159 tonnes in 1940, a level not matched after the war until 1955. By 1960, however, it had quintupled to 45 200 tonnes (Figure 5.1), making up 40 % of Japan's total output (Arima, 1979). In 1951, to increase production of acetaldehyde, the factory changed the oxidiser of acetaldehyde production from manganese to iron (Miyazawa, 1996). This production change and related technical improvement are considered to have increased methylmercury waste from the factory (Miyazawa, 1996). Nishimura and Okamoto (2001) estimate a more than eight-fold increase from 1951 to 1959.

In 1952, the factory succeeded in producing octanol from acetaldehyde (Miyazawa, 1996). Japan had previously relied on imports of octanol, an important ingredient in plastics. As a result, the factory increased production of acetaldehyde and by 1959, the factory accounted for 85 % of Japan's octanol output (Hashimoto, 2000). As a consequence the methylmercury waste from the factory also increased.

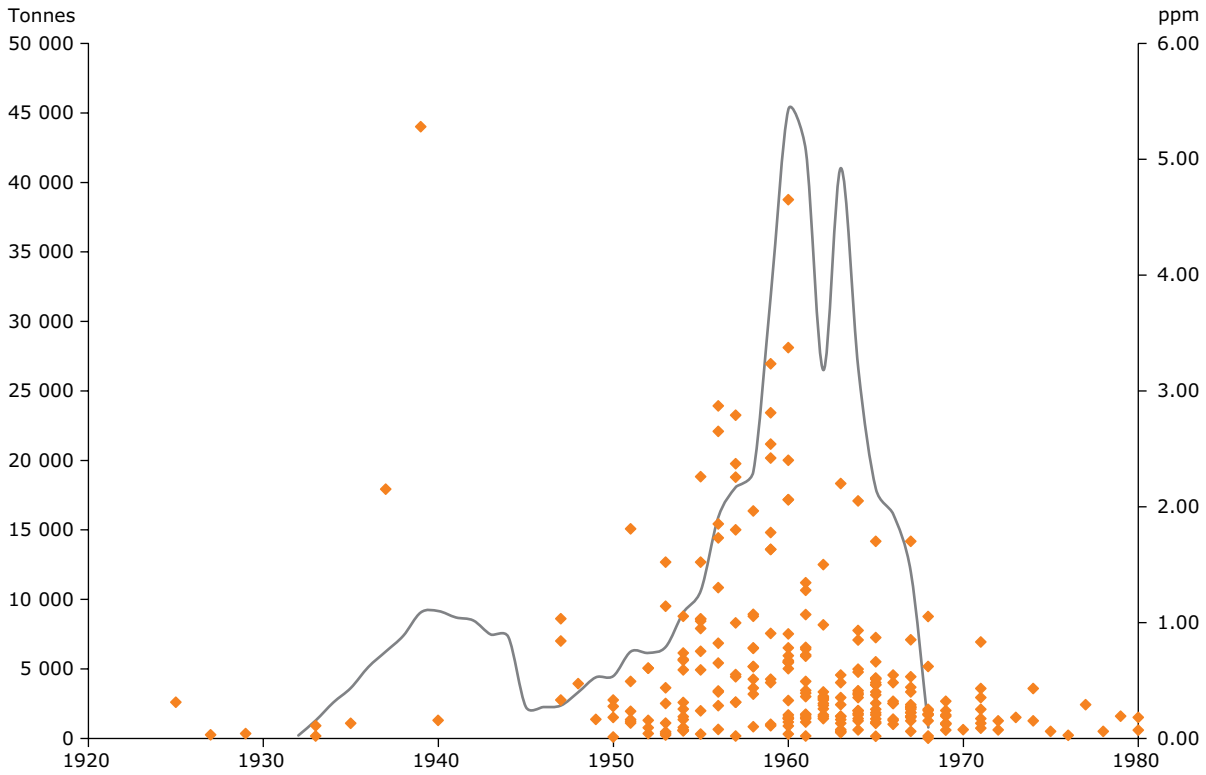
The economic significance of this production was considerable. Japan had recorded a trade deficit since the end of the Second World War (Ministry of Finance, 2011) and plastic products were key Japanese exports at that time helping to reduce its deficit. As the most advanced chemical company during those decades, the Chisso factory clearly had an important economic role.

Minamata city grew with the factory, 'reaching a population of 20 000 in 1921, 30 000 in 1941, 40 000 in 1948, and a peak of 50 000 in 1956' (George, 2001). The factory was a main employer in the city: at least 3 811 of Minamata's 19 819 workers were employed at the factory in 1960 (Ui, 1968). In addition, the factory not only paid half of the local taxes in Minamata but also provided public facilities, such as a factory hospital (Ui, 1968). Hikoshichi Hashimoto, who developed the method to produce acetaldehyde, went on to serve four terms as Minamata's mayor (1950–1958 and 1962–1970) after managing the factory during the Second World War (George, 2001). In these circumstances, Minamata was known as Chisso's 'castle town' (after the capital cities of the feudal lords who controlled much of the lives of its citizens) (Harada, 2004).

The first hints of what was to become Minamata disease may be apparent in reports of the factory's impact on the local fishery (Harada, 2004). Around 1925–1926, the company began to receive requests for compensation from the fishing cooperative. On the condition that no further complaints would ever be lodged, Chisso paid a small amount of 'sympathy money'. The issue of fishery damage arose again in 1943 due to carbide residue from acetylene production and another compensation contract was concluded. And after the war the issue of fishery damage resurfaced in 1949 but compensation negotiations reached no conclusion and the issues faded.

The fishermen knew that it had become more difficult to catch fish; that barnacles did not attach themselves to boats moored near the factory waste outfall; and that fish could not live in water from the outfall. But, the factory would not listen to them, replying that these facts were 'not scientific, not supported by data' (Harada, 2004), although the fishing cooperative

Figure 5.1 Acetaldehyde production at the Chisso factory (line) and methylmercury concentrations in the umbilical cords of residents around the Shiranui Sea (dots)



Source: Reprinted from Science of the Total Environment, vol. 408, 272–276, Yorifuji, T., Kashima, S., Tsuda, T. and Harada, M., 'What has methylmercury in umbilical cords told us? — Minamata disease', Copyright (2009), with permission from Elsevier.

collected and showed detailed data of the fishery damage (described below). In addition, neither the company nor the government assisted in identifying an appropriate scientific protocol for researching the fishermen's significant concerns.

From around 1950, strange phenomena started occurring around Minamata Bay (Harada, 2005). Fishermen witnessed huge numbers of fish rising to the surface and swimming around as though they were crazy. Sea birds that had become unable to fly were seen crouching on the shores of the bay. Oysters and cockles were washed up onto the beach rotting with their shells open, emitting a horrible stench. During this period, according to the data collected by the fishing cooperative, total fish catch of 459 225 kg on average in the period 1950–1953 dropped to 172 305 kg in 1955 and 95 599 kg in 1956 (Harada, 2004).

In 1952, fishermen requested Kumamoto Prefecture to address the situation. The Fisheries Division at Kumamoto Prefecture asked the factory about the discharge treatment and the factory submitted documentation, which reported that mercury was used in the process of producing acetic acid (a substance produced after acetaldehyde) (Chisso factory, 1996). Reiji Miyoshi at the Fisheries Division inspected the factory five months later and reported that the discharge should be analysed (Miyazawa, 1996). However, Kumamoto Prefecture did not conduct a further survey of the fishery damage or the discharge and the fishery damage continued. Moreover, neither Kumamoto Prefecture nor any other group (including the research group at Kumamoto University) ever used the factory documentation indicating mercury use to identify the etiologic agent for Minamata disease or its production mechanism.

Around 1953, local cats, which ate great quantities of fish, began exhibiting strange behaviour: drooling and staggering about, undergoing convulsions or running in circles as though they were mad, or leaping up into the air and charging forward (Harada, 2004). Eventually, fishermen had no more cats. In August, 1954, a local newspaper reported that fishermen in a village (Modo) were annoyed by the increase in mice due to the annihilation of cats (Kumamoto Nichinichi Shinbun, 1954). These strange occurrences were an omen of what would happen next to humans. Watching the 'dancing cats', people began to feel uneasy. Indeed, a few patients with neurological symptoms of unknown origin were detected during this period and a subsequent study revealed that the first patient was traced back to 1942 (Nishigaki and Harada, 1975).

Cats eating great quantities of fish from the polluted Minamata Bay went mad and died after strange dancing and convulsions.

Photo: © istockphoto/Taner Yildirim

On 21 April 1956, a paediatrician at Chisso Hospital, Kaneki Noda, examined a girl aged five years and 11 months. The girl had difficulty in walking and speaking; she appeared to be in a drunken state, unsteady on her feet and slurring her words. She was hospitalised two days later. On the same day, her sister, aged two years and 11 months, developed the same symptoms and she was hospitalised on 29 April. Subsequently, Dr Noda learned of other patients with similar symptoms in the neighbourhood. He officially notified the Minamata Public Health Centre on 1 May 1956 (Harada, 1995 and 2004; Miyazawa, 1996).

Hasuo Ito, the Director of the Public Health Centre, interviewed the children's mother in detail about the disease. He then made a report to the Health Department at Kumamoto Prefecture (Ito, 1996). A newspaper in Kyushu Area, the south-western area of Japan, reported the disease on the 8 May (Miyazawa, 1996). On 28 May, the Minamata Doctors Association, the Public Health Centre, Chisso Hospital, the municipal hospital, and the city government established the Minamata Strange Disease Countermeasures Committee

(Harada, 2004). Subsequently, doctors including Dr Noda and Hajime Hosokawa, the Director of Chisso Hospital, identified numerous new cases. According to the report of Dr Hosokawa, 30 cases including 11 deaths were identified by 29 August (Hosokawa, 1996).

The first official patients lived in a secluded spot at the end of a tiny inlet off Minamata Bay, where five or six families huddled together on the narrow strand. They were so close to the sea that they could have cast a fishing line from the windows at high tide. They were people who lived as one with nature. Because the outbreak of the disease was spontaneous and occurred among neighbours, the doctors at the factory and officials at the Public Health Centre suspected that they were dealing with a contagious disease and moved the patients to an isolation ward. Officials from the Public Health Centre went to the patients' homes and made a show of spraying them with disinfectant.

Transferring the patients to an isolation ward may have indicated an intention on the part of doctors and the city — out of good will or for political reasons — to mitigate the residents' anxiety and to exempt the patients from medical expenses. The patients hated it, however, and it fostered discrimination against them. They were shunned by other community members and experienced years of discrimination (Harada, 2004).

This was the beginning of Minamata disease.

5.2.2 The cause: fish and shellfish contaminated by factory discharge (1956–1957)

In response to the request of the Minamata Strange Disease Countermeasures Committee, Kumamoto University School of Medicine established a research group including various medical departments on 24 August 1956 (MDRG, 1966). In the epidemiological section, Shoji Kitamura and his group conducted both descriptive and analytic epidemiological studies (Kitamura et al., 1957). In the descriptive study, the time sequence of cases was evaluated on a spot map, which indicated that the disease was not contagious. The analytical study revealed a relationship between the family occupation (fishing) and the disease, and a dose-response relationship between eating fish caught in Minamata Bay and the disease. Prof. Kitamura concluded that the disease could be induced by continuous exposure to a common factor, which seemed to be contaminated fish in Minamata Bay.

On 3 November 1956, the Research Group on Minamata Disease reported that the disease was not contagious but rather a food poisoning incident resulting from intake of fish contaminated by a heavy metal in Minamata Bay. It also reported that the factory's effluent was considered the cause of contamination (Kumamoto University, 1956). Furthermore, in the second debrief session on 26 February 1957, the Research Group recommended prohibiting fishing or applying the Food Sanitation Act (Miyazawa, 1996). The Act could be used to take actions against food poisoning, such as prohibiting the sale or distribution of food. An individual disobeying such a prohibition could face criminal punishment.

In November 1956, the Scientific Research Team of the Ministry of Health and Welfare of Japan (MHWJ) started an epidemiological investigation in Minamata (Matsuda et al., 1996). In March 1957, they demonstrated a relationship between the family occupation (fishing) and the disease, consistent with the Kumamoto University Research Group's epidemiological study. Moreover, they showed that families living closer to Minamata harbour, where factory effluent was discharged, were affected more than families living further away. For example, all seven families in the Tsukinoura area included at least one patient, while 33 % (7/24) of families included a patient in the Yudo area. Noting that sea water and mud in Minamata Bay was strongly affected by the factory effluent, the Research Team inferred that fish caught in Minamata Bay were contaminated by the effluent. They concluded that the disease could be induced by contaminated fish in Minamata Bay and that the factory and its effluent should be fully investigated to elucidate the disease's mechanism.

In response to these findings, the local government of Kumamoto Prefecture considered applying the Food Sanitation Act in March 1957 (Kumamoto Prefecture, 1996b) because Shizuoka Prefecture had used the Act to address an episode of shellfish food poisoning in 1950 (Shizuoka Prefecture, 1996). The local government had the authority to decide whether to apply the Act but asked for an opinion from the national government on 16 August (Kumamoto Prefecture, 1996a). On 11 September 1957, Masayoshi Yamaguchi, the Chief of the Public Health Bureau of MHWJ replied to the local government as follows (bold added) (MHWJ, 1996a):

I. 'We recommend that you should continue your policy of warning against the ingestion of fish and shellfish caught in a specified area of Minamata Bay because it may lead to the

occurrence of the unknown disease of the central nervous system.'

II. 'There was no clear evidence that **all** fish and **all** shellfish are poisoned in the specified area in Minamata Bay. Therefore, we have decided that it is impossible to apply Provision 4-2 of the Food Sanitation Act to all the fish and shellfish to be caught in that area.'

Japan's Food Sanitation Act provides that a local government's public health centre must investigate food poisoning outbreaks in detail and take measures in response. When a cause (e.g. an institution or food) is identified, the exposed area and residents must be investigated and the sale or distribution of the cause prohibited. In Minamata, despite the risk outlined in paragraph I of the reply, consumption of contaminated fish was not prohibited based on the reasoning in paragraph II. Although the Minamata Public Health Centre, like the Minamata Strange Disease Countermeasures Committee, identified severely affected patients from the start of its work, it did not further investigate the area and the exposed residents epidemiologically. Residents continued to eat contaminated fish without effective information.

In 1990, the government of Japan asserted that another reason it did not apply the Food Sanitation Act in 1957 was that the etiological agent (methylmercury) had not been identified in 1957 (Environment Agency et al., 1999). While the etiologic agent may not have been clearly identified in 1957, however, the cause/transmission (ingesting fish caught in Minamata Bay) was identified in 1956. Indeed, fish in Minamata Bay were recognised as food causing Minamata disease even in records of food poisoning published by MHWJ in 1956 (Department of Environmental Sanitation, 1957).

It is perhaps surprising that the governments of Shizuoka and Kumamoto Prefectures responded differently to their respective food poisoning episodes, although each had the same strength of evidence that shellfish or fish were contaminated with an unknown etiologic agent. Shizuoka Prefecture decided itself to apply the Food Sanitation Act, while Kumamoto Prefecture asked MHWJ and finally did not apply the Act. Miyazawa (1996) has argued that Kumamoto Prefecture's response reflected the Prefecture's concern about the compensation claims that Chisso factory would have faced if the Act had been invoked.

The early epidemiological studies identified the causes of the health impacts but concentrated solely on severely affected patients and did not investigate the health status of residents in affected areas, as required by the Act. This caused serious problems later. Since the government undertook no effective countermeasure, subsequent research sought to discover the etiologic agent or its mechanism of production in university laboratory settings rather than using epidemiological studies to find moderate cases or investigate health in local settings. During this period, local residents were almost unaware of the finding that the fish and shellfish were contaminated. Although some residents probably knew of the contamination from newspapers and their own experiences, poor fishermen in particular could not stop fishing, as this was their only means of survival.

Whereas affected fishermen lacked political power locally and nationally, Chisso factory was supported by the local government and Ministry of International Trade and Industry of Japan (MITIJ) at that time. Subsequent events clearly indicate that Chisso factory had considerable influence in Japanese industry and society. The Chief of the Department of Environmental Health who recommended not invoking the act in his reply to local government in 1957 had taken measures beyond the law to address a polio outbreak in the 1950s, importing vaccine from Russia. When a lawyer later asked him why he had avoided prohibiting fish consumption in the Minamata disease outbreak in accordance with the Food Sanitation Act despite taking measures beyond the law in the polio outbreak, he replied 'Chisso never existed behind the polio outbreak'.

In the late-1950s, the officers of MITIJ sent weekly demands to the Water Quality Maintenance Section of the Economic Planning Agency of Japan (EPAJ) that wastewater bans should never be implemented. The MITIJ officers urged their counterparts to 'stick it out' and 'offer opposition to the ban', stressing that 'Japanese economic growth would never be realised if such a big industry, Chisso, were stopped. Never stop it!' (Hashimoto, 2000).

5.2.3 Organic mercury theory (1958–1959)

With neither the factory nor the Government taking appropriate measures to control the outbreak, exposure continued and spread. Following a research meeting on 15 February 1958, Masayoshi Yamaguchi, Chief of the Public Health Bureau of MHWJ reported to other ministries and local governments on 7 July 1958 that: 'Minamata disease was caused by intake of contaminated fish and shellfish. The discharge from Minamata factory

(Chisso factory) affected Minamata Bay. The same chemical toxicant (which the discharge of which affected the bay) was considered to poison the fish and shellfish' (MHWJ, 1996b).

A local newspaper reported the news, describing it as the first MHWJ statement to mention the factory as a cause (Kumamoto Nichinichi Shinbun, 1958). Yamaguchi, who made the statement, explained later in court that MHWJ had reported to Kumamoto Prefecture because it expected the Prefecture to take control measures based on the Food Sanitation Act (Miyazawa, 1996). However, the Act was not invoked.

Following the MHWJ statement the factory took steps to dilute the discharge containing methylmercury. In September 1958 it changed the drainage route of acetaldehyde production from Minamata Bay to Minamata River (Harada, 1995 and 2004; Miyazawa, 1996), where some effluents (such as phosphoric acid effluent, carbide residue) were already discharged (Nishimura and Okamoto, 2001). Several other factors probably also influenced the factory's decision to act (Miyazawa, 1996): the factory had learned of Kumamoto University's focus on mercury; high mercury concentrations had been detected in shellfish in Minamata Bay; the factory wanted to increase acetaldehyde production; and everyone knew that residents continued to eat fish.

Hajime Hosokawa, Director of Chisso hospital, objected to the plan. He also noted that 'if patients were detected in the area around the Minamata River, it would prove that the discharge was the cause' (Miyazawa, 1996). However, the plan was executed — without the knowledge of local residents. Exposure subsequently spread not only in Minamata Bay but along the entire coast of Shiranui Sea. Fish and cats began to die in other villages (Harada, 1995). And from 1959 onwards, patients with similar neurological symptoms were identified among the residents of other villages around the Shiranui Sea (Kumamoto Nichinichi Shinbun, 1959; Ninomiya et al., 1995; Yorifuji et al., 2008).

Meanwhile, researchers at Kumamoto University's School of Medicine continued their efforts to find the etiologic agent of Minamata Disease and its biological mechanism of action. This was not easy because the Research Group knew nothing about the interior of Chisso factory (Harada, 2005): what was produced, how it was produced, what substances were used and which processes. At that time the Research Group received no assistance from the engineers at Chisso factory or even from the organic chemistry sector of Kumamoto University's School of Engineering.

The Research Group identified various possible etiologic agents — manganese, thallium and selenium — but when fed to cats these substances did not produce effects comparable to organic mercury (Takeuchi et al., 1960). Although mercury was the first etiologic agent considered, Shoji Kitamura of the Research Group has recollected with regret that 'Mercury was taken off the list on the assumption that such an expensive material would never be thrown away in the sea' (Harada, 2004).

Douglas McAlpine, a British neurologist, visited Minamata on 13 and 14 March 1958. He examined 15 Minamata disease patients and made a very valuable observation, noting that symptoms such as constriction of the visual field, impaired hearing and ataxia closely resembled those of methylmercury poisoning reported by Hunter et al. (1940).

McAlpine reported his observations in the journal Lancet in September 1958 (McAlpine and Araki, 1958). In his paper, he pointed out that the disease was caused by eating fish caught in Minamata Bay as well as the toxic action of a chemical compound contained in the effluent from Chisso factory (McAlpine and Araki, 1958). Moreover, he listed methylmercury as one of the metals which could induce Minamata disease. This was the first time that methylmercury was identified as a potential etiologic agent. McAlpine's observations were important but before he could report them to a Japanese Society of Neurology Conference he was stopped by a professor of Kumamoto University on the grounds that too many theories would be confusing (Harada, 2004).

Meanwhile, another researcher, Tadao Takeuchi, also suspected that the etiological agent was organic mercury since he also saw similarities to so-called Hunter-Russell syndrome (Hunter et al., 1940; Hunter and Russell, 1954). Takeuchi et al. (1960) extracted significant levels of mercury (not organic mercury) from patients' organs at autopsy and also succeeded in inducing similar neurological symptoms in cats by feeding them organic mercury.

Shoji Kitamura and his Research Group likewise extracted large quantities of mercury from mud and shellfish in Minamata Bay (Kitamura et al., 1960b), and noted that concentrations decreased as the distance from the factory increased. They extracted mercury from experimentally affected cats at autopsy and ascertained that mercury levels

increased in shellfish bred in the bay, demonstrating that mercury was accumulating internally (Kitamura et al., 1960b). On 22 July 1959, researchers finally concluded that the etiological agent was mercury based on the clinical characteristics and animal experiments (Kumamoto Nichinichi Shinbun, 1959; Kumamoto University, 1996).

During this period, researchers at Kumamoto University School of Medicine suspected that the mercury concentrations were a byproduct of vinyl chloride production. This is because when they had asked the factory in 1957, the factory had only mentioned vinyl chloride among organic synthetic compounds that the factory produced, and mercuric chloride was actually used as a catalyst in the production process (Miyazawa, 1996). In addition, they noticed that vinyl chloride output growth paralleled the increase in patients (Takeuchi et al., 1960). Although focusing on the vinyl chloride process, the Kumamoto University Research Group was unable to show how inorganic mercury in the waste from producing vinyl chloride changed to organic mercury.

Leonard Kurland of the National Institute of Health (NIH) in the US visited Minamata in September 1958 and examined patients. In a subsequent article, he supported the Kumamoto University's conclusion that the etiologic agent was organic mercury in *World Neurology* and also focused on vinyl chloride production (Kurland et al., 1960). However, a local newspaper, the *Minamata Times*, published by Masao Shino, a Minamata citizen, already noted on 10 December 1959 that mercuric salt was used as a catalyst in producing acetaldehyde and suspected the relationship between the acetaldehyde production and the disease (Minamata Times, 1996). This information must have been leaked from workers inside the factory.

On 7 October 1959, Hajime Hosokawa, Director of Chisso Hospital, succeeded in inducing Minamata disease in a cat, labelled number 400, which had been given waste water from acetaldehyde production daily for 78 days (Harada, 2004). This important finding, which was not made public, clearly shows that the waste from acetaldehyde production actually contained organic mercury. If the Kumamoto University Research Group had known of this finding it could have made great progress. Instead, when Dr Hosokawa reported the cat number 400 result to the factory, the findings were kept secret and the factory prohibited further studies (Miyazawa, 1996). When allowed to restart experiments in 1960, Hosokawa found that cats given waste water from acetaldehyde production also manifested disease. However, he resigned from the factory in April 1962, without being able to make the results public.

Later in 1962, Jun Ui, a postgraduate engineering student, and Shisei Kuwabara, a photographer, visited a doctor at the Chisso factory (Mishima, 1992) and found a note concerning the results of the experiments on cats. Kuhabara photographed this evidence when the doctor was out of the room. They subsequently showed the photograph to Dr Hosokawa who acknowledged its authenticity. Ui later related these facts along with other details about Minamata disease in the monthly magazine Goka and the information played an important role in the first Minamata disease lawsuit (Tomita, 1965). Despite suffering from lung cancer, Dr Hosokawa testified in the first Minamata disease lawsuit in 1970 from his hospital bed, making two key points (Mishima, 1992): first, cat number 400 had definitely demonstrated symptoms of Minamata disease; second, his recommendation that the factory waste should not be shifted from Minamata Bay to the mouth of the Minamata River had been ignored. He died later that year.

Wastewater containing methylmercury from Chisso factory was led directly into Minamata Bay.

Photo: By Eugene Smith © Aileen M. Smith

Based on the Kumamoto University Research Group's July 1959 report, organic mercury was recognised as the etiological agent by the Minamata Food Poisoning Committee organised by MHWJ on 12 November 1959 (MFPC, 1996). However, there was no mention of the source of the contamination, Chisso factory. Indeed, before the Committee announced that organic mercury was an etiologic agent, the section chief of the MHWJ Environmental Sanitation Department told the Committee's representative not to conclude that the factory was a cause because it was not 'scientifically' proved (Miyazawa, 1996). And after the Committee had reported its opinion to the Minister of Health and Welfare, it was suddenly dissolved. No official reason was ever given.

An inter-ministerial meeting was held on the day before the Minamata Food Poisoning Committee's announcement. There, an MITIJ representative told researchers and other officers that: 'No similar patients have been observed around chemical factories with the same system as Chisso. If the operation by Chisso were causal, we would find such patients around those factories. Furthermore, the mercury used in the Chisso factory as a catalyst is inorganic. The causative agent that you have identified is organic mercury. No means by which inorganic mercury could be converted to organic mercury has been identified. We cannot accept the explanation that waste water from Chisso contains the etiologic agent of Minamata disease' (Hashimoto, 2000).

In September 1959, Chisso factory likewise refuted the organic mercury theory based on the absence of similar disease at other factories; difficulties explaining the abrupt increase in patients since 1954 and uncertainty regarding the organic chemical reaction mechanism (Minamata factory, 1996). This was despite the fact that a factory researcher had already demonstrated that organic mercury was synthesised in the production of acetaldehyde in 1951 (Arima, 1979).

The factory also claimed that it was difficult to trust the Kumamoto University Research Group because it had considered other theories (manganese, thallium or selenium) before identifying organic mercury (Minamata factory, 1996). In line with Chisso's counterargument, Raisaku Kiyoura, a professor at Tokyo Institute of Technology, claimed that mercury concentrations in Minamata Bay were not higher than those in other areas (Harada, 2004). Furthermore, Takeji Ohshima, the Executive Director of the Japanese Association of Chemical Industries, suggested that the cause might be explosives dumped into Minamata Bay by the Japanese military (Harada, 2004).

The debate suggests that Chisso factory intentionally, although inconsistently, used reductionist argumentation to postpone action. On one hand, despite the abundance of evidence that had already existed since 1956, Chisso factory contended that the only way to prove causality between its production processes and the organic mercury concentrations was to demonstrate, via a reductionist approach, the chemical mechanism linking the two. On the other, it criticised the researchers at Kumamoto University for applying a reductionist approach by considering other possible metals first. In addition, the consistency argument (relating to the absence of similar disease in other areas and abrupt increases in patient numbers) was also intentionally used to postpone action.

Recent analysis provides the following explanations for the absence of similar disease near comparable factories. First, the factor's output of acetaldehyde was the highest in Japan at that time, accounting for one third or a quarter of national production (Hashimoto, 2000). Second, methylmercury by-product per unit of acetaldehyde production was higher than at other factories due to technical improvements to increase acetaldehyde production (Hashimoto, 2000; Miyazawa, 1996). Third, the factory's proximity to the sea meant that the chloride ion concentration of industrial water was high, which changed the methylmercury byproduct to volatile methylmercuric chloride (Hashimoto, 2000; Nishimura and Okamoto, 2001). It was then discharged when the acetaldehyde was purified by distillation.

Organic mercury represented the most credible explanation but was resisted by Chisso, the chemical industry and the MITIJ. The MITIJ therefore ordered Chisso to return the drainage outfall of acetaldehyde production to Minamata Bay (from the Minamata River) and to install wastewater treatment equipment within the year (Harada, 2004). The factory therefore established a purifying system for the contaminated water in December 1959 (Arima, 1979; Harada, 2004).

Most residents believed that the discharge of the etiological agent would soon cease. However, the system installed, as should have been known by Chisso at the time of installation, was completely ineffective at removing methylmercury (Irukayama, 1969). It was installed only to give the appearance of action by the company and researchers at Kumamoto University were deceived by being given a fake sample from Chisso factory (Miyazawa, 1996). Unsurprisingly, mercury concentration in fish and

shellfish in Minamata Bay failed to decline after the system was installed (Irukayama, 1969). To make matters worse, the effluents also continued to be discharged into Shiranui Sea (Irukayama, 1969). As a result, residents not only in Minamata Bay but also in the other villages around Shiranui Sea continued to be exposed.

5.2.4 Detecting the organic mercury production process and social recognition of Minamata disease (1960–1963)

Although researchers were now satisfied that organic mercury was the etiologic agent of Minamata disease, no steps were taken to control the poisoning. Researchers concentrated on identifying organic mercury in organisms or finding the mechanism by which it was produced. On 14 February 1960, Makio Uchida, a professor at Kumamoto University, extracted organic mercury in a shellfish in Minamata Bay (Harada, 2004; Uchida et al., 1960). Subsequently, researchers at Kumamoto University made further important findings: confirming that short-chain organic mercury was toxic, extracting methylmercury sulfide from shellfish, and inducing Minamata disease in cats and mice using the substance (Harada, 2004; Sebe et al., 1961; Uchida et al., 1960).

In February 1960, the Minamata Disease General Investigation Committee was established to replace the dissolved MHWJ committee and research Minamata disease (MDGIC, 1996). Many of the discussions in the Committee centred on possible objections to the organic mercury theory and served only to obscure the theory (George, 2001). Its last meeting was held in March 1961. Finally, in March 1962, Fisheries Agency abandoned research regarding Minamata disease (Arima, 1979). After that, no research activities were conducted by government agencies until 1968.

In April 1960, the Minamata Disease Research Council, known as the 'Tamiya Committee', was established (Miyazawa, 1996). Its chair was Takeo Tamiya, President of the Japanese Medical Association, and all members were from universities in Tokyo. Primarily sponsored by the Chisso factory, the Tamiya Committee attempted to obscure the organic mercury theory. The Committee wished to involve Kumamoto University but Kansuke Sera, Dean of Kumamoto University School of Medicine, refused the request (George, 2001), which was a remarkable and noteworthy act.

Researchers on the supposedly 'authoritative' Tamiya Committee disputed Kumamoto University's organic mercury theory by arguing that other factors were responsible (Harada, 2004). On 13 April 1960, Raisaku Kiyoura published a newspaper article promoting his theory that a group of organic chemicals called amines were responsible ([4]). He claimed that amines, not mercury, were detected in shellfish that caused Minamata disease in cats. Any detailed examination would have demonstrated the dubious medical validity of that counter-theory but the mass media were enthusiastic (Harada, 2004).

The next year, Kikuji Tokita, a professor of Toho University, proposed that eating rotten fish was the cause and the etiologic agent was again suggested to be amines. However, people in Minamata, despite their poverty, were able to eat as much fresh fish as they wanted. Anyone who visited Minamata and observed the life of its people would understand immediately that his theory was wrong. In his paper, the name of Chisso factory, Takeji Ohshima (who had suggested that dumped munitions were responsible) and Raisaku Kiyoura were listed in the acknowledgments. George (2001) notes the emphasis placed on the 'line of attack-that scientists from the "centre" could be trusted over those from "hick" universities on the periphery'. Importantly, all researchers recognised that fish were a cause.

During this period, researchers at the Department of Internal Medicine at Kumamoto University School of Medicine conducted a large investigation to locate unidentified Minamata disease sufferers and determine whether Minamata disease occurred in a chronic form and, if so, what its diagnostic features were (Tokuomi et al., 1962). They targeted 1 831 residents in affected areas, of whom 1 152 (62.9 %) participated, and used a questionnaire to identify participants who needed further physical examination. The study identified 131 participants with neurological signs similar to Minamata disease (Kumamoto Nichinichi Shinbun, 1962), although only 24 of these had severe symptoms. Finally, the Screening Council for Minamata Disease Patients (described in Section 5.4.2) recognised two of cases as having Minamata disease (Miyazawa, 1996 and 2007).

Although this investigation could be seen as an important step towards fully describing Minamata disease (e.g. the nature, threshold, frequency and severity of symptoms; the scale of poisoning; and the prognosis) in practice the investigation could not go far beyond the boundaries of earlier

([4]) An amine is any derivative of ammonia in which one or more hydrogen atoms are replaced by alkyl or aryl groups.

epidemiological studies. This is probably because researchers at Kumamoto University, keen to protect the organic mercury theory from a steady flow of criticism at that time, focused on the typical and severe cases of organic mercury (Hunter-Russell syndrome) (Miyazawa, 2007), even though this was contrary to the primary object of their investigation. They did not use non-exposed areas as controls to compare prevalence and they did not follow up with the 131 participants identified as having similar symptoms to Minamata disease. Finally, when publishing their findings in March 1962 (Tokuomi et al., 1962), the researchers gave the impression that Minamata disease was no longer a problem, observing that 'Minamata disease seems to have terminated at last.'

Meanwhile, in 1960 the Kumamoto Prefecture Institute for Health Research investigated the mercury concentration in hair samples from 1 645 healthy fishermen from around Shiranui Sea (Doi and Matsushima, 1996; Matsushima and Mizoguchi, 1996). It was the first large survey using hair samples. The distribution of a high concentration (0–920 ppm) of mercury among the hair samples indicated that the contamination had spread throughout entire Shiranui Sea. The mercury content in Minamata was the highest (a median of 30 ppm) but the mercury content in Goshonoura (median 21.5 ppm) on the other side of the Shiranui Sea was also about 10 times higher than that of residents in the non-exposed city of Kumamoto (median 2.1 ppm) (Doi and Matsushima, 1996; Matsushima and Mizoguchi, 1996; Ninomiya et al., 2005). Among 199 residents examined in Minamata, 61 residents (30.7 %) had hair mercury concentration greater than 50 ppm, while even in Goshonoura 153 residents (13.2 %) among 1 160 examined had those levels of mercury.

Two other investigations were conducted up to 1962 (Doi et al., 1996). Although the investigators claimed that further follow-up studies were needed because the contamination source was not removed and the mercury concentration in hair samples was high, Kumamoto Prefecture decided to stop the investigation in 1962 (Miyazawa, 1996). Furthermore, the health status of the fishermen who provided hair samples was never followed up and the fishermen were never informed of the mercury concentration results.

After Masachika Kutsuna replaced Kansuke Sera as Dean in April 1961, Kumamoto University School of Medicine adopted a conciliatory attitude to Chisso factory (Miyazawa, 1996). It joined the Tamiya Committee and began to receive research funding from Chisso factory and the Tamiya Committee. Indeed, when Kumamoto University published a volume of their research reports in 1966, Chisso factory was listed in the acknowledgments (MDRG, 1966). From that time, Minamata disease became a sensitive problem in Kumamoto University. When a local news paper reported that Prof. Irukayama extracted methylmercury chloride from the sludge of the factory (Kumamoto Nichinichi Shinbun, 1963), Dean Kutsuna reprimanded him and called Chisso factory to apologise for the news (Miyazawa, 1996). Later on, an instruction was handed down in the School of Medicine: 'you can do experimental research about Minamata disease but do not conduct clinical research.' It was said that clinical research 'is not research but rather work conducted by social activist or Prefecture Government' (Harada, 2004) because if researchers included human beings, they naturally became involved in various social problems surrounding Minamata disease.

Finally in 1962, Katsuro Irukayama, a professor at Kumamoto University School of Medicine succeeded in extracting methylmercury chloride from the sludge of the acetaldehyde production process in the factory (Irukayama et al., 1962). Although it was not disclosed, Chisso factory laboratory also extracted methylmercury chloride from the sludge (Miyazawa, 1996). This showed that methylmercury was a by-product of acetaldehyde production and present in discharges from the factory. However, the Food Sanitation Act was not applied as a result of these findings, nor was the factory regulated in any other manner.

Important scientific findings continued. In addition to the success extracting methylmercury chloride from the sludge of the factory, in 1962 an unusual occurrence of cerebral palsy infants was diagnosed as resulting from methylmercury intoxication during fetal life (Harada, 2004). However, the public's attention began to shift away from Minamata Disease. Chisso factory paid 'mimaikin' ('sympathy money') to patients in 1959 meaning that many, including researchers, believed that the issue was settled. Furthermore, from 1962 to 1963, there was a big dispute at Chisso over workers' pay (Harada, 2004). The issue of Minamata disease began to be forgotten except among sufferers and their families.

5.2.5 Niigata Minamata disease and proof of the causal relationship (1964–1968)

Although the source of contamination, the causal food, the etiologic agent and the process creating the methylmercury had all been identied, the government

did not regulate fish consumption or factory waste at Chisso. In January 1965, similar methylmercury food poisoning occurred in Niigata, causing 'Niigata Minamata disease' (Niigata Prefecture, 2007; Saito, 2009). The factory responsible (Showa Denko) operated in the same way as Chisso in Minamata, with methylmercury being discharged during acetaldehyde production. Although the damage was less than Minamata, over 1 500 individuals (Niigata Prefecture, 2007) were needlessly affected. Once more, cats started dancing and dying from madness in Niigata as a harbinger to the human consequences that were to follow (Harada, 2004).

From the beginning, MITIJ and Chisso had disputed that the factory was the cause of Minamata disease. A compelling argument, in their eyes, had been the observation that 'No similar patients have been observed around other production plants with the same system as Chisso. If the operation by Chisso was causal, we would find such patients around these other factories' (Hashimoto, 2000). At that time, Chisso was by far the largest producer of acetaldehyde, and Showa Denko was the second most important (Harada, 2004). The appearance of Minamata disease in the vicinity of Showa Denko was a powerful refutation of their argument.

A legal case relating to Niigata Minamata disease went on trial in 1967 and on 26 September 1968 the government of Japan finally agreed that there was the causal relationship between wastewater from Chisso (and Showa Denko) and Minamata disease (MHWJ, 1996c). By then, however, this admission was immaterial as acetaldehyde was no longer necessary and production had stopped by May 1968 (Arima, 1979). Twelve years had passed since the institution and food contaminant had been identified. In total, 488 tonnes of mercury were discharged into the sea from 1932 to 1968 (Miyazawa, 1996).

5.3 Congenital Minamata disease: intrauterine methylmercury poisoning

Minamata disease can be considered a typical example of industrial pollution (Ui, 1968) for several reasons. First is the manner of the outbreak. Minamata disease is a form of food poisoning (and indeed carried through the food chain) as a result of environmental pollution. Second, it is a classic example of how decisions supposedly based on factual judgements were influenced by political, financial, legal and even psychological factors (such as hierarchies within society, within and between scientific disciplines and between different wings of government, and supposed 'loss of face' in admitting error). Corruption also played a role.

However there is another reason why it will long be remembered: the discovery of congenital Minamata disease. Before congenital Minamata disease was proven, it was believed that the womb protected the foetus from poisons. This was the first clear-cut case of chemical poisoning transmitted through the placenta to the foetus. A paragraph from Harada (2005) conveys the normal attitudes towards congenital Minamata disease at that time (1961):

> When conducting a survey in the area of frequent outbreak of Minamata disease, I came upon two brothers on a veranda. Their symptoms were exactly the same; so I assumed that they must both have Minamata disease. However, their mother said, 'The 9 year-old contracted Minamata disease when he was 3 years and 6 months, but the 5 year-old has cerebral palsy.' 'Why?' I asked, to which she responded, 'The younger one has never eaten fish; he was born this way, so it's not Minamata disease.' I was convinced right away. This is because we used to believe at that time that the placenta would not let poisons pass though. Then the mother added, 'That's what doctors say. Both my husband and this older child got Minamata disease. My husband died in 1954. I ate the same fish. At the time the younger boy was inside of me. I think the reason I have few symptoms of the disease is because this child absorbed all the mercury that I ate.' At the time I thought this was just the fancy of an amateur who knew nothing about medical science. But time proved that she was correct. Her words were prophetic in speaking of the suffering of the foetus because after that time we had many experiences of congenital cases.

Many infants born after 1955 showed symptoms resembling those of cerebral palsy in the affected areas (Kitamura et al., 1959 and 1960a). Shoji Kitamura mentioned in 1959, 'It is possible that the substance causing the poisoning was transferred to the infants through the placenta or mother's milk, producing symptoms similar to those of Minamata disease' (Kitamura et al., 1959). Careful clinical and epidemiological studies were conducted (Harada, 1978). All the patients displayed similar symptoms (Harada, 2005) including mental retardation, disturbed coordination, deformities of limbs, poor reflexes, poor nutrition and impaired growth. Most were hyperactive, suffered from muscular spasms and uncontrollable slow writhing, had squints,

One of the symptoms of Minamata disease is deformed limbs.

Photo: By Eugene Smith © Aileen M. Smith

produced excess saliva resulting in drooling, and were subject to sudden mood changes.

Epidemiologically, the patients were coincident with Minamata disease both in timing and location (Harada, 1964). Their mothers consumed a large amount of fish and exhibited mild symptoms of Minamata disease. Furthermore, 13 infants (6.9 %) among 188 infants born during the period 1955–1958 suffered from severe cerebral-palsy-like symptoms in the three most heavily contaminated areas (Harada, 1964). Since the overall incidence of cerebral palsy in Japan was 0.2–0.6 % at that time, this clearly showed that this incidence of cerebral-palsy-like infants (congenital Minamata disease patients) was very high (Harada, 1964).

Despite these clinical and epidemiological features, it took a long time for congenital Minamata disease to be accepted as a fact. Mothers of children seeking assistance with medical costs were told that they would only be helped once some children had died, had been autopsied, and the nature of the illness had been confirmed (Harada, 2004). Finally, two autopsies of infants confirmed methylmercury intoxication during foetal life. Then, in December 1962, 17 patients were officially diagnosed with congenital Minamata disease (Miyazawa, 1996). Later research revealed that the disease existed in a broader region, and 66 cases including 13 deaths were identified by Harada (2005 and 2007). However, no other epidemiological studies to investigate the existence of congenital Minamata disease have ever been conducted.

One reason why it took five to eight years to confirm congenital Minamata disease is that researchers had never previously seen a case of poisoning through the placenta (Harada, 2005). In addition, researchers who became convinced that the disease was being transferred from mother to foetus were told (for example by the Screening Council for Minamata Disease Patients and city officials) that they had no proof (Harada, 2005). Because organic mercury was only recognised as the etiologic agent in 1959, mercury levels in hair or umbilical cord blood were not previously measured at birth. However, in 1968 Masazumi Harada realised that the Japanese tradition of preserving the umbilical cord might make it possible to measure methylmercury concentrations in preserved umbilical cords as an indicator of foetal exposure. He collected umbilical cords among the residents around Shiranui Sea and was able to demonstrate a correlation between acetaldehyde production in Chisso factory and the concentration of methylmercury in umbilical cords (Figure 5.1) (Nishigaki and Harada, 1975; Yorifuji et al., 2009a). This supported the hypothesis that methylmercury affected foetuses in the uterus via the placenta.

A continuing problem relating to intrauterine exposure to methylmercury is the effects of low to moderate exposure (Harada and Tajiri, 2009), i.e. exposure that is below the level that produces the full effects but is nevertheless debilitating. While symptoms of congenital Minamata disease can be similar to cerebral palsy, some individuals exposed to a high umbilical cord mercury level did not show exactly these symptoms — and were therefore disregarded — despite showing other mental disabilities, behavioural anomalies or other cerebral dysfunctions. They were missed because of the failure to implement proactive epidemiological investigation targeting residents exposed to methylmercury via the uterus.

It is well established that methylmercury concentrations in congenital Minamata disease and Minamata disease patients are higher than in healthy individuals. But we now know that methylmercury concentrations in other mentally retarded groups are also higher than in healthy people (Harada et al., 1999). Further investigation of mental retardation cases revealed clumsiness in finger movement and other light motor dysfunctions. The ongoing developments regarding the effects of low to moderate exposure among residents underline the continuing failure to investigate the consequences of Minamata thoroughly. Further follow-up

studies are needed targeting congenital Minamata disease patients and more moderate exposure to methylmercury in the uterus. Nothing can reverse the history of exposure but much could and should be done to mitigate and learn from its effects.

5.4 Chaos implementing the Minamata disease accreditation system (1968 to present)

After the Japanese government accepted the causal relationship between Chisso factory and Minamata disease in 1968, attention shifted to the 'accreditation' of the disease in individual patients, in order to determine compensation claims. The payment of compensation can be grouped into four phases. First, Chisso paid 'sympathy money' without accepting responsibility. Second, 1971 witnessed an early application of the precautionary principle, when accreditation criteria were relaxed and applications for compensation soared. Third, the government introduced far harsher accreditation criteria in 1977. Fourth, a period of 'political settlements' took place from 1995/96 until the present, with the government and Chisso factory attempting to settle the conflict by paying lump sums (not as compensation) without changing the strict criteria or recognising affected individuals as official patients.

All of this was done without formally defining Minamata disease and legal cases still continue because the geographic and temporal boundaries for claimants still lack an agreed evidential basis.

5.4.1 Accreditation system

As of 2013, the Minamata disease accreditation system remains based on Japan's Pollution-Related Health Damage Compensation Act. This involves passive assessment based on applications by patients to become accredited (Minamata Disease Museum, 2007; Ministry of Environment, 2006), rather than the active surveillance system based on the Food Sanitation Act, described earlier.

The Judgment Committee for Minamata Disease Accreditation (an advisory body to the Governor of the Prefecture) determines whether 'the applicant is a Minamata disease patient' based on the results of a medical examination (Minamata Disease Museum, 2007; Ministry of Environment, 2006). Committee meetings are held in the cities of Kumamoto and Kagoshima, nearly 70 km from Minamata, and committee members do not directly examine the applicants, regardless of their proximity. The Committee consists largely of neurologists, with a few pathologists, ophthalmologists (eye disorder experts) or otolaryngologists (ear and throat experts). Having considered a case, the Committee makes an assessment by unanimous decision and submits it to the Governor of the Prefecture. If the judgment is positive then the Governor accredits the applicant as an officially recognised Minamata disease patient. Compensation is not available without accreditation, which can take a long time. For example one patient accreditation took 25 years (Miyazawa, 1996), imposing a significant burden on the applicant.

The Environmental Agency of Japan (EAJ), a predecessor of the Ministry of Environment of Japan, determined the official position that patients with a 'probability of 50 % or more' of having the disease are to be accredited as Minamata disease patients (Ministry of Environment, 2006). As such, a quantitative approach is supposed to be applied. In practice, however, the Committee for Accreditation uses a qualitative diagnostic method, based on whether the symptoms match those documented for the 'Hunter-Russell' syndrome.

As described below, the criteria for accreditation have been revised several times since their introduction in 1959 (Yorifuji et al., 2013), with profound implications for those affected by Minamata disease.

5.4.2 History of the accreditation system

Prior to 1969, the initial disease accreditation system, the 'Screening Council for Minamata Disease Patients', was used to identify patients who deserved low 'mimaikin' ('sympathy money') payments from Chisso factory (Harada, 2004; Minamata Disease Museum, 2007). This was not considered compensation, nor was it done based on any law because Chisso factory insisted that it was not proven that it was the cause of Minamata disease (Miyazawa, 2007).

The settlements, in effect decided by Chisso factory, were very small. A death resulted in a lump sum payment of JPY 300 000 (about EUR 2 900 today) while affected adults received JPY 100 000 (about EUR 960) annually and children JPY 30 000 (about EUR 290) annually (Minamata Disease Museum, 2007). The contract provided that 'the patients relinquish their claim to further compensation even if it is decided in the future that Minamata disease is caused by Chisso's effluents' (Harada, 2004). In its judgement at the First Minamata Disease Lawsuit in 1973, the court nullified these agreements as a

breach of the common good (Minamata Disease Museum, 2007).

By 1969, eighty-nine Minamata disease patients, excluding those with congenital Minamata disease, had been accredited through this system (Minamata Disease Museum, 2007). At that time, patients suffered not only from the disease itself but also faced discrimination from Minamata citizens in part because accredited patients could obtain compensation money. This climate in Minamata deterred residents from seeking accreditation. Instead, some sought to hide their neurological symptoms (Miyazawa, 2007).

In the 1960s, large-scale incidences of health effects from environmental pollution were identified elsewhere in Japan, for example, methylmercury poisoning in Niigata (Niigata Prefecture, 2007), air pollution in urban areas (Yoshida et al., 1966), cadmium poisoning in Toyama (Osawa et al., 2001), and arsenic poisoning in Miyazaki (Tsuchiya, 1977). This lead to a change in the public mood. The Japanese government was forced to take active measures to prevent further cases, and make better provision for patient support and compensation. In 1969, the Act on Special Measures for Pollution-Related Health Damage Relief (later changed to the Pollution-Related Health Damage Compensation Act) was created. It came into effect the following year (Minamata Disease Museum, 2007). Subsequently, the EAJ was established in July 1971.

As a result of the Act, the Screening Council for Minamata Disease Patients was replaced by the Judgment Committee for Minamata Disease Accreditation (hereafter, 'Committee for Accreditation') at the end of 1969 and this continues to be the responsible body (Minamata Disease Museum, 2007).

On 7 August, 1971, the 'Administrative Vice Director of the EAJ Notice' was published, marking the first real policy change since 1956 (JSPN, 1997). This Notice specified that if it appeared 'clear that a patient had been affected by the consumption of fish and shellfish containing organic mercury, the cause of [his/her neurological signs (characteristic of methylmercury poisoning)] should be presumed to be Minamata disease, even if other causes were conceivable' (George, 2001). The approach reflected the usual thinking with respect to food poisoning and was not dissimilar to the precautionary principle concept, as developed in Europe in the following decade. The Notice listed neurological signs such as constriction of the visual field, ataxia (loss of bodily coordination), hearing loss and paresthesia (a disabling tingling sensation, 'pins and needles', on both sides of the body) and did not require combinations of these neurological signs for Minamata disease to be confirmed.

On 20 March 1973, the Kumamoto District Court ordered Chisso factory to pay compensation to Minamata disease patients engaged in a lawsuit against the company (Minamata Disease Museum, 2007). The patients then signed a compensation agreement with Chisso (Minamata Disease Museum, 2007). This resulted in dramatic increase of the number of accreditation applications.

Meanwhile, in 1971, the Department of Neuropsychiatry at Kumamoto University School of Medicine undertook the first and largest cross-sectional population-based investigation to evaluate the prevalence of neurological signs of Minamata disease among local residents (Tatetsu et al., 1972). Leonard Kurland had first suggested this study to the Japanese Society of Neurology but the proposal was rejected and the Department of Internal Medicine therefore did not cooperate in the study.

In the study, three areas were selected for investigation in Kumamoto Prefecture: Minamata (a high-exposure area), Goshonoura (a medium-exposure area) and Ariake (a low-exposure, reference area). The findings demonstrated the severe effects of methylmercury on residents in Minamata and even in Goshonoura. It provoked debate about the 'Third Minamata disease' (following those experienced in Minamata and Niigata) since even in the reference area there were residents with neurological signs of Minamata disease (Miyazawa, 1996), although the prevalence was not so high.

The analysis was highly plausible because the Ariake and Minamata areas are connected by sea (Map 5.1) and fishermen often went to the Shiranui Sea to catch fish. Unfortunately, the findings were made public before the investigation was complete and created a sensation. People stopped buying fish or shellfish caught in Ariake area, causing Ariake's fishermen to protest against the Prefecture and Kumamoto University. A doctor at Kyushu University diagnosed those with neurological symptoms in Ariake as not having the disease, which caused distrust of Kumamoto University researchers. These pressures meant that the research was terminated after only two thirds of the programme had been completed.

Recent publications in international journals (Yorifuji et al., 2008, 2009b, 2010, 2011 and in press) have

shown, however, that even the incomplete findings of the Kumamoto University investigation were valuable and should have been more fully utilised. They fill out the details of Minamata disease: the kinds of symptoms, their frequency and thresholds and the types of residents affected. Unfortunately, this information has never been used for diagnosis or compensation from the 1970s to the present.

The third change in EAJ policy came in 1977 when, following the sensational news of the 'Third Minamata disease', and also a dramatic increase of accreditation applications, the 'precautionary' approach of the 1971 EAJ Notice, based on the notion of food poisoning, was reversed. A new and more rigid set of accreditation criteria for Minamata disease the '1977 Criteria' were established (Minamata Disease Museum, 2007; Ministry of Environment, 2006; Yorifuji et al., 2013) and remain in force today. Subsequently, there was a rise in the number of patients who had methylmercury-related symptoms but were not formally accredited. This is because the 1977 Criteria once more require a combination of neurological signs (Minamata Disease Medical Research Group, 1995, JSPN, 1997). They also provide that in addition to taking into account a person's exposure history, paraesthesia would now be regarded as a necessary but insufficient criterion for accreditation. Unlike the 1971 Criteria, the occurrence of paraesthesia 'alone' would not result in accreditation.

In 1978, it was decided that Kumamoto Prefecture should issue debt to support Chisso factory in paying compensation money (Ministry of Environment, 2006). This meant that the authorities now had a potential conflict of interest between their duty to the patients and their financial situation. It became increasingly apparent that the accreditation system was an important defensive barrier — not only for Chisso but also for the government (Miyazawa, 2007).

The 1977 Criteria became a continuing source of dispute. In August 1985 the Fukuoka High Court decided that the criteria for accreditation should be relaxed again, allowing more people to qualify (Minamata Disease Museum, 2007). In response, in October 1985, the EAJ summoned eight medical specialists to reconsider the 1977 Criteria (Miyazawa, 2007). Their 'expert opinion' stated that the Criteria remained 'valid' and once more asserted that it was not certain that paraesthesia occurred in isolation in Minamata disease and should not, therefore, be used to accredit patients (JSPN, 1997). Only neurologists were present at the meeting, which was both closed and brief (seven hours). The meeting minutes were not published and no medical evidence was given in public to support their conclusion. It seems inconceivable that they would not have been aware of the prominent research of Bakir et al. (1973), which provides evidence that paresthesia can occur in isolation following methylmercury exposure (JSPN, 1999).

By the early 1990s many accreditation applications (including new applicants and those who had reapplied) and lawsuits remained outstanding, in part because the 1977 Criteria were strict. Indeed, of the 944 patients who satisfied the diagnostic criteria and should have been accredited according to the 1977 Criteria, only 205 patients were in fact accredited by 1981 (Miyai, 1997). Even by 1992, only about one third of the qualified applicants had been accredited (316/944) (Miyai, 1999).

The EAJ therefore asked the Japanese Central Council for Environmental Pollution Control, an advisory body to the EAJ Director, to consider the issue in 1991 (Ministry of Environment, 2006). A 14 member Minamata disease working group was set up, comprising nine members with medical backgrounds and five with legal expertise. Based on the working group's advice, the Council stated that its accreditation of Minamata disease was in accordance with the 1985 'expert opinion' and proposed a medical care project to support exposed residents who had signs of the disease but were not accredited as Minamata disease patients.

In 1995–1996, under a condition that there was no liability on government, a reconciliation (the so-called 'first political solution') was reached based on the Central Council's recognition and the medical care project. According to the reconciliation, instead of issuing further Minamata disease accreditation, Chisso would make a lump sum payment to patients with methylmercury-related symptoms living in the exposed areas but these patients must withdraw any legal action or claim against Chisso (McCurry, 2006). In this reconciliation, about 10 000 patients received lump sum payments of JPY 2.6 million (about EUR 25 000 today) as relief money (not compensation) because neither the government nor Chisso admitted liability and they were not formally recognised as 'Minamata disease patients' (Miyazawa, 2007). Accordingly, this situation could be considered as repeating the 'mimaikin' ('sympathy money') payments in 1959. The number of accreditation applicants fell to zero but one legal action continued in Osaka, where some residents born in Minamata had previously moved.

By this time the Japanese Society of Psychiatry and Neurology (JSPN) — an independent academic society consisting mainly of psychiatrists and neurologists — was becoming increasingly involved. In 1998, it examined whether the medical specialists in the EAJ commission in 1975 had used any medical evidence when creating the 1977 Criteria. It concluded that they did not. Moreover, the JSPN judged the 1977 criteria to be medically invalid based on an evaluation of the data gathered in the Kumamoto University study of 1971 (JSPN, 1998). Subsequently, in a 1999 review, JSPN strongly criticised the 1985 'expert opinion', stating that there was no scientific evidence for the 1985 'expert opinion'; that the 'experts' were selected to justify the 1977 Criteria and the position of the EAJ; and that the 'experts' were guilty of pandering to the government's desires (JSPN, 1999).

Throughout the history of Minamata disease, key decisions have been characterised by very little transparency and much secrecy, making it hard to evaluate their reasoning. In 2003, after the first political settlement, JSPN (2003) analysed the minutes of the Central Committee working group's discussions in 1991, which were disclosed under the Access to Government Information Act ([5]). It concluded that the working group members did not have the medical evidence to support the 1977 criteria; that the 1985 'expert opinion' was not a medical assessment but a government opinion; and that the meeting minutes made it evident that the working group's discussion had been conducted with the sole aim of producing an opinion that complied with the EAJ's view. These facts had, until then, been hidden from the public.

In October 2004, the Japanese Supreme Court decided on the case involving Osaka residents, confirming the liability of the national and Kumamoto Prefecture governments for damage caused by methylmercury poisoning in the Minamata area (McCurry, 2006; Minamata Disease Museum, 2007; Nagashima, 2005). Like the Fukuoka High Court in its 1985 decision, the Japanese Supreme Court also ruled that the 1977 criteria should be relaxed (McCurry, 2006; Nagashima, 2005). Remarkably, the EAJ has still not changed its attitude on the criteria. As a result of the Court's ruling, the number of accreditation applicants began to grow again, exceeding 6 000 by 2008 and 8 000 in 2010) (Minamata Disease Museum, 2010).

In 2009, an Act on Special Measures (the so-called 'second political solution') was passed without changing the strict 1977 criteria. It was determined that Chisso should pay lump sums of JPY 2.1 million (about EUR 20 000) to patients who at least have paresthesia and who had lived in defined affected areas for at least one year during a defined period of time (Kumamoto Prefecture, 2010). Similar to the first reconciliation in 1995–1996, the lump sum money is not compensation but relief money because neither the government nor Chisso admits liability and the patients are not formally recognised as 'Minamata disease patients'.

It is expected that most of the applicants involved in litigation will withdraw their actions. Residents who have never applied for compensation but have neurological symptoms are also able to apply for lump sums. However, the outcome is perplexing. While 2 273 patients were officially recognised as Minamata disease patients in the affected prefectures, there are also at least several tens of thousands of exposed patients with neurological signs characteristic of methylmercury poisoning who have not been formally recognised as Minamata disease patients and not properly compensated.

Despite the enactment of the Act on Special Measures, several lawsuits are still under way because the Act defined the affected area and time period without investigating or defining evidential criteria. It is known that exposed residents with neurological signs characteristic of methylmercury poisoning are observed in areas other than those defined in the Act (Kumamoto Nichinichi Shinbun, 2010a). The approach used in the Act has shortcomings. First, it is inappropriate to define 'areas' based on the notion of food poisoning; instead, exposed persons with relevant symptoms should be counted as patients. Furthermore, the problem will persist because individuals who were exposed in the uterus but were not severely enough affected to be recognised as 'congenital' Minamata disease patients are not covered by the Act.

5.4.3 Criminal charges

One criminal case was put forward against Chisso in 1975 although a number of civil trials were raised against the factory. The report by Hunter et al. (1940) was the best known in Japan, although the danger of using organic mercury in the production had been common knowledge long before 7 May 1932, when Chisso began to discharge effluents containing organic mercury

([5]) The Aarhus Convention and 'right to know' laws have made public access to data and decision-making an issue of critical importance.

into Minamata Bay (Iriguchi 2012). However, the accused stressed that toxicity of methylmercury was not well known before 1956 when the first patient was notified by the Minamata Public Health Center. In 1979, the Kumamoto District Court sentenced the ex-president and the ex-factory-head to two years in prison with three years suspension of sentence. The Japanese Supreme Court accepted the court's ruling as final in 1988.

5.5 What are the lessons of the Minamata disease story?

5.5.1 Medical, scientific and public health lessons

Respond to the signals of sentinel wildlife
As a result of the pollution route, at Minamata harm to wildlife was observed before human harm. Fish and cats died strangely before the first patients were observed in Minamata and surrounding areas, and the same pattern occurred at Niigata. Subsequently, those who lived and worked close to nature and who ate the local fish were the first to suffer from pollution. The earliest cases identified lived close enough to the sea that they could fish from their windows. They were people who lived at one with nature.

This suggests that, as a general rule, when wildlife impacts are observed we should ensure that we understand the epidemiology, identify the source of the problem and take action to prevent human suffering. Minamata showed that effective action (preventing the discharge of pollution) was possible even before the first patients were notified. Early actions are justified by our responsibility to protect the environment — but they can also avoid subsequent harm to humans.

Prevention is possible and essential
As Hajime Hosokawa (Director of Chisso hospital) pointed out 'prevention is far more important than relief.' (Harada, 2004). It was already known in 1921 that organic mercury was synthesised in the production of acetaldehyde (Ishihara, 2002; Vogt and Nieuwland, 1921). And a researcher at Chisso factory had demonstrated that organic mercury was synthesised in the production of acetaldehyde in 1951 (Arima, 1979). Furthermore, intoxication due to occupational exposure to organic mercury was reported in the 1930s in Europe (Ishihara, 2002; Koelsch, 1937; Zangger, 1930). While these reports were published in Europe, especially in Germany (where Chisso had strong links since the 1920s), this is no excuse for a diligent company to be unaware of such important risks. Prevention was possible before the hideous consequences were first identified in 1956.

Early epidemiological studies are valuable
Early epidemiological studies, 'good enough' for their purpose can play a key role in preventing and minimising future harm. The Kumamoto University research group's early study in 1956 demonstrated that eating fish caught in Minamata Bay was a cause of harm and this conclusion has never changed. Minamata is a classic example of how spurious demands for more precision with respect to the cause of harm resulted in unnecessary delay and continuing exposure ('analysis by paralysis'). It was three years before the etiologic agent was found and six years before the mechanism by which methylmercury was produced was (re)discovered. As an important principle, this shows that prompt countermeasures should be conducted when the cause is identified and should not be postponed until an etiological agent or the biological mechanism of action is identified.

In-depth epidemiological studies are also valuable
While early epidemiological studies should have been heeded, far more could have been done to reduce harm if there had been more early epidemiological effort focusing on the features of the disease (such as the threshold, frequency and severity of symptoms; the scale of poisoning; and the prognosis). The first systematic epidemiological study of the features of the disease was not conducted until 1971. An investigation by the Department of Internal Medicine in 1960 only focused on the severest cases and did not follow up with the participants. Similarly the Kumamoto Prefecture Institute for Health Research's investigation of mercury concentrations in 1 645 healthy fishermen in 1960 was not able to follow up the participants. Follow-up studies would have revealed the developments of neurological symptoms or the dose-response relationship between exposure and symptoms.

Instead, the initial emphasis was placed on clinical manifestation (Hunter-Russell syndrome) and became bogged down in legal dispute about what was and was not Minamata disease. However, after organic mercury or fish was identified as a cause, causal criteria (i.e. cause and disease) should have been used in subsequent epidemiological studies. They should also have been used from the outset to certify patients and determine entitlement to compensation.

Demands for excessive levels of scientific proof can exacerbate harm
The history of Minamata disease provides many examples of spurious obstacles and inappropriate

burdens of proof, which prevented speedy and effective action. The demand for high levels of scientific proof (i.e. 'clear evidence' or 'beyond the reasonable doubt') was used as spurious cover, allowing Chisso to delay the search for what turned out to be a simple alternative production technology that avoided methylmercury pollution. Epidemiology demonstrated that poisoning was caused by contaminated fish and the factory discharge in 1956. This was unintentionally confirmed when the drainage route was altered in 1958 causing new victims in the new discharge area. There was a tendency for many stakeholders to accept that high evidential burden was required to justify taking preventative action.

Be wary of deliberately manufactured doubt
Regulators (and others) should be attuned to 'manufacturing of doubt' by those with sufficient means and an incentive to maintain the status quo. At various points, alternative explanations arose for the harm, such as metals, dumped explosives or amines in rotten fish and these were exploited to the full by Chisso and its supporters in government.

Of course, a plurality of viewpoints is essential for scientific analysis. Indeed, during the early stages of the disaster various metals were considered as possible causes but these were dropped when the evidence did not stand up to scrutiny. Ironically, this openness to consider alternative explanations was then used by Chisso to criticise the researchers when they concluded that methymercury was the likely cause.

Several characteristics distinguish those manufacturing doubt, often by proxy, from those promoting genuine open debate. Often they demand high levels of proof for results that demand action from the vested interest but accept low levels of proof (or standards of analysis) for their alternative hypothesis, which may be the object of criticism from scientific peers. They also fail to consider the pros and cons of alternative courses of action judged from the perspective of society as a whole and that of the wider environment.

Look beneath the tip of the iceberg
Throughout the history of Minamata disease, researchers have consistently discovered more subtle effects, at lower exposure concentrations. However neurologists, who occupied a dominant position in the process ([6]), became fixated on qualitative diagnostic method and a set of symptoms (Hunter-Russell syndrome), which were used to determine the disease's presence. This insistence restricted greater understanding of the disease in all its manifestations.

The legalistic approach to accreditation and compensation compounded the problems, encouraging constant premature attempts to define formally what Minamata disease is and is not. A number of scientists involved in the compensation process ended up having their reputations damaged by defending rigid criteria that became increasingly indefensible as time progressed.

Congenital Minamata disease
The uterus is part of the environment: to pollute the exterior environment is to pollute the uterus and thereby to pollute future life (Harada, 2005). Contrary to previous assumptions, we now know that the biological barrier of the uterus (and also the blood brain barrier) cannot be assumed to prevent the transfer of substances not found in the natural world or high exposure to substances that are naturally of low concentrations. The former are synthetic chemical compounds created by man. The latter are things we dig up from the earth, concentrate, process and use in great quantities.

On a practical note, the Minamata disaster has demonstrated the utility of umbilical cords for assessing pollution and shows that simple methods of preservation of biological tissue are sufficient to capture the disease's history (Miller, 1976).

5.5.2 Social lessons

A narrow focus on economic growth subverts society's wellbeing
The economic and political power of one factory or stakeholder can dominate public health interests in a context that is strongly oriented to promoting economic growth. In this case, the factory was Chisso, which had a great influence in Japanese industry and society in the late 1950s and 1960s. Economic growth was the top priority in Chisso and in Japanese society more broadly.

It is noteworthy that after years of scientific evidence of harm to humans, it was only when the

([6]) The excessive dominance of one discipline in a multidisciplinary dispute was noted in *Late lessons from early warnings* Volume 1 (EEA, 2001).

production process was no longer needed that the factory changed its processes and the government altered its stance regarding the harm. In Minamata, acetaldehyde production became unnecessary and stopped on 18 May 1968. Only after this date did the Japanese government officially accept the causal relationship between wastewater from Chisso factory and Minamata disease.

Discrimination perpetuates harm
The Minamata story reveals discrimination in various forms. There was discrimination against the fishermen of South Kyushu, who were poor and situated far from Tokyo, the geographical and political centre of Japan. It is notable that when the discharge from paper manufacturing in Tokyo caused fishery damage in 1958, the Tokyo metropolitan government halted the factory's production (Hashimoto, 2000).

Furthermore, when the disease occurred it was initially considered infectious. Patients were shunned and avoided by other community members and experienced years of discrimination. Fear of this actually prevented patients from coming forward. They also experienced discrimination after they obtained compensation money.

Don't 'shoot the messenger' who brings 'inconvenient truths'
After the Minamata Food Poisoning Committee published its organic mercury theory, the Committee was suddenly dissolved without stated reason. With hindsight, it is evident that their conclusions were not welcome to the authorities. Indeed, researchers at Kumamoto University were criticised as being a 'hick' university by the 'centre' after they proposed the organic mercury theory.

Stakeholders can supress science
Science can be absorbed and suppressed by the stakeholders such as industries and public authorities. Hajime Hosokawa, Director of Chisso hospital, demonstrated that giving a cat wastewater from acetaldehyde production induced Minamata disease. These results were suppressed by the factory. Later, Kumamoto University School of Medicine, which had initially analysed organic mercury as a cause of Minamata disease, joined the (pro-industry, later discredited) Tamiya Committee and began to receive research funds from Chisso factory and the Tamiya Committee. From that time, Minamata disease became a sensitive research issue at Kumamoto University.

Similarly, the Japanese Central Council in 1991 (JSPN, 2003) was not neutral and its discussions were directed towards complying with the EAJ's views. This fact was hidden from the public. Furthermore, JSPN also pointed out that biased distribution of public research funding played an important role in e.g. controlling researchers (JSPN, 2000).

Information must be transparent and broadly communicated
It is important to be transparent with information and to communicate it widely so that events are not repeated elsewhere. With better management of information, Niigata Minamata disease could have been avoided altogether.

While transparency and communications have improved in many advanced industrial economies, the translocation of manufacturing capacity elsewhere means that the consequences of product manufacture may no longer be transparent to consumers in advanced economies. Moreover, there are cases where information is not communicated to the public. Indeed, in Minamata, none of the information generated was publicly communicated to residents during the contamination period.

5.5.3 Inter-disciplinary lessons

Value of lay and local knowledge
Lay and local knowledge should not be ignored. The fishermen knew that fish could not live in water from the outfall from Chisso factory before Minamata disease occurred. Minamata citizens knew that mercuric salt was used to produce acetaldehyde in 1959. Finally, as described in the conversation between Masazumi Harada and the mother of congenital Minamata disease patient, the mother had deduced that neurological signs observed in her son were due to Minamata disease, at a time when this was assumed to be impossible by experts. The value of lay and local knowledge is one of the 'Twelve late lessons' of Volume 1 (EEA, 2001) ([7]).

Interdisciplinary barriers and the absence of open discussion augment harm
In the Minamata case, a lack of open discussion delayed preventive actions, obscured the features

([7]) See also the report from EEA workshop on Lay, local traditional knowledge and citizen science, June 2011 (http://lltk.ew.eea.europa.eu/about/lltk-and-citizen-science-meeting-report.pdf).

of the disease and postponed its resolution. Researchers at Kumamoto University did not know what was produced at the factory, how it was produced, or what substances were used in which processes. The Research Group did not receive assistance from the engineers at Chisso factory, or from the organic chemistry sector of School of Engineering at Kumamoto University. Even within medicine, epidemiology was considered an inferior discipline and neurologists did not apply epidemiological thinking in certifying patients. Among the universities, it was said that scientists from the 'centre' could be trusted over those from 'hick' universities on the periphery.

The Japanese medical community misunderstood or was unfamiliar with the Food Sanitation Act, which must have bolstered the government's position. According to the Food Sanitation Act, doctors who recognise food poisoning must notify the local health centre, which must investigate the problem. No doctors (both clinicians and researchers) notified this outbreak as food poisoning in Kumamoto Prefecture. Instead, they continued to search for the etiologic agents. Had doctors treated and identified this as food poisoning, the government would have had difficulty not applying the Act.

Even the ministry responsible for health policy was unfamiliar with the Act. In 1990, the MHWJ argued that the government did not apply the Act because the etiological agent (methylmercury) had not been identified in 1957. In fact, the Act should have been applied when cause/transmission was identified. The different approach employed in the food poisoning case in Shizuoka in 1950 suggests that not applying the Act in Minamata was a political choice.

Finally, there was little direct discussion between stakeholders. In Japan, even in 2010, policymaking decisions do not involve stakeholders such as patients' organisations. However, EAJ and the exposed patients do share some common perspectives and conclusions regarding Minamata disease. The protracted legal action might have been shortened via direct dialogue between EAJ and the exposed patients. The fact that experts at the university, the health centre and Chisso were not willing to hear and consider the opinions of lay and local people are symptoms of the same problem. Recently, there are some measures toward Minamata's regeneration, such as making Minamata city a model city for the environment, facilitating waste reduction and recycling etc. (Minamata Disease Museum, 2007). In particular, the word 'Moyainaoshi' (the re-establishment of emotional ties or reconciliation) is often used to strengthen interpersonal ties so that citizens can speak up in public about Minamata disease issues. Despite such efforts, there is still little discussion about how to support patients, investigating the exposed patients etc. to solve the real problem of Minamata disease. It can be said that Japan still faces a problem of democracy (George, 2001).

Table 5.1 Early warnings and actions

1908	The Nihon Carbide factory was established in Minamata
1921	Methylmercury synthesised during acetaldehyde production in Germany
1921	The Chisso factory in Minamata Bay bought German patent and began using carbide and acetylene to manufacture a wide range of chemicals
1925–1926	The company began to receive requests for compensation from the fishing cooperative. On the condition that no further complaints would ever be lodged, Chisso paid a small amount of 'sympathy money'
1932	The Chisso factory in Minamata began to produce acetaldehyde from acetylene gas, using mercury as a catalyst
1930–1937	Mercury poisoning in German factories using acetaldehyde
1943	The issue of fishery damage arose again due to carbide residue from acetylene production and another compensation contract was concluded
1949	More fishery damage negotiations failed. Chisso said the catch data were 'not scientific'
1950	Fishermen around Minamata Bay witnessed huge numbers of fish rising to the surface and swimming around as though crazy. Sea birds were unable to fly. Oysters and cockles were washed up onto the beach rotting with their shells open. Barnacles did not attach themselves to boats fishing near factory outlet
1951	Chisso increased production of acetaldehyde and related methylmercury pollution from the factory
1952	Upon request from local fishermen, the Fisheries Division of the Kumamoto Prefecture inspected the factory and Chisso documents about mercury use and discharge. As a result they reported that the discharge should be analysed. Kumamoto Prefecture and Chisso failed to do this and fishery damage continued
1953	Local cats, which ate great quantities of fish, went mad and died after strange dancing and convulsions
1956	1 May: First official notification of strange disease to the Minamata Public Health Centre. 28 May: Minamata Strange Disease Countermeasures Committee was organised by Chisso Hospital, local doctors and Minamata Public Health Centre. 30 cases including 11 deaths were identified. Kumamoto University Research Group reported that the disease was not contagious (as was claimed at first) but rather a food poisoning from eating fish contaminated by a heavy metal in Minamata Bay

Table 5.1 Early warnings and actions (cont.)

1957	Kumamoto University Research Group recommended prohibiting fishing under the Food Sanitation Act. Japanese Ministry of Health and Welfare Research Group from Tokyo confirms local conclusions and recommends full investigation of Chisso effluents. In response to these findings, the local government of Kumamoto Prefecture considered applying the Food Sanitation Act in March 1957, although the Chief of the Public Health Bureau of the Ministry of Health and Welfare of Japan replied to the local government that it is impossible to apply the Food Sanitation Act. Then, the local government abandoned the application
1958	Chisso changed the drainage route of acetaldehyde production from Minamata Bay to Minamata River
1958	UK neurologist Douglas McAlpine examined 15 Minamata disease patients and reported his observations in The Lancet, listing methylmercury as one of the metals which could induce Minamata disease. This was the first time in a scientific paper that methylmercury was identified as possible cause. McAlpine was prevented from presenting his findings to the Japanese Society of Neurology
1959	On 7 October 1959, Chisso Hospital director Hajime Hosokawa fed factory effluent to cats and induced Minamata disease but this was suppressed until a compensation case in 1970
1959	Organic mercury was recognised as the etiological agent by the Minamata Food Poisoning Committee organised by the Ministry of Health and Welfare of Japan on 12 November 1959. However, there was no mention of the source of the contamination, Chisso factory. Indeed, the section chief of the Ministry of Health and Welfare's Environmental Sanitation Department asks for Chisso effluent case to be removed from report as it was not 'scientifically' proven. After the Committee had reported its opinion to the Minister of Health and Welfare, it was suddenly dissolved
1959	In December 1959, Chisso factory established an ineffective purifying system for the contaminated water
1960	Kumamoto Prefecture Institute for Health Research investigated the mercury concentration in hair samples from 1 645 healthy fishermen from around Shiranui Sea. Results indicated that the contamination had spread throughout the entire Shiranui Sea
1962	An unusual occurrence of cerebral palsy infants was officially recognised as congenital Minamata Disease. Professor Katsuro Irukayama succeeded in extracting methylmercury chloride from the sludge of the acetaldehyde production process in the factory but the Fisheries Agency abandoned research on Minamata disease. No research activities were conducted by government agencies until 1968
1965	Methylmercury food poisoning occurred in Niigata, causing 'Niigata Minamata disease'. The factory responsible (Showa Denko) operated in the same way as Chisso in Minamata
1968	In May, Chisso stopped its acetaldehyde production for commercial reasons. In total, 488 tonnes of mercury were discharged into the sea from 1932 to 1968. After that, on 26 September, Japan's government accepts causal link between wastewater from Chisso (and Showa Denko) and Minamata disease
1969–	Government compensation arrangements applied 'chaotically'. Private compensation cases for Minamata victims begin
1971	On 7 August, 1971, the 'Administrative Vice Director of the EAJ Notice' was published, marking the first real policy change since 1956. The Department of Neuropsychiatry at Kumamoto University School of Medicine undertook the first and largest cross-sectional population-based investigation to evaluate the prevalence of neurological signs of Minamata disease among local residents
1972	The United Nations Conference on the Human Environment was held in Stockholm. Two Minamata disease patients attended and created much public awareness
1973	Kumamoto District Court ordered Chisso factory to pay compensation to Minamata disease patients engaged in a lawsuit against the company
1975	From 1968, Masazumi Harada collected umbilical cords (traditionally preserved in Japan) from residents around Shiranui Sea and demonstrated a link between acetaldehyde production in Chisso factory and methylmercury; supporting the hypothesis that methylmercury could affect foetuses
1977	A new and more rigid set of government accreditation criteria for Minamata disease required a combination of neurological signs: the '1977 criteria'. These remain in force today. Subsequently, there was a rise in the number of patients who had methylmercury-related symptoms but were not formally accredited
1978	Kumamoto local government issues debt to help Chisso pay compensation
1995–1996	In the 'first political solution' the government and Chisso factory attempted to settle the conflict by paying lump sums (not as compensation) in 10 000 cases without changing the strict criteria or recognising affected individuals as official patients
2004	The Japanese Supreme Court confirmed the liability of the national and Kumamoto Prefecture governments for damage caused by methylmercury poisoning in the Minamata area. The court also ruled that the 1977 criteria should be relaxed
2009	The Act on Special Measures (the so-called 'second political solution') was passed without changing the strict 1977 criteria. Chisso to pay lump sums to patients, not as compensation but relief money because neither the government nor Chisso admits liability and the patients are not formally recognised as 'Minamata disease patients'
2009–	UNEP initiates a global mercury phase-out and works to develop a global legally binding instrument on mercury, planned for signature in Japan in 2013
2013–	Private law suits still continue

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From Minamata to global health risk

Philippe Grandjean ([8]) ([9])

While the Minamata incident was being elucidated, other methylmercury poisonings occurred elsewhere due to extensive use of mercury fungicides and improper labelling. Treated seed grain was mistakenly used for bread-making, and the most serious poisoning incident happened in Iraq during a famine in 1970-1971 (Bakir et al., 1973). A widely cited report on 93 poisoned Iraqi adults reported that facial paraesthesia was the earliest clinical sign of poisoning and showed a clear dose-dependence (Bakir et al., 1973). However, the study was small in comparison with the officially recorded 6 500 hospitalisations, of whom 459 died (Bakir et al., 1973), and the amount of treated grain used (100 000 tonnes) would suggest that many more may have been poisoned. The first author of the science report, Farhan Bakir, was later recognised as Saddam Hussein's personal physician, then in exile along with at least one other Iraqi co-author (Giles, 2003; Hightower, 2009). As no useful dose-response data were available from Minamata, the Iraqi data were used for many years as the main documentation for risk assessment. Given the history of the poisonings, one can assume that methylmercury toxicity was at least not exaggerated (Grandjean et al., 2010).

Attention turned to neurotoxicity during brain development as a result of an experimental study: rats exposed during early development showed adverse effects that were not apparent at first, but later became obvious as deranged behaviour in the mature animals (Spyker et al., 1972). This report clearly supported the Minamata evidence as well as a Swedish report 20 years earlier that described mental retardation in two children exposed to methylmercury from treated grain (Engleson and Herner, 1952).

There was another surprise when Swedish researchers examined the chemical fate of mercury in a simple aquarium: methylmercury was formed from inorganic mercury compounds in the aquarium sediment. None was formed after prior autoclaving of the sediment, suggesting that microorganisms played a role (Jensen and Jernelov, 1967). Although these processes were of little significance in Minamata, where methylmercury was formed in the acetaldehyde plant (Grandjean et al., 2010), methylation of mercury suddenly became a world-wide problem. Widespread use of methylmercury for seed dressing or as a fungicide in paper mills was already known to cause local pollution of waterways and coastal waters. Now it turned out that any release of mercury could be converted into the dangerous methylmercury molecule. Studies in North America verified that bio-accumulation took place, with the highest concentrations at the top of the food chains (Fimreite, 1974). Although the first studies were contradicted and explained away, methylmercury contamination of fish emerged as a worldwide concern. Many rivers and lakes were already so polluted with mercury that fish advisories against eating sports fish were issued, especially in countries like Canada, Sweden and the US Advisories against eating locally caught fish now affect over 16 million lake acres and 1.3 million river miles in the US (US EPA, 2007).

Mercury must always have been a natural component of life on the planet, but pollution has released large amounts to the biosphere. Mercury analyses of preserved hair, teeth, and feathers from Arctic indicator species show that current levels are about ten times those in pre-industrial times (Dietz et al., 2009).

After the discovery that exposures to lead at levels considered to be 'low' could cause damage to brain development (Needleman et al., 1979), researchers suspected that methylmercury might have similar effects and may not be safe at common levels of exposure. Some of the most highly exposed populations were indigenous groups. In Canada, a study of 234 Cree children showed abnormal tendon reflexes with increased mercury concentrations in maternal hair which reflected exposure during pregnancy (McKeown-Eyssen et al., 1983). Soon after, a larger study from New Zealand showed that increased levels of mercury in mothers' hair during pregnancy were associated with delayed brain development of their children (Kjellström et al., 1986; Kjellström et al., 1989). The results were published after peer review by the Swedish Environmental Protection Agency, but were ignored for formal reasons by other regulatory authorities, allegedly because the reports had not appeared in a peer-reviewed scientific journal.

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Two large prospective studies were then initiated in the mid-1980s. The largest consisted of 1 000 children from the Faroe Islands and concluded that low-level methylmercury exposure during prenatal development was associated with deficits in several brain functions at school age; clear deficits were apparent well below a previously proposed safe level of 10 μg/g for mercury concentration in maternal hair (Grandjean et al., 1997). On the other hand, largely non-positive findings were initially reported in children from a similar study in the Seychelles (Myers et al., 2003), and the apparent disagreement was perceived as a controversy and fuelled a debate on uncertainty (Grandjean, 1999), with resonance in trade journals, internet sites, commercial campaigns, and even an editorial in the *Wall Street Journal*. Additional longitudinal data later appeared from Japan, Poland and the US in support of the Faroes conclusions (Jedrychowski et al., 2006; Lederman et al., 2008; Murata et al., 2006; Oken et al., 2008). Although less weighty, several cross-sectional studies also supported the existence of low-level exposure neurotoxicity (Grandjean et al., 2005).

The reasons for the apparent lack of mercury effects in the Seychelles could be that beneficial nutrients in fish might obliterate or dampen the mercury toxicity (Clarkson and Strain, 2003). New research from the Seychelles has recently shown that cognitive development in children was associated neither with maternal fish intake nor with methylmercury exposure, when each of them was considered separately. If maternal fish intake and mercury were included in the statistical analysis at the same time, then fish intake was clearly beneficial, while mercury had negative effects (Strain et al., 2008). Also, in the Faroes, the mercury toxicity became more prominent after adjustment for the beneficial effects of fish intake during pregnancy (Budtz-Jorgensen et al., 2007).

Because of the apparent disagreement between the two major studies and the public health implications of mercury, the US White House in 1998 called for an international workshop with 30 invited experts, who were asked to critically examine the scientific evidence. They emphasised a variety of possible uncertainties. The conclusions stated that 'there are inadequate data … to draw meaningful conclusions at this time' (NIEHS, 1998). Despite the possibility that subclinical toxicity could easily be missed and underestimated, the workshop experts were quite optimistic: 'Measurement error can impact significantly on both the estimated levels of effect and the decision on the level of exposure at which an effect is detected because of the potential for misclassification. However, the data presented in the workshop suggest that the precision of measurements of methylmercury in hair or cord blood is very good.' The experts recommended further research.

At the request of the US Congress, a new expert panel was then convened by the National Research Council (NRC, 2000) to determine whether an exposure limit of 0.1 μg/kg bodyweight per day was appropriate, as proposed by the US Environmental Protection Agency (EPA) on the basis of the data from Iraq. The committee supported the US EPA limit, but recommended that it should be based on the data from the Faroes study (which agreed with the overall evidence including New Zealand and Seychelles).

This recommendation would seem justified and appropriate, but may not be sufficiently protective. First, the exposure limit should address the problem that mercury toxicity may be masked by increased intake of essential nutrients from seafood that promote brain development (Budtz-Jorgensen et al., 2007). If this adjustment is not made, mercury would seem less toxic than it really is. Second, all the calculations have assumed that the mercury exposures are precise, but any imprecision in exposure assessments will result in misclassification and a likely underestimate of the real mercury toxicity. If this factor is taken into account, the exposure limit should be decreased by about 50 % (Grandjean and Budtz-Jorgensen, 2007).

Thus, the first likely cases of developmental methylmercury poisoning were already described in 1952 and subsequently reported from Minamata; replication in laboratory animals was published in 1972; and the first prospective population study of prenatal methylmercury toxicity due to contaminated seafood in humans was published in 1986. However, scientific consensus on prenatal vulnerability was hampered by focusing on scientific details rather than public health implications, and international agreement on the need for protection against prenatal exposures was only reached in 2002, i.e. 50 years after the first medical report that methylmercury can damage brain development.

Environmental methylation of mercury in sediment was discovered accidentally, since systematic studies of mercury's environmental fate were not conducted, and initial studies focused on total mercury concentration, not on the methylmercury compound responsible for brain toxicity. Recognition of contamination of food chains and environmental bioaccumulation of methylmercury was therefore also delayed by several decades.

Following the publication of new data on the adverse effects of low-level exposures to methylmercury,

Important early warnings of methylmercury toxicity

1952	First report on developmental methylmercury neurotoxicity in two infants
1960	Mental retardation in Minamata associated with maternal seafood diet
1955–1972	Poisoning epidemics from use of methylmercury-treated seed grain for baking and cooking
1967	Demonstration of mercury methylation in sediments
1972	Experimental study of delayed effects due to developmental neurotoxicity
1978	Exposure limit based on toxicity in adults
1986	First epidemiology report on adverse effects in children related to maternal fish intake during pregnancy in New Zealand
1997	Confirmation from the Faroe Islands on adverse effects in children from methylmercury in maternal seafood intake during pregnancy
1998	White House workshop of 30 scientists identifies uncertainties in evidence
2000	US National Research Council supports exposure limit based on Faroes data
2004	European Food Safety Authority recommends that exposures be minimised

regulatory agencies requested scientific scrutiny. Expert committees emphasised uncertainties and weaknesses in the available data. Less attention was paid to the question of what could have been known, given the research methods and possibilities, and whether developmental neurotoxicity at low doses could be ruled out. The reports also generally ignored that the imprecision of the measurements most likely resulted in an underestimate of the true effects. Instead, more research was recommended. The insistence on solid evidence promoted by polluters and regulatory agencies therefore agreed with a desire among researchers to expand scientific activities in this area. However, the wish to obtain a more complete proof had the untoward effect of delaying corrective action.

In a commentary on the regulatory delays in dealing with methylmercury poisoning in Minamata, Professor Jun Ui wrote (quoted from D'Itri and D'Itri, 1978): 'It might be a coincidence, but a strange, parallel relationship was observed between the actual symptoms of Minamata Disease and the reactions of these formal organisations. A constriction of the visual field was common among all organizations. Ataxia, a loss of coordination between various parts of the body, was often exhibited in contradictions between the measures taken by various parts of the government. There was also a loss of sensation as the appeal of the victims went unheard and there was little effort to grasp the situation as a whole. Many organisations also reacted with spasmic convulsions when they faced the problem. This was followed by mental retardation and forgetfulness.' It seems that memory loss, narrow-mindedness, and lack of coordination also affected the planning and the interpretation of environmental research on methylmercury in a more general sense.

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Mercury science and policy since Minamata: four insights for policy

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Introduction

The events at Minamata, as well as other serious instances of high-dose exposure, showed the extremely toxic potential of mercury. Furthermore, beginning in the 1960s, widespread environmental contamination by mercury beyond locally-contaminated areas began to be measured by scientists and addressed by policymakers. Mercury emerged through the late 20th century as a substance known to pose risks at locations far from its release, and at low doses (UNEP, 2002). In the 21st century, policies continue to be developed to address the global spread of mercury, and scientists and policymakers are becoming increasingly aware of the complexities of the links between human activities such as energy production and connections between mercury and other environmental and health issues. The case history of mercury beyond Minamata provides four major and partially overlapping insights into the application of scientific knowledge to political efforts to deal with environmental and human health hazards, for both scientists and policymakers.

These are the need to:

- conduct research into 'blind spots';
- encourage policy-relevant scientific assessments;
- design policies that can be adapted to changing knowledge; and
- acknowledge and manage interactions between different risk issues.

Conduct research into 'blind spots'

A first insight from the mercury case, for the scientific community, is the need to conduct policy-relevant research into 'blind spots,' and be open for that research to challenge the dominant scientific understanding of a problem. (The scientific inertia that can lead to a focus on conventional paradigms is also illustrated in Chapter 26 on science for precautionary decision-making). The conventional wisdom in the 1970s was that mercury was essentially a local problem. The World Health Organization illustrated this view by stating: 'In the global cycle, most of the mercury is derived from natural sources whereas the local cycle is predominantly concerned with man-made release' (WHO and UNEP, 1976). A lawsuit by US swordfish distributors also challenged US Environmental Protection Agency (US EPA) mercury limits for fish, based on the understanding that anthropogenic mercury remained in directly-contaminated areas. The swordfish distributors argued that mercury in fish was naturally-occurring and thus should not be regulated as a contaminated product (US Court of Appeals, 1980). However, there was early evidence that mercury from human activities could be at least as important as natural mercury in remote areas, as reported by the US EPA in 1973: 'Mercury from burning coal is dispersed widely, and may enter the aquatic or terrestrial environment far from the point of discharge. Since mercury discharged in this way is of the same order of magnitude as the total of mercury mined in the world, it appears advisable to try to develop a technology to remove mercury either from the coals or from stack gases' (Klein, 1973). Despite these early warnings, mercury continued to enter the global environment at an increasing rate.

The understanding that mercury was a global problem began to emerge as predominant, at least scientifically, in the 1980s and early 1990s. Scientific research demonstrated clearly that industrial contaminants, including mercury and persistent organic pollutants (POPs), were present at elevated levels in areas far from their sources (Selin, 2010). A review of environmental concentrations of mercury in the Arctic environment in 1997 by the Arctic Monitoring and Assessment Programme (AMAP) noted that circumpolar levels of mercury were increasing in lake and ocean sediments and in the livers and kidneys of marine mammals (AMAP, 1997). While research about the degree of anthropogenic relative to natural contamination was still uncertain, AMAP urged Arctic countries to develop international mechanisms to address mercury contamination. The changed scientific understanding of mercury as a substance that travels long distances and poses risks far from its release points slowly began to shape policies to address mercury internationally.

Scientific understanding of the mercury problem continues to evolve. While international policy is now addressing the widespread spatial scale of mercury as a global problem, scientific research is also illuminating the multiple timescales under which mercury affects humans and the environment (Selin, 2011). Mercury mobilised from fossil sources continues to circulate in the land-atmosphere-ocean system over timescales longer than those considered by policies. It will take an estimated

3 000–10 000 years for mercury so mobilised to return to deep-ocean sediments (Mason and Sheu, 2002; Selin et al., 2008). A corollary to this is that only about one third of mercury currently entering ecosystems comes from direct anthropogenic activity. Another third results from natural sources, and the remainder is legacy mercury, previously emitted from anthropogenic sources, continuing to circulate between the land, ocean and atmosphere. This means that human perturbations to the global mercury cycle are very long-lived, and the Earth system will recover only slowly from historical mercury contamination. On the other hand, some environments may respond very quickly to decreases in mercury input (Harris et al., 2007). One example is the Northeast US, where, coincident with and likely as a result of regional policies to reduce emissions, concentrations of mercury in fish declined from 1999 to 2004 (Hutcheson et al., 2006). Monitoring the continuing impacts of mercury in ecosystems, and potential improvements resulting from policies, will require this evolving scientific understanding of the environmental timescales of mercury to be taken into account.

Encourage policy-relevant scientific assessments

A second insight from the interface with science and policymaking, relevant to both scientists and policymakers, is that scientific information on new risks can influence the policy process through targeted, international scientific assessments. These are widely applied to inform international and global policymaking on environmental issues such as ozone depletion and climate change (Eckley, 2001; Mitchell et al., 2006). In the case of mercury, they provide a mechanism for new scientific understanding to be taken up and addressed. This suggests that scientists should participate actively in international assessment processes.

An example of the influence of scientific assessments on mercury policy occurred in the late 20th century. The process that led to the 1997 AMAP assessment, discussed above, was a critical factor in bringing mercury contamination of remote areas to public attention (Selin and Selin, 2008). Partially influenced by the AMAP work, the Convention on Long-range Transboundary Air Pollution (CLRTAP), a regional agreement among the countries of the United Nations Economic Commission for Europe (including western and eastern European countries, Russia, the United States and Canada), was one of the first international bodies to express interest in addressing mercury. Following their own scientific assessment process (UNECE, 1995), CLRTAP countries negotiated a protocol on heavy metals, which was adopted in 1998 and entered into force in 2003. The protocol requires parties to reduce emissions of three heavy metals (lead and cadmium as well as mercury) below 1990 levels (or, alternately, below the year of their choice between 1985 and 1995), and to apply limit values and best available techniques to control major sources.

Another example of scientific assessments of mercury influencing policy came when, in response to growing global concerns about mercury, a mercury assessment organised by the United Nations Environment Programme (UNEP) concluded that there was 'sufficient evidence of significant global adverse impacts to warrant international action to reduce the risks to human health and/or the environment arising from the release of mercury into the environment' (UNEP, 2002). Despite this strong statement, policy actions following this pronouncement proceeded slowly, as there were strong political interests both in favour of and against a mercury treaty (Selin and Selin, 2006). However, in 2009, partly as a result of a change in the position of the United States (after a change in presidential administration), countries agreed to begin negotiations on a global, legally-binding instrument to address mercury. The process towards a global mercury treaty began in 2009 with the goal of completing negotiations in 2013. This delay shows that even with strong scientific assessments, gaining international consensus, balancing political and stakeholder interests, is a lengthy process, and can be slow to respond to new scientific information and understanding of the problem.

Design policies to adapt to changing knowledge

A third insight, for policymakers, is that policymaking processes and policies should be designed so that specific policies can be revised and adapted to reflect new information and changing scientific understanding. Early policies, focused on local contamination of mercury, did not address its long-range impacts, and new institutional frameworks such as a mercury treaty are thus needed to address mercury as a global problem. As noted above, gaining political agreement for new institutions can delay action for years to decades. As scientific work continues to reveal new information and paradigms change, flexible policies might be able to respond more quickly.

Even today, few policies address the full complexity of mercury in the environment. While there are several forms of mercury emitted from ecosystems

— long-lived forms that transport globally and other forms that cause local impacts — policies generally treat all forms of mercury in the same way. A recent review of the effectiveness of the CLRTAP heavy metals protocol noted that pollution control techniques specified by the protocol primarily address the forms that cause local impacts, raising questions about how effective mercury controls are at addressing the forms that cause global contamination (van der Gon et al., 2005). In addition, there are few links between policies that address emissions and those that address exposure and impacts (Selin, 2011).

Policies for minimising mercury exposure through dietary advice for fish consumers are one area where interventions have been expanded, formalised and revised over time. In 2003, the Joint FAO/WHO Expert Committee on Food Additives (JECFA) revised its provisional tolerable weekly intake (PWTI) of methylmercury from 3.3 μg kg–1 bodyweight per week down to 1.6 μg kg–1 bodyweight per week, specifically to protect against developmental toxicity for childbearing women. Some countries have also set domestic standards. For example, the United Kingdom Food Standards Agency recommends that pregnant women and children under 16 avoid eating shark, marlin and swordfish, and minimise their consumption of tuna to four medium-sized cans or two steaks per week (United Kingdom Food Standards Agency, 2004). In Sweden, pregnant or nursing women are advised to avoid eating fish high in mercury more than two or three times a year (Sweden National Food Agency (Livsmedelsverket), 2011). Some research has shown that the consequence of dietary advice focused on methylmercury has been an overall decrease in fish consumption by pregnant women (Burger and Gochfeld, 2008). There is growing scientific understanding of the benefits of n-3 polyunsaturated fatty acids (n-3 PUFAs), nutrients present in fish and shellfish, on prenatal development (Mahaffey et al., 2011). Some fish, however, such as mackerel or herring, are high in n-3 PUFAs and low in mercury, suggesting that dietary advice could better reflect fish choices to maximise benefit and minimise risk (Mahaffey et al., 2011). However, sensitive populations continue to be exposed to high levels of methylmercury, suggesting the potential for improved risk management (Mahaffey et al., 2009).

Acknowledge and manage interactions between risk issues

As a fourth and final insight, for both scientists and policymakers, the mercury case shows that acknowledging and managing both environmental and societal connections between different risk issues can be critical. From an environmental perspective, in addition to the potential benefits of fish consumption noted above, other pollutants such as PCBs may be present in different kinds of fish, complicating efforts to provide dietary advice (Mahaffey et al., 2011). Climate and other environmental changes can affect the mercury problem by changing environmental pathways of contamination (AMAP, 2011).

Societal issues such as economic development also intersect with the mercury issue. While global emissions of mercury have remained relatively constant since 1990 (Pacyna and Pacyna, 2002; Pacyna et al., 2006; Pacyna et al., 2009), this reflects increases in Asia, resulting from rapid industrialisation and the increasing use of coal, compensating for decreases in North America and Europe. Future Asian economic development, particularly in China, could lead to dramatic increase in mercury emissions from that region (Streets et al., 2009). In addition, increasing gold prices can lead to increased use of mercury in artisanal and small-scale gold mining (Spiegel and Veiga, 2005).

Governance-related connections have additional importance. Actions in the 1980s to reduce air pollution had substantial co-benefits for mercury reductions in Europe and North America, as traditional air pollutant controls can potentially achieve > 90 % emission reductions for mercury (Northeast States for Coordinated Air Use Management, 2010). Information about the long-range transport and low-dose effects of other substances, such as persistent organic pollutants, have also helped to improve the scientific understanding of mercury risks (Selin, 2010). Finding ways to harness and encourage co-benefits, while mitigating shared risks, is a complex and continuing challenge.

Concluding remarks

After decades of science and policy actions, mercury still poses significant challenges to society. A major reason is that conceptions of the mercury problem were initially limited, and scientific and policy understanding has continued to expand and increase in complexity, increasing complexity unfortunately being the rule rather than the exception in addressing environmental risks.

The case of mercury shows that both scientists and policymakers can play an important role in risk management, through the four major insights

summarised above. Scientists should encourage early research into 'blind spots' expanding understanding of environmental complexity. Policymakers should support, and scientists should participate in, targeted and international scientific assessments for policy. Policymakers should also be conscious that scientific information can and will change and design policies accordingly. Finally, both scientists and policymakers would benefit from acknowledging the full complexities and links between environmental risks. Understanding and managing multiple, linked environmental and human stressors is a primary challenge for sustainability.

While it is tempting to assume that our current understanding of the mercury problem represents a comprehensive picture of the real world, history suggests that both our understanding of the problem and our strategies to address it probably continue to have blind spots. A substantial area of uncertainty, for example, is the mechanism by which mercury is converted to methylmercury in the ocean. Additional connections with other risks — both environmental and social — are likely to be identified in the future. Drawing lessons from the mercury case, by encouraging expanded research paradigms, supporting scientific assessments, designing dynamic policies, and exploring and taking advantage of cross-issue connections — would help societies to better address risks and surprises in the future.

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Mercury in fish — the need for better information about contaminant exposures

Argelia Castaño

There is no doubt about adverse effects of mercury in highly exposed populations, but the question is where to put an acceptable level for the general population? The major source of methyl-mercury is fish and particularly large marine fishes like tuna, sharks and swordfish. Fish and marine products are rich in unsaturated fatty acids which reduce the risk of cardiovascular disease and therefore are beneficial for health. Cardio-vascular disease is related to high consumption of red meat and dairy products and low intake of vegetables and fruits. Public health authorities are therefore recommending a Mediterranean diet with a high proportion of fish, marine products, vegetables and fruit as a way to reduce cardiovascular disease burden.

However, the authorities are facing the dilemma of balancing the benefits of fish consumption with the assumed adverse effects of low level methyl-mercury exposures. Should the policymakers advise against fish consumption because of contaminants or are the negative effects of the contaminant burden still minor compared with the positive effects of a healthier diet? The issue is even more serious for indigenous populations, for example in the Arctic region, which traditionally have a diet based on marine species (seals, whales) which today have high levels of contaminants. Should we recommend these populations to change their diet to a Western life style diet with the accompanying new health problems like obesity and diabetes? The contaminants are there — we have to accept this although we should try to reduce the exposure by all means — but we have to be pragmatic in establishing the safe levels and not exaggerate the risks in the light of the obvious benefits.

Security and confidence are the driving forces for decision-making. For mercury we have a good knowledge base from experimental and epidemiological studies connecting body burdens and adverse effects. What is critical to assess is the exposure. How much mercury are we exposed to in our daily lives and from where is it coming?

The decision-makers need robust information before they can decide on mitigation strategies. The European Commission within the frame of Environment & Health action plan 2004-2010, has funded a project to standardise protocols for human biomonitoring in Europe ([10]). The protocols and methodologies that were developed are now being tested in a pilot study co-funded by 17 EU Member States with contribution from the EU LIFE+ programme ([11]). This study, which will be reported by the end of 2012, will provide an insight into mercury levels in children and their mothers in Europe, measured for the first time under strictly standardised and harmonised conditions.

It will then be possible to map human mercury exposure at a European level with real and comparable numbers, even though the sample cannot be considered representative at national levels. For individual Member States this mapping provides an important benchmark which could assist in national mitigation strategies. Information of this kind is essential both for developing a European position for international negotiations concerning the implementation of the Global Mercury Treaty currently being progressed by UNEP and for helping national authorities and consumers to make better-informed choices about healthy diets.

([10]) http://www.eu-hbm.info/cophes.
([11]) http://www.eu-hbm.info/democophes.