

Perspectives on Niigata, Japan

Methyl mercury exposure and poisoning at Niigata, Japan

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The following three articles describe one of the most dramatic pollution episodes to occur in the past century, but one about which relatively little information is available in the English scientific literature. It is the consequence of an industrial disaster that occurred at Niigata, Japan in the mid 1960s. The Niigata pollution episode has not been as well documented as the one that occurred at Minamata. However, it is of equal or even greater interest to both scientists and policy makers since information on exposure was obtained on mothers and children either during the pregnancy or immediately after the births.

Niigata and Minamata are the only two sites where clinically apparent human methyl mercury (MeHg) poisoning has been confirmed due to fish consumption. There have been anecdotal reports and media assertions that such poisoning may have occurred in China along the Songhua River, on Indian reservations in Canada, and along the Amazon basin in South America, but there are no confirmed case reports in the peer reviewed literature to document these assertions. The pollution at both Minamata and Niigata was massive. At Minamata, 200 tons of Hg was used as a catalyst to produce acetaldehyde between 1949-1953 and large amounts of waste were discharged into Minamata Bay (1). In addition, some of the waste was probably in the form of organic mercury. The quantity of mercury discharged at Niigata is unknown. The affected children in most cases both at Minamata and Niigata were in families who made a living by fishing. These families consumed large quantities of fish frequently. Although all fish contain at least small amounts of MeHg, fish at both Minamata and Niigata had up to 40 ppm of MeHg, as did local shellfish (2). This differs from the average MeHg concentration in fish from Seychelles and most western countries such as the United States where it is less than 0.5 ppm (3-4).

There have been reports of prenatal and postnatal MeHg poisoning elsewhere in the world from sources other than fish. These include MeHg treated grain in Sweden (5) and Iraq (6) and consumption of MeHg contaminated pork in New Mexico (7). The source of exposure is probably of little significance when there is actual poisoning, but the situation may be different when dealing with low-level exposures, especially from fish consumption. Fish contain beneficial nutrients such as n-3 fatty acids and various other micronutrients, including concentrations of selenium. N-3 fatty acids are important for brain development and selenium is known to interact with MeHg and may alter its toxicity. Consequently, the benefits of fish consumption need to be weighed against the potential for toxicity. In addition, some sources of exposure such as sea mammals may also expose the individual to other toxicants such as polychlorinated

biphenyls (PCBs) and other organohalogen compounds. These are known to adversely influence brain development (8-9).

Although Minamata and Niigata have similarities, they also have differences. The disaster at Minamata was not well known until Eugene Smith brought it to worldwide attention in the early 1970s with his outstanding photojournalism (10). His documentation of the Minamata poisoning made it a name known widely and one that became synonymous with industrial pollution and corporate greed and insensitivity. At the time the Minamata poisoning was taking place, there were several other factories in Japan using the same process to produce acetaldehyde, but only one was documented as also causing human poisoning. That factory was the Showa Denka plant upriver from Niigata.

The pollution and its consequences at Niigata are not as well known as those at Minamata and only limited details have been published previously in the English scientific literature. Most of what has been described previously is in two books on Minamata disease (11-12). However, for those citizens consuming fish from the polluted Agano River in Niigata, the consequences were equally serious. Given the large number of commercial fishermen working on the Agano River at that time, the number of individuals who consumed contaminated fish and thus were exposed to MeHg was probably very high.

The Niigata pollution was similar to that at Minamata in that waste from acetaldehyde production, using a process requiring mercury as a catalyst, was discharged directly into a nearby body of water. The waste discharged from the factory contaminated the local aquatic ecosystem and its fish. The Agano River runs through Niigata and the factory was located about 65 kilometres upstream. However, the pollution at Niigata differed in one important aspect: the availability of data on mercury exposure. At Minamata, several years elapsed before the cause of Minamata disease was identified as MeHg poisoning and numerous other pollutants were also present. Manganese, copper, iron, selenium, lead, arsenic, and zinc were all measured to be at high levels near the Minamata pier and first considered as possible causes of the disorder. By the time MeHg was identified as the main cause of Minamata disease, it was too late to determine the actual mercury exposure of the victims. Consequently there are no acute exposure data from Minamata. After the outbreak at Minamata though, the signs and symptoms of MeHg poisoning became widely known in Japan. Niigata physicians recognized the cause of their patients' symptoms early and were able to measure exposure. They then instituted public health measures, warned the public, reduced further exposure and likely prevented a greater tragedy. Whether

other industrial pollutants were present in the Agano River is not known.

Although the first case report of prenatal MeHg poisoning came from Sweden (5), it was Harada who brought prenatal poisoning to scientific attention (13). He described 22 children from Minamata that he believed had congenital poisoning. The youngest of his patients was over a year old when first examined. Later other individuals from Minamata were determined to have prenatal poisoning based on their histories and physical findings, but the total number of individuals reported to have prenatal poisoning from Japan is fewer than 60. Although early recognition of the poisoning at Niigata resulted in exposures being measured either during pregnancy or shortly thereafter, only one prenatal case was described. Her prenatal exposure was measured at 293 ppm in maternal hair about one month after she was born (14).

In Japan, determining the facts surrounding MeHg exposure has been complicated by at least four factors. First, because the cause of the disorder was unknown initially, it was referred to as Minamata disease. That term has unfortunately continued to be used and the authors of the current papers refer to their Niigata patients as having Niigata Minamata Disease. When an illness is named a disease, it suggests that it is an act of nature or even that the patient's family, lifestyle or habits might have contributed. In contrast, when one says a patient was poisoned, others ask how and by whom?

Second, because it was an industrial poisoning, official recognition as a victim carried with it monetary compensation. This led to official definitions of who might be a victim and requirements that they have a documented disability. Those who felt they had been poisoned were required to apply for official recognition and undergo a medical evaluation. To date nearly 20 000 individuals have applied for recognition, but official recognition has been granted to only about 3000. In addition, the official criteria excluded paraesthesias since they cannot be objectively proven. However, paraesthesias are known to be among the earliest symptoms of MeHg poisoning, are present in nearly all adult poisoning cases, and can be more incapacitating than motor deficits (15). The most clearly documented outcome of the Niigata episode was damage to the visual system. Examinations of 325 patients demonstrated deficits in several functions: diminished visual acuity, visual field constriction, critical flicker frequency abnormalities, and abnormal scotopic visual evoked potentials (16). Although there are reports suggesting a high incidence of mild degrees of cerebral palsy following the Minamata epidemic, documentation is limited. Nearly all of the prenatally and postnatally exposed individuals certified as victims have displayed substantial neurological involvement.

Thirdly, Japan was encouraging industrialization during that time period and the large chemical companies were important to the local and national economy and politically were very influential. Fourthly, and perhaps most crucial, the culture in Japan is one of respect for authority and sensitivity to stigma. Having Minamata disease was and still is considered a stigma and carrying the label can have profound affects on a person's family and work relationships. Victims from Hiroshima, Nagasaki, Minamata and Niigata and their offspring all hide their histories. Since many victims hide their disability to avoid the social consequences, they refuse to apply for official recognition or even to be examined by health care authorities.

These factors taken together make interpretation of the data from Japan challenging and add importance to the data from Niigata that is presented here. We sincerely hope that the investigators in Niigata will continue to study this unique cohort.

Competing interests None declared.

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