Chapter 3
Past, Present, and Future of Mercury Pollution Issues

Abstract In this chapter, countermeasures and solutions to methylmercury poisoning (MPM) and current and outstanding issues on the mercury pollution are discussed. Correct information and data, public access to information, establishment of company ethics, rapid response by governments, societal responsibility, and penalties on responsible organizations are necessary for preventing the similar kind of pollution and human hazard. Recent studies which show the relationship between the anthropogenic emissions of mercury and the risk to human health, especially to fetuses and infants, are summarized. Finally, outstanding issues on MPM that are in connection with the global mercury cycle are discussed.

Keywords Anthropogenic mercury emission · Global mercury cycle · Low-dose exposure · Mental retardation · Neurodevelopmental deficits · Root causes

3.1 Lessons from MPM

If we can clarify the root causes of poisoning events such as MPM, then we can make effective countermeasures. From this viewpoint, summarization of students' opinions regarding the causes of MPM (the Sect. 2.20) may lead to antipollution measures as shown below:

1. Correct information and data and public access to information
2. Establishment of company ethics
3. Rapid response and approaches to give priority to human rights of citizens rather than to protecting profits of companies by governments
4. Societal responsibility where all people recognize the importance of public welfare and environments
5. Strict and justifiable penalties on organizations (e.g., companies, governments) that have caused/permitted environmental pollution

Over 60 years have passed after the initial acknowledgment of MPM. Many people are still suffering from this disease. In modern Japan, we have experienced many environmental pollution issues from the Ashio Copper Mine mineral pollution incident in the 1880s–1980s to the Fukushima nuclear power plant accident in 2011. Especially during the rapid economic growth period from the late 1950s through the

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1970s, four major pollution diseases including MPM, methylmercury poisoning which occurred in Niigata (Niigata "Minamata disease"), Yokkaichi asthma, and ouch-ouch disease had occurred sequentially. It seems that these environmental pollution-related diseases share key characteristics. It is necessary to investigate this root cause for prevention of recurrence.

A few students' reports stated that the unclear etiology at the initial MPM stage outbreak had resulted in the disease expansion. Even without exact information on the causative agent, if banning of fisheries and selling marine products was enforced by the late 1956 or 1957, the expansion of MPM must have been avoided. At that time, the national and local governments knew already that the disease had occurred through ingesting local seafood based on epidemiological surveys conducted by KU. Kumamoto Prefecture (KP), however, never applied the Food Sanitation Act, even though KP had sufficient authority to apply this Act independently and to ban fisheries and selling by themselves. The national and local governments maintained the stance that they would not act until the identification of the causative agent. The inexcusable fault to allow the expansion of MPM lies with the national and local governments.

On the other hand, Chisso noticed that their own factory was producing toxic material by 1958 at the latest, when the discharge system for plant effluents was changed; nevertheless they continued the acetaldehyde production, resulting in harming the life and health of residents. The behavior of Chisso should be charged with a crime not a fault. Such criminal companies which prioritize own profits rather than public welfare can exist in all times and countries. Governments might support such companies rather than the public. In these cases, prosecution should function to protect social justice and public health. In the MPM incident, however, the police and prosecutor never took action; instead they charged patients who had made a violent protest to Chisso with assault immediately and severely. I can't help thinking that MPM occurred in the economy-first policy of Japan society in the years following the World War II and such atmosphere continues today. In order to avoid pollution disasters such as MPM, it is necessary to inspect why MPM occurred and to assess where and who take responsibility.

I have to refer to the approach of the pollution research. Members of KU have played a central role in the research to determine the cause of MPM. They reached a conclusion of the truth in 1959 by the continuous, sincere efforts. Meanwhile, a few researchers made objections to this conclusion based on unfounded inferences in support of Chisso's opinion. Professor Tetsuzo Kitagawa of Yokohama National University also disturbed the resolution of methylmercury poisoning incident occurred in Niigata by showing erroneous data. Such patronized scholars avoided the truth and occasionally twisted data in favor of the criminal companies to receive benefits. Their attitude resulted in the lowering of professional integrity among scientists and in eternal dishonor upon themselves.

In Japan, studies on environmental pollution tend not to be favored by researchers, or even if researchers in universities or research institutes of governments want to carry on researches, superiors occasionally disrupt such subjects in fear of large impacts on the society and pressures from companies or governments. Such trends, however, are decreasing in recent days, accompanied by the increased awareness of the environment. Researchers of universities and research institutes should take on environmental pollution problems for the welfare of humanity.

Environmental pollution affairs often cause controversies among residents, patients, company managers, factory laborers, government officers, and researchers. Even among researchers, severe debates have sometimes arisen at minor things. At that time, it is important to open all data and materials which would serve as their claims and then to discuss with each other scientifically and objectively.

We have to recognize the serious pollution like the MPM incident destroys nature, humans, and societies and how long time it takes to recover. Controversies were generated not only between patients and responsible companies but also between patients and residents. A part of residents living in the MPM-prone area who had depended on Chisso are economically alienated patients and referred to mildly symptomatic patients who had requested compensations disparagingly as "fake patients." There were also severe controversies among patients over the method of negotiation with Chisso and the governments. In 1994, the mayor of Minamata City, Masazumi Yoshii, apologized publicly for the patients about the administrative actions during the period of the MPM outbreak that had intended to maintain the operation of the plant, and then he pursued a policy "Moyai-naoshi" in Japanese, which means "reconnection" between individuals as well as between humans and environments. In recent years, the effects seem to be realized gradually, and I am encouraged by the changing attitudes toward the environment not only in Japan but also in the world.

3.2 Global Mercury Cycle and Low-Dose Exposure

3.2.1 Global Mercury Cycle

In recent years, greater attention has been given on the global mercury cycle (Downs et al. 1998). UNEP (2013) which estimated that total anthropogenic emissions of mercury to the atmosphere in 2010 were 1960 t (30% of total mercury), while another 10% came from natural geological sources, and the rest (60%) were from "reemissions" of previously released mercury that had built up over decades and centuries in surface soils and oceans. Artisanal and small-scale gold mining (727 t in 2010, 37% of total anthropogenic emissions) and coal burning (474 t, 24%) were

the largest components of anthropogenic emissions in 2010, followed by cement production (173 t), large-scale gold production (97 t), and consumer product waste (96 t). Gworek et al. (2016) also showed the similar levels of mercury emission (total emission = 5000–6600 t year^{-1}; anthropogenic emission = ~2200 t year^{-1}) by citing many papers. Asia contributes almost half of global anthropogenic emissions, accounting 40% of the total emission by East and Southeast Asia, in which China accounts three-quarters (UNEP 2013). Fu et al. (2015) estimated that the annual emission of anthropogenic gaseous elemental mercury (GEM) over the past decade in China was 632–1138 t and that anthropogenic GEM increased by 2.4–2.5% year^{-1}. Emissions of anthropogenic mercury to the air have peaked in the 1970s, have declined over the following two decades, and then have been stable between 1990 and 2005; however, since then, there were indications of increases in emissions from fossil fuel combustion and metal and cement production (UNEP 2013). It is widely recognized that increases in industrial mercury emissions in recent years result in elevated levels of mercury concentrations in the atmosphere.

The anthropogenic mercury emissions were considered to contribute significantly to the burden of mercury in precipitation from the findings that the increases of mercury in precipitation are global (Downs et al. 1998). Mercury in the atmosphere is transported into the aquatic environments through wet and dry depositions and then constitutes a main source of mercury to aquatic biota in the absence of point source discharges. On the other hand, UNEP (2013) listed mercury sources which are released directly into the aquatic environments, that is, point sources from industrial sites (185 t year^{-1} including 93 t year^{-1} of nonferrous metal production, 89 t year^{-1} of consumer product waste), sources from contaminated sites (8–33 t year^{-1}), artisanal and small-scale gold mining (800 t year^{-1}), and soil erosion following deforestation (800 t year^{-1}). These estimates remain uncertain; however, the total releases were calculated to be more than 1000 t year^{-1}, which exceeded estimated natural releases, 150–960 t year^{-1}. Amos et al. (2013) showed that atmosphere and surface ocean (0–54 m) are enriched by 2.6- and 2.3-fold, respectively, in comparison with the preindustrial (1840) reservoir mass using a simulation model.

UNEP (2013) also showed the pathways and fate of mercury in the oceans. Pathways by which mercury reaches the oceans include direct deposition from the atmosphere (3700 t in 2008), supply from hydrothermal vents (<600 t) or from groundwater, and remobilization from sediment (100–800 t) and inflow from rivers (380 t except for 2420 t which is trapped in estuaries). Anthropogenic emissions and releases over time have increased mercury loads in the oceans, resulting in the doubled amount of mercury in the top 100 m of the world's oceans in the last 100 years. Accumulated inorganic mercury can be converted into methylmercury by bacterial activities as described in Sect. 2.13. In freshwater and coastal environments, methylation occurs primarily in sediments, while in the open ocean, this takes place largely

at intermediate depths, between 200 m and 1000 m in the water column. Approximately 300 t of methylmercury is produced in the oceans in a year, 80 t is transported from other sources to the oceans, 240 t is removed by photochemical reaction or by microbial activity, and then 40 t per year is taken up into marine food webs.

3.2.2 Bioaccumulation of Mercury and Effects on Public Health

Among mercury species, methylmercury is accumulated selectively by phytoplankton, accounting $10^{5.5}$-fold bioaccumulation rate between water and phytoplankton, subsequently bioaccumulating methylmercury approximately by fourfold, and then followed by subsequent bioaccumulation to planktivorous fish at similar rate (Mason et al. 1995). Thus, the pathway of methylmercury to fish is dominated by the food chain, resulting in highest levels of methylmercury in top predators and in posing risks to humans through consumption of contaminated seafood.

Due to the increased anthropogenic emissions of mercury, mercury concentrations in Arctic marine animals have increased by 12 times since the preindustrial period (UNEP 2013). Downs et al. (1998) also described that established background levels for fish (0.2–1.0 μg g^{-1}) exceeded the preindustrial level of 0.15 μg g^{-1}, suggesting anthropogenic origin. Gworek et al. (2016) showed ranges of the mercury concentration in various sea fish collected from ten areas in the world. Maximum values obtained from six areas exceeded 0.5 μg g^{-1} dry wt, while the values from four areas exceeded 1.0 μg g^{-1}. These results suggest that the effects of anthropogenic mercury expanded over the whole world.

As described in the previous sections (2.3, 2.4 and 2.11), methylmercury is a neurotoxicant, and its severe toxicity to residents was first recognized in Minamata. They are considered to consume seafood containing high concentrations of methylmercury exceeding 10 ppm (μg g^{-1} dry wt) usually or 100 ppm occasionally, which are considered to be accumulated through the process of food chains. Since the late twentieth century, it has been recognized that elevated levels of mercury in normal environments pose increasing concerns on human health hazards by low-dose mercury exposure. Zahir et al. (2005) summarized the effects of low-dose mercury toxicity in Table 3.1. The effects extend over not only in the nervous system but also in motor, renal, cardiovascular, immune, and reproductive systems of all age groups. Among the effects listed, special concerns have been focused on mental retardation of fetuses and infants. Populations who routinely consume fish or particular fish species have high exposure to methylmercury. Mothers consuming diet containing methylmercury pass the toxicant to fetuses through the blood-brain barrier and to

Table 3.1 Effect of low-dose mercury toxicity on various organ systems (Zahir et al. 2005)

1. Nervous system	
Adults	Memory loss including Alzheimer-like dementia, deficit in attention, hypoesthesia, ataxia, dysarthria, subclinical finger tremor, impairment of hearing and vision, sensory disturbances, and increased fatigue
Children/infants	Deficit in language (late talking) and memory deficit in attention, and autism
2. Motor system	
Adults	Disruption of fine motor function, decreased muscular strength, and increased tiredness
Children/infants	Late walking
3. Renal system	Increases plasma creatinine level
4. Cardiovascular system	Alters normal cardiovascular homoeostasis
5. Immune system	Decreases overall immunity of the body, exacerbates lupus-like autoimmunity, multiple sclerosis, autoimmune thyroiditis, and atopic eczema
6. Reproductive system	Decreases rate of fertility in both males and females, and birth of abnormal offspring

infants through breast milk. Exposure of methylmercury to children under age 14 is two to three times high because of higher food intake per unit weight. The developing brain is susceptive and vulnerable to methylmercury; therefore, even low-dose exposure may damage the brain of fetuses and infants.

Biomarkers of methylmercury exposure, such as total mercury levels in hair or blood, are regarded as more accurate measures of human exposure than dietary assessment because methylmercury concentrations vary both between and within fish species and because recall of specific species may be imprecise (Groth 2010). Karagas et al. (2012) defined "low-dose mercury exposures" as mean measured mercury levels of <4 μg g^{-1} in maternal hair, 20 μg L^{-1} in cord blood, or <12 μg L^{-1} in adult blood.

Gribble et al. (2016) summarized major cohort studies examining early-life mercury exposure and neurodevelopment in children. Four of nine populations showed the positive association. First, in the Faroe Islands where residents consumed pilot whale meat, neurodevelopmental deficits (i.e., language, attention, memory, and visuospatial and motor functions) were found at birth and early school years. An upper limit for "safe" exposure in terms of the maternal hair mercury concentration was considered to be 10 ppm (Grandjean et al. 1997, 2014). Second, in Massachusetts, USA, higher mercury levels of maternal hair (the range of maternal hair mercury = 0.02–2.38 ppm) were associated with lower infant cognition at 6 months of age (Oken et al. 2005). Third, in Seychelles, an Indian Ocean archipelago, neurodevelopmental performance at 30 months infants decreased with increased methylmercury exposure (mean maternal hair methylmercury = 5.9 ppm), although in the initial studies (Davidson et al. 1998; Myers et al. 2003), no effects were observed

(Davidson et al. 2008). Lastly, in Italy, mercury in hair of 7-year-old children was fairly correlated with mercury in maternal hair at delivery and was strongly correlated with child's seafood consumption. Children born from mothers with hair mercury levels ≥2.0 ppm had lower intelligence quotient (IQ) than children born from women with lower mercury levels. (Deroma et al. 2013).

3.2.3 Tolerable Methylmercury Intake and Recommendations for Public Health

Since the 1980s when concerns about low-dose exposure were raised, a lot of efforts have been paid to identify the tolerable methylmercury intake for protecting human health. The US Environmental Protection Agency (EPA) requested the National Academy of Sciences to prepare recommendations on the appropriate reference dose for mercury exposure. Then, the National Research Council (2000) assessed preceding studies which examined children who experienced methylmercury exposures in utero at concentrations relevant to current and concluded that (1) neurodevelopmental deficits are the well-documented effects, (2) mercury concentrations in cord blood would be expected to correlate most closely with fetal brain mercury concentrations during late gestation, (3) the preferred estimate of a benchmark dose level (BMDL) is 58 ppb (μg L^{-1}) of mercury in cord blood (corresponding to 12 ppm Hg in hair) based primarily on the Faroe Islands study and secondary on the New Zealand and Seychelles studies, and (4) the value of EPA's current methylmercury exposure reference dose (RfD), 0.1 μg kg^{-1} d^{-1} (0.7 μg kg^{-1} $week^{-1}$), is a scientifically justifiable level for the protection of public health. RfD was calculated by

$$\mathrm{RfD} = \left(C \times b \times V\right) / \left(A \times f \times bw \times \mathrm{UF}\right),$$

where:

C = concentration in blood (44 μg L^{-1})
b = elimination constant (0.014 $days^{-1}$)
V = volume of blood in the body (5 L)
A = absorption factor (expressed as a unitless decimal fraction of 0.95)
f = fraction of daily intake taken up by blood (unitless, 0.05)
bw = body-weight default value of 60 kg for an adult female
UF = uncertainty factor: mathematical adjustments for reasons of safety when knowledge is incomplete (10)

On the other hand, the Joint Expert Committee on Food Additives and Contaminants (JECFA) which was organized by the Food and Agriculture Organization (FAO) and the World Health Organization (WHO) provided recommendations on methylmercury in 2003 as follows: (1) 14 μg kg^{-1} maternal hair mer-

cury was recommended as BMDL based on the Faroe Islands and Seychelles studies, and (2) a provisional tolerable weekly intake (PTWI) of 1.6 μg kg^{-1} $week^{-1}$ methylmercury was considered sufficient to protect developing fetuses (WHO 2004).

The Ministry of Health, Labour and Welfare, Japan (MHLW), requested a risk assessment of methylmercury in seafood to the Food Safety Committee (FSC) in the Cabinet Office. FSC (2005) calculated PTWI using the following equation:

$$\begin{aligned}\mathrm{PTWI} = \mathrm{RfD} \times 7 &= \left\{ \left(C \times b \times V \right) / \left(A \times f \times bw \times UF \right) \right\} \times 7 \\ &= \left\{ \left(44 \times 0.14 \times 5.4 \right) / \left(0.95 \times 0.05 \times 60 \times 4 \right) \right\} \times 7 \\ &= 2.0 \ \mu\mathrm{g\ kg^{-1}\ week^{-1}}\end{aligned}$$

The difference in PTWI among three organizations (EPA, 0.7 μg kg^{-1} $week^{-1}$; FAO/WHO, 1.6 μg kg^{-1} $week^{-1}$; MHLW, 2.0 μg kg^{-1} $week^{-1}$) resulted mainly from the values of the uncertainty factor adopted (10.0, 6.4, and 4.0, respectively). This means that the difference in the PTWI value reflects the difference of view to food safety among organizations, that is, EPA imposed a high standard for avoiding low-dose methylmercury hazards, whereas MHLW set a laxest standard.

MHLW (2005a) summarized data on the concentrations of total mercury in 529 food materials including 495 raw aquatic fish/animals and 34 processed sea-/freshwater food. As I explained in Sect. 2, we can evaluate the toxicity of aquatic organisms using values of total mercury, because methylmercury usually accounts for the majority of total mercury in aquatic organisms. Among these materials, 31 fish/animals show >1.0 ppm (μg g^{-1} wet wt) in the maximum value. Except two materials of which identification is unclear, data of 29 fish/animals are shown in Fig. 3.1. These materials consisted of 23 fish species/species groups, 5 whale species that belong to *Odontoceti*, and 1 crustacean species. All species are carnivores. Excluding American lobster, all species are large sized and piscivorous, indicating that they are the highest on the food chain and are ready to accumulate methylmercury. In Fig. 3.1, the highest value, 37.0 ppm (μg g^{-1} wet wt), was found in a specimen of the bottlenose dolphin (no. 1, *Tursiops truncatus*). This species also ranked at the top of the mean value (20.8 ppm). High maximum and mean values are also found in other whale species such as the short-finned pilot whale (no. 3, maximum = 8.9 ppm; mean = 7.1 ppm), the sperm whale (no. 5, 4.6 ppm, 2.1 ppm), the Baird's beaked whale (no. 9, 2.6 ppm, 1.2 ppm), and the Dall's porpoise (no. 25, 1.2 ppm, 1.0 ppm). Several marlin (nos. 2, 7, 11, 13) and tuna (nos. 2, 4, 6, 10, 17, 22) species almost always showed high mercury concentrations (marlin, range of maximum values = 1.7–9.3 ppm, range of mean values = 0.6–1.2 ppm; tuna, 1.3–6.1 ppm, 0.2–0.7 ppm). Similar to these fish, two species belonging to the genus *Scomberomorus* (nos. 14, 15) which actively swim in the pelagic zone showed high mercury concentrations (max = 1.6–1.7 ppm, mean = 0.3–0.7 ppm). On the other hand, there are a variety of demersal/sedentary fish which have high mercury concentrations, such as tilefish (no. 6, max = 3.7 ppm, mean = 1.2 ppm), the splendid alfonsino (no. 12, max = 2.2 ppm, mean = 0.7 ppm), three species of grouper (nos. 23, 24, 29, max = 1.0–1.3 ppm, mean = 0.5–1.1 ppm), and two species of halibut (nos. 16, 27,

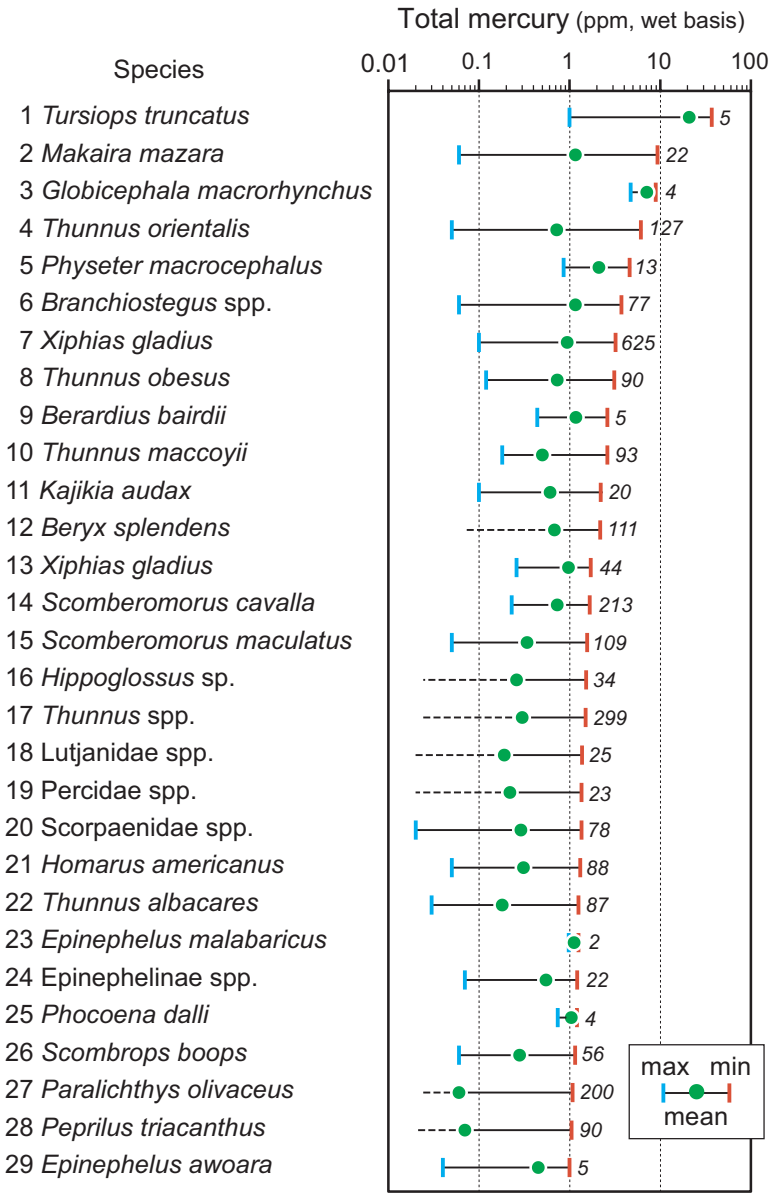

Fig. 3.1 Aquatic organisms collected from waters around Japan and other localities, showing high levels in the maximum value (>1.0 ppm = μg g^{-1} wet wt). Minimum, maximum, and mean values and the sample size (italics in the figure) are indicated. Solid lines show the range of values, while broken lines are used when minimum values are unknown. Among the numbers that precede, the scientific names in the figure, nos. 1–5, 8–10, 12, 13, 22, 23, 25–27, and 29 animals, were collected from waters around Japan, while nos. 6, 7, 11, 14–21, 24, and 28 animals were collected from other oceans. English names are as follows: 1, bottlenose dolphin; 2, Indo-Pacific blue marlin; 3, short-finned pilot shale; 4, bluefin tuna; 5, sperm whale; 6, tilefish; 7, swordfish; 8, bigeye tuna; 9, Baird's beaked whale; 10, southern bluefin tuna; 11, marlin; 12, alfonsino; 13, swordfish; 14, king mackerel; 15, Spanish mackerel; 16, halibut; 17, tuna; 18, snapper; 19, perch (freshwater); 20, scorpionfish; 21, American lobster; 22, yellowfin tuna; 23, Malabar grouper; 24, grouper; 25, Dall's porpoise; 26, gnomefish; 27, bastard halibut; 28, butterfish; and 29, banded grouper (Data source: MHLW 2005a)

Table 3.2 Three kinds of aquatic products categorized by FDA and EPA, USA, for avoiding low-dose methylmercury hazards (EPA 2007)

A. Best choices
Anchovy, Atlantic croaker, Atlantic mackerel, black sea bass, butterfish, catfish, clam, cod, crab, crawfish founder, haddock, hake, herring, lobster (American and spiny), mullet, oyster, Pacific chub mackerel, perch (freshwater and ocean), pickerel, plaice, pollock, salmon, sardine, scallop, shad, shrimp, skate, smelt, sole, squid, tilapia, trout (freshwater), tuna (canned light, including skipjack), whitefish, whiting
B. Good choices
Bluefish, buffalo fish, carp, Chilean sea bass/Patagonian toothfish, grouper, halibut, mahi mahi/dolphinfish, monkfish, rockfish, sablefish, sheepshead, snapper, Spanish mackerel, striped bass (ocean), tilefish (Atlantic Ocean), albacore tuna/white tuna (canned and fresh/frozen), yellowfin tuna, weakfish/sea trout, white croaker/Pacific croaker
C. Choices to avoid
King mackerel, marlin, orange roughy, shark, swordfish, tilefish (Gulf of Mexico), bigeye tuna

max = 1.1–1.5 ppm). Among these species, the splendid alfonsino is known as a deepwater fish (depth, > 200 m), while perch (no. 19, max = 1.4 ppm) is a freshwater fish. These data show that carnivorous aquatic animals can accumulate methylmercury irrespective of their habitat and the taxonomic group to which they belong.

Based on the results on mercury concentrations in aquatic products, the Food and Drug Administration (FDA) and EPA in the USA issued advice regarding amounts of seafood ingested (EPA 2007). This advice was geared toward helping women who are pregnant or may become pregnant as well as breastfeeding mothers and parents of young children. FDA/EPA categorized aquatic products into three groups (Table 3.2) and recommended that (1) people can eat 227 g to 340 g a week of fish in the "best choices" category, (2) people can eat 113 g a week of fish in the "good choices" category, and (3) people should not eat fish in the "choices to avoid" category or feed them to young children.

The Japanese government published a document about the mercury risk of consuming seafood (MHLW 2005b). The summary is as follows: (1) fish and shellfish are an important diet; (2) mercury contaminations in fish and shellfish are very small, but some species contain higher levels in the mercury concentration; (3) reported mercury effects on fetuses are minor ("no more than degree" in original), for example, auditory response is delayed by less than one-thousandth second, and its intake through normal meals will not affect the baby's future life; (4) this advice is intended not to ask pregnant women to avoid fish and shellfish that contain high levels of mercury; and (5) women who are or may be pregnant should follow the recommendations in order to reduce the health risk to the minimum. MHLW (2005b) also categorized aquatic products having risks into four groups (A–D, Table 3.3) mainly based on mean values shown in Fig. 3.1 as well as on 2.0 μg kg^{-1} $week^{-1}$ of PTWI which was established by themselves (FSC 2005). The objects of the recommendations are women who are or may be pregnant, while lactating women, infants, children, and adults are removed from consideration, because MHLW (2005b) considered that they are not affected by normal consumption of any aquatic food. The

Table 3.3 Recommendations for pregnant women on consumption of aquatic products concerning mercury contamination by MHLW (2005b)

Target species	Recommendations
A. Bottlenose dolphin (1)	
Pregnant women should restrict the consumption of meat within 80 g (average amount per one meal) per 2 months	
B. Short-finned pilot whale (2)	
Pregnant women should restrict the consumption of meat within 80 g per 2 weeks.	
C. Alfonsino (12), swordfish (7), bluefin tuna (4), bigeye tuna (8), finely-striate *Buccinum* (30), Baird' beaked whale (9), sperm whale (5)	
Pregnant women should restrict the consumption of meat within 80 g per 1 week.	
D. Yellowback seabream (31), marlin (11), Hilgendorf saucord (32), southern bluefin tuna (10), blue shark (33), Dall's porpoise (25), Japanese bluefish (34)	
Pregnant women should restrict the consumption of meat within 160 g per 1 week.	

See Fig. 3.1 for scientific names of species that are assigned numbers from 1 to 25 in parentheses. Scientific names of species that are assigned numbers 30–34 are as follows: 30, *Buccinum striatissimum*; 31, *Dentex tumifrons*; 32, *Helicolenus hilgendorfii*; 33, *Prionace glauca*; and 34, *Scombrops gilberti*

observed maximum concentrations of whales, marlins, and tunas, however, were at the same level as those found in Minamata around 1960, suggesting that if these food was consumed by humans freely, even adults would become the acute type of MPM. The document includes the sentence "you can eat other tune species (yellowfin tuna, Albacore and juvenile bluefin tuna) than listed ones and canned tuna without any particular restriction." Reasoning of this recommendation is supposed to be relatively low mean values of the mercury concentration in these species; however, we can find a high maximum value, 1.25 ppm, in yellowfin tuna. The observed mean and maximum values are usually affected by the sample size and locality collected; we should be careful when listing risky species. Thus, the recommendation of MHLW seems to emphasize the advantage of consuming aquatic food and to dispel concerns about consuming aquatic food by underrepresenting the risk of mercury. Should we think to follow international standards not the laxest standard (2.0 μg kg^{-1} $week^{-1}$) that MHLW established by themselves, to list risky aquatic organisms not at a species level but at a trophic level, and to recommend avoiding the consumption of highly risky organisms during the gestational period?

Consumption of seafood provides risks and benefits for humans. Seafood is the primary source of protein for many populations. It has many nutrients including eicosapentaenoic acid (EPA) and docosahexaenoic acid (DHA), which give benefit by reducing heart diseases. Therefore, balanced assessments of the risks and benefits of seafood are required. In general, as trophic level increases, methylmercury

concentrations in marine animals increase, whereas there is no relationship between trophic level and nutrient concentrations, suggesting that consuming lower trophic level seafood can minimize the risk of mercury exposure without reducing the benefits of nutrient intake.

As noted in Sect. 3.2.2, there is competent evidence for the fetal neurotoxicity of methylmercury at low-dose exposure (Gribble et al. 2016). That is, the positive association of mercury with neurodevelopment was found in four of nine populations, while five populations did not show any evidence (Kjellström et al. 1989; Davidson et al. 1998; Myers et al. 2003; Valent et al. 2013; Tatsuta et al. 2014; Strain et al. 2015). Further studies are necessary to clarify the relationship between low-dose mercury exposure and health hazards and to make appropriate suggestions regarding the acceptable dose level.

3.3 Outstanding Issues

Dr. Masazumi Harada, who contributed to the study on MPM as well as to MPM patient care, published a book entitled *Minamata Disease Not Settled Yet* in 1985. In those days, many patients were not acknowledged officially and sought redress from the court. As of 2017, legal battles over MPM still persist. This is mainly due to a strict criterion for diagnosing MPM which was established by the national government in 1977. In this criterion, multiple symptoms among (i) sensory disturbance, (ii) ataxia, (iii) equilibrium disturbance, (iv) visual field constriction, (v) central visual disturbance, (vi) central auditory disturbance, and (ii) others are needed for the diagnosis of MPM, resulting in discarding many patients. Such a conflict seems to be caused from a lack of understanding the whole context of victims through detailed surveys by the government. Urgent and thorough surveys are necessary, although long overdue. Besides, pathology of MPM should be elucidated further. If methylmercury damages the vascular system and/or acts as an endocrine-disrupting agent as suggested by Shiraki (1979), lesions will extend over the whole body resulting in a variety of symptoms, and even tiny amounts of methylmercury may affect the whole human body. Such studies may provide a useful clue as to resolving low-dose mercury issues in the global mercury cycle.

Gworek et al. (2016) described that the average residence time of mercury in oceanic waters, 20–30 years, is much longer than that in the atmosphere (0.8–2 years), resulting in that the mercury discharged into the ocean is removed from there much more slowly than the mercury emitted into the atmosphere and that an increase in the mercury concentration level in oceanic waters will be very slow and may take hundreds of years. This fact means that the mercury concentration will continue to increase gradually, even if anthropogenic emissions of mercury to the atmosphere stop right now. Methylmercury concentrations in some aquatic animals have reached the threshold level to cause neurodevelopmental deficits in fetuses and infants already; therefore, we have to minimize the mercury emissions as soon as possible.

In this context, it is gratifying that the "Minamata Convention on Mercury" entered in force on August 16, 2017. Its major highlights include a ban on new mercury mines, the phasing out of existing ones, the phasing out and phasing down of mercury use in a number of products and processes, control measures on emissions to air and on releases to land and water, and the regulation of the informal sector of artisanal and small-scale gold mining.

An American photographer, William Eugene Smith, took excellent pictures of MPM patients. Among these, a picture titled "Tomoko Uemura in Her Bath," which depicted a mother cradling her severely deformed, naked daughter in a traditional Japanese bathroom, is impressive. The mother called Tomoko as "Takara-ko" in Japanese which means "treasure daughter" for the reason that the mother was able to keep her health because Tomoko had drawn all mercury and accumulated it into Tomoko's own body and that her six children grew up healthy and helped each other as a result of learning the importance of mercy from assisting their elder sister (Harada 1985). Tomoko passed away on December 12, 1977, at the age of 20. We can provide a description of the assailant who took the life of Tomoko. On the other hand, presently, we may be living in the world full of toxicants including mercury. In this world, we have a high probability of becoming a victim, and at the same time, we can be an assailant. Let's consider what we can do under such circumstances.

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