

## Methylmercury and Human Embryonic Development <sup>[1]</sup>

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Methylmercury (MeHg) is an organic form of mercury that can damage the developing brains of human fetuses. Women who consume methylmercury during [pregnancy](#) <sup>[2]</sup> can bear children who have neurological issues because methylmercury has toxic effects on the nervous system during embryonic development. During the third week of [gestation](#) <sup>[3]</sup>, the human nervous system begins to form in the embryo. During this gestational period, the embryo's nervous system is particularly susceptible to the influence of neurotoxins like methylmercury that can result in abnormalities. Furthermore, the fetal brain can incur damage despite the lack of signs of poisoning in the pregnant woman. In children, defects due to methylmercury can result in deficits in attention, behavior, cognition, and motor skills.

Methylmercury has both natural and synthetic sources. Natural sources of mercury include volcanic emissions, geologic deposits, and oceanic evaporation. Humans have introduced mercury into the environment with alkali and metal processing, coal-burning, medical waste, and gold and mercury mining. When mercury is released into the air, it circulates in the atmosphere until it is brought down through rain or snowfall, and then it can flow into bodies of water like lakes and streams.

Once in water, bacteria transform the mercury into its more toxic form, methylmercury. In this form, it is absorbed by aquatic plants and animals. Fish can absorb methylmercury from water through their gills and by eating other [fish](#) <sup>[4]</sup> that possess concentrations of methylmercury. Consequently, the concentrations of methylmercury in exposed fish's tissues increases over time, with [fish](#) <sup>[4]</sup> high on the food chain possessing higher concentrations of methylmercury than [fish](#) <sup>[4]</sup> low on the food chain.

Humans expose themselves to methylmercury when they eat contaminated [fish](#) <sup>[4]</sup>. When pregnant women eat contaminated [fish](#) <sup>[4]</sup>, their embryos or fetuses also are exposed to the methylmercury, and they can develop congenital Minamata disease. In 1959, researchers attributed Minamata symptoms to poisoning by methylmercury found in the water of Minamata Bay in Kyushu, Japan. Congenital Minamata disease enabled researchers to identify the poisonous effects of methylmercury, which those near Kyushu consumed when they ate local seafood. Symptoms associated with Minamata disease include: lack of eye coordination, convulsions, neck instability, mental [retardation](#) <sup>[5]</sup>, reflex, growth, and cerebellar deficits, hyperkinesis, hypersalivation, hyperkinesia, dysarthria, strabismus, [microcephaly](#) <sup>[6]</sup>, and paroxysmal symptoms. The congenital disease gets its name from the Minamata Bay, where many cases of intrauterine methylmercury poisoning occurred due to the contamination of water by a nearby acetaldehyde factory of the Chisso Corporation.

The contamination of the water began in 1932 when the factory discharged wastewater that contained methylmercury into Minamata Bay, a practice they continued until 1968. It took approximately ten years from the time of the initial investigations into the causes of the disease for the Japanese government to officially endorse the causal relationship between the

wastewater containing mercury dumped into the bay Chisso factory and Minamata disease, thereby compelling Chisso to cease dumping methylmercury into the water.

Many neurophysiological and mental conditions are associated with prenatal exposure to methylmercury. Hideyo Matsumoto, Goyo Koya, and Tadao Takeuchi at Kumamoto University in Kumamoto, Japan, described two case reports of infants from the Minamata Bay area with cerebral palsy that had been identified in 1960 as being afflicted with Minamata disease. In their clinical and pathological findings, conducted while the infants lived and after they died, the researchers described the infants as underdeveloped, unable to move purposefully, having poor mental development, and experiencing convulsions. At the time that the infants were observed, one child was one year and four months old and the other child was three years and seven months old. The children died at two years and six months old, and six years and three months old, respectively.

Matsumoto, Koya, and Takeuchi observed how methylmercury impacted the [central nervous system](#) [7] and degenerated and decreased the number of neurons in the brains of those afflicted. In particular, the researchers noted a disappearance of granule and pyramidal cells, elimination of [Purkinje cells](#) [8], a narrowing of the molecular layer, reduced central [white matter](#) [9], diffuse atrophy of the folia in the [cerebellum](#) [10]'s hemispheres, and lack of myelination in the pons and medulla oblongata both areas of the [hindbrain](#) [11]. [Purkinje cells](#) [8] are necessary for the functioning of the [cerebellum](#) [10], which coordinates movements and maintains posture. Methylmercury prenatal poisoning yields babies with symptoms similar to those in individuals with cerebral palsy.

From 1971 to 1972, a population in rural Iraq experienced methylmercury poisoning after they consumed bread tainted with the chemical. Case studies conducted in the area detailed the neurological impact of methylmercury on infant children who had been exposed to methylmercury as embryos. In 1972, Ben Choi, Lowell Lapham, Laman Amin-Zaki, and T. Salam described the pathological changes in the brain of two infants born during the outbreak of methylmercury poisoning. The researchers were doctors from the University of Baghdad in Baghdad, Iraq, and from the University of Rochester Medical Center in Rochester, New York. They reported that one infant died after thirty-three days, and the other soon after birth. Both infants had high methylmercury content in their blood. The team's post mortem examinations of the infants' brains revealed widespread abnormal neuronal migration in the cerebral and cerebellar [white matter](#) [9]. They also found disorganization and misalignment of cerebral cortex neurons.

In 1973, Laman Amin-Zaki, Sami Elhassam, Mohamed A. Majeed, Thomas W. Clarkson, Richard A. Doherty, and Michael Greenwood examined the clinical manifestations of methylmercury poisoning in six of fifteen mother-infant pairs. Those researchers also were from the University of Baghdad and the University of Rochester. The infants they studied had been exposed to methylmercury *in utero* as embryos after their mothers had eaten bread contaminated with methylmercury during the early stages of [pregnancy](#) [2]. The researchers collected blood tests of both the mothers and the infants to measure the extent to which they had been exposed to methylmercury. They found that the infants had higher concentrations of methylmercury in their blood than did their mothers, indicating that methylmercury easily transferred from pregnant women to their fetuses or embryos. They noted that the infants with prenatal exposure to methylmercury displayed fretfulness, irritability and excessive crying, impaired vision or blindness, impaired hearing, weak muscles, and impairment of mental development. They also noted that the mothers displayed symptoms of poisoning.

From 1986 to 1987, a research team studied methylmercury concentrations in the blood of umbilical cords. The team included Philippe Grandjean, Pal Weihe, Roberta White, Frodi Debes, Shunichi Araki, Kazuhito Yokoyama, Katsuyuki Murata, Nicolina Sorensen, Rasmus Dahl, and Poul Jorgensen. The researchers were from [Boston University](#) [12] in Boston Massachusetts, Tokyo University in Tokyo, Japan, Odense University in Odense, Denmark, and some were government researchers from Denmark and from the Faroe Islands. The team assembled a cohort of 1022 consecutive singleton births in the Faroe Islands. They had 917 of the seven year-olds undergo neurobehavioral examinations. The researchers used [umbilical cord](#) [13] blood to discern mercury concentration. They found that mercury in the cord blood was significantly related to lower scores on attention, language, and memory tests, and to a lesser degree visuo-spatial and motor functions involving coordination, speed, and tactile processing.

In the early 2000s, a team of US researchers studied the impact of methylmercury on intelligence. The team included Daniel Axelrad with the US Environmental Protection Agency (EPA) headquartered in Washington D.C., David Bellinger, Louise Ryan from [Harvard University](#) [14] in Cambridge, Massachusetts, and Tracey Woodruff from the EPA in San Francisco, California. The team tested neurologic functions to assess childhood intelligence quotients (IQs) of six, seven, and nine year-olds from the Faroe Islands, from New Zealand, and from Seychelles study cohorts. They observed a decreases in IQ of 0.18 IQ points for every part per million mercury in maternal hair.

In addition to the case studies, to further investigate the mechanisms by which methylmercury disrupts embryonic [neural development](#) [15], researchers experimented with methylmercury and animal embryos. In 2000, Kyoko Miura, Seiichiro Himeno, Nori Koide, and Nobumasa Imura from Wako University and Kitasato University, both in Tokyo, Japan, found that [chick](#) [16] embryos exposed to methylmercury had less than normal microtubule mass in nerve fibers. Methylmercury also altered surface membrane structure, and it inhibited nerve fiber growth in the dorsal root ganglion, a cluster of neural bodies that carry signals to the spinal cord in [chick](#) [16] embryos. In 2002, a team led by Elaine Faustman from the University of Washington in Seattle, Washington, used primary [rat](#) [17] embryo [central nervous system](#) [7] (CNS) cells and exposed them to methylmercury. Those cells could not transition to different stages of the cell cycle, and they died.

Two years later, researchers from various universities in Italy observed that [chick](#) [16] embryos

exposed to methylmercury had degenerative damage on [Purkinje cells](#) [8], [granule cells](#) [18], and astrocytes, the latter of which function in the CNS's function and development, along with impairment of the blood-brain-barrier. In a study published in 2006, a group of researchers throughout the US examined the influence of methylmercury on neuronal cells in rats. They noted that methylmercury altered the cell cycle, preventing cerebellar and cortical neurons from dividing and proliferating.

Also in 2006, Christin Bland and Matthew Rand at the University of Vermont in Burlington, Vermont, showed that methylmercury alters Notch signaling between developing [nerve cells](#) [19] in used fruit fly embryos. The Notch signaling pathway is a mechanism within and between cells in developing organisms that affects cell fate, proliferation, migration, and neurite growth. Also in 2006, Christoffer Tamm's research team at Uppsala University in Uppsala, Sweden, used a neural stem cell line from mice and primary embryonic cortical neural [stem cells](#) [20] from rats to investigate the effect of methylmercury on those cells. The team found that when exposed to methylmercury, the cells die, which then reduces the number of progenitor cells in a cell population.

In 2008, researchers from several universities across Brazil and the US observed that in mice exposed to methylmercury as embryos had impaired Glutathione (GSH) antioxidant systems as newborns. This effect can damage a newborn's aerobic metabolism. Researchers from Egypt and the US observed in 2011 that zebrafish embryos exposed to methylmercury at low concentrations had delayed neuronal development, whereas higher concentrations killed the embryos, which failed to develop complete neural tubes. Then in 2014, Gregory Engel and Rand at the University of Vermont, using fruit fly embryos, discerned that methylmercury disrupts the production of a key protein in developing muscles, which then disrupts the signals between motor neurons and those muscles.

In 2004, the US Federal Drug Administration, headquartered in Silver Spring, Maryland, and the US Environmental Protection Agency, headquartered in Washington D.C., issued a joint statement. The statement advised people that to prevent adverse exposure to methylmercury, people should eat no more than twelve ounces of commercially-caught [fish](#) [4] per week and no more than six ounces of locally-caught [fish](#) [4] per week.

Prenatal exposure to methylmercury can produce various harmful neurological effects during embryonic development, and consumption of [fish](#) [4] contaminated with mercury is the primary cause of human ingestion of methylmercury. By the early decades of the twenty-first century, researchers had yet to determine, a dose-effect but they noted that people should reduce their exposure to methylmercury. The neurotoxin can transfer to an embryo through the [placenta](#) [21] and because the embryo is highly susceptible to neurotoxicity, the damage to the developing nervous system *in utero* can be severe.

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## Subject

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Articles <sup>[56]</sup>

## Last Modified

Wednesday, July 4, 2018 - 04:40

## DC Date Accessioned

Monday, April 18, 2016 - 07:00

## DC Date Available

Monday, April 18, 2016 - 07:00

## DC Date Created

2016-04-18

## DC Date Created Standard

Monday, March 7, 2016 - 07:00

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- [55] <https://embryo.asu.edu/topics/disorders>
- [56] <https://embryo.asu.edu/formats/articles>