MERCURY AND THE DEVELOPING BRAIN

CLEAR THE AIR

Mercury and the Developing Brain

Introduction

Children are most vulnerable to mercury exposure, whether exposed in utero or as young children. Mercury affects the developing brain, causing neurological problems that manifest themselves as vision and hearing difficulties, delays in the development of motor skills and language acquisition, and later, lowered IQ points, problems with memory and attention deficits. These developmental deficits may translate into a wide range of learning difficulties once children are in school.

This report explains the sources of mercury in the environment and how people are exposed. The physical changes that occur in the developing brain due to mercury exposure during pregnancy are described along with how these changes later translate into learning difficulties in school. The report estimates the societal and economic impacts of mercury exposure in terms of the cost of special education in the U.S. and the societal benefits of reducing mercury emissions.

The information in this report is timely. Numerous policy options are being considered by state, federal and international lawmakers to reduce mercury emissions to the environment. Stringent regulations, implemented as quickly as possible, must be enacted to help reduce the level of mercury exposure to children.

Methylmercury Exposure in the U.S.

Methylmercury is a neurotoxin – a substance that damages, destroys, or impairs the functioning of nerve tissue. In the U.S., the general population is exposed to various forms of mercury through inhalation, consumption of contaminated food or water, and exposure to substances containing mercury, such as vaccines. Different chemical types of mercury can adversely affect several organ systems, with the severity of effects depending largely on the magnitude and timing of the exposure (i.e., during fetal development or as a child or adult).[1] Outside of occupational settings, methylmercury is the most toxic form of mercury to which humans are regularly exposed and this form of mercury is the focus of the health impacts discussed in this report. Exposure to methylmercury in the U.S. is almost exclusively from eating fish and shellfish.

Methylmercury poses the greatest hazard to the developing fetus. It passes easily through the placenta and impairs the development of the brain and nervous system. Prenatal methylmercury exposure from maternal consumption of fish can cause later neurodevelopmental effects in children.[2] Infants appear normal during the first few months of life, but later display subtle effects. These effects include poor performance on neurobehavioral tests, particularly on tests of attention, fine motor function, language, visual-spatial abilities (e.g., drawing) and memory. These children will likely have to struggle to keep up in school and might require remedial classes or special education.[3]

Methylmercury exposure prior to pregnancy is as critical as exposure during pregnancy because methylmercury stays in the body for

Where Does Methylmercury in Fish Come From?

Mercury (Hg) is emitted into the atmosphere from both natural (e.g., volcanic activity) and industrial sources, with the emissions from industrial sources far exceeding the natural sources. In the U.S., coal-fired power plants are the largest unregulated source of mercury emissions by far, responsible for approximately 41 percent of the country's industrial emissions. Other large domestic sources of mercury emissions like municipal waste combustors have already become subject to regulation. Because of this, the proportion of U.S. mercury emissions attributable to coal-burning power plants is increasing. Moreover, in the absence of controls and with increasing energy production and coal use projected for the U.S., mercury emissions from these sources will increase.

Mercury is converted to other forms of mercury in the atmosphere and ultimately is deposited from the atmosphere in rain and snow and by being attached to small particles. (Figure 1.) After being deposited, mercury can be converted by bacteria in the aquatic ecosystem to methylmercury, a form that is especially toxic to humans and wildlife. Fish absorb methylmercury from the water as it passes over their gills and as they feed on other organisms. As larger fish eat smaller ones, methylmercury concentrations increase in the bigger fish, a process known as bioaccumulation.

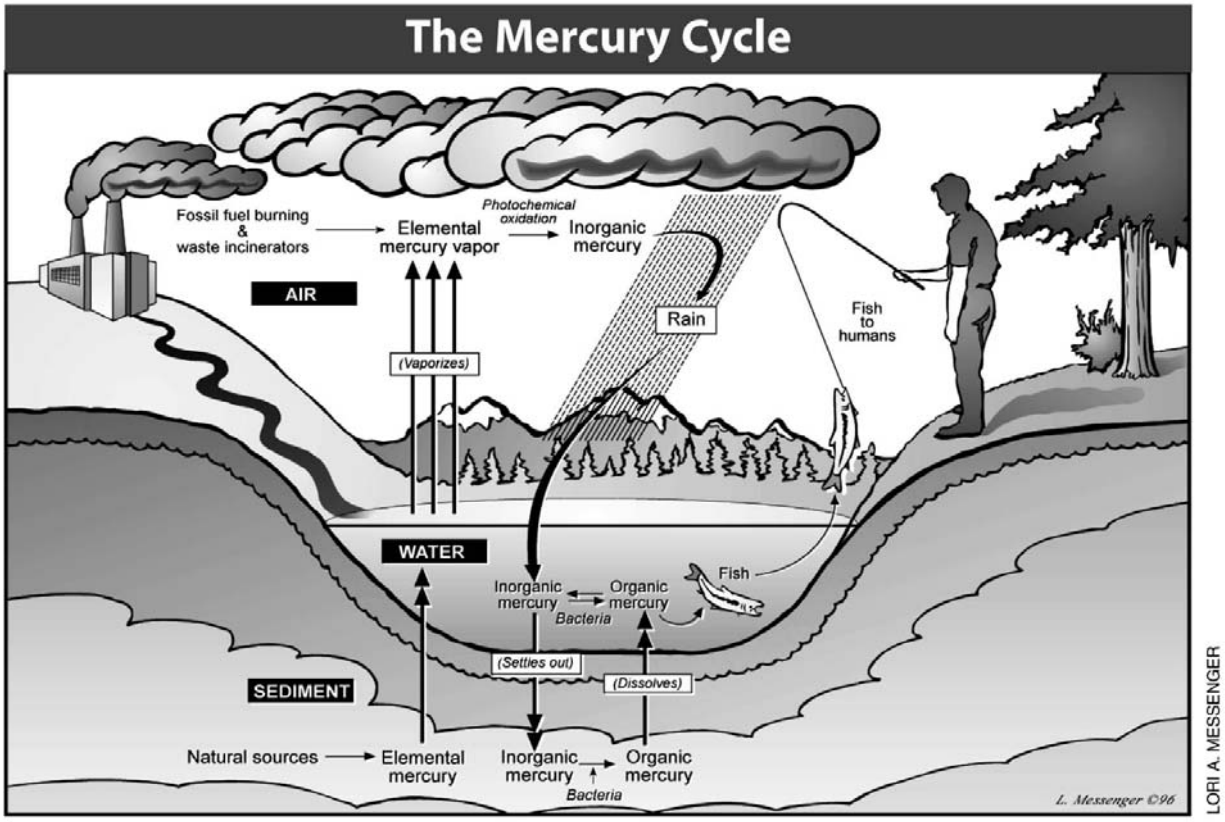
Consequently, larger predator fish usually have higher concentrations of methylmercury from eating contaminated prey. Humans, birds and other wildlife that eat fish are exposed to methylmercury in this way.

months and is slowly excreted. Many of the critical stages of brain and nervous system development occur during the first two months after conception and since many women do not know they are pregnant during that time, the fetus may be exposed to high levels of methylmercury. Because of the risk methylmercury poses to the developing fetus, women of childbearing age (i.e., 15 to 44 years of age) who might become pregnant and pregnant women are the most important members of the population in terms of mercury exposure.[4]

Infants and children are also at risk. Infants may ingest methylmercury from breast milk and children are exposed through their diet. Children and infants may be more sensitive to the effects of methylmercury because their nervous systems continue to develop until about age 16. Children also have higher methylmercury exposures than adults because a child eats more food relative to his or her body weight than an adult does. As a result, they have a higher risk for adverse health effects.[5]

There is also evidence in humans and animals that exposure to methylmercury can have adverse effects on the developing and adult cardiovascular system, blood pressure regulation, heart-rate variability, and heart disease.[6]

Figure 1. The Mercury Cycle

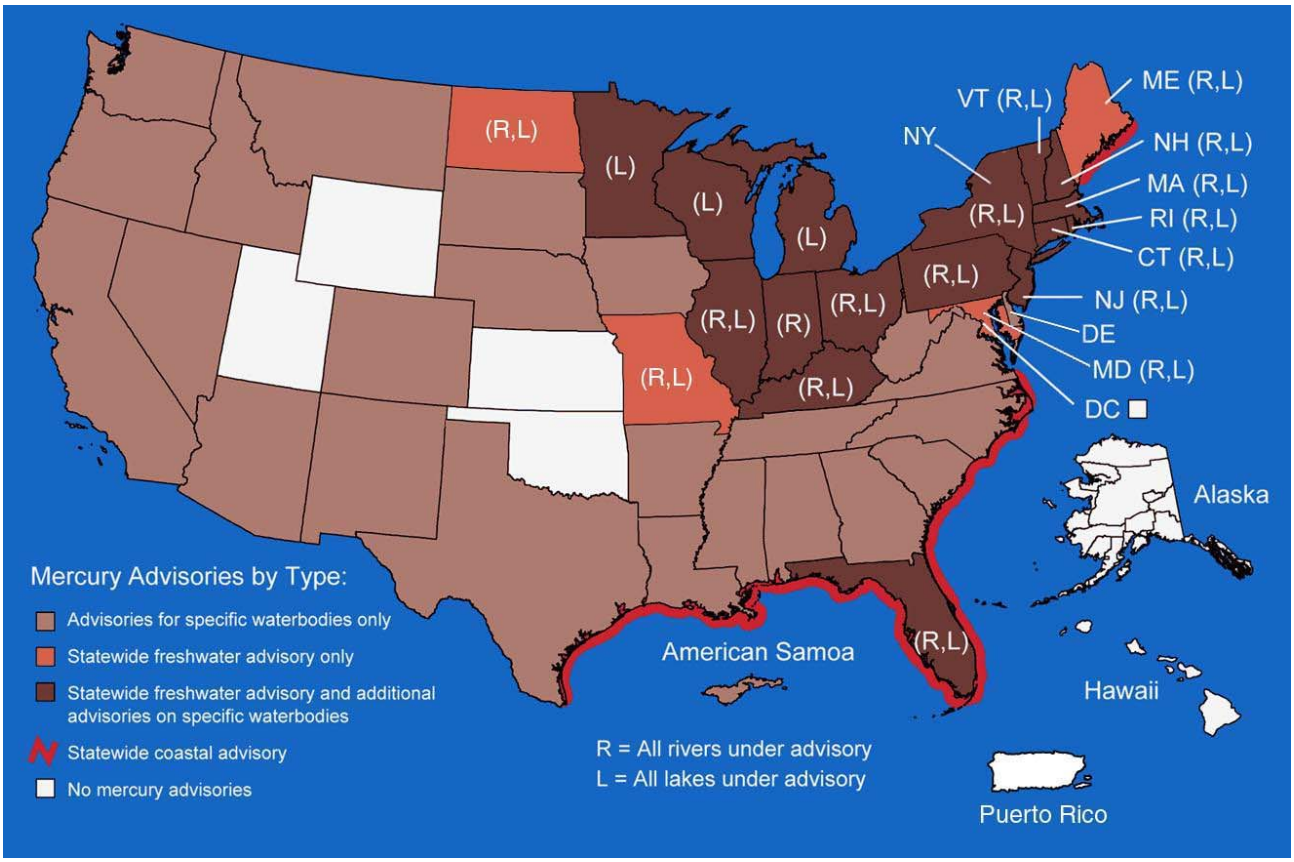


Elevated levels of methylmercury in fish have prompted concerns about the public health hazards from methylmercury exposure. Despite the known nutritional and health benefits from eating fish, in 2003, public health agencies in 45 states issued fish consumption advisories warning citizens to limit how often they eat certain types of fish because the fish are contaminated with high levels of mercury.[7] Twenty-one states have issued mercury advisories for fish in *every inland lake or river*.[8] (Figure 2.)

The U.S. Food and Drug Administration and EPA specifically warn pregnant women, women of childbearing age, nursing mothers, and young children not to eat shark, swordfish, king mackerel, or tilefish. The advisory warns the same populations to limit their consumption of albacore "white tuna" or tuna steaks to six ounces or less per week and fish that have lower levels of mercury, such as shrimp, canned light tuna, salmon, pollock, and catfish, to 12 ounces or less per week. (Six ounces of fish is an average cooked meal, about the size of a can of tuna.[9])

In 2004, EPA indicated that 1 in 6 women of childbearing age has mercury levels in her blood above EPA's current health threshold.[10] Nationally, this means that as many as 630,000 of the four million babies born each year – or 15 out of every 100 babies - are at risk of developmental problems due to mercury exposure in utero.[11]

Figure 2. Mercury Fish Consumption Advisories



Methylmercury and Brain Development

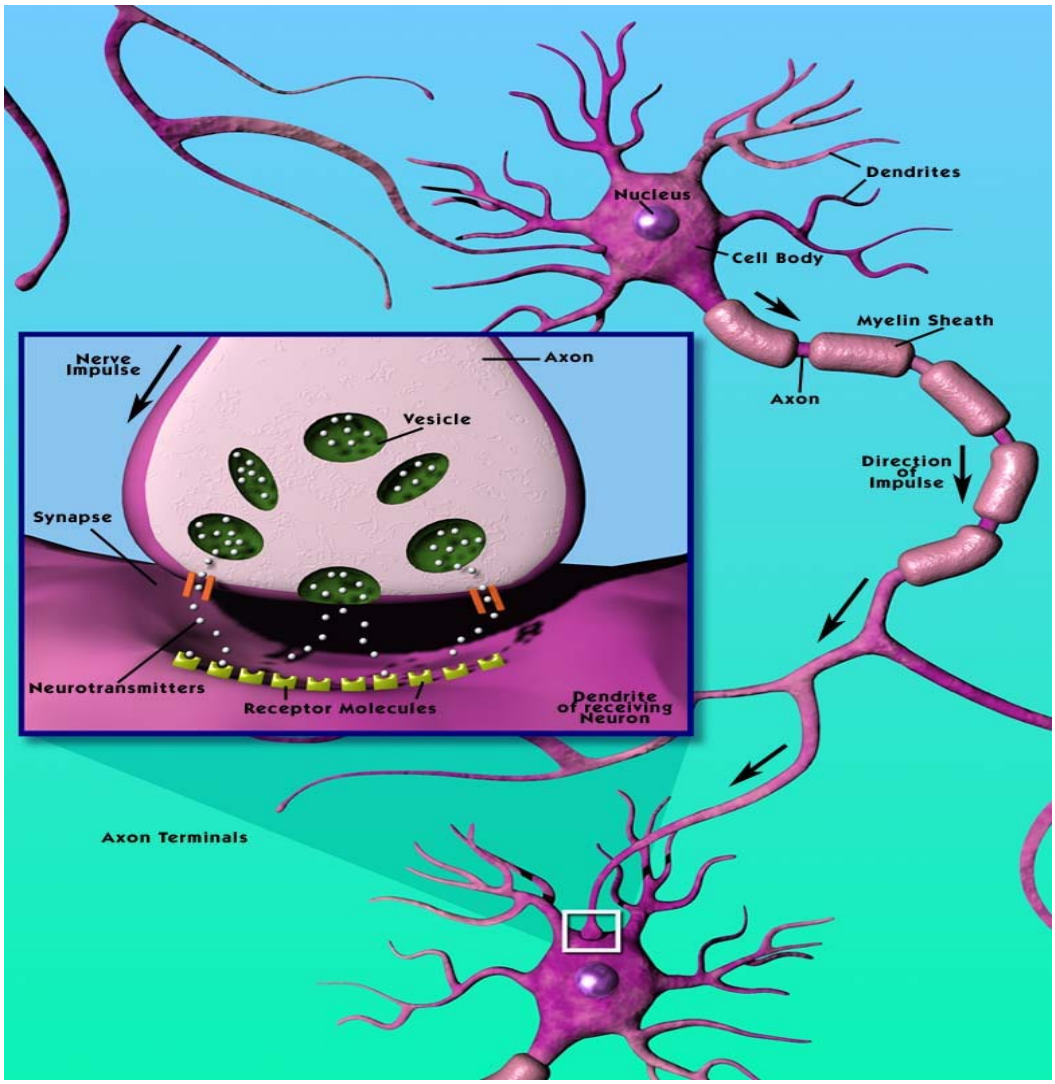
Understanding how the brain forms is essential to our understanding of how methylmercury affects brain development. The detrimental effects of methylmercury exposure that become evident in a young child are thought to result from changes that occur during the formation and organization of brain cells in the developing embryo.

The fully formed brain, with its elaborate and precisely balanced network of at least 100 billion nerve cells, begins developing a mere three to four weeks after conception. During the nine months of pregnancy, the production of nerve cells is so great that at times as many as 50,000 cells are created each second to provide the necessary number for the adult brain. Brain development is so complex that at least half of our genetic code is devoted to producing this organ. While genetics largely determine brain development, environmental factors are also involved from almost the beginning of embryonic life. These environmental factors include the health and nutritional status of the mother and her contact with potentially toxic substances like alcohol, tobacco and other drugs. Methylmercury exposure at this critical time of fetal growth is also an environmental factor that can have a detrimental effect on brain development.

Neurons Wire the Brain

The building block of the brain and central nervous system is the nerve cell, or neuron. A neuron is a specialized cell that transmits information to other nerve cells, muscles or glands. (Figure 3.) Neurons consist of a cell body containing the nucleus and an axon, an electricity-conducting fiber that conducts the nerve signal away from the cell body. The axon gives rise to many smaller axon branches before ending at nerve terminals. Another extension of the cell body includes dendrites, which extend from the neuron cell body like branches of a tree and receive the incoming signal from other neurons. Their function is to conduct a nerve impulse toward the cell body. Synapses are the actual gaps between neurons through which nerve impulses travel, like the gap in spark plugs. Hollow tubes called microtubules run the length of the neuron and function not only as the "skeleton" or supporting structure for the cell but act as conveyor belts for transporting cellular components within the cell.

Figure 3. The Healthy Neuron



How Neurons Work

Neurons signal by transmitting electrical impulses along their axons. A layer of specialized fatty cells produce the myelin sheath, which insulates the axon and helps speed the transmission of electrical impulses. The electrical impulses are transmitted at speeds up to several hundred miles per hour. The impulses are formed by a complex interaction of electrically charged sodium and potassium atoms, which move in and out of the neuron through the cell membrane, creating a tiny voltage change. At the end of the axon, the voltage change causes the release of calcium atoms that in turn signal the release of specific chemical messengers, called neurotransmitters. The neurotransmitters cross the synapse and bind to receptors on the dendrite of the target neuron, starting an electrical impulse in it. Thus, the impulses are transmitted from neuron to neuron in a domino effect – faster than a lightening strike.

Neurons and Development of the Central Nervous System

Neurons are initially produced in the neural tube – the primitive brain that will form the hindbrain, midbrain and forebrain. The neurons produced in the neural tube migrate in a precise and complex sequence to a final destination in the brain which is determined both by genetics and by the activity of various proteins that influence the type of neuron that will be formed. Neuron migration does not occur at a constant rate throughout development, but occurs in "waves" depending on cell type. The neurons form each of the brain's specific structures and grow long distances to find and connect, through the formation of synapses, to other specialized neurons. Although neurons retain the ability to make new synapses throughout life, the developmental period is critical for the basic formation of the intricate and specific circuits of the nervous system. In addition, throughout all stages of development the number of neurons is reduced through a process known as apoptosis, or programmed cell death. This process is necessary as only about one-half of the neurons generated during development are needed in the adult brain.

All neurons in the central nervous system pass through this same sequence of events. However, neurons in different regions of the brain mature at different times. Each region of the brain has an individual timetable of development that is fixed and cannot be delayed. Periods of intense neuron proliferation have been shown in most brain regions in humans. The timing of such growth spurts can occur over a short period of time, frequently over a period of only a few hours. The migration of neurons to their correct destination in the brain must occur in proper sequence and at the proper time if normal brain development is to result. Should migration of any subset of neurons be delayed or interfered with, subsequent neurons will either be blocked or will pass the delayed neurons and lead to displacement of these cells in the brain. Normal function requires that certain cells with certain characteristics are located in the correct location. Different learning or behavorial effects may result from exposure to the same agent at different times in brain development, depending on the location in the brain where susceptible neurodevelopmental events are taking place at the time of the exposure.

How Methylmercury Affects the Brain

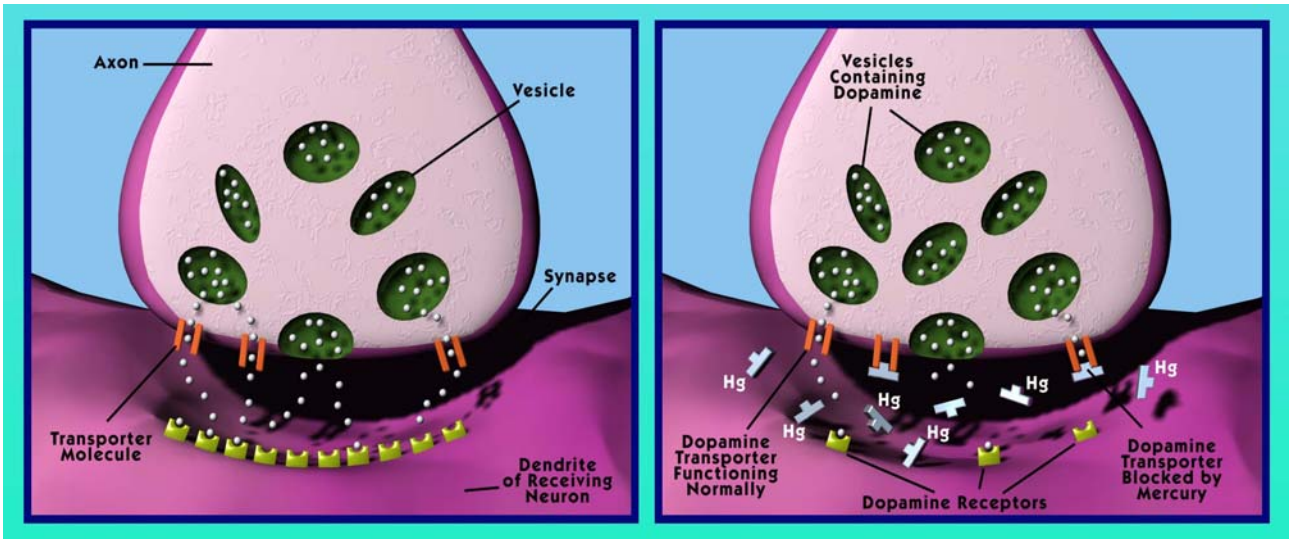
Numerous studies have confirmed that methylmercury exposure changes the function and structure of the central nervous system. In adults, the damage is typically localized in specific areas of the brain, but when exposure to methylmercury occurs in utero or at an early age, the damage to the central nervous system is generalized and widespread. There are several ways by which methylmercury causes developmental damage to the central nervous system which makes the fetus and the young brain far more vulnerable than the adult brain. The mechanisms by which methylmercury affects neurodevelopment are listed below.

- **Methylmercury decreases the transmission of impulses across the synapse.**

Methylmercury decreases the electrical activity of neurons by interfering with the transfer of sodium across the cell membrane. It also blocks the release of calcium ions, thus preventing the release of neurotransmitters such as dopamine. (Figure 4.) Dopamine is a critical neurotransmitter that is necessary for a wide variety of functions such as attention, thinking and motor skills.

Parkinson's disease, mood disorders and deficits in attention, motor control and perception have all been linked to dysfunction of the dopamine system. Research in animals has shown that prenatal exposure to methylmercury resulted in loss of motor control when the exposure occurred during the critical time period when dopamine neurons were developing into mature cells. The dose of mercury in this animal study was comparable to the amount of methylmercury found in the brains of infants from fish-eating populations.

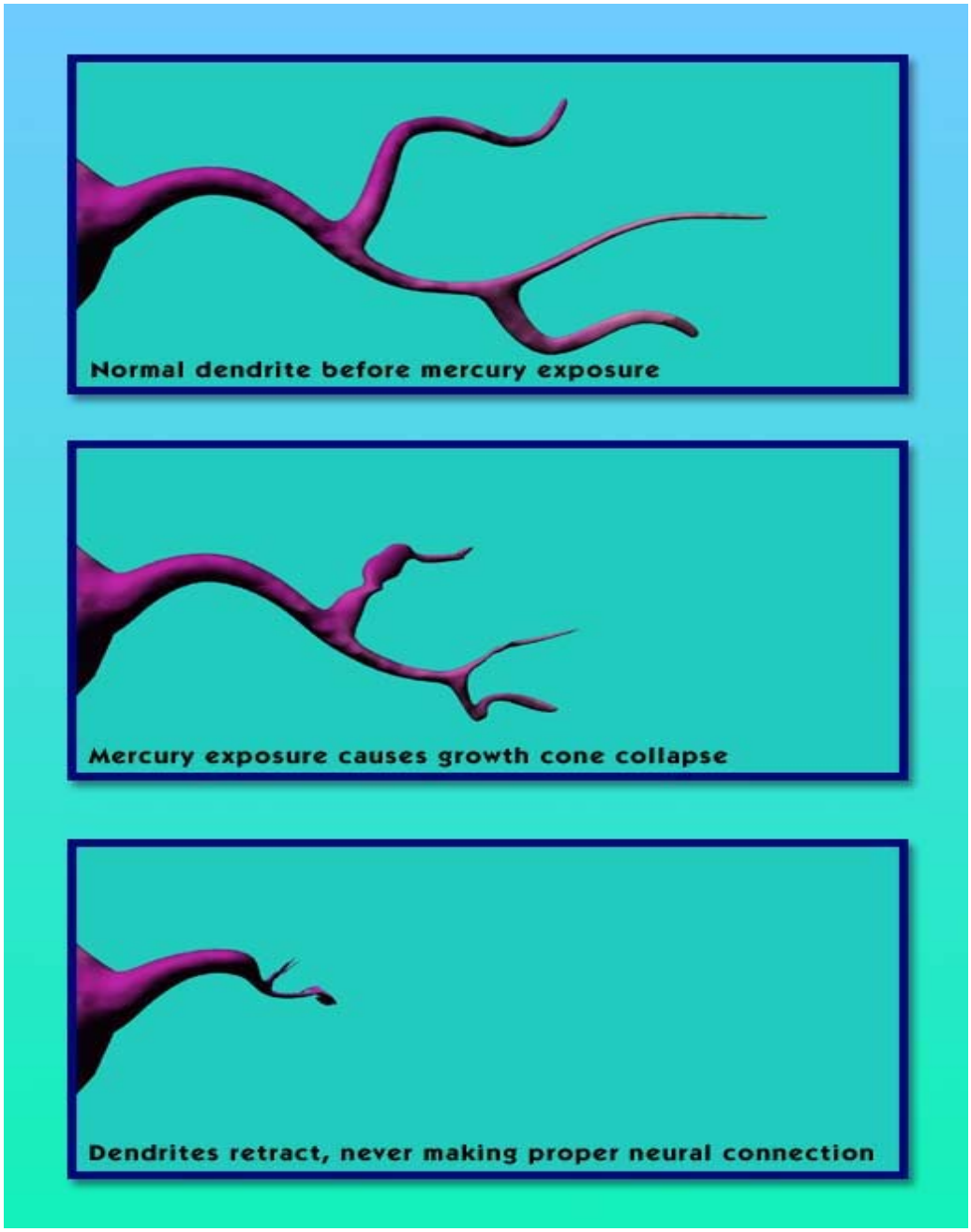
Figure 4. Methylmercury blocks the release of neurotransmitters such as dopamine.



Methylmercury hinders the formation of axons and dendrites.

To complete the circuit of the central nervous system, axons must stretch out to their final destination to meet the dendrites of their connecting neurons. Growth cones - special cells at the tip of the axon - receive chemical signals that tell the growth cone whether to move forward, stop or change direction. Laboratory studies with immature neurons have demonstrated that methylmercury prevents the growth of axons and dendrites by causing the growth cones and the nerve extensions to retract, thus never making the proper connections. (Figure 5.)

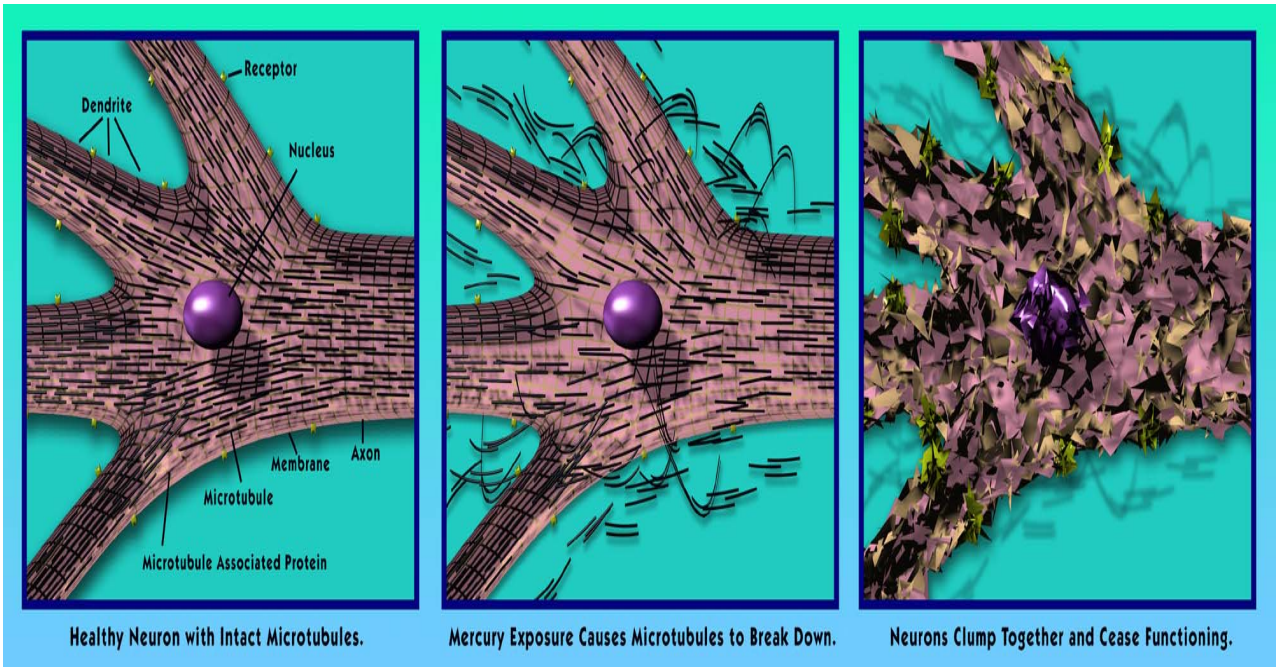
Figure 5. Methylmercury prevents the growth of axons and dendrites.



- **Methylmercury targets the cell structure and disturbs neuron migration.**

A very important feature of prenatal methylmercury exposure is the affect methylmercury has on the cell microtubules. These structures not only provide the structural support for the cell, they play a critical role in cell production, neuron migration and the extension and stabilization of axons. Exposure to methylmercury is known to cause microtubules to break down and leave the neuron stripped of its protective membrane. These neurons clump together forming aggregates that cannot function normally. The result is abnormal arrangement of neurons in the brain. Interference with microtubules causes brain lesions that are consistent with those found in infants exposed in utero to methylmercury during the early stages of pregnancy.

Figure 6. Methylmercury targets the cell structure and disturbs neuron migration.



- **Methylmercury stops cell division and the formation of new neurons.**

Prenatal methylmercury exposure is associated with reduced brain size and weight and a reduced number of neurons. Methylmercury impairs cell production by stopping the process of cell division and interfering with the genes that regulate the cell growth cycle.

- **Methylmercury induces apoptosis (programmed cell death).**

While apoptosis is a necessary process during the development of the brain, methylmercury induces this process with the result that the cells die at the wrong time.

Effects of Methylmercury Exposure in Schoolchildren

The most severe effects of methylmercury exposure were reported after high-dose poisoning episodes in Japan beginning in the 1950s and in Iraq in the 1970s. Children exposed to methylmercury in utero suffered severe adverse effects including mental retardation, cerebral palsy, deafness, blindness and dysarthria (a speech disorder that is due to a weakness or lack of coordination of the speech muscles). Sensory and motor impairment were also documented in adults. While poisoning episodes like those in Iraq and Japan are rare, chronic low-dose methylmercury exposure from maternal consumption of fish is common and has been associated with more subtle neurodevelopmental effects in children.

In the 1990s, methylmercury effects were studied in newborns and children in three large investigations in the Faroe Islands, the Seychelles Islands and New Zealand, as well as in other smaller studies in French Guiana and the Amazon.[12] The Seychelles Islands study consisted of 779 mother-infant pairs where the mothers consumed 12 meals per week of fish with low methylmercury levels. Neurodevelopmental tests were performed on the children at approximately 6 months, 1½ years, 2½ years, 5½ years, and 9 years. No effects were observed. The Faroe Islands study included about 900 mother-infant pairs where the mothers consumed 1-3 meals per week of fish with low average methylmercury levels and 1 meal a month of pilot whale meat containing high average methylmercury levels. Neurodevelopmental tests were conducted at 7 years of age. Dysfunctions in language, attention, and memory were observed in the Faroes children.

In the New Zealand study, 38 children of mothers with high mercury levels were paired with children from mothers with low mercury levels. At 6 years of age, 237 children were assessed, using tests similar to those used in the Seychelles study. Similar to the Faroe Islands study, the New Zealand children appeared entirely normal during the first few months of life, but later displayed adverse effects as documented by a battery of neurodevelopmental tests.

Scientists are not sure why the results of the studies differ. Maternal exposure to methylmercury in the studies was comparable and is unlikely to account for the different findings.[13] Among the other differences in the studies that may account for the different findings are differential genetic susceptibility of the population and different patterns of methylmercury exposure (continuous v. episodic).[14] In considering these differences however, the National Research Council advised the U.S. EPA, as a matter of prudent health practice, to use the results of the Faroe Islands and other positive studies for assessing the public health risk of mercury exposure in the U.S.

The tests administered in the Faroe Islands study were neuropsychological tests designed to assess specific brain functions (e.g., attention, motor performance and memory). By comparison, tests administered in the New Zealand study yielded scores that represented the performance of the brain over a number of domains (e.g., IQ). All of the tests assessed functions that are important for a child's ability to learn, remember and succeed in school. The Faroe Islands and New Zealand studies showed a significant correlation between impairment in the areas of language, attention and memory, and prenatal methylmercury exposure.[15] Table 1 describes the tests given

in the Faroe Islands and New Zealand and how poor scores on these tests are relevant to school performance.

In 2000, the National Research Council of the National Academies of Sciences reviewed all of the available scientific data on human exposure to methylmercury. It concluded that neurobehavorial deficits of the magnitude reported in the Faroe Islands and New Zealand (as measured by the test results) are likely to be associated with increases in the number of children who have to struggle to keep up in a normal classroom or who may require remedial classes or special education.[16] Data from the Faroe Islands also indicates that adverse health effects associated with methylmercury exposure may not be reversible, increasing the significance of these effects.[17]

Table I. Tests Administered to Children to Assess the Effects of Prenatal Methylmercury Exposure

Mercury Study	Test Administered	Function Assessed by Test	Test Relevance to School Performance
Faroe Islands	Finger Tapping (Oscillation) Test	This procedure measures motor speed and performance. By examining performance on both sides of the body, inferences may be drawn regarding possible disorder of the motor nerves.	Difficulty with fine motor skills such as writing.
	Continuous Performance Test Reaction Time	Vigilance, attention, information processing speed. Identifies children with potential learning disabilities.	Intelligence, school behavior and performance especially in the areas of sustained attention and distractibility.
	Bender Copying Errors	Diagnoses the maturity of visual-motor perception in young children. Identifies problems with visual motor perceptual functioning, such as ability to process and interpret visual information about where objects are in space.	Math performance, classroom performance (e.g., shifting gaze between objects at a distance(writing on the board), to close-up objects (a book at the desk).
	Boston Naming Test	Expressive vocabulary. Specifically assesses confrontational naming skills – child has to identify line drawings of common objects under time pressure. Cues are given if child cannot respond spontaneously.	Reading, school performance.
	California Verbal Learning Test: Delayed Recall	Memory. Assesses only one domain of function, but in	Learning ability, school performance

		considerable depth.	
New Zealand	Test of Language Development (TOLD)	Language development. Identifies children who are significantly below their peers in language proficiency.	Literacy skills, learning, school performance.
	Wechsler Intelligence Scale for Children-Revised Performance IQ	Performance IQ e.g., visuospatial, sustained attention, sequential memory. Assesses learning style to determine relative strengths and weaknesses, levels of cognitive functioning, assists in the determination of a learning disability.	Learning, school performance.
	Wechsler Intelligence Scale fro Children-Revised Full-Scale IQ	Full-scale IQ e.g., performance IQ plus verbal processing and expressive vocabulary.	Learning, school performance.
	McCarthy Perceptual Performance	Measures cognitive performance such as performance IQ, visuospatial, memory. Visuospatial perception is the ability to reach for objects in space and to shift our gaze from one point to another.	Learning, school performance.
	McCarthy Motor Test	Measures gross and fine motor skills.	Difficulty with fine motor skills such as writing.

Societal and Economic Impacts of Methylmercury Exposure

The exposure of the developing child to methylmercury may well translate into lifelong impacts on brain function. While no one can say for certain how many children will suffer neurodevelopmental impairments from methylmercury exposure, EPA has indicated that 1 in 6 women of childbearing age has mercury levels in her blood above EPA's current health threshold.[18] Nationally, this means that as many as 630,000 of the four million babies born each year are at risk of developmental problems due to mercury exposure in utero.[19]

What do these staggering numbers mean for childhood development, for our education system and for our society? Developmental and learning disabilities, including loss of IQ points, have negative impacts not only on individuals, but also have long-term consequences for the population and society as a whole.[20] Chemical contamination of the brain – the ultimate pollution – affects not only the educational attainment, economic performance and income of the individual, but it also has an impact on the performance of the economy as a whole through its affect of society's potential production and rate of technical progress and overall productivity.[21]

Lowered IQ has a documented relationship with economic outcomes such as lifetime earnings.[22] Even small decrements in IQ have been linked with lower wages and earnings. Two recent studies have attempted to calculate the societal cost of methylmercury exposure in the U.S and the related economic benefits of reducing such exposure. The Center for Children's Health and the Environment at the Mt. Sinai School of Medicine concluded that exposure to methylmercury causes lifelong loss of intelligence in hundreds of thousands of American babies born each year, and that this loss of intelligence exacts a significant economic cost to American society - a cost that is estimated to be in the hundreds of million dollars each year.[23]

In a different study, the Northeast States for Coordinated Air Use Management (NESCAUM) in collaboration with the Harvard School of Public Health quantified how decreasing mercury emissions from coal-fired power plants would result in less methylmercury exposure and consequently, IQ point gains for the population of children born each year.[24] According to this study, a 70% decrease in coal-fired power plant mercury emissions by 2018 would result in benefits to society of between $119 million to $288 million every year. Consequently, a reduction in emissions of more than 70% would result in even greater benefits. Extrapolating these results, we estimate that a 90% reduction in emissions would result in benefits to society worth more than $370 million per year.

Effects on IQ however, may be just the tip of the iceberg. As researchers point out, IQ effects may be the easiest to quantify and put a dollar value on, but they may not be the most serious in terms of life and career outcomes. Toxicants like methylmercury that affect the nervous system, alter a person's ability to plan, organize and initiate ideas and may induce problems with attention, distractibility, impulsive behavior and inability to handle stress and disappointments. These effects could be far more serious with respect to success in school and life.[25]

According to the Learning Disabilities Association of America, 15 percent of the U.S. population, or one in seven Americans, has some type of learning disability. Among

school-age children, more than 6 percent are currently receiving special education services because of learning disabilities – that's almost 3 million students![26]

In 1999-2000, the cost of educating students with disabilities represented over 21 percent of the spending on all elementary and secondary educational services in the U.S. According to the Special Education Expenditure Project:[27]

- In 1999-2000, the total spending to provide a combination of regular and special education services to students with disabilities amounted to $77.3 billion, or an average of $12,474 per student.

- The additional expenditure to educate the average student with a disability is estimated to be about $5,918 per student each year, or 1.9 times the costs of educating a student without special needs.

- Federal funding to local education agencies (including Medicaid funds) covers only 12 percent of the additional expenditure on special education students.

It is difficult to estimate the cost of learning disabilities due to methylmercury exposure. Of the 630,000 babies born each year at risk for developmental effects due to mercury exposure, researchers don't know how many children will actually suffer adverse effects because of different exposure levels and the timing of exposure relative to brain development in individual children. However, in its review of methylmercury effects, the National Research Council concluded that more than 60,000 children in the U.S. each year - those born to mothers with the highest fish consumption during pregnancy - will likely struggle to keep up in a normal classroom or may require remedial classes or special education.[28] Assuming 60,000 students at each grade level (K-12) at cost of $5,918 per student per year, one estimate of special education costs for methylmercury exposure is $4.6 billion per year. This estimate is illustrative of the potential enormous costs of special education due to mercury exposure during pregnancy.

Stopping Mercury Pollution at the Source

To decrease human exposure to methylmercury, mercury contamination in fish must be reduced. In order to reduce mercury contamination in fish, mercury emissions that deposit from the atmosphere into our waterways must be reduced. The largest and last unregulated industrial source of mercury emissions in the U.S. is coal-fired power plants. Mercury emissions from these power plants must be reduced to the maximum extent possible, as quickly as possible. The Clean Air Act requires EPA to issue "maximum achievable control technology" (MACT) standards for coal-fired power plants. Power plants would have to comply with these rules in 2008. Independent tests indicate that 90 percent removal of mercury will be achievable and affordable by that time.[29,30] In fact, some power plants are reducing their emissions by more than 90 percent right now.[31] There is no need to wait 15 or more years to reduce mercury pollution from these sources as new industry-supported legislative proposals would allow.

Strong regulations must require that each power plant reduce its emissions and not allow the "trading" of mercury emission "credits". Emissions trading means that some power plants may not have to reduce

their emissions at all. Instead, they could buy mercury emission "credits" from other power plants and do nothing to stop contamination of local lakes and streams. Some plants could even *increase* their mercury emissions. Because mercury trading could lead to toxic hotspots where mercury contamination increases, EPA must bar trading in mercury emissions. A final regulation to address mercury pollution from power plants is expected from EPA in 2005. EPA's 2004 proposed regulation generated enormous public debate, and was criticized by children's health advocates and the public health community as weak and not protective enough of the health risks to unborn babies, infants, and children.

In addition, because of the global circulation of mercury in the atmosphere, a binding global treaty that requires all nations to reduce their mercury use and emissions must be negotiated. This global agreement must complement - not replace - our national efforts to reduce mercury emissions in this country as required by the Clean Air Act. During February 2005 meetings of the United Nations Environment Programme (UNEP), the United States delegation opposed a proposal by several governments, including members of the European Union, for a legally binding pact to ban mercury. Instead, U.S. diplomats called for voluntary public-private partnerships to reduce mercury levels. The option of a binding treaty will be revisited by the UNEP governing council in 2007. The U.S. must step up in a leadership role and commit to reducing mercury use and emissions at home and abroad.

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