The Effect of Prenatal Mercury Exposure

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ABSTRACT

The purpose of this study was to investigate the relationship between maternal mercury intake and its effect on prenatal central nervous system development. There has been growing concern that developmental defects result from neurotoxic environmental exposure. One hypothesis and an alternative are set forth: 1) prenatal mercury exposure will have no effect on the developing central nervous system, and 2) prenatal mercury exposure will result in abnormalities of the developing central nervous system; the degree of developmental defects will increase with increasing exposure to mercury. Data from cohort studies confirm mercury’s toxicity, evidenced by abnormalities of the developing central nervous system with varying levels of mercury exposure.

Introduction

There has been an increasing awareness of the potential toxicity of mercury resulting in the concern regarding human dietary exposure and the role methyl mercury may play in neurological abnormalities (Bakir et al. 1973). Because human exposure to mercury is primarily through consumption of fish, populations that rely on fish as a major food source may obtain levels thought to have an adverse affect on the developing central nervous system (Hylander and Goodsite 2006). Harada’s (1978) study provided affirmation that the developing fetus is vulnerable to the toxic effects of methyl mercury, evidenced by mental retardation and cerebral palsy. Numerous studies have been conducted to confirm if the effects that are thought to exist actually occur, and to search for any consistent relationship between neurological abnormalities and mercury exposure. The purpose of this study is to investigate the relationship between maternal mercury intake and its effect on prenatal central nervous system development. It is postulated that prenatal mercury exposure will have no effect on the developing central nervous system. Alternately, if a relationship is found the degree of central nervous system developmental defects will increase with increasing mercury exposure. According to Hylander and Goodsite (2006), mercury is toxic, is of no biological benefit, and little is know about its effects on human health.

Mercury is the only metal that is liquid at room temperature (Clarkson 1998, Zalups 2000). Being a silver liquid, elemental mercury was given the Latin name “hydrargyrum” and the atomic symbol, Hg. Mercury is subdivided into two broad categories, organic and inorganic.

Inorganic mercury includes elemental mercury. The liquid form of mercury is not easily absorbed, therefore, poses no great health risk (Clarkson et al. 2007). Mercury liquid is highly volatile and can be readily transformed to mercury vapor (Zalups 2000). Elemental mercury can undergo oxidation (the loss of electrons) to form the two major states of mercury. The first oxidation state, where mercury has lost one electron, is called mercurous mercury (Hg+). The second oxidation state, where mercury has lost two electrons, is called mercuric mercury or divalent mercury (Hg++) (Clarkson et al. 2007). The mercurous mercury is an ionic compound containing two atoms of mercury which separate releasing one atom of divalent mercury and one atom of elemental mercury (Clarkson and Magos 2006, Clarkson et al. 2007).

Clarkson et al. (2007) defines organic mercury as “those compounds in which divalent mercury is covalently bound to one or two carbon atoms.” There are two classes of organic mercury, those that rapidly separate into constituent parts forming inorganic mercury and those that do not. Those that do not break down are comprised of the short-chain alkyl mercurials and include methyl and ethyl mercury (Clarkson et al. 2007). Methyl and ethyl mercury compounds move freely in the human body and are potential source of damage to the brain (Clarkson and Magos 2006, Clarkson et al. 2007). Public
awareness and concern regarding the potential impact of mercury on the brain and central nervous system arose from the environmental disaster at Minamata, Japan.

In the mid-1950’s, Japanese fishermen and their families suffered from incoordination, constricted visual fields, and numbness in their extremities (Clarkson 2002). The source of the poisonings was traced to a factory that manufactured acetaldehyde. The industrial waste from the process was derived from inorganic compounds of mercury, used as a catalyst during the manufacturing process, which were converted to methyl mercury and eventually discharged into Minamata Bay (Clarkson 1998, 2002). Once in the bay the converted mercury bioaccumulated through the food chain where the fish accumulated concentrations many times greater than is in their food, proving lethal when consumed by the villagers.

The discovery of biomethylation and bioaccumulation drew attention to the impact of mercury on the environment. Methyl mercury was detected in all species of fish, the waterfowl and land animals that consumed the fish. The source was the biomethylation of inorganic mercury by microorganisms in the sediment of the aquatic environment (Clarkson 2002). After biomethylation, methyl mercury bioaccumulates through the food chain to top fish predators. In bodies of fresh water, species such as pike and bass have some of the highest levels of methyl mercury; in the ocean the shark contains the highest levels (Clarkson 2002, Clarkson and Magos 2006).

Today the main source of mercury exposure is by ingestion of methyl mercury contaminated fish (Clarkson 1998). This is when bioaccumulation and biomagnification become significant because the toxicant and the food together are absorbed in the gastrointestinal tract (Klaassen et al. 1996). Generally, the mucous layer that covers the intestinal mucosa limits the absorption of toxins. However, toxicants that are lipid soluble and smaller in particle size enhance absorption (Sreedhuran 2004). Liquid mercury’s larger particle size explains why it is poorly absorbed and relatively harmless (Sreedhuran 2004, Klaassen et al. 1996). The lipid soluble characteristic of methyl mercury is an exception because rather than enhance absorption, absorption is actually slow. In view of the fact that methyl mercury does not readily dissolve in the gastrointestinal fluids of the stomach, absorption is thought to occur primarily in the intestines. During the absorption process mercury crosses the intestinal membranes and enters the bloodstream (Klaassen et al. 1996).

Once a toxicant is absorbed into the bloodstream it can be rapidly distributed to the tissues and organs throughout the body. The rate of mercury diffusion into the bloodstream is determined by the rate of blood flow and diffusion out of the capillary bed (Klaassen et al. 1996). Mercury vapor is oxidized by the red blood cells before it is free to circulate throughout the body tissues. Oxidation of mercury vapor in the fetal liver limits the amount of mercury that reaches the fetal brain. The mercuric mercury that reaches the brain attaches to the selenide species of selenium forming mercuric selenide, HgSe. Mercuric selenide is not soluble so it resides in the brain for years. The mechanisms of distribution and deposition of primary importance for this study result from the ingestion and absorption of methyl mercury. From the bloodstream it is distributed to all tissues readily crossing the blood-brain barrier and placenta (Clarkson et al. 2007).

There are two transport mechanisms that are thought to account for the mobility of methyl mercury in the body. One is for mercury to enter the cells on large amino acid carriers as a complex with cysteine and homocysteine. The other is for mercury to exit from cells on endogenous glutathione carriers as a complex with glutathione. These mechanisms are important to understand when selecting the type of indicator for determining mercury levels in the body organs and tissues.

The indicator media for fetal brain exposure are placental tissue, (umbilical) cord blood, and maternal blood and scalp hair. About 99% of the mercury in plasma is bound to albumin, and the remaining 1% is methyl mercury cysteine. When blood is used as an indicator, it is assumed that the mobile methyl mercury species maintains a steady percentage (Clarkson et al. 2007). While this may be the case with one individual, percentages will differ across populations. Doi and Tangawa (1983) claim that binding affinities for methyl mercury will differ between individuals because hemoglobin proteins are genetically determined; for the same reason blood brain ratios will also differ. Bartell et al. (2000) attributes the variation in levels of the mobile methyl mercury species in individuals to differences in hair
to blood ratios. Once mercury maintains a steady distribution within the body, hair levels are about 250 times greater than in whole blood (Cernichiari et al. 2007).

Clarkson and colleagues (2007) claim that, “based on what we know about the mechanisms of methyl mercury transport, maternal scalp hair offers the best functional index of fetal brain levels.” Hair is useful in that it provides a historical record of methyl mercury exposure revealing the start and end of exposure and past blood concentrations (Cox et al. 1989). Amino acids are important for the formation of keratin during hair follicle growth. The neutral amino acid carriers transport amino acids and the methyl mercury cysteine complex into the hair follicle. The hair formed contains the cysteine to which methyl mercury binds, hence, its accumulation in the hair (Cernichiari et al. 2007, Yu et al. 1993). The actual uptake of amino acids in growing hair accounts for the hair to plasma concentration ratio of 250:1 (Cernichiari et al. 2007).

**Method**

Review of literature began with a search for human population studies involving children that were prenatally exposed to methyl mercury. Many large cohort studies as well as small group studies exist. For this research project it was important to find studies that accurately reflected measured mercury concentrations during the gestation period. Maternal hair mercury levels were selected because exposure could be determined for a specific period of time and blood levels only indicate recent exposure (Myers et al. 2000). Scrutinizing studies was confounded by the various methods that authors compile and analyze data for literature review. So study selection was further narrowed to how the actual data reflected any continuum of toxicity; this could be any level of exposure versus no effects to varying degree of exposure versus a continuum of neurological effects. Based on these criteria five studies were selected for review.

**Review of Studies**

Two studies, which investigated the effects of prenatal exposure to methyl mercury, resulted from the epidemic poisoning in Iraq (Marsh et al. 1987, Amin-Zaki et al. 1979). The poisonings occurred as a result of the ingestion of bread prepared from methyl mercury contaminated wheat. Each study selected infant/mother pairs in which the mother consumed the contaminated bread during pregnancy.

The purpose of the 81-infant/mother pairs study conducted by Marsh and his colleagues (1987) was to determine a dose-response relationship between maternal hair concentrations and the frequency of effects in children. Effects were measured by psychomotor retardation, seizures, and neurological signs. Infants were examined and a scoring system was formulated from the neurologic results. Assessment included but was not limited to, cranial nerve signs, strength, deep tendon reflexes, coordination, dexterity, and primitive reflexes. When absolutely normal, the neurological score assigned was 0, when minimal signs were identified scores of 0 to 3 were assigned, the highest score in the most severely affected children was 11. The developmental examination included developmental milestones (walking and talking), mental development and seizures; these were labeled signs and symptoms.

The second study conducted by Amin-Zaki and colleagues (1979) evaluated the progress of 32-infant/mother pairs. Clinical observation of the infants was conducted over a five-year period to determine central nervous systems damage and/or the development of neurological signs over time. The cohort was divided into two groups; one group of 14 that exhibited early clinical manifestations with ten cases of cerebral palsy and another group of 18 that exhibited no early clinical manifestations. A control group of infant/mother pairs was designated from the same rural area; the infants were of the same sex and age distribution as those of the study cohort. Mothers of the controls had methyl mercury concentrations of 5 ppm or less. Survivorship in these controls was important for the assessment of mercury’s role in study cohort versus control deaths.

Three studies, which investigated the effects of prenatal exposure to methyl mercury, focused on populations that consume fish (McKeown-Eyssen et al. 1983, Myers et al. 1995, Marsh et al. 1995). Each study selected infant/mother pairs in which the mother consumed either freshwater or marine fish during gestation.
In Ontario, Canada, 234 children were assessed for neurological abnormalities after prenatal exposure when their mothers consumed freshwater fish. This study attempted to formulate a relationship between neurological function and exposure. The children in the Quebec study were assessed for physical, mental, and neurological development. The neurological examination included assessment of tendon reflexes, muscle tone, coordination, the persistence of Babinski response, and a summary of the presence or absence of neurological abnormality. Based on the degree of neurological findings most closely related to exposure, children within this study were classified as cases or controls, then these two groups were compared and a relationship between the degree of neurological abnormalities and prenatal exposure index was established (McKeown-Eyssen et al. 1983).

In a study of 740-infant/mother pairs in the Republic of Seychelles it was postulated that maternal hair mercury levels less than 30 ppm was related to development. In this population mothers consumed marine fish. Significant factors for neurological examination included but were not limited to cranial nerves, strength, muscle tone, deep tendon reflexes, and developmental milestone (rolling, sitting, pulling to a stand, and standing). The relationship between neurodevelopment and fetal exposure was examined in 6 ½ month old infants (Myers et al. 1995).

The Peruvian study was similar to the Seychellois study in that exposure to methyl mercury was through the consumption of marine fish. This study also intended to verify the relationship of any determinable defects with maternal exposure. The study assessed muscle tone, limb strength, reflexes, motor and mental retardation. The study was conducted between 1981 and 1984, and as of August 1985 no other study had been published investigating the effects of maternal fish consumption during pregnancy (Marsh et al. 1995).

**Results of Studies**

In the study conducted by Marsh et al. (1987) there was a wide range of maternal hair levels of mercury spanning from 1 ppm to 674 ppm. A summary of levels of mercury and outcomes are found on Table 1.
The Effect of Prenatal Mercury Exposure

<table>
<thead>
<tr>
<th>Study</th>
<th>Source of Mercury</th>
<th>Levels of Mercury</th>
<th>Outcomes</th>
</tr>
</thead>
<tbody>
<tr>
<td>Fetal Methylmercury Poisoning Relationship Between Concentration in Single Strands of Maternal Hair and Child Effects</td>
<td>Bread prepared from methylmercury contaminated wheat</td>
<td>&lt;50 ppm</td>
<td>6 children abnormal scores; no definite neurological signs</td>
</tr>
<tr>
<td>Marsh et al. (1987)</td>
<td></td>
<td>404 ≥ 443 ppm</td>
<td>4 children most severely affected; seizures with severe psychomotor retardation</td>
</tr>
<tr>
<td></td>
<td></td>
<td>1-674 ppm</td>
<td>20 children with abnormal scores, &gt;3</td>
</tr>
<tr>
<td>Fetal Methylmercury Poisoning Clinical Observations Over 5 Years</td>
<td>Bread prepared from methylmercury contaminated wheat</td>
<td>400-532 ppm</td>
<td>Group 1: 14 cases neurological signs (hyperactive deep tendon reflexes, positive Babinski’s responses) and developmental delays 10 cases of cerebral palsy in group</td>
</tr>
<tr>
<td>Amin-Zaki et al. (1979)</td>
<td></td>
<td>371-532 ppm</td>
<td></td>
</tr>
<tr>
<td></td>
<td></td>
<td>32-400 ppm</td>
<td>Group 2: 18 cases no early signs of CNS damage; follow-up revealed similar signs and symptoms as those found in 4 mild cases from group 1; no cases of cerebral palsy in group</td>
</tr>
<tr>
<td>Methyl Mercurv Exposure in Northern Quebec II. Neurologic Findings in Children</td>
<td>Fresh water fish</td>
<td>Average indices 6 ppm</td>
<td>Overall 4% had mild abnormalities (neurologic abnormalities, abnormal tendon reflexes, muscle tone disorders, and delayed motor development) with no clinical significance</td>
</tr>
<tr>
<td>McKeown-Eyssen et al. (1983)</td>
<td></td>
<td>&gt;20 ppm</td>
<td>6% of children</td>
</tr>
<tr>
<td>Main Neurodevelopmental Study of Seychellois Children Following in Utero Exposure to Methylmercury from a Maternal Fish Diet; Outcome at Six Months</td>
<td>Marine Fish</td>
<td>Range 0.5 ppm to 26.7 ppm</td>
<td>735 children; neurologic score normal in 710 and abnormal or questionable in 25 (child’s attention and interaction with environment, cranial nerves, strength, and age appropriate abilities</td>
</tr>
<tr>
<td>Myers et al. (1995)</td>
<td>Median 5.9 ppm</td>
<td></td>
<td>733 children; limb tone score normal in 720 and abnormal in 13</td>
</tr>
<tr>
<td></td>
<td></td>
<td></td>
<td>734 children; deep tendon reflexes normal in 718 and abnormal in 16</td>
</tr>
<tr>
<td>Fetal Methylmercury Study in a Peruvian Fish-Eating Population</td>
<td>Marine Fish</td>
<td>0.9-5.0 ppm</td>
<td>44 in group; 4 with retarded speech, 1 motor retardation, and 1 mental retardation</td>
</tr>
<tr>
<td>Marsh et al. (1995)</td>
<td></td>
<td>5.1-8.8 ppm</td>
<td>43 in group; 4 with retarded speech, 2 abnormal reflexes, 1 motor retardation, and 1 mental retardation</td>
</tr>
<tr>
<td></td>
<td></td>
<td>9.0-28.5 ppm</td>
<td>44 in group; 5 with retarded speech, 2 abnormal muscle tone, 1 limb weakness, 4 abnormal reflexes, 3 motor retardation, and 2 with mental retardation</td>
</tr>
</tbody>
</table>

Seven of the children exposed to ≥ 78 ppm had seizures and retarded early development. No seizures were reported in those with less exposure. Four of the children had severe psychomotor retardation (mental and speech retardation), scoring 11, and all within the exposure range of 404-443 ppm. Remaining children with increased neurological scores had abnormal muscle tone, reflexes and developmental delay. Less affected children did not have neurological signs but were delayed in walking.
and/or talking. Individual scores, from raw data provided in the study, exhibited a wide range of variability. Neurological score plotted against maternal hair concentration reveal a clear dose-response relationship (figure 1). When mercury exposure was related to signs and symptoms by sex, affects were greater in boys than in girls (figure 2). When this study cohort reached school age inquiries were made of their readiness to attend school. In some cases the mothers deemed their children to be not ready for school. In other cases the teachers sent the child home telling them to return the following school year.

![Mercury Concentration in Maternal Hair vs. Neurological Score of Offspring](image1.png)

**Figure 1.** Data from Table 1. Marsh et al. (1987) The index of fetal exposure is the maximum maternal mercury concentration during gestation and offspring neurological score is based on signs found during physical examination.

![Maternal Hair Mercury Related to Signs and Symptoms by Sex](image2.png)

**Figure 2.** Data from Table 2. Marsh et al. (1987) Mean scores for eight exposure groups plotted against maternal mercury concentration reveal a dose response relationship, with larger effects for boys.
The five-year study (Amin-Zaki et al. 1979) like the previous study demonstrated manifestations of varying degrees of neurological damage (table 1). Four of the children in the first group had abnormal reflexes, mild brain damage, and little or no language development. The ten more severely affected children had abnormal reflexes, lacked body strength, had difficulty swallowing and chewing, were incontinent of urine and stool, were irritable, and startled easily. Five years later the first group’s neurological signs remained unchanged and the second group demonstrated neurological signs, delayed psychomotor development, and abnormal reflexes. Although mild, brain damage in this group was similar to that of the first group. Three years into the study nine of the infants died, compared to two deaths in the control group. Five of the children that died had cerebral palsy and all nine of the cohort deaths were related to intercurrent infections.

In the Quebec study, neurologic abnormality occurred in only about four percent of children of both sexes (table 1). Abnormalities in muscle tone, Babinski reflexes, and coordination were found in equal frequency in boys and girls. Abnormality of the tendon reflexes was the most frequent finding, 11% of boys and 12% of girls. This was the only abnormality in neurological function that was associated with the effects of methyl mercury, and significant only for boys. Comparison of the 15 boys with abnormal muscle tone or reflexes with the 82 normal controls, neurologic abnormalities increased seven times for each 10 µg/g increase of the prenatal exposure index. Finally, although the percentage of abnormality of muscle tone or reflexes against prenatal exposure index, in boys, does not demonstrate a uniform increase (figure 3), it is clear that frequency of abnormality increases with increased exposure (McKeown-Eyssen et al. 1983).

![Abnormality of Muscle Tone or Reflexes, in Boys](image)

Figure 3. Data from Table 7. McKeown-Eyssen et al. (1983) The frequency of abnormal muscle tone or reflexes versus prenatal hair index, for boys, is positively related to the index of prenatal mercury exposure.

In the Seychellois study findings were mild, isolated, and generally not associated with exposure. Examination outcomes are described in table 1. Only 3.4% scored abnormal or questionable on the overall examination, 1.8% had an abnormal limb tone score and 2.2% had an abnormal deep tendon reflex score. A higher rate of abnormal limb tone was found in the Seychellois male children. No relationship between exposure and neurological development could be found; no adverse neurological effects were observed with low dose exposure (< 30 ppm) as postulated. Myers and his peers suggest that
although no relationship is seen at 6 ½ months of age, neurological effects related to low dose exposure may be easier to detect as these children age (1995).

The Peruvian fish eating population (Marsh et al. 1995) revealed no significant relationship between maternal hair mercury concentrations and neurological abnormalities (table 1). Peak maternal hair mercury levels ranged from 1.2 ppm to 30.0 ppm. It was determined that there was no difference between mean and peak hair mercury levels because of the steady maternal diet of marine fish. When infant signs (abnormal for age on examination) are plotted against mean hair methyl mercury concentrations, signs only gradually increase with increasing levels of mercury exposure (figure 4). Decreased tendon reflexes were the most common sign, however, they did not occur with other neurological abnormality nor did they occur throughout all exposure groups. Two boys in the highest exposure group had isolated signs of increased tone or tendon reflexes. Although there was a slight increase in isolated signs in the most highly exposed group, there was not enough of an increase to support a significant relationship between mercury hair concentrations and neurological signs.

![Of 131 Infants-Signs Related to Means Maternal Hair MeHg Level](image)

**Figure 4.** Data from Table 4. Marsh et al. (1995) Frequency of infant neurological signs based on group mean maternal hair mercury concentrations.

**Discussion/Conclusion**
The earliest indications of the vulnerability of the developing central nervous system came from a report from Minamata where infants born after 1955 presented symptoms of severe brain damage. No abnormality was observed at birth; however, six months later the children began to show mild abnormalities, which developed into severe neurological and mental symptoms (Harada 1978). The cases of poisoning in Iraq further verify that prenatal exposure can result in severe brain damage. Children in the Iraq studies suffered from a continuum of neurological abnormalities and developmental retardation ranging from abnormal reflexes to severe retardation with cerebral palsy.

Even in children with no initial early signs of damage, follow up examination revealed developmental delays and mild neurological abnormalities. Other studies also indicate a latent period between ingestion and the first appearance of symptoms (Bakir et al. 1973). When prenatal exposure was related to maternal hair mercury concentrations, a dose-response relationship between hair mercury levels versus abnormal neurological findings emerged. Clearly an elevated maternal dose predicts the probability of effects in offspring. Data indicated that the abnormalities were a result of the consumptions of enough methyl mercury to produce maternal hair concentrations as low as 10 ppm (Marsh et al. 1987).
From the Minamata and Iraq cases it is evident that adverse neurological effects drastically increased with increasing fetal exposure. Children in the fish consuming population studies demonstrated extremely contrasting results. The purpose of these studies was to ascertain whether low maternal mercury exposure could result in any measure of neurological damage. Abnormal muscle tone and deep tendon reflexes were predominant in the Quebec study. The study did not support a dose-response relationship; however, it did support a connection between neurological abnormalities and methyl mercury levels in maternal hair and these findings supported the findings in the Iraq studies. The children in the Peruvian study and the Seychelles study did not appear to suffer any adverse effects. It was unclear how definitive these finding were so subsequent studies were conducted. Pilot studies found nothing supporting the suggestion that exposure to methyl mercury from fish consumption may be associated with undesirable neurological effects (Myers et al. 2000).

Today the most common human exposure to methyl mercury is through the consumption of fish. Exposure to methyl mercury from fish consumption is different from methyl mercury poisoning. Normal fish consumption involves exposure to small repeated doses over an extended period of time versus the high degree of exposure during a relatively short period of time. The Minamata poisonings resulted from high exposure during a short time period; keeping in mind the high degree of pollution in the waters of Minamata Bay. The Iraq poisonings involved a wide range of doses and the exposure time was also relatively short. In all of these cases exposure was measured in severe outcomes such as cerebral palsy and mental retardation.

Does constant low dose exposure to methyl mercury have subtle yet real effects on the developing central nervous system? The studies of fish eating populations have not demonstrated a consistent picture of the lowest prenatal levels that may pose a measurable risk to the developing central nervous system. There are many factors that could account for the differences in outcomes between these studies: differences in testing strategies, differences in genetic vulnerabilities, the source of mercury, or environmental pollution. The Seychellois study and Peruvian study found no relationship between neurological abnormalities and exposure yet data clearly indicated a slight increase in abnormality as exposure increased. Data from the Peruvian study clearly demonstrated that infant signs gradually increased with increasing levels of mercury exposure, and these increases were observed when maternal hair concentrations were as low as 10 ppm.

One primary difference between all studies was the use of peak versus mean exposure measures. Marsh and colleagues (1987) used peak hair mercury values for their study and a clear relationship emerged from the data. They were able to do this because exposure time was short and they were able to calculate true peak values from maternal hair mercury levels based solely on gestation period. The other studies used mean hair mercury values. They were unable to assess mercury hair levels for such a clearly defined period of time; before gestation women in the fish consuming populations already had maintained mercury levels from their steady diet of fish. It is not clear which method of measure is the most reliable, however, it would seem that peak values provide more accurate documentation of exposure.

This study set forth searching for the lack of a relationship between prenatal mercury exposure and its effect on the developing central nervous system. It was postulated if a relationship was found that the degree of central nervous system developmental defects would increase with increasing mercury exposure. It is difficult to ascertain prenatal dose because dose can only be estimated by indirect measures. The most reliable indicator media source is maternal hair mercury, being the index of concentration of methyl mercury to the fetal brain. The dangers of methyl mercury have been demonstrated in the Minamata environmental disaster where high levels of pollution resulted in adverse neurological defects. Data from the Iraq studies demonstrated a strong relationship, adverse neurological effects drastically increasing with increasing fetal exposure. Further the Iraq study provided evidence for permanent damage and support for mercury’s role in the progression of development. The Quebec study provided evidence for abnormalities in muscle tone and reflexes, specifically in boys, although overall the study substantiates the Iraq studies. Finally, although no relationship between exposure and neurological abnormalities was found in the Seychellois study and Peruvian study data clearly indicated a slight
increase in abnormality as exposure increased. One thing consistent across the studies investigated is that effects (even the most subtle) are detected in a range of 10 to 20 ppm in maternal hair.

An extremely significant similarity between all of the studies was the lack of adequate control groups. Generally, experimental design requires the use of a control group to compare with the experimental group. They are valuable because they reduce error and bias and aid in eliminating alternate explanations. While human population studies yield a wealth of data they generally lack adequate, if any, controls. The control group must be identical to the experimental group except for the casual agent. In the studies presented for review the general population was also exposed to methyl mercury so researchers were unable to compile a pool of controls from the same general population from which the cohort had been drawn. Normally, the lack of controls results in the inability to generalize findings to people universally, therefore, findings are restricted to the study groups.

Finally, studies of fish consuming populations do not provide convincing evidence of any relationship between maternal hair concentrations and neurological abnormalities. Plainly, a minimal exposure dose of methyl mercury is required to elicit abnormalities of the developing central nervous system. The size of this dose remains a mystery because any measure of fetal brain exposure is determined solely by indirect measures such as maternal hair, cord blood, maternal blood and scalp hair. It is puzzling that the effects of mercury can take several months to appear. Two studies demonstrated the latent effects of methyl mercury exposure (Harada 1978, Amin-Zaki 1979). Taking these two factors into account, how reassuring can the absence of adverse effects in fish consuming populations really be? To substantiate any risk from low dose exposure it would be beneficial to conduct longitudinal studies assessing neurological development of the children within the fish consuming cohort groups.
Works Cited


