Assessing mercury contamination in the Amazon Basin - 2001
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1. Abstract

Anthropogenic activities, such as gold mining and soil erosion, seem to play an important role in the recent enrichment of mercury to aquatic sediments of the Amazon. Soil erosion may have a more regional effect, while the effect of gold mining is more local. High Hg-methylating capacities are found in aquatic systems of the Amazon. Floating macrophyte mats seem to play an important role in the methylation of inorganic mercury to methylmercury. Riverine humans in the Amazon are exposed to methylmercury through the ingestion of contaminated fish. Hair mercury levels in riverine human populations are commonly above 10 μg/g, which is the threshold limit for adverse neurological effects to the fetus. Recent studies in the Amazon have shown relations between adverse neurological performance and methylmercury. Finally, predicted hair mercury concentrations in riverside human populations were compared to observed values. The results indicate that human exposure and health risks, associated with fish ingestion, may be assessed via the predictive model.

2. Introduction

The gold rush in the Amazon region and in other developing countries in the 1980s increased the interest on the environmental cycle of mercury in the tropics. Studies in the Amazon have shown high total mercury concentrations in fish and human hair in the vicinity of gold mining areas (Akagi et al. 1995; Malm et al., 1990; Pfeiffer et al., 1988). However, other studies from pristine areas have shown mercury concentrations in fish and human hair samples similar to those from gold mining areas (Forsberg et al., 1994). More recently, high mercury concentrations were found in the mineral horizons of soils in the Amazon (Roulet et al., 1998a). Soil erosion, intensified by human activities like forest clearing, agriculture and gold mining, was shown to be an important Hg source for local aquatic systems (Roulet et al., 2000). The contribution from other Hg sources such as forest burning has also been a subject for debate.

Although the relative importance of the different mercury sources in the Amazon is uncertain, available data show that mercury deposition in river sediments and floodplain soils has increased in the recent past. This is concerning, because mercury may be transformed to the highly toxic and readily bioavailable mono-methylmercury in aquatic systems. Methylmercury tends to accumulate into the lower levels of the aquatic food chain and is then biomagnified up through the trophic levels of the food chain (WHO, 1990).

Few studies in the Amazon have measured methylation potentials of mercury (Hg^{++}). Initial studies focused on Hg-methylation in river and lake sediments (Guimaraes et al., 1995), but the highest net methylation potentials were later found below the water surface in the submerged roots of dense floating macrophyte mats (Guimaraes et al., 2000; Lemos et al., 1999; Mauro et al., 1999). Flooded forest soils and river impoundments have also shown to have higher methylation potentials than river sediments (Guimaraes et al., 2000).

The biotransformation of inorganic mercury (Hg^{++}) into methylmercury makes human exposure possible through consumption of contaminated fish (WHO, 1990). In the Amazon where fish is a dietary mainstay for many riverine human populations, mercury levels commonly range between 10 and 20 μg Hg / g hair (Akagi et al., 1995; Boshio and Henshel, 1995; Grandjean et al., 1999; Lebel et al., 1998). Field studies in the Amazon have recently reported manifestations of methylmercury toxicity in the visual and psychomotor performance in riverside human populations (Dolbec et al., 2000; Grandjean et al., 1999; Lebel et al., 1996, 1998). The large amount of data on mercury concentrations in fish and human hair samples gave me the opportunity to test a predictive

model summarized by WHO (1990). My results indicate that the predictive model may be used to assess human exposure and human health risks to methylmercury.

This paper will try to define relevant background concentrations and sources of mercury in the Amazon region. Furthermore, the formation of methylmercury in aquatic and semi-aquatic systems of the Amazon will be discussed. I will also refer to the literature on possible toxicological consequences of methylmercury on human populations. Finally, the validity of a predictive model will be assessed. However, some western guidelines on mercury levels in various environmental compartments will be given first.

3. Discussion

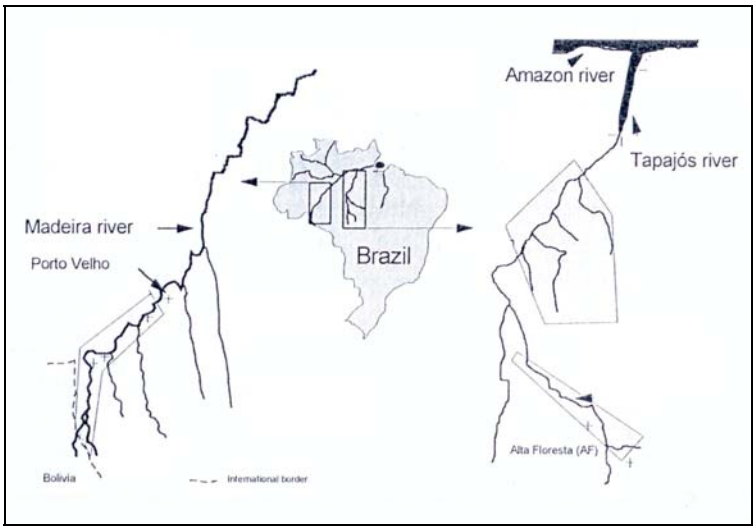
3.1. Western guidelines

Western guidelines, suggest that natural Hg levels are 1-3 ng / m^3 in air, 1-2 ng / l in the dissolved phase in surface waters, 2-25 ng / l in rainwater and less than 100 ng / g in sediments remote from natural Hg mineral deposits and wastewater sources (Porcella et al., 1997). Furthermore, average mercury concentration in most fish is less than 0.2 μg/g (USDHH, 1994).

Mercury is a naturally occurring metal that is ubiquitous in the environment. The major source of atmospheric mercury has been reported to be global degassing of mineral mercury from the lithosphere and hydrosphere at a rate of 2,700-6,000 tons/year (WHO, 1990). Estimates on anthropogenic releases of mercury to the atmosphere normally ranged from 2,000 to 4,500 tons/year (WHO, 1990). Furthermore, weathering of mercury-bearing minerals in igneous rocks is estimated to release about 800 tons of mercury per year to surface waters on a global base (WHO, 1990).

3.2. Map of main study area in the Amazon

Fig. 1: Map of Brazil showing the Madeira River and the Tapajos River



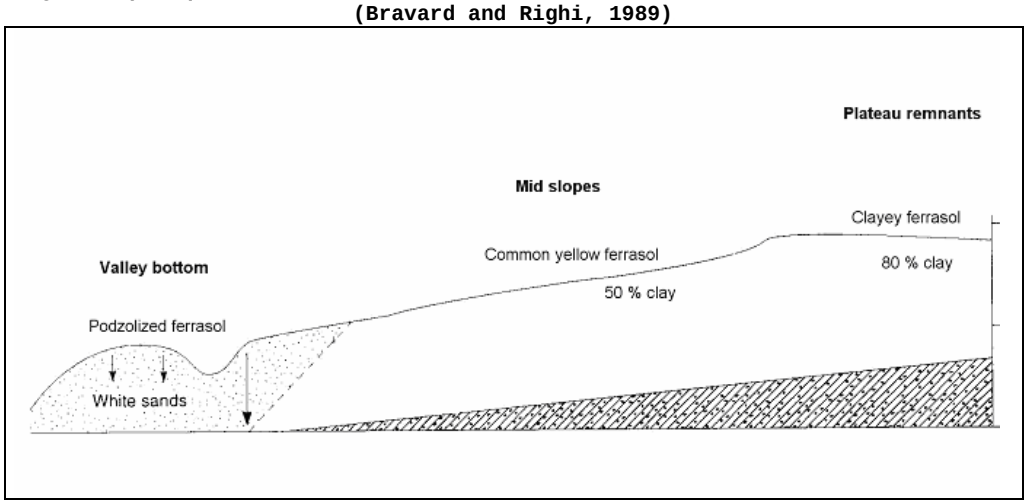
The area of interest is the Amazon region of Brazil. I will mainly focus on two areas of the Amazon, the Tapajos River and Madeira River (Fig. 1). The shaded areas on the map show gold mining areas. River samples from the Madeira and Tapajos River were taken

downstream from gold mining areas.

3.3. Background concentrations of mercury in the Amazon region

According to Lucas et al. (1996), sandification and podozolization processes control the evolution on slopes. In the Amazon this is seen as a clay gradient from high clay concentrations on plateau remnants to low clay concentrations at the valley bottom (Fig. 1). This is interesting because mercury is not homogeneously distributed over the various grain size fractions in soils and sediments, but is generally found in the fine-grained fractions consisting mainly of clay minerals.

Fig 2: Toposequence of ferrasols in central Amazonia, Brazil. (Bravard and Righi, 1989)



In the very clayey ferrasols, typical of plateau remnants, total mercury concentrations varied between 212 to 439 ng Hg / g d.w. (Forsberg et al., 1999; Lechler et al., 2000; Roulet et al., 1998b). In mid slope ferrasols total mercury concentrations varied between 136 to 210 ng Hg / g d.w. (Roulet et al., 1998b; Malm et al., 1995). Finally, total mercury concentrations in podzolized ferrasols were *circa* 43 to 83 ng Hg / g d.w (Roulet al., 1998b).

Background mercury concentrations in surface sediment varied from 100 to 250 Hg / g d.w. in the Tapajos River (Roulet et al., 1998). In the Madeira River, from Porto Velho and 400 km downstream, mercury concentrations in sediments ranged from 245 to 439 ng Hg / g d.w. (Lechler et al., 2000). Although sample sites were downstream from gold mining locations no systematic downstream Hg-trends were observed in sediments or filtered water from the Madeira and Tapajos River (Lechler et al., 2000; Roulet et al., 1998, respectively).

Dissolved mercury concentrations in the Madeira, Tapajos and Amazon River were 0.5-3.7, 0.4-2.8 and 1.31 ng Hg / l, respectively (Lechler et al., 2000; Roulet et al. 1998a; Roulet et

al. 1998a, respectively). Furthermore, mercury concentration in the fine particulate matter of the Tapajos River were 0.28-13.3 ng / l, which is one order of magnitude lower than previously reported Hg-concentrations in Amazonian waters (Roulet et al. 1998a).

Average total mercury concentration was 3.05 ng m^{-3} in the air over the Amazon region (Artaxo et al., 2000). However, over pristine areas mercury concentrations ranged between 0,5–2 ng m^{-3} and over gold mining areas mercury concentrations were as high as 14.8 ng m^{-3}.

In summery, mercury concentrations in Amazonian soils, sediments and air are high compared to global averages. Mercury bounded to fine particulate matter in rivers is also higher than global values. Dissolved mercury concentrations in the Madeira and Tapajos River are similar to global averages.

3.4. Anthropogenic sources of mercury in the Amazon

I will comment on 3 important mercury sources arising directly or indirectly from anthropogenic activities in the Amazon. The sources are forest burning, gold mining and soil erosion.

3.4.1. Forest burning

Viega et al. (1994) estimated that the burning of forest biomass in the Amazon released 90 tons Hg / year into the atmosphere and suggested that the burning of forest biomass was the major source of atmospheric mercury emissions in the Amazon. This was disputed by Lacerda (1995) who calculated atmospheric mercury emissions from forest burning in the Amazon to be 17 tons Hg / year. However, both these estimates are based on assumed Hg concentrations, and not on actual values observed in the various compartments of the Amazonian forest.

Roulet et al. (1998a) measured mercury concentrations in forest biomass from three forests situated in French Guyana and Brazil. The average emission factor from forest combustion for burning of primary forest is 273 g Hg / km^2 and 370 g Hg / km^2 for when the cumulative impact of slash and burn agriculture (cycles of 3 fires over 10 years) was accounted for. Annual deforestation rates for the Amazon region ranged from 22000 km^2 / year to 34000 km^2 / year (Fearnside, 1991 and Myers, 1991, respectively). Based on the data for initial burning of primary forest and annual deforestation rates it is estimated that forest burning releases 6 to 9 tons of Hg / year (Roulet et al. 1998a).

3.4.2. Gold mining

There are two distinct periods of precious-metal mining in South America. The first period took place in Colonial America (1550-1880). The second period was more recent and has been taking place for the last 20 years.

In the first period both gold and silver were extracted by similar amalgamation techniques (patio process). However, mainly gold was extracted in the second period. Although mining techniques were very similar, they were regional differences between the two periods. Precious metal mining in Colonial America mainly took place in the Andes whereas the recent gold rush mainly took place in the Amazon region.

Emission factors (EF) for mercury in Colonial and today's mining processes are estimated to be 1.5 kg Hg / kg precious metal (Pfeiffer and Lacerda, 1988). Recent calculations, based on precious-metal production records and emission factors for mercury, show that *circa* 200,000 tons of mercury has been released to the environment between 1550 and 1880 (Nriagu, 1993). During the recent period it is estimated that 2000 tons of mercury has been released into the environment over the last 20 years (Pfeiffer et al., 1988). This results in an annual input of 100 tons Hg / year into the environment.

Roughly half of the mercury emitted from gold mining activities is released to the atmosphere and the other half is released to the rivers (Pfeiffer et al., 1988).

Atmospheric release of mercury from gold mining activities may account for 63% of the mercury in the air over the Amazon (Artaxo et al., 2000). The intense convection in the Amazon together with the flat terrain and long residence times for atmospheric mercury makes regional atmospheric Hg transport quite efficient. Atmospheric transport of mercury may also result in the export of mercury to other parts of the world.

Some of the mercury released into the aquatic system of the Amazon may be lost through sedimentation and some may be converted to mercuric mercury (Hg^{++}), which in turn could be biotransformed to the highly bioavailable methylmercury.

Mercury used in gold mining activities in the Amazon is often blamed for the high Hg-concentrations found in fish and humans. However, there is no clear scientific evidence that this is the case in Brazil, as studies from pristine areas have shown Hg concentrations in fish and human hair samples similar or even higher to those from gold mining areas (Forsberg et al., 1994).

However, results from a personal study in Guyana showed that hair mercury concentrations in an Amerindian population (Micobie Village) near a gold mining area were significantly higher, when

compared to a control Amerindian population (Moraikobai Village). Mean ±SD hair mercury concentrations were and 15.4 ±0.9 μg/g (n=47) and 5.6 ±0.4 μg/g (n=44), respectively. Both villages were situated on the banks of black-water river systems and both villages were from the same tribes. Apart from the intense gold mining activities in Micobie River, no other anthropogenic activities were identified.

3.4.3. Soil erosion

Recent studies indicate that soil erosion may influence the natural biogeochemical cycle of mercury in the Amazon (Forsberg et al., 1999; Lechler et al., 2000; Roulet et al., 2000). The term soil erosion is used when anthropogenic activities such as deforestation, cultivation and mining result in sol erosion.

Roulet et al. (2000) analyzed vertical profiles of sediments from the Tapajos River for mercury, textural indicators (water content and dry density), mineralogical indicators (iron and aluminum associated with oxyhydroxides and aluminumsilicates) and organic indicators (carbon, nitrogen, C/N ratio). The results demonstrate that soil erosion is responsible for an overall enrichment of recent sediments by fine clay particles rich in mercury. Furthermore, the mercury levels in the sediments of the Tapajos River had the same relationship with aluminosilicates of soils. Additionally, the activity of lead-210 suggested that surficial sediments originated from eroded soils. A preliminary dating indicated that the environmental changes recorded in the sediment began sometime between the 1950s and 1970s. This coincides with the colonization of the Brazilian Amazon.
Direct relationship between arsenic (As) and Hg were also found in sediments of the Madeira River, suggesting that As and Hg originated from a soils (Lechler et al., 2000)

Studies in the Tapajos and Madeira region have shown a lack of temporal and spatial Hg trends downstream of gold mining areas. (Lechler et al., 2000; Roulet et al., 1998b). This suggests that the mercury found in the various environmental compartments is related to a regional geological source rather than a local anthropogenic source. The geological source seems to be the naturally occurring mercury in soils.

In summery, anthropogenic activities in the Amazon, especially gold mining and soil erosion, seem to be important sources of Hg contamination to the aquatic environment. Natural soils may represent the largest reservoir of mercury in the Amazon region.

3.5. Methylation of mercury in the Amazon

2 issues will be addressed in this section. First, I will comment on a common analytical method for net methylation rates of mercury in sediments, as this has been a subject to some criticism. Then, I will refer to the literature on net methylation rates in the aquatic environment of the Amazon.

3.5.1. Common analytical method

Despite the toxicological significance of Hg methylation, this step of the Hg cycle is still poorly understood. Net Hg-methylation is dependant on the balance between methylation and demethylation, which are influenced in a complex and variable manner by an array of biological and physio-chemical parameters like, pH, oxygen, sulphate, Hg and methylmercury concentration and availability, and bacterial activity.

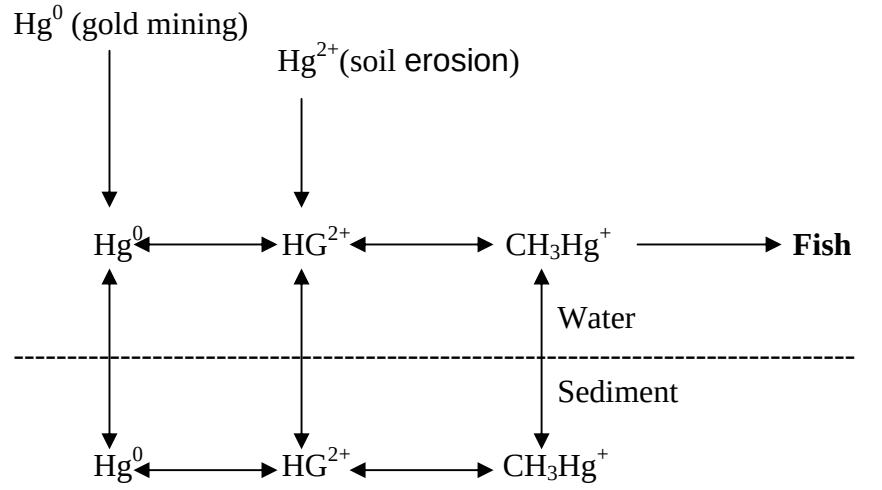
Typically, researchers add $^{203}Hg^{++}$ to biologically active untreated sediment and sterilized sediment. They observe that methyl-^{203}Hg formation occurs predominantly in biologically active sediments. Experiments under anoxic conditions with molybdate (MnO_4^{--}), a specific inhibitor of sulphate reducing bacteria (SRB), suggest that SRB are the main methylators of bioavailable Hg^{++}. However, critics argue that the mercury species added may not reflect the natural species found in sediments. The added mercury could be more or less bioavailable than natural mercury species. Also, solutions added could change the biology and chemistry of sediments, e.g. MnO_4^{--} may act as an oxidizing agent or react with Hg^{++}. High concentrations of Hg^{++} are sometimes added to samples, allowing only mercury resistant species, which do not predominate in the aquatic environment, to survive. Sterilization methods may also change chemistry as well as biology in sediments. Interestingly, the 28 mM sulphate concentration in seawater makes it difficult to explain the ubiquitous occurrence of methylmercury in the marine biota as results indicate that methylmercury production, by SRB, stops at 5 mM sulphate (Weber, 1993).

3.5.2. Methylation rates in the Amazon

The eco-toxicological effect of inorganic mercury inputs to the aquatic environment is dependent on its bioavailabilty. The water-soluble fraction of inorganic mercury is sometimes used as a measure for its bioavailabilty. The aqueous solubility of elemental mercury, i.e. from gold mining activities, is low (0.025 ppm at 20 ^{o}C). However, low pH and oxidizing agents in the aquatic environment may enhance mercury's solubility. The water-soluble fraction in soils is also thought to be low, however no studies addressing this issue were found for the Amazon region. In future

studies, it would be interesting to relate the soluble fraction of inorganic mercury in soils to soil erosion. A schematic representation of possible distribution routes of mercury in the aquatic system is given in Fig. 3.

Fig. 3: Schematic representation of the most important inputs and common distribution routes of mercury leading to bioaccumulation of methylmercury in fish in the Amazon.



Hg-methylation rates are usually associated with sediments. However, submerged roots of floating macrophyte mats are efficient traps for suspended particles and have a high surface area for the fixation of periphyton and bacteria. Guimaraes et al. (2000) showed that methylation potentials in untreated macrophyte roots were higher than in macrophyte roots stripped from associated solids, and methylation potentials were higher in solids stripped from macrophyte roots than in river sediments. Experiments with stimulation and inhibition of sulphate reduction activity suggested that SRB in macrophyte roots as well as in sediments are the main methylating bacteria (Guimaraes et al., 1999; Mauro et al., 1999).

Furthermore, data from various studies showed that on average methylation potentials in the submerged parts of macrophytes were much higher than in underlying lake sediments at the same sites (Guimaraes et al., 2000). Guimaraes et al., 2000 concluded that average net Hg-methylation in sediments and aquatic macrophytes were, 0.6% and 13.8%, respectively. However, part of the range between aquatic macrophytes and sediments could arise, from variations in the amount of added total Hg from study to study, caused mainly by the use of ^{203}Hg solutions with different ages and belonging to different ^{203}Hg lots.

High methylmercury concentrations were also found in filtered water sampled in floating macrophyte mats (Guimaraes et al., 2000; Mauro et al., 1999). These findings are interesting because high methylmercury concentrations in filtered water are highly bioavailable compared to methylmercury bounded to sediment particles. Additionally, the roots of floating macrophytes are an essential source of food and shelter for large populations of fish and invertebrates. Commonly, methylmercury is bioaccumulated in the bottom level of the food chain and is then biomagnified up through the trophic levels of the aquatic food chain.

Guimaraes et al. (2000) also showed that flooded soils and semi aquatic sediments had higher hg-methylation potentials, than river sediments. The high net Hg-methylation potentials found in newly flooded soils are interesting, because vast areas of the Amazon are flooded in annual cycles. River impoundment is similar to flooded soils and also shows high Hg-methylation rates. Hg levels in reservoir fishes are frequently high, even in the absences of aquatic point source of mercury (Porvari, 1995).

In summery, our understanding of *in situ* Hg-methylation is still limited partly because of inadequate research techniques. The formation of methylmercury in the Amazon region seems to differ from temperate regions by having multiple substrates, e.g. sediments, floating macrophyte mats and flooded soils. Also, the unique nature of aquatic and semi-aquatic systems of the Amazon seems to favor net-methylmercury formation.

3.6. Human health risks due to methylmercury ingestion

Methylmercury in humans is often related to the ingestion of methylmercury-contaminated fish. Mainly two disasters, the Minimata and Iraqi incident, have shown that human health risks are associated with methylmercury ingestion. These two disasters and more recent studies will be discussed before concentrating on the possible adverse human health effects in the Amazon due to ingestion of methylmercury contaminated fish. However, some important toxic effects of methylmercury will be discussed first.

3.6.1. Toxic effect of methylmercury

Methylmercury is usually ingested and 95% of the ingested methylmercury is absorbed in the gastrointestinal tract. Methylmercury distributes readily to all tissues, including the brain and fetus, after absorption from the gastrointestinal tract. The uniform tissue distribution is due to methylmercury's ability to cross diffusion barriers and penetrate all membranes without difficulty. Although

distribution is generally uniform, the highest levels are found in the kidney. It is believed that methylmercury is transformed to inorganic mercury in cells of must tissues, including the brain.
The fecal (bilary) pathway is the predominant excretory route for methylmercury. In humans, nearly all of the mercury in the feces after organic administration is of the inorganic form. Methylmercury is secreted in the bile and can be reabsorbed in the intestine.

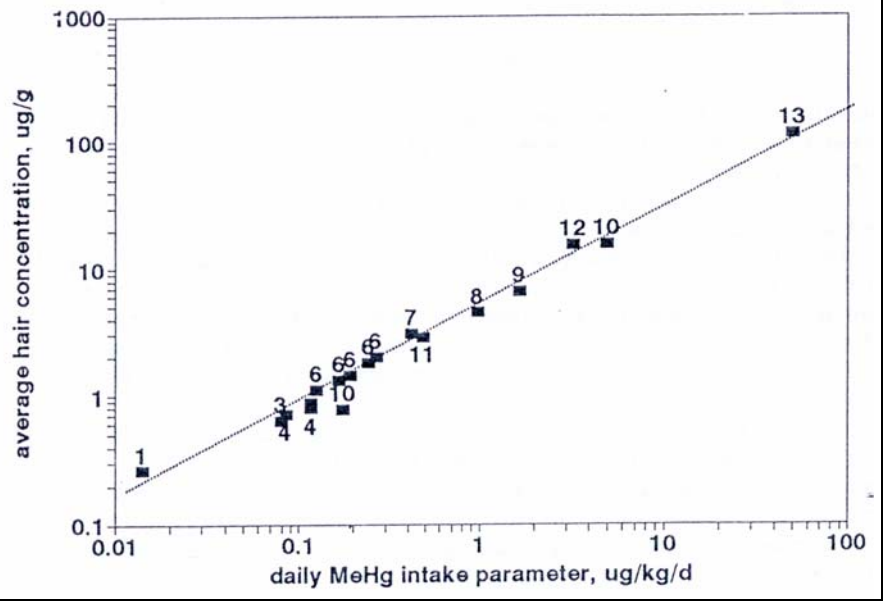
The toxicity of methylmercury is partly related to its ability to diffuse across cell membranes and partly due to its high affinity for thiol groups (SH^-).
Methylmercury is thought to cross the blood brain barrier by binding to L-cysteine complexes in the blood. The Methylmercury-L-cysteine complex is then transported into the brain via the methionine uptake mechanism, which is a neutral amino acid carrier (USDHH, 1994). The developing brain undergoes a complex series of proliferation, differentiation and migration of neurons and glia. It is thought that methylmercury disrupts these processes by binding to tubilin-SH, causing the impairment of spindle function during cell division. One should also bear in mind that the binding of mercury to thiol groups might also lead to the dysfunction of enzymes and proteins through structural changes.

Apart from the neurotoxic effect of methylmercury on human populations, other toxic effects have been reported. In Greenland it was shown that the frequency of sister-chromatid exchanges, in Eskimos, increased with increasing blood mercury levels (Wulf et al., 1986). Recently, a study from the Amazon region showed significant cytotoxic effects in a riverine population exposed to methylmercury through fish ingestion (Amorim et al., 2000). The mitotic index in peripheral lymphocytes declined with increasing mercury levels. Furthermore, the frequency of polyploides and chromatid breaks in lymphocytes increased with increasing mercury levels. These findings suggest that spindle function was disrupted during mitosis. Methylmercury have also been shown to result in T-cell apoptosis by depleting thiol reserves, which predisposes cells to generate more reactive oxygen species and at the same time activates death-signalling pathways (Shenker and Shapiro, 1998). Further studies by Shenker and Shapiro (1999) lead them to propose that the target organelle for methylmercury is the mitochondrion and confirmed that induction of oxidative stress leads to activation of death-signaling pathways. The above findings indicate that mercury has a common mechanism of action, which is the disruption of spindle function. Methylmercury may be regarded as neurotoxic, genotoxic, and immunotoxic. However, only the neurotoxic aspect of methylmercury will receive further attention.

3.6.2. Human studies outside of the Amazon

The Minimata bay incident in the late 1950's showed for the first time that fish was an important source of methylmercury to humans (WHO, 1990). This relation has since been supported in many studies (Fig. 4).

Fig.4: Relationship between average hair mercury concentrations from epidemiological studies of fish-eating populations and their respective average mercury dietary intake rates. Numbers 1-13 represents the various studies. (Lipfert, 1988).



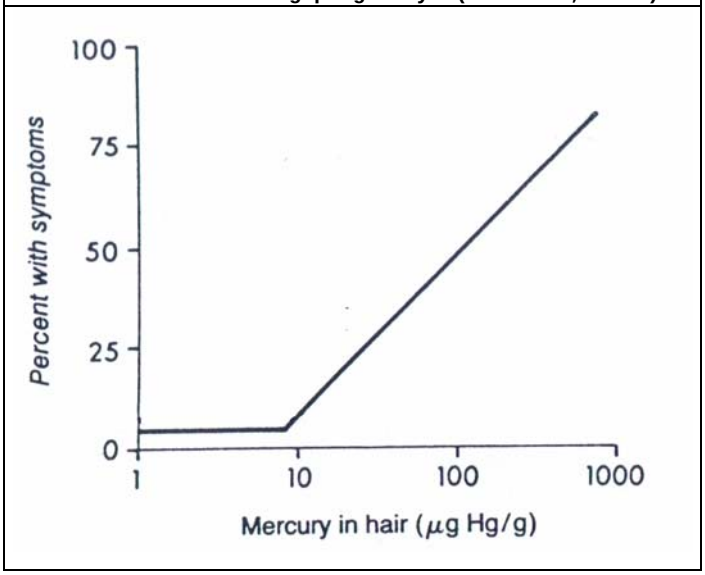
Furthermore, it was recognized that the fetus could be severely affected even when the mother was asymptomatic (WHO, 1990). Clinical findings in symptomatic patients with prenatal exposure were similar and included microcephaly, mental retardation, cerebral palsy, seizures and deficits of hearing and vision. The findings with postnatal exposure were similar, but the severity was often less and seemed to vary with dosage and age. Very little data was obtained about less severe forms of prenatal exposure and none about postnatal exposure.

There were many uncertainties surrounding the Minimata episode, i.e. the cause was not identified until years after exposure. Consequently, maximum exposure had to be estimated. The interpretations of exposures were further complicated by inadequate assay methods.

During the winter of 1971–1972, a widespread outbreak of methylmercury poisoning occurred in Iraq. Methylmercury treated seed grain was consumed rather than planted. There were 6530 patients of all ages admitted to the hospital and 459 known deaths (WHO, 1990 ref. to Bakir et al., 1973). Paresthesias were the first clinical symptoms reported by patients. The first clinical finding was ataxia. Furthermore, a number of children who were both prenatally and postnatally ex-

posed, showed clinical findings similar to those from the Minimata incident (WHO, 1990 ref. to Amin-Zaki et al., 1976). Studies also showed that continued breast-feeding by mothers with elevated blood mercury levels resulted in the infants' blood mercury levels falling slower than expected. When maternal and infants blood mercury levels were compared at birth, the infants had higher blood mercury levels than those from the mothers (WHO, 1990 ref. to Amin-Zaki et al., 1976). These studies also showed that the most severely affected offspring had been exposed to methylmercury during the second trimester and that male offspring were more severely affected than female offspring. A dose-response curve for the association between prenatal exposure and attainment of developmental milestones (walking unaided before or after 18 months of age and using two meaningful words before or after the age of 24 months) and neurological findings were determined (Cox et al., 1989). The dose-response curve suggested that prenatal exposure as low as 10 ppm peak mercury in maternal hair might be associated with adverse fetal consequences (Fig. 5).

Fig. 5: Percentage of children, from the Iraqi study, with motor retardation plotted against the maximum maternal hair during pregnancy. (Clarkson, 1990)



This was concerning because methylmercury is naturally occurring in fish and communities relying heavily on fish are exposed to methylmercury, which easily results in hair mercury concentrations above 10 ppm (WHO, 1990).

However, the Iraqi study had some limitations. Interviews of the mother were done through interpreters at a mean child age of 30 months. Birth dates were ascertained in relation to other events, since they are not important in Arabic culture. The background rate of neurological abnormalities in

the population was unknown. Most of the positive responses (i.e. delays in onset of walking) were observed for maternal hair levels above 60 ppm. Only 3 out of 24 children with positive responses were born to mothers with hair levels below 59 ppm. Actually, this data was used to generate Fig. 5. It was also unclear how applicable the data from an acute seed-grain poisoning episode were to a long time low-level exposure from a dietary source (i.e. fish). Small amounts of methylmercury consumed from fish over a longer time period could alter the way the human body handles it. Interestingly, at low Methylmercury-intake rates, hair methylmercury is about 15 times daily intake, but at very high intake rates the ratio approaches 2 (Lipfert, 1997). Furthermore, selenium and amino acids in fish could also influence mercury toxicity. Selenium may decrease the toxic effects of mercury and amino acids may compete with methylmercury for transport into the brain (WHO, 1990). Long-chain polyunsaturated fatty acids are high in fish and believed to be important in brain development and may inhibit adverse effects from low-level methylmercury exposure (Myers et al., 2000).

After the Iraqi incident, studies concentrated mainly on children who were prenatally exposed to mercury. The first studies were inconclusive mainly because of inadequate procedures. An example is the New Zealand study, where a dietary survey of 11,000 women provided an opportunity to study prenatal methylmercury exposure. 73 of the women had hair mercury levels above 6 ppm with a mean hair mercury concentration of 8 ppm. Their children were enrolled in a study when these were 4 years of age (Kjellstrom et al., 1986). It was concluded that there was a significant dose-response relationship between mean hair mercury during pregnancy and results of the Denver Developmental Screening Test. However, there was a mismatching of age and ethnicity. Pacific Island children were compared to Europeans, and controls were older and had a better chance to pass the DDST. Marsh et al. (1994) suggested that the mixing of ethnic groups and difference in age could account for the differences in DDST score. Sample size is also important when study subtle effects on populations.

Two well-designed studies are the Seychelles Child developmental Study and the Faroe Island study. Both studies were initiated independently in the late 1980’s.

The Seychelles study consisted of 740 children who were followed and evaluated at 6.5, 19, 29, and 66, and 96 months (Myers et al. 2000). Median maternal hair mercury concentration was 7 ppm during pregnancy (Myers et al., 2000). The composition of test batteries was neurological, developmental and psychological. No adverse associations between prenatal or postnatal mercury exposures were found in the Seychelles study (Myers et al. 2000). However, clear relations were found

between mercury concentration in the brains of stillbirths and maternal hair mercury concentrations in the Seychelles study (Cernichiari et al., 1995).

The Faroese diet is high in seafood including both fish and pilot whale. Average mercury concentration in pilot whale was 3.3 μg/g (Grandjean and Weihe, 1993). The Faroe study consisted of 1000 children born during a 21-month period. The index of prenatal exposure was total mercury in umbilical cord blood and in maternal hair during pregnancy. Median mercury concentration in umbilical cord blood was 24.2 μg/l (Grandjean et al., 1994). This value corresponds to maternal hair mercury concentration of 6 μg/g (Grandjean et al., 1994).

During the early years developmental data were recorded, and at approximately 7 years of age, 917 of the children underwent detailed neurobehavioral examination (Grandjean et al., 1997). Neuropsychological tests included Finger Tapping; Hand-Eye Coordination; reaction time on a Continuous Performance Test; Wechsler Intelligence Scale for Children-Revised Digit Spans, Similarities, and Block Designs; Bender Visual Motor Gestalt Test; Boston Naming Test; and California Verbal Learning Test (Children). Clinical examination and neurophysiological testing did not reveal any mercury-related abnormalities. However, mercury-related neuropsychological dysfunctions were most pronounced in the domains of language, attention, and memory, and to a lesser extent in visuospatial and motor functions. These associations remained after adjustment for covariates and after exclusion of children with maternal hair mercury concentrations above 10 ppm.

3.6.3. Human health studies in the Amazon

Mean mercury concentrations in fish are typically 0.36 ppm from the Madeira River and 0.30 ppm from the Tapajos River (Table 2). Over 90% of the mercury present in Amazon fish is methylmercury (Akagi et al., 1994). Furthermore, riverine human populations, *ribeirinhos*, in the Amazon rely heavily on fish as a food source and observed mean fish meals per day ranged between 1.8 and 1.9 (Boischio and Henshel, 2000). Field research with 607 individuals showed that daily fish consumption rates resulted in a log normal distribution with a median of 200g (Boischio and Henshel, 2000). As a result of fish ingestion riverside human hair methylmercury concentrations typically vary between 10 and 20 ppm (Lebel et al., 1996). This is concerning as the Faroe study has shown neuropsychological dysfunctions in children prenatally exposed to methylmercury at maternal hair mercury concentrations below 10 ppm during pregnancy. Unfortunately, prenatal studies similar the Faroe and the Seychelles Study have yet to be done for the Amazon region. However, studies relating mercury exposure to neuropsychological and motor performance in adults and children are present.

An adult study was carried out in a village on the Tapajos River on 91 inhabitants (15-81 years), whose hair mercury levels were inferior to 50 ppm (Lebel et al., 1998). Mean total hair mercury were 23.9 ±9.3 for fishermen, 14.3 ±9.4 for other men and 12.6 ±7.0 for women. Performance on a neurofunctional test battery and clinical manifestations of nervous system dysfunction were examined in relation to hair mercury concentrations. Near visual contrast sensitivity and manual dexterity, adjusted for age, decreased significantly with hair mercury levels ($P < 0.05$), while there was a tendency for muscular fatigue to increase and muscular strength to decrease in women. For the most part, clinical examinations were normal, however, hair mercury levels were significantly higher ($P < 0.05$) for persons who presented disorganized movements on an alternating movement task and for persons with restricted visual fields.

Another adult study from the same river system showed similar results to that of Lebel et al. (1998). Dolbec et al. (2000) examined 84 individuals between 15 and 79 years of age. Median hair mercury concentration was 9 ppm. Methylmercury accounted for more than 90% of the total mercury. Psychomotor performance was evaluated using the Santa Ana manual dexterity test, the grooved pegboard fine motor test and the fingertapping motor speed test. The Santa Ana manual dexterity test was similar to the manual dexterity test used by Lebel et al. (1998). Diminished performance on the Santa Ana manual dexterity test, the grooved pegboard test and the fingertapping test was associated with increasing hair mercury levels. Interestingly, the grooved pegboard test was chosen, because a similar test was done on methylmercury exposed non-human primates in a laboratory by Rice (1989). After 6 years of exposure, from birth, to 50 μg methylmercury/kg/day, the monkeys showed diminished performance in retrieving raisins from a recessed grid.

Grandjean et al. (1999) examined 351 of 420 eligible children between 7 and 12 years of age in four comparable Amazonian villages. In three Tapajos villages with the highest exposures, more than 80% of 246 children had hair mercury concentrations above 10 ppm. Neuropsychological tests of motor function, attention, and visuospatial performance showed decrements associated with the hair mercury concentrations. Especially on the Santa Ana form board and the Stanford-Binet copying tests, similar associations were also apparent in the 105 children from the village with the lowest exposures, where all but two children had hair mercury concentrations below 10 ppm. Although average exposure levels may not have changed during recent years, prenatal exposure levels are unknown, and exact dose relationships cannot be generated from this cross-sectional study. However, the current mercury pollution seems sufficiently severe to cause adverse effects on brain development.

In summery, there is still some uncertainty surrounding the 10 ppm safety limit for pregnant women. Many of the uncertainties arise from differences in test procedures from study to study. However, the Faroese study suggests that the present safety limit for pregnant women should be adjusted to a lower value. Studies from the Amazon show that hair mercury concentrations, between 10 and 20 ppm, are sufficient to cause adverse neurological performance.

3.7. Relations between mercury in fish and mercury in humans in the Amazon

Human exposure to methylmercury is through fish ingestion in the Amazon region. This gives us an opportunity to evaluate a predictive model, which relates methylmercury concentrations edible fish to methylmercury concentrations in human hair (WHO, 1990).

A few aspects of the single-compartment model will be given first. Then data acquired from the literature will be used to calculate average mercury concentrations in hair and fish samples. Finally, a comparison between predicted average methylmercury concentrations and observed values will be given. The areas of interest will be the Madeira and the Tapajos River.

3.7.1. The single compartment model

Relationships between mercury concentration in fish and human hair are based on a single-compartment model summarized by the International Programme on Chemical Safety for methylmercury (WHO, 1990). The elimination of methylmercury generally follows first order kinetics since excretion is directly proportional to body burden (Nielsen and Andersen, 1991). Duration of exposure may affect the excretion process of methylmercury. A two-compartment model was established by Rice et al. (1989) for a single oral dose study in monkeys. In this study an initial rapid elimination phase was followed by a slower elimination phase. However, following continuous dosing for 2 years, a single-compartment model was considered a more reasonable fit for the data.

The single compartment model suggests that continuous exposure of methylmercury results in a steady state, where intake equals excretion after approximately 5 half times. The half time for whole-body methylmercury is estimated to be 70 days, thus steady state is attained approximately after 1 year (WHO, 1990). An important prediction of the single-compartment model is that constant dietary exposure to methylmercury for a period of several years should not result in any greater accumulation than after one year of exposure. Calculations based on the single-compartment model assume that all of the mercury ingested is methylmercury; 95% of the mercury intake is absorbed through the intestines; 5% of the absorbed mercury goes to the blood compart-

ment; the blood volume is 5 liters; the ratio of methylmercury between blood and hair is 1:250; and the elimination constant for methylmercury is 0.01 $days^{-1}$ (Who, 1990). The validity of the single-compartment model is supported by the reasonable agreement between predicted and observed blood concentrations of methylmercury in single-dose tracer studies, single-dose fish intake experiments, and studies involving the extended controlled intake of methylmercury from fish (WHO, 1990).

3.7.2. Fish consumption patterns in the Amazon

Commonly, few fish species represent more than 50% of the fish caught (Castilhos et al., 1998). Catches also show a 1:1 relationship between piscivorous and non-piscivorous species. The same relation is assumed for human consumption. Furthermore, piscivorous fish usually have higher mercury concentrations than omnivorous and herbivorous fish (Table 1).

Tabel 1: Mercury concentrations in piscivorous, omnivorous and herbivorous fish from the Madeira River.

Mercury concentrations in fish (μg/g)			References
Piscivorous	Omnivorous	Herbivorous	
0.69 (n=438)	0.45 (n=294)	0.15 (n=164)	Boischio and Henshel, 2000

A survey on fish ingestion rates in *ribeirinhos* resulted in a median fish ingestion rate of 200g fish per day.

Cultural patterns may influence fish consumption patterns (Boischio and Henshel, 2000). Interestingly, local culture defines certain fish to worsen vulnerable stages of life, i.e. illness, pregnancy and breast-feeding. The Aruana fish species is considered the safest fish for consumption and is often consumed during pregnancy and breast-feeding. However, mean mercury concentration for this species is 1.44 μg/g, which is very high (Boischio and Henshel, 2000).

Riverside people of the Amazon, *ribeirinhos*, also prefer scaled fish rather than non-scaled fish, i.e. catfish (Boischio and Henshel, 2000). In contrast urban populations seem to consume more catfish. Interestingly, scaled fish are generally low in mercury compared to non-scaled fish. The implications are that urban populations are likely to be eating a low amount of fish with high mercury levels, whereas the *ribeirinhos* are eating large amounts of fish with lower mercury levels. Fish consumption patterns also seem to differ between the dry season and the rainy season. Piscivorous fish are more frequently consumed in the wet season, while herbivorous and omnivorous fish are more frequently consumed in the dry season (Lebel et al., 1997). This is reflected in higher hair mercury concentrations in humans in the rainy season, when compared to the dry season (Lebel et al., 1997).

3.7.3. Observed mercury concentrations in fish and human hair

Selective studies on certain types of fish or humans with certain occupations, i.e. piscivorous fish or fishermen, were excluded. These types of studies do not represent a normal population, but only a selective part of a population.

Table 2: Mean mercury concentrations in fish from the Madeira and Tapajos River, Brazil.

Study area	Mean Hg conc. (µg/g fish)		Reference
Madeira River			
	0.39	(n= 576)	Boischio et al., 2000
	0.36	(n= 245)	Boischio et al., 1995
	0.34	(n= 125)	Malm et al., 1995a
Average	**0.36**	(n= 946)	
Tapajos River			
	0.22	(n= 69)	Lima et al., 2000
	0.29	(n= 181)	Lebel et al., 1997
	0.38	(n= 65)	Castilhos et al., 1998
Average	**0.30**	(n= 315)	

Data summarized in Table 2 show that observed average methylmercury concentrations in fish samples are similar for the Madeira and the Tapajos River, 0.36 and 0.3 µg/g, respectively. Generally, over 90% of the mercury present in fish from the Amazon region is methylmercury (Akagi et al. 1994).

Table 3: Mean mercury concentrations in human hair from the Madeira and Tapajos River, Brazil.

Study area	Mean Hg conc. (µg/g hair)		Reference
Madeira River			
	17	(n= 237)	Boischio et al. 1995
	12.6	(n=90)	Boischio and Henshel, 2000
Average	**14.8**	(n=327)	
Tapajos River			
	13.5	(n=98)	Amorim et al., 2000
	9.0	(n=84)	Dolbec et al., 2000
	17.5	(n=91)	Lebel et al. 1998
	17	(n=432)	Malm et al., 1997
	16.3	(n=136)	Akagi et al., 1993
	9.2	(n=96)	Lebel et al., 1997
Average	**13.8**	(n= 937)	

Data summarized in Table 3 show that average human hair mercury concentrations in *ribeirinhos* populations along the Madeira and the Tapajos River are similar, 14.8 and 13.8, respectively (Table 3). Generally, over 90% of the mercury present in human hair samples of *ribeirinhos* populations

along the Madeira and Tapajos River is methylmercury (Kehrig et al., 1997). Only humans with no occupational association to gold mining activities were accounted for.

3.7.4. Predicted mercury concentrations in humans

In order to use the single-compartment model human populations must be in a steady state with regards to methylmercury. I assume that this is the case. The interconversion between mercury in fish and mercury in human hair was done as summarized by WHO (1990).

Hg daily intake (μg) = daily fish consumption (g) χ Hg conc. in fish (equation 1)

[Hg] in human blood (μg/l) = 0.95 χ daily fish consumption (g) (equation 2)

[Hg] in human hair (μg/g) = (250/1060) χ [Hg] in human blood (equation 3)

Interestingly, predicted mercury concentrations in humans from both rivers are similar to observed vales (Table 4). This is further indication that methylmercury exposure in *ribeirinhos* is through the ingestion of methylmercury-contaminated fish. The results also suggest that methylmercury concentrations in fish could be used to assess human exposure and thus human health risks to methylmercury.

Table 4: Comparison of predicted mercury concentrations in human hair with observed values. The area of interest is the Madeira and Tapajos River, Brazil.

	Mercury concentrations in hair (μg/g)	
	Predicted	**Observed**
Madeira River	16.1	14.8
Tapajos River	13.4	13.8

4. Conclusion

Anthropogenic activities (i.e. gold mining and soil erosion) seem to play an important role in the recent enrichment of mercury to the aquatic environment of the Amazon. Additionally, the Amazonian hydrographic basin seems to have a very high methylating capacity for mercury. This is concerning because methylmercury tends to bioaccumulate in the aquatic food chain and studies on riverine communities, relying heavily on fish, are showing relations between adverse neurological performance and methylmercury. The use of a predictive model has also shown relations between methylmercury in fish and methylmercury in humans.

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