ACADEMIC
PRESS

Available online at www.sciencedirect.com

SCIENCE DIRECT

Ecotoxicology and Environmental Safety 56 (2003) 174–179

Ecotoxicology and Environmental Safety

http://www.elsevier.com/locate/ecoenv

Cases of mercury exposure, bioavailability, and absorption

Michael Gochfeld

Environmental and Community Medicine, UMDNJ–Robert Wood Johnson Medical School and Consortium for Risk Evaluation with Stakeholder Participation (CRESP), Environmental and Occupational Health Sciences Institute (EOHSI), Piscataway, NJ 08854, USA

Accepted 20 March 2003

Abstract

Mercury is a unique element that, unlike many metals, has no essential biological function. It is liquid at room temperature and is 13.6 times heavier than water. Its unique physical properties have been exploited for a variety of uses such as in mercury switches, thermostats, thermometers, and other instruments. Its ability to amalgamate with gold and silver are used in mining these precious metals and as a dental restorative. Its toxic properties have been exploited for medications, preservatives, antiseptics, and pesticides. For these reasons there have been many industrial uses of mercury, and occupational exposures of workers and industrial emissions and effluents contaminating air, water, soil, and ultimately food chains have long been a matter of great public health concern. This paper examines briefly six cases representing various forms of exposure to different species of mercury, and indicates the methodological issues in estimating exposure, bioavailability and absorption; these cases include Minamata disease in Japan, organic mercury poisoning in Iraq, methylmercury (MeHg) exposure in the Amazon, dimethylmercury (PMM) in the laboratory, an elemental mercury spill in Cajamarca, Peru, and a mercury-contaminated building in Hoboken, NJ, USA. Other scenarios that are not described include occupational exposure to mercury salts, mercurial preservatives in vaccines, cultural and ritualistic uses of mercury, and mercury in dental amalgams.

Keywords: Mercury Bioavailability; Methylmercury; Dimethylmercury; Elemental mercury; Fish consumption

1. Background

Mercury is at the same time among the best known and most puzzling metals in the environment. It occurs in all media in several species, both organic and inorganic. It is relatively uncommon in the Earth's crust, from which it is liberated by natural processes such as erosion and vulcanism as well as by mining. Anthropogenic activities both intentional (mercury manufacture and disposal) and unintentional (fossil fuel combustion) contribute most of the mercury in the environment. The mercury species differ greatly in properties, but all are toxic. Elemental mercury (Hg^0) is a familiar, dense, silvery liquid at room temperature. It volatilizes readily, emitting mercury vapor, which is readily absorbed in the lungs; however, elemental mercury is very poorly absorbed through the skin or gastrointestinal (GI) tract. Inorganic mercurial salts (Hg^+ and Hg^{2+}) vary in solubility and absorptive properties. Most organic mercurial compounds are readily absorbed through lungs and GI tract, and some are readily absorbed through the skin. All mercury compounds are toxic to humans and animals, but the organic forms, particularly methylmercury and dimethylmercury, have the highest toxicity. Methylmercury is the form found most widely in nature, and it bioaccumulates in the food chain. It is the form to which most human exposure occurs.

2. Mercury in the environment

2.1. Inorganic mercury

Exposure to elemental mercury occurs in certain workplaces, in health care facilities, and occasionally in homes. The droplets of mercury are attractive to humans, and children have been known to bring mercury home to play with. Cultural practices such as Santeria also result in household exposures to elemental

---

E-mail address: gochfeld@eohsi.rutgers.edu.

0147-6513/03/$ - see front matter © 2003 Elsevier Inc. All rights reserved.
doi:10.1016/S0147-6513(03)00060-5

mercury. Breakage of thermometers and spills from gas meters during their removal are infrequent but important sources of mercury. When such spills occur it is important that they be cleaned up quickly, because droplets of mercury continue to emit vapor for a long time. The exposure is almost exclusively through inhalation of vapor.

Hg^0 vapor in the atmosphere is subject to long-range transport. Hg^0 is slightly soluble in water (0.08 mg/L at 25°C) (ATSDR, 2000). Therefore a small fraction of Hg^0 vapor can be washed out of the atmosphere during precipitation events. The more likely fate of Hg^0, however, is eventual oxidation to Hg^{2+} by reaction with atmospheric oxidants such as oxygen, ozone, and chlorine. Once converted to the Hg^{2+} form, the Hg is much more soluble and more subject to washout from the atmosphere with precipitation. This is called wet deposition and is a major source of mercury input to the environment. A small amount of the mercury may adhere to fine particles in the atmosphere and may fall out without rainfall as dry deposition (Fitzgerald, 1995).

Today, the major sources of mercury for the general environment include the incineration of wastes and the burning of coal to produce electricity. Much of this inorganic mercury reaches aquatic systems (either as direct fall out to water surfaces or as indirect fall out to land with subsequent runoff). In bodies of water it settles onto the sediment surface or algal mats, where it is converted by bacterial action from inorganic forms into the highly toxic methylmercury.

2.2. Methylmercury

Elemental and inorganic mercury are deposited on watersheds or directly on bodies of water. This mercury settles to the sediment, where some of it reacts with sulfate to form an insoluble mercuric sulfide precipitate, whereas a small percentage is biomethylated by bacteria. This MeHg is readily bioavailable and biomagnifies up the food chain so that fish at higher trophic levels regularly have mercury concentrations a million-fold greater than the water in which they live. Fish consumption poses the only significant source of dietary exposure to MeHg for most people, and conversely 75–95% of the mercury in fish is in the MeHg form.

Methylmercury exposure over a period of years resulted in the outbreak known as Minamata disease (case 6.1) and continues to be a source of mercury poisoning among fish-eating peoples of the Amazon (case 6.2). People who eat large amounts of fish (even species with relatively low mercury content) can accumulate sufficient levels of MeHg to cause symptoms, and pregnant women can transfer to a fetus amounts of MeHg that are sufficient to impair nervous system development.

Table 1
Absorption of mercury species by routes

	Elemental (Hg^0)	Ionic/salts (Hg_i)	Organic
Lungs	Almost complete	Variable	Almost complete
GI tract	Negligible	Variable	Almost complete
Dermal	Negligible	Negligible	Moderate to high

3. Bioavailability and absorption

As used in this paper, bioavailability refers to the ability of an organism to extract mercury from the environmental matrix (external bioavailability). Aquatic organisms readily take up MeHg from the water column. In higher organisms, that ingest mercury-containing foods, bioavailability refers to processes within the GI tract by which mercury is liberated from the matrix. Mercury compounds that enter the GI tract or the lungs do not necessarily gain access to the bloodstream or reach critical target organs. The amount that is transferred depends on both bioavailability and absorption.

External bioavailability is mainly a property of the matrix, whereas absorption is mainly a property of the organ. The GI tract and the lungs differ in their absorptive properties for each species of mercury, and absorption may vary by age, frequency of meals and other dietary variables. It is generally recognized that elemental mercury vapor is readily absorbed through the lungs (50–100%), but that absorption of liquid elemental mercury from the GI tract is $<1\%$ (see Table 1).

In contrast, MeHg is readily absorbed from the GI tract (close to 100%) and from the lungs. Once in the bloodstream, mercury is subject to very complex and inadequately understood processes involving binding and redox cycling, both extracellularly and intracellularly. Physiologically based pharmacokinetic models have been applied to mercury, to estimate the exchange among organ compartments. In vivo methylation and demethylation may also occur. These complex processes influence the amounts of mercury exchanged between the blood compartment and various organs, including the brain (internal bioavailability).

4. Human exposure

Humans are exposed to Hg^0 primarily by inhalation and to MeHg primarily by ingestion. With relatively few exceptions, the general public has very low exposure to elemental mercury, and these exposures are often related to deliberate uses (e.g., occupational, dental, and perhaps folk/cultural practices). The health consequences of widespread mercury exposure from dental amalgams is currently highly controversial. For most people, the greatest problem is with MeHg to which the

Table 2
Sources and estimates of daily human exposures to various forms of mercury[a]

Source of exposure	MeHg (μg/day)	Inorganic Hg salts (Hg^{2+}) (μg/day)	Elemental Hg (Hg^0) (μg/day)
Food (nonfish)	Negligible	0.9	Negligible
Commercial fish	6	<1	Negligible
Sport fish	No population-based data available	No population-based data available	Negligible
Drinking water	Negligible	Most public supplies $\ll 4$ μg, but some wells >4 μg	Negligible
Outdoor air	Negligible	Negligible	0.2–0.4 μg
Indoor air	Negligible	Negligible	Cultural, recreational and accidental uses in a few homes
Soil ingestion	Negligible	Contaminated sites may >3 μg	Negligible
Dental amalgams	Negligible	Negligible	$\ll 1$ μg but occupational exposure occurs

[a] Modified from New Jersey Mercury Task Force (2001). Negligible is defined as <0.1 μg/day.

most significant exposure comes from eating fish. There is a large variation in the amount of fish that people consume as well as in the concentrations of mercury in different kinds of fish (Stern et al., 2001). The major sources and estimates of human exposures to various forms of mercury are given in Table 2.

5. Analysis of mercury

The analytical approaches to mercury will be dealt with in greater detail elsewhere. As with all environmental chemistry a comprehensive quality assurance program must be developed and adhered to, so as, to have confidence in the results and their interpretation. Quality assurance should be in place before sampling begins and must extend through quality control of the analyses all the way to quality control of the data.

5.1. *Mercury speciation*

Because of the variation in toxicity and the different biological and preventive implications, it is often desirable to know what species of mercury (at least MeHg vs. inorganic mercury) is present in a sample. Experience has shown that in aquatic ecosystems, including fish, most of the mercury (75–95% in fish) is in the form of methylmercury. Therefore, because speciation is not a trivial process, it is customary for most purposes to analyze total mercury and assume that it is in the methyl form. In other matrices (soil, ground water, waste), the proportion of MeHg is usually quite low (generally $<1\%$). However, in some cases the presence and amount of MeHg can be significant for risk assessment and must be determined. Improved and efficient speciation methods are therefore highly desirable.

5.2. *Laboratory analyses*

A variety of techniques are available for analyzing total mercury, including cold-vapor atomic absorption spectrometry (CVAAS), gold foil capture, and inductively coupled mass spectrometry (ICP-MS). Standard CVAAS provides reliable results in the ppb range. However, this requires destructive sample preparation (usually thermal and acid), which may lose volatile mercury. Several new devices that allow direct pyrolysis of liquid or solid samples offer the promise of measuring ppt concentrations.

X-ray fluorescence allows a nondestructive analysis of mercury in solid tissues. It can be used to scan along a hair to provide segmental analysis, which can indicate the time sequence of exposure.

5.3. *Field analyses*

Portable equipment is required for field analyses and is generally restricted to measuring levels of mercury in the air. Several commercially available devices exist for real-time monitoring of mercury in air. The standard of measure has been the so-called NIOSH method, which is based on an 8-h sampling of air.

The Jerome analyzer is small, easily transported, and useful in highly contaminated areas (e.g., indoor spills). Its detection level is ~ 1 ppm, and it is quite accurate >10 ppm. Mercury in air is sucked into the pump onto a gold foil, where it forms an amalgam. Periodically (every 10 s), the foil is rapidly heated to drive off the mercury vapor through the detector. This is useful for occupational exposure and for highly contaminated environments. However, because household levels >0.1 μg/m^3 are considered unsafe, the Jerome is not sufficiently sensitive to provide information at that level.

The Lumex is a portable instrument that is larger than the Jerome and does not use gold foil or a carrier gas. It has a long cell through which air carries mercury vapor past the detector. It can provide almost instantaneous readings (every 3 s) and can average readings. It is sensitive down to 2 ng/m^3, with an effective range of 10–2000 ng/m^3. It can also be used in the laboratory with a direct pyrolysis attachment.

A semiportable instrument, the Tekran is much larger than the previous instruments and requires a compressed carrier gas. It is somewhat more sensitive and stable than the others, but is not readily portable. It can be used to measure ambient mercury in air in the 1 ng/m^3 range.

6. Case studies

6.1. Minamata disease in Japan

Methylmercury poisoning was first recognized in Minamata, Japan around 1960. Hundreds of fisherfolk and their families were severely poisoned during the 1950s by MeHg that bioaccumulated in fish as a result of release of mercury to the bay from a local chemical plant. By 1956 the affected population recognized that their poisoning was due to fish, and abandoned eating fish from the bay. However, cases continued to accumulate, and by 1960 the serious and mysterious affliction, affecting both adults and infants, was recognized as MeHg poisoning, a hitherto unrecognized disease. The Minamata epidemic, first reported by Kurland et al. in 1960, has been studied more recently by Eto (2000), Futatsuka et al. (2000), and Kondo (2000). The cause was mercury effluent from the Chisso chemical plant's acetaldehyde production. However, not until 1968 was the plant forced to curtail mercury releases, and not until the 1990s was the bay sufficiently clean to allow fishing to resume.

High-level exposure produced serious neurological disease in adults, but the most dramatic manifestation was congenital Minamata disease in infants born to mothers with high mercury levels. These babies were born with severe cerebral palsy, blindness, and profound mental retardation. Many of these victims survived into their 20s, and many of the adult victims survive to this day. Autopsies of victims revealed destructive lesions of the cerebellum and in various parts of the cortex. Peripheral neuropathy also occurred.

Unfortunately there was negligible mercury monitoring at the time the outbreak was discovered, so that it provided no information for risk assessment, and there was no routine monitoring for biomarkers of exposure or affect. The second epidemic appeared in 1965 along the Agano River, Niigata prefecture, but again biomonitoring was inadequate to illuminate issues of external or internal bioavailability.

6.2. Organic mercury poisoning in Iraq

Epidemics of organic mercury poisoning from consumption of grain treated with organomercurial fungicides have also occurred in Iraq and Guatemala. A family in New Mexico was poisoned by eating pork from their pigs, which they had fed on fungicide-treated grain.

By the time the severe Iraq outbreak occurred in 1971, epidemiologists and toxicologists were alert and analytical results (mainly hair mercury) were obtained and used in risk assessment (Amin-Zaki et al., 1974). This resulted in calculation by the US Environmental Protection Agency (US EPA, 1997) of a reference dose of 0.3 μg/kg/day for adults. More recently, supported by a comprehensive review by the National Research Council (NRC, 2000), EPA has developed a reference dose of 0.1 μg/kg/day, sufficient to protect the most sensitive endpoint—fetal neurobehavioral development. Hair mercury was the main biomarker.

6.3. Methylmercury exposure in the Amazon

In the Amazon Basin, mercury contamination of fish has been recognized as a problem affecting many of the indigenous people who live along the tributaries and rely on fish as a major source of protein. Mercury has been released during gold mining operations, but the largest sources of mercury may be deforestation and damming of rivers, which makes naturally occurring mercury in the soil accessible to biomethylation by aquatic organisms.

Extensive studies have been conducted in the Amazon on mercury exposure and neurobehavioral effects. Using segmental hair analysis, Dolbec et al. (2001) were able to document that hair mercury levels among Amerindian women varied with the season. During the wet season when herbivorous fish predominated in the diet, levels were lower than in the dry season, when herbivores made up $<50\%$ of the diet. They also found a positive association with reported fish consumption, however, paradoxically, the proportion of hair mercury that was inorganic increased with increasing fish consumption. They also reported cytogenetic damage in lymphocytes in women with hair mercury <50 μg/g (ppm).

Guimaraes et al. (2000) demonstrated spatial and seasonal variation in biomethylation of mercury using an isotope method. Using ^{203}Hg as a tracer, they showed that MeHg was formed in the superficial layer of flooded soil or sediment and on the surface of aquatic plants (macrophytes). Up to 9% of the available mercury was methylated. The discovery that mercury can be methylated even in relatively "dry" forests, rather than in bodies of water is highly significant, particularly because much of the Amazonian forests is seasonally flooded. They concluded that "flooded forests and macrophyte mats are specific features of the Amazon and are important links between Hg inputs from natural and manmade sources and MeHg exposure of local populations through fish intake".

In a cross-sectional study, Dolbec et al. (2000) studied neurobehavioral performance of 84 residents along the Rio Tapajos, using hair mercury as their exposure metric. The mean percentage of meals containing fish was 62%, and the median hair mercury was 9 μg/g (~95% was organic mercury). There was a negative correlation between hair mercury and neurobehavioral performance, particularly in fine motor coordination, and the relationship was stronger than with blood mercury, indicating the benefits of using hair levels that track exposure over a period of months. However, only 8–16% of the variance in performance was explained by the hair Hg. Cytogenetic damage also correlated with hair mercury levels (Amorim et al., 2000).

6.4. Dimethylmercury in the laboratory

Dr. Karen Wetterhahn, professor of chemistry at Dartmouth College (Hanover, NH, USA), was one of the world's leading researchers on the intracellular redox cycling of metals. In August 1996 she used dimethylmercury (DMM) to calibrate an instrument. In January of 1997 she began to notice difficulty speaking and an awkward gait. Tunnel vision ensued. Within 3 weeks she had progressed to coma from which she never awakened despite heroic therapeutic efforts (Nierenberg et al., 1998). Diagnosis was based on extremely high blood mercury levels, and the timing of her one-time exposure was verified by segmental hair analysis. During the 3 weeks of lucidity, when the diagnosis of mercury poisoning was found, she recalled that she had spilled several drops of DMM on her gloves. This was the only time she had used this compound. After cleanup she disposed of the gloves. However, DMM readily penetrates latex gloves and is readily absorbed through the skin (Blayney, 2001).

6.5. An elemental mercury spill in Catamarca, Peru

In June 2000, a contractor truck carrying flasks of elemental mercury from a gold mine in the Peruvian mountains spilled mercury along a stretch of road, contaminating several villages along the way. The driver was ill and in a hurry to reach a clinic. As his truck sped down the mountain road, the load shifted and a flask containing an estimated 300 pounds of elemental mercury leaked. Small amounts of mercury contaminated the road shoulder, but most of the mercury leaked out in the towns where he stopped. Some villagers collected the droplets of mercury to use in cultural practices about which little was known by health officials. An international team conducted air monitoring outdoors as well as indoors, using the Lumex and the Jerome analyzers. Many homes exceeded 1 ppm. The Lumex proved satisfactory for identifying outdoor hot spots that were not visually apparent, as well as for monitoring the indoor cleanup and allowing people to return to their homes.

Urine monitoring for mercury indicated that most people (even in homes with elevated mercury levels) had urine concentrations $<20\,\mu g/L$. However, some people exceeded this value and underwent chelation therapy.

6.6. A mercury-contaminated building in Hoboken, NJ, USA

In the early 1990s a cooperative of 20 artists purchased a former industrial building from a tool-and-dye company. It later became known that General Electric Corporation had formerly owned the factory and manufactured mercury vapor lamps there. The building was highly contaminated with mercury. The artists converted the building into apartments and studios. Shortly after moving in, some residents discovered droplets of elemental mercury. Public health authorities were notified and the building was soon found to have high levels of mercury vapor. Of the 29 people tested, 19 had mercury levels $>20\,\mu g/L$, and several showed otherwise unexplained decrements in neurobehavioral performance (Fiedler et al., 1999). Two subjects had such extremely low mercury levels that they were suspected of diluting their urine to avoid being evacuated. Paradoxically this was discovered because the mercury/creatinine ratio was extremely high because the denominator as well as the numerator were reduced. Air sampling indicated that virtually all apartments had mercury levels exceeding $1\,\mu g/m^3$, whereas about a third exceeded the occupational standard of $50\,\mu g/m^3$ (Orloff et al., 1997). Moreover, mercury levels correlated with air levels in apartments and duration of residency.

6.7. Summary

Table 3 is an exposure matrix summarizing the pathways of exposure to various mercury compounds. Cases 6.1–6.6 represent primarily the air inhalation and food ingestion routes.

7. Conclusions

The cases illustrate exposure to both elemental and organic mercury compounds in air and food, uptake through lungs, GI tract, and skin, and elevations of mercury in blood, urine and hair. The choice of the most useful biomarker depends on the species of metal (e.g., hair is useful for organic but less for elemental exposure), and the question being asked, is current or

Table 3
Pathways of exposure to various species of mercury

Route\medium	Air	Soil	Water	Food	Other
Inhalation	+ + + Mainly Hg^0; household, cultural, occupational Cajamarca, Peru; Hoboken, NJ	Mainly Hg_i in dust	+ + Hg^0 in ground water during cooking and showering	Not a pathway	Amalgam from oral cavity (often in 200 ng/m^3 range)
Ingest	+ Airborne deposition on food, mainly occupational	+ + + Main route for toddlers	+ + Some water supplies contaminated by historic agricultural use	+ + + Mainly MeHg in fish; also fungicides on grain Minamata, Japan; Iraq; Amazon	Amalgam (minute quantities of Hg^0 with negligible GI absorption)
Skin	0	+ Some organic compounds	+ Some organic compounds	0	DMM Many historic medications laboratory case
Injection					Thimerosal in vaccines

Hg^0, elemental mercury; Hg_i, ionic species of mercury; MeHg, methylmercury; DMM, dimethylmercury.

historic exposure of concern. Identification of the metal species, its bioavailability in the medium, and the levels of a biomarker, can estimate or validate risk assessments.

References

Amin-Zaki, L., Elhassani, S., Majeed, M.A., Clarkson, T.W., Doherty, R.A., Greenwood, M., 1974. Intra-uterine methylmercury poisoning in Iraq. Pediatrics 54 (5), 587–595.

Amorim, M.I., Mergler, D., Bahia, M.O., Dubeau, H., Miranda, D., Lebel, J., Burbano, R.R., Lucotte, M., 2000. Cytogenetic damage related to low levels of methyl mercury contamination in the Brazilian Amazon. An. Acad. Bras. Cienc. 72 (4), 497–507.

ATSDR, 2000. Toxicologic Profile for Mercury. Agency for Toxic Substances and Disease Registry, Centers for Disease Control and Prevention, Atlanta.

Blayney, M.B., 2001. The need for empirically derived permeation data for personal protective equipment: the death of Dr. Karen E. Wetterhahn. Appl. Occup. Environ. Hyg. 16 (2), 233–236.

Dolbec, J., Mergler, D., Sousa Passos, C.J., Sousa de Morais, S., Lebel, J., 2000. Methylmercury exposure affects motor performance of a riverine population of the Tapajos River, Brazilian Amazon. Int. Arch. Occup. Environ. Health 73 (3), 195–203.

Dolbec, J., Mergler, D., Larribe, F., Roulet, M., Lebel, J., Lucotte, M., 2001. Sequential analysis of hair mercury levels in relation to fish diet of an Amazonian population, Brazil. Sci. Total Environ. 271 (1–3), 87–97.

Eto, K., 2000. Minamata disease. Neuropathology 20 (Suppl.), S14–19.

Fiedler, N., Udasin, I., Gochfeld, M., Buckler, G., Kelly McNeil, K., Kipen, H., 1999. Neuropsychological and stress evaluation of a residential mercury exposure. Environ. Health Perspect. 107 (5), 343–347.

Fitzgerald, W., 1995. Is mercury increasing in the atmosphere? The need for an atmospheric mercury network (AMNET). Water, Air and Soil Pollution 80 (1), 245–254.

Futatsuka, M., Kitano, T., Shono, M., Fukuda, Y., Ushijima, K., Inaoka, T., Nagano, M., Wakamiya, J., Miyamoto, K., 2000. Health surveillance in the population living in a methyl mercury-polluted area over a long period. Environ. Res. 83 (2), 83–92.

Guimaraes, J.R., Roulet, M., Lucotte, M., Mergler, D., 2000. Mercury methylation along a lake-forest transect in the Tapajos river floodplain, Brazilian Amazon: seasonal and vertical variations. Sci. Total Environ. 261 (1–3), 1–98.

Kondo, K., 2000. Congenital Minamata disease: warnings from Japan's experience. J. Child Neurol. 15 (7), 458–464.

Kurland, L.T., Faro, S.N., Siedler, H., 1960. Minamata disease. World Neurol 1 (4), 370–395.

New Jersey Mercury Task Force, 2001. Mercury in New Jersey. New Jersey Department of Environmental Protection, Trenton, NJ.

Nierenberg, D.W., Nordgren, R.E., Chang, M.B., Siegler, R.W., Blayney, M.B., Hochberg, F., Toribara, T.Y., Cernichiari, E., Clarkson, T., 1998. Delayed cerebellar disease and death after accidental exposure to dimethylmercury. N. Engl. J. Med. 338 (23), 1672–1676.

NRC, 2000. Toxicological Effects of Methylmercury. National Research Council, National Academy Press, Washington, DC.

Orloff, K.G., Ulirsch, G., Wilder, L., Block, A., Fagliano, J., Pasqualo, J., 1997. Human exposure to elemental mercury in a contaminated residential building. Arch. Environ. Health. 52 (3), 169–172.

Stern, A.H., Gochfeld, M., Weisel, C., Burger, J., 2001. Mercury and methylmercury exposure in the New Jersey pregnant population. Arch. Environ. Health 56 (1), 4–10.

US EPA, 1997. Report to Congress: Mercury. US Environmental Protection Agency, Washington DC.