資料Ｎｏ．２－２

UNITED NATIONS
ENVIRONMENT PROGRAMME

CHEMICALS

UNEP

GLOBAL MERCURY ASSESSMENT

Issued by UNEP Chemicals

Geneva, Switzerland

December 2002

Disclaimer:

This publication is intended to serve as a guide. While the information provided is believed to be accurate, UNEP disclaims any responsibility for possible inaccuracies or omissions and consequences that may flow from them. Neither UNEP nor any individual involved in the preparation of this publication shall be liable for any injury, loss, damage or prejudice of any kind that may be caused by persons who have acted based on their understanding of the information contained in this publication.

The designation employed and the presentation of material in this publication do not imply any expression of any opinion whatsoever on the part of the United Nations or UNEP concerning the legal status of any country, territory, city or area or any of its authorities, or concerning any definition of frontiers or boundaries.

This publication is produced within the framework of the
Inter-Organization Programme for the Sound Management of Chemicals (IOMC).

*The **Inter-Organization Programme for the Sound Management of Chemicals (IOMC)**, was established in 1995 by UNEP, ILO, FAO, WHO, UNIDO and OECD (Participating Organizations), following recommendations made by the 1992 UN Conference on Environment and Development to strengthen cooperation and increase coordination in the field of chemical safety. In January 1998, UNITAR formally joined the IOMC as a Participating Organization. The purpose of the IOMC is to promote coordination of the policies and activities pursued by the Participating Organizations, jointly or separately, to achieve the sound management of chemicals in relation to human health and the environment.*

This document is available from:

UNEP Chemicals
11-13, chemin des Anémones
CH-1219 Châtelaine, Geneva
Switzerland
Phone: +41 22 917 1234
Fax: +41 22 797 3460
E-mail: chemicals@unep.ch
Website : http://www.chem.unep.ch

UNEP Chemicals is a part of UNEP's Technology, Industry and Economics Division

Table of Contents

Summary of the report

CHAPTER 1 - Introduction

36. This report responds to the request of the Governing Council (GC) of the United Nations Environment Programme (UNEP), through GC decision 21/5, that UNEP undertake a global assessment of mercury and mercury compounds, in cooperation with other members of the Inter-Organization Programme for the Sound Management of Chemicals (IOMC), to be presented to the Governing Council at its 22nd session in 2003. The assessment would include contributions from Governments, intergovernmental and non-governmental organizations and the private sector, and cover a number of specific elements defined in the GC decision. These elements are covered as far as possible in the different chapters of the report.

37. As part of the implementation of GC decision 21/5, UNEP established a Global Mercury Assessment Working Group to assist in the drafting and finalization of this report, first through a comment round by mail, then through a meeting of the Working Group, which took place 9-13 September 2002 in Geneva, Switzerland. The Working Group was open-ended and consisted of members nominated by Governments, intergovernmental organizations and non-governmental organizations.

38. This report will be forwarded to the Governing Council for consideration at its 22nd session in February 2003. By having initiated the development of this assessment report, the Governing Council will have a better basis for considering if any international action on mercury is called for in order to promote environmentally sound management of mercury and its compounds. The report will contribute to increased awareness and understanding among decision makers of the major issues related to mercury and its compounds, thereby facilitating the debate on the issue at the next session of the Governing Council.

CHAPTER 2 – Chemistry

39. Mercury occurs naturally in the environment and exists in a large number of forms. Like lead or cadmium, mercury is a constituent element of the earth, a heavy metal. In pure form, it is known alternatively as "elemental" or "metallic" mercury (also expressed as Hg(0) or Hg^0). Mercury is rarely found in nature as the pure, liquid metal, but rather within compounds and inorganic salts. Mercury can be bound to other compounds as monovalent or divalent mercury (also expressed as Hg(I) and Hg(II) or Hg^{2+}, respectively). Many inorganic and organic compounds of mercury can be formed from Hg(II).

40. Elemental mercury is a shiny, silver-white metal that is a liquid at room temperature and is traditionally used in thermometers and some electrical switches. If not enclosed, at room temperature some of the metallic mercury will evaporate and form mercury vapours. Mercury vapours are colourless and odourless. The higher the temperature, the more vapours will be released from liquid metallic mercury. Some people who have breathed mercury vapours report a metallic taste in their mouths.

41. Mercury is mined as mercuric sulphide (cinnabar ore). Through history, deposits of cinnabar have been the source ores for commercial mining of metallic mercury. The metallic form is refined from mercuric sulphide ore by heating the ore to temperatures above 540 ° C. This vaporises the mercury in the ore, and the vapours are then captured and cooled to form the liquid metal mercury.

42. Inorganic mercuric compounds include mercuric sulphide (HgS), mercuric oxide (HgO) and mercuric chloride ($HgCl_2$). These mercury compounds are also called mercury salts. Most inorganic mercury compounds are white powders or crystals, except for mercuric sulphide, which is red and turns black after exposure to light. Some mercury salts (such as $HgCl_2$) are sufficiently volatile to exist as an atmospheric gas. However, the water solubility and chemical reactivity of these inorganic (or divalent) mercury gases lead to much more rapid deposition from the atmosphere than for elemental mercury. This results in sig-

nificantly shorter atmospheric lifetimes for these divalent mercury gases than for the elemental mercury gas.

43. When mercury combines with carbon, the compounds formed are called "organic" mercury compounds or organomercurials. There is a potentially large number of organic mercury compounds (such as dimethylmercury, phenylmercury, ethylmercury and methylmercury); however, by far the most common organic mercury compound in the environment is methylmercury. Like the inorganic mercury compounds, both methylmercury and phenylmercury exist as "salts" (for example, methylmercuric chloride or phenylmercuric acetate). When pure, most forms of methylmercury and phenylmercury are white crystalline solids. Dimethylmercury, however, is a colourless liquid.

44. Several forms of mercury occur naturally in the environment. The most common natural forms of mercury found in the environment are metallic mercury, mercuric sulphide, mercuric chloride, and methylmercury. Some micro-organisms and natural processes can change the mercury in the environment from one form to another.

45. Elemental mercury in the atmosphere can undergo transformation into inorganic mercury forms, providing a significant pathway for deposition of emitted elemental mercury.

46. The most common organic mercury compound that micro-organisms and natural processes generate from other forms is methylmercury. Methylmercury is of particular concern because it can build up (bioaccumulate and biomagnify) in many edible freshwater and saltwater fish and marine mammals to levels that are many thousands of times greater than levels in the surrounding water.

47. Methylmercury can be formed in the environment by microbial metabolism (biotic processes), such as by certain bacteria, and by chemical processes that do not involve living organisms (abiotic processes). Although, it is generally believed that its formation in nature is predominantly due to biotic processes. Significant direct anthropogenic (or human generated) sources of methylmercury are currently not known, although historic sources have existed. Indirectly, however, anthropogenic releases contribute to the methylmercury levels found in nature because of the transformation of other forms. Examples of direct release of organic mercury compounds are the Minamata methylmercury-poisoning event that occurred in the 1950's where organic mercury by-products of industrial-scale acetaldehyde production were discharged in the local bay, and the Iraqi poisoning events where wheat treated with a seed dressing containing organic mercury compounds were used for bread. Also, new research has shown that methylmercury can be released directly from municipal waste landfills (Lindberg *et al.*, 2001) and sewage treatment plants (Sommar *et al.*, 1999), but the general significance of this source is still uncertain.

48. Being an element, mercury cannot be broken down or degraded into harmless substances. Mercury may change between different states and species in its cycle, but its simplest form is elemental mercury, which itself is harmful to humans and the environment. Once mercury has been liberated from either ores or from fossil fuel and mineral deposits hidden in the earth's crust and released into the biosphere, it can be highly mobile, cycling between the earth's surface and the atmosphere. The earth's surface soils, water bodies and bottom sediments are thought to be the primary biospheric sinks for mercury.

Mercury exists in the following main states under natural conditions

- As metallic vapour and liquid/elemental mercury;
- Bound in mercury containing minerals (solid);
- As ions in solution or bound in ionic compounds (inorganic and organic salts);
- As soluble ion complexes;
- As gaseous or dissolved non-ionic organic compounds;
- Bound to inorganic or organic particles/matter by ionic, electrophilic or lipophilic adsorption.

Significance of mercury speciation

49. The different forms mercury exists in (such as elemental mercury vapour, methylmercury or mercuric chloride) are commonly designated "species". As mentioned above, the main groups of mercury species are elemental mercury, inorganic and organic mercury forms. Speciation is the term commonly used to represent the distribution of a quantity of mercury among various species.

50. Speciation plays an important part in the toxicity and exposure of mercury to living organisms. Among other things, the species influence:

- The physical availability for exposure - if mercury is tightly bound to in-absorbable material, it cannot be readily taken up (e.g. into the blood stream of the organism);
- The internal transport inside the organism to the tissue on which it has toxic effects - for example the crossing of the intestinal membrane or the blood-brain barrier;
- Its toxicity (partly due to the above mentioned);
- Its accumulation, bio-modification, detoxification in – and excretion from – the tissues;
- Its bio-magnification on its way up the trophic levels of the food chain (an important feature particularly for methylmercury).

51. Speciation also influences the transport of mercury within and between environmental compartments including the atmosphere and oceans, among others. For example, the speciation is a determining factor for how far from the source mercury emitted to air is transported. Mercury adsorbed on particles and ionic (e.g. divalent) mercury compounds will fall on land and water mainly in the vicinity of the sources (local to regional distances), while elemental mercury vapour is transported on a hemispherical/global scale making mercury emissions a global concern. Another example is the so-called "polar sunrise mercury depletion incidence", where the transformation of elemental mercury to divalent mercury is influenced by increased solar activity and the presence of ice crystals, resulting in a substantial increase in mercury deposition during a three month period (approximately March to June).

52. Moreover, speciation is very important for the controllability of mercury emissions to air. For example, emissions of inorganic mercuric compounds (such as mercuric chloride) are captured reasonably well by some control devices (such as wet-scrubbers), while capture of elemental mercury tends to be low for most emission control devices.

CHAPTER 3 – Toxicology

53. The toxicity of mercury depends on its chemical form, and thus symptoms and signs are rather different in exposure to elemental mercury, inorganic mercury compounds, or organic mercury compounds (notably alkylmercury compounds such as methylmercury and ethylmercury salts, and dimethylmercury). The sources of exposure are also markedly different for the different forms of mercury. For alkylmercury compounds, among which methylmercury is by far the most important, the major source of exposure is diet, especially fish and other seafood. For elemental mercury vapour, the most important source for the general population is dental amalgam, but exposure at work may in some situations exceed this by many times. For inorganic mercury compounds, diet is the most important source for the majority of people. However, for some segments of populations, use of skin-lightening creams and soaps that contain mercury, and use of mercury for cultural/ritualistic purposes or in traditional medicine, can also result in substantial exposures to inorganic or elemental mercury.

54. While it is fully recognised that mercury and its compounds are highly toxic substances for which potential impacts should be considered carefully, there is ongoing debate on how toxic these substances, especially methylmercury, are. New findings during the last decade indicate that toxic effects may be taking place at lower concentrations than previously thought, and potentially larger parts of the global population may be affected. As the mechanisms of subtle toxic effects – and proving whether such effects are taking place – are extremely complex issues, a complete understanding has so far not been reached on this very important question.

Methylmercury

55. Of the organic mercury compounds, methylmercury occupies a special position in that large populations are exposed to it, and its toxicity is better characterized than that of other organic mercury compounds. Within the group of organic mercury compounds, alkylmercury compounds (especially ethylmercury and methylmercury) are thought to be rather similar as to toxicity (and also historical use as pesticides), while other organic mercury compounds, such as phenylmercury, resemble more inorganic mercury in their toxicity.

56. Methylmercury is a well-documented neurotoxicant, which may in particular cause adverse effects on the developing brain. Moreover, this compound readily passes both the placental barrier and the blood-brain barrier, therefore, exposures during pregnancy are of highest concern. Also, some studies suggest that even small increases in methylmercury exposures may cause adverse effects on the cardiovascular system, thereby leading to increased mortality. Given the importance of cardiovascular diseases worldwide, these findings, although yet to be confirmed, suggest that methylmercury exposures need close attention and additional follow-up. Moreover, methylmercury compounds are considered possibly carcinogenic to humans (group 2B) according to the International Agency for Research on Cancer (IARC, 1993), based on their overall evaluation.

Elemental mercury and inorganic mercury compounds

57. The main route of exposure for elemental mercury is by inhalation of the vapours. About 80 percent of inhaled vapours are absorbed by the lung tissues. This vapour also easily penetrates the blood-brain barrier and is a well-documented neurotoxicant. Intestinal absorption of elemental mercury is low. Elemental mercury can be oxidized in body tissues to the inorganic divalent form.

58. Neurological and behavioural disorders in humans have been observed following inhalation of elemental mercury vapour. Specific symptoms include tremors, emotional lability, insomnia, memory loss, neuromuscular changes, and headaches. In addition, there are effects on the kidney and thyroid. High exposures have also resulted in death. With regard to carcinogenicity, the overall evaluation, according to IARC (1993), is that metallic mercury and inorganic mercury compounds are not classifiable as to carcinogenicity to humans (group 3). A critical effect on which risk assessment could be based is therefore the neurotoxic effects, for example the induction of tremor. The effects on the kidneys (the renal tubule) should also be considered; they are the key endpoint in exposure to inorganic mercury compounds. The effect may well be reversible, but as the exposure to the general population tends to be continuous, the effect may still be relevant.

Summary of effect levels

59. To put the level of exposures for methylmercury in perspective, for the most widely accepted non-lethal adverse effect (neurodevelopmental effects), the United States (US) National Research Council (NRC, 2000) has estimated the benchmark dose (BMD) to be 58 micrograms per litre (µg/l) total mercury in cord blood (or 10 micrograms per gram (µg/g) total mercury in maternal hair) using data from the Faroe Islands study of human mercury exposures (Grandjean et al., 1997). This BMD level is the lower 95% confidence limit for the exposure level that causes a doubling of a 5% prevalence of abnormal neurological performance (developmental delays in attention, verbal memory and language) in children exposed in-utero in the Faroe Islands study. These are the tissue levels estimated to result from an average daily intake of about 1 µg methylmercury per kg body weight per day (1 µg/kg body weight per day).

60. Other adverse effects have been seen in humans with less reliability or at much higher exposures. For methylmercury, effects have been seen on the adult nervous system, on cardiovascular disease, on cancer incidence and on genotoxicity. Also, effects have been reported on heart rate variability and blood pressure in 7 year-old children exposed prenatally, and on cardiovascular mortality in adults. For elemental mercury and inorganic mercury compounds, effects have been seen on: the excretion of low molecular weight proteins; on enzymes associated with thyroid function; on spontaneous abortion rates; genotoxicity; respiratory system; gastrointestinal (digestion) system; liver; immune system; and the skin.

Dietary considerations

61. Fish are an extremely important component of the human diet in many parts of the world and provide nutrients (such as protein, omega-3 fatty acids and others) that are not easily replaced. Mercury is a major threat to this food supply. Certainly, fish with low methylmercury levels are intrinsically more healthful for consumers than fish with higher levels of methylmercury, if all other factors are equal.

62. There is limited laboratory evidence suggesting that several dietary components might reduce (e.g. selenium, vitamin E, omega-3 fatty acids) or enhance (e.g. alcohol) mercury's toxicity for some end-points. However, conclusions cannot be drawn from these data at this time.

CHAPTER 4 - Current mercury exposure and risk evaluations for human health

63. As mentioned earlier, the general population is primarily exposed to methylmercury through the diet (especially fish) and to elemental mercury vapours due to dental amalgams. Depending on local mercury pollution load, substantial additional contributions to the intake of total mercury can occur through air and water. Also, personal use of skin-lightening creams and soaps, mercury use for religious, cultural and ritualistic purposes, the presence of mercury in some traditional medicines (such as certain traditional Asian remedies) and mercury in the home or working environment can result in substantial elevations of human mercury exposure. For example, elevated air levels in homes have resulted from mercury spills from some old gas meters and other types of spills. Also, elevated mercury levels in the working environment have been reported for example in chlor-alkali plants, mercury mines, thermometer factories, refineries and dental clinics, as well as in mining and manufacturing of gold extracted with mercury. Additional exposures result from the use of Thimerosal/Thiomersal (ethylmercury thiosalicylate) as a preservative in some vaccines and other pharmaceuticals. The relative impacts of mercury from local pollution, occupational exposure, certain cultural and ritualistic practices and some traditional medicines may today vary considerably between countries and regions in the world, and are significant in some regions.

64. The chapter gives examples of data on total mercury and methylmercury exposures primarily from fish diets, but also other sources in different parts of the world, including Sweden, Finland, the United States of America (USA), the Arctic, Japan, China, Indonesia, Papua New Guinea, Thailand, Republic of Korea, Philippines, the Amazonas and French Guyana. For example, in a study of a representative group of about 1700 women in the USA (aged 16-49 years) for years 1999-2000, about 8 percent of the women had mercury concentrations in blood and hair exceeding the levels corresponding to the US EPA's reference dose (an estimate of a safe dose). As shown in the chapter, data indicate exposures are generally higher in Greenland, Japan and some other areas as compared to the USA.

65. In some of these countries and areas, local and regional mercury depositions have affected the mercury contamination levels over the years and countermeasures have been taken during the last decades to reduce national emissions. Mercury emissions are, however, distributed over long distances in the atmosphere and oceans. This means that even countries with minimal mercury emissions, and other areas situated remotely from dense human activity, may be adversely affected. For example, high mercury exposures have been observed in the Arctic far distances from any significant sources.

66. Data on mercury concentrations in fish have been submitted from a number of nations and international organisations. Additionally, many investigations of mercury levels in fish are reported in the literature. Submitted data, giving examples of mercury concentrations in fish from various locations in the world, are summarised in the chapter. The mercury concentrations in various fish species are generally from about 0.05 to 1.4 milligrams of mercury per kilogram of fish tissue (mg/kg) depending on factors such as pH and redox potential of the water, and species, age and size of the fish. Since mercury biomagnifies in the aquatic food web, fish higher on the food chain (or of higher trophic level) tend to have higher levels of mercury. Hence, large predatory fish, such as king mackeral, pike, shark, swordfish, walleye, barracuda, large tuna (as opposed to the small tuna usually used for canned tuna), scabbard and marlin, as well as seals and toothed whales, contain the highest concentrations. The available data indicate that mercury is present all over the globe (especially in fish) in concentrations that adversely affect

human beings and wildlife. These levels have led to consumption advisories (for fish, and sometimes marine mammals) in a number of countries, warning people, especially sensitive subgroups (such as pregnant women and young children), to limit or avoid consumption of certain types of fish from various waterbodies. Moderate consumption of fish (with low mercury levels) is not likely to result in exposures of concern. However, people who consume higher amounts of contaminated fish or marine mammals may be highly exposed to mercury and are therefore at risk.

CHAPTER 5 – Impacts of mercury on the environment

Build-up of mercury in food webs

67. A very important factor in the impacts of mercury to the environment is its ability to build up in organisms and up along the food chain. Although all forms of mercury can accumulate to some degree, methylmercury is absorbed and accumulates to a greater extent than other forms. Inorganic mercury can also be absorbed, but is generally taken up at a slower rate and with lower efficiency than is methylmercury. The biomagnification of methylmercury has a most significant influence on the impact on animals and humans. Fish appear to bind methylmercury strongly, nearly 100 percent of mercury that bioaccumulates in predator fish is methylmercury. Most of the methylmercury in fish tissue is covalently bound to protein sulfhydryl groups. This binding results in a long half-life for elimination (about two years). As a consequence, there is a selective enrichment of methylmercury (relative to inorganic mercury) as one moves from one trophic level to the next higher trophic level.

Bioaccumulation and biomagnification

The term **bioaccumulation** refers to the net accumulation over time of metals within an organism from both biotic (other organisms) and abiotic (soil, air, and water) sources.

The term **biomagnification** refers to the progressive build up of some heavy metals (and some other persistent substances) by successive trophic levels – meaning that it relates to the concentration ratio in a tissue of a predator organism as compared to that in its prey (AMAP, 1998).

68. In contrast to other mercury compounds the elimination of methylmercury from fish is very slow. Given steady environmental concentrations, mercury concentrations in individuals of a given fish species tend to increase with age as a result of the slow elimination of methylmercury and increased intake due to changes in trophic position that often occur as fish grow to larger sizes (i.e., the increased fish-eating and the consumption of larger prey items). Therefore, older fish typically have higher mercury concentrations in the tissues than younger fish of the same species.

69. The mercury concentrations are lowest in the smaller, non-predatory fish and can increase many-fold on the way up the food chain. Apart from the concentration in food, other factors affect the bioaccumulation of mercury. Of most importance are the rates of methylation and demethylation by mercury methylating bacteria (e.g., sulphate reducers). When all of these factors are combined, the net methylation rate can strongly influence the amount of methylmercury that is produced and available for accumulation and retention by aquatic organisms. As described in chapter 2, several parameters in the aquatic environment influence the methylation of mercury and thereby its biomagnification. While much is generally known about mercury bioaccumulation and biomagnification, the process is extremely complex and involves complicated biogeochemical cycling and ecological interactions. As a result, although accumulation/magnification can be observed, the extent of mercury biomagnification in fish is not easily predicted across different sites.

70. At the top levels of the aquatic food web are fish-eating species, such as humans, seabirds, seals and otters. The larger wildlife species (such as eagles, seals) prey on fish that are also predators, such as trout and salmon, whereas smaller fish-eating wildlife (such as kingfishers) tend to feed on the smaller forage fish. In a study of fur-bearing animals in Wisconsin, the species with the highest tissue levels of

mercury were otter and mink, which are top mammalian predators in the aquatic food chain. Top avian predators of aquatic food chains include raptors such as the osprey and bald eagle. Thus, mercury is transferred and accumulated through several food web levels (US EPA, 1997). Aquatic food webs tend to have more levels than terrestrial webs, where wildlife predators rarely feed on each other, and therefore the aquatic biomagnification typically reaches higher values.

Mercury compounds toxic to wildlife

71. Methylmercury is a central nervous system toxin, and the kidneys are the organs most vulnerable to damage from inorganic mercury. Severe neurological effects were already seen in animals in the notorious case from Minamata, Japan, prior to the recognition of the human poisonings, where birds experienced severe difficulty in flying, and exhibited other grossly abnormal behaviour. Significant effects on reproduction are also attributed to mercury, and methylmercury poses a particular risk to the developing fetus since it readily crosses the placental barrier and can damage the developing nervous system.

72. In birds, adverse effects of mercury on reproduction can occur at egg concentrations as low as 0.05 to 2.0 mg/kg (wet weight). Eggs of certain Canadian species are already in this range, and concentrations in the eggs of several other Canadian species continue to increase and are approaching these levels.

73. The levels of mercury in Arctic ringed seals and beluga whales have increased by 2 to 4 times over the last 25 years in some areas of the Canadian Arctic and Greenland. In warmer waters as well, predatory marine mammals may also be at risk. In a study of Hong Kong's population of hump-backed dolphins, mercury was identified as a particular health hazard, more than other heavy metals.

Vulnerable ecosystems

74. Recent evidence suggests that mercury is responsible for a reduction of micro-biological activity vital to the terrestrial food chain in soils over large parts of Europe – and potentially in many other places in the world with similar soil characteristics. Preliminary critical limits to prevent ecological effects due to mercury in organic soils have been set at 0.07-0.3 mg/kg for the total mercury content in soil.

75. On the global scale, the Arctic region has been in focus recently because of the long-range transport of mercury. However, impacts from mercury are by no means restricted to the Arctic region of the world. The same food web characteristics - and a similar dependence on a mercury contaminated food source - are found in specific ecosystems and human communities in many countries of the world, particularly in places where a fish diet is predominant.

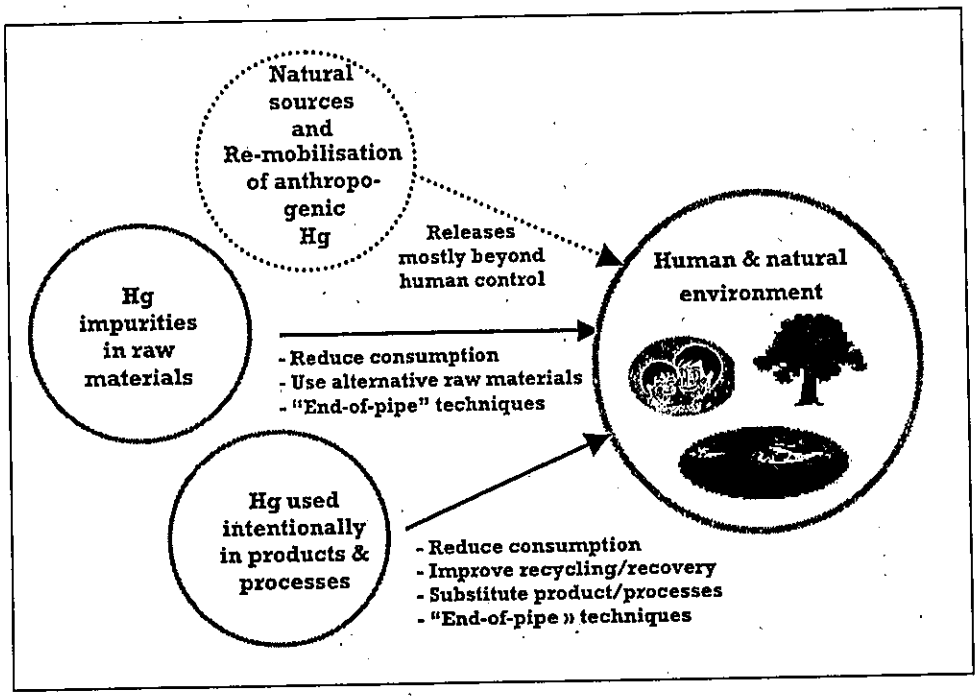
76. Rising water levels associated with global climate change may also have implications for the methylation of mercury and its accumulation in fish. For example, there are indications of increased formation of methylmercury in small, warm lakes and in many newly flooded areas.

CHAPTER 6 – Sources and cycling of mercury to the global environment

77. The releases of mercury to the biosphere can be grouped in four categories:

- Natural sources - releases due to natural mobilisation of naturally occurring mercury from the Earth's crust, such as volcanic activity and weathering of rocks;
- Current anthropogenic (associated with human activity) releases from the mobilisation of mercury impurities in raw materials such as fossil fuels – particularly coal, and to a lesser extent gas and oil – and other extracted, treated and recycled minerals;
- Current anthropogenic releases resulting from mercury used intentionally in products and processes, due to releases during manufacturing, leaks, disposal or incineration of spent products or other releases;
- Re-mobilisation of historic anthropogenic mercury releases previously deposited in soils, sediments, water bodies, landfills and waste/tailings piles.

78. The figure below shows these release categories with main types of possible control mechanisms.



79. The recipients of mercury releases to the environment include the atmosphere, water environments (aquatic) and soil environments (terrestrial). There are continuing interactions – fluxes of mercury – between these compartments. The speciation – the chemical form – of the released mercury varies depending on the source types and other factors. This also influences the impacts on human health and environment as different mercury species have different toxicity.

80. Given the understanding of the global mercury cycle, current releases add to the global pool of mercury in the biosphere – mercury that is continuously mobilised, deposited on land and water surfaces, and re-mobilised. Being an element, mercury is persistent – it cannot be broken down to less toxic substances in the environment. The only long-term sinks for removal of mercury from the biosphere are deep-sea sediments and, to a certain extent, controlled landfills, in cases where the mercury is physio-chemically immobilised and remains undisturbed by anthropogenic or natural activity (climatic and geological). This also implies that even as the anthropogenic releases of mercury are gradually eliminated, decreases in some mercury concentrations – and related environmental improvements – will occur only slowly, most likely over several decades or longer. However, improvements may occur more quickly in specific locations or regions that are largely impacted by local or regional sources.

Local releases – global effects

81. The origins of atmospheric mercury deposition (flow of mercury from air to land and oceans) are local and regional as well as hemispherical or global. Several large studies have supported the conclusion that, in addition to local sources (such as chlor-alkali production, coal combustion and waste incineration facilities), the general background concentration of mercury in the global atmosphere contributes significantly to the mercury burden at most locations. Similarly, virtually any local source contributes to the background concentration – the global mercury pool in the biosphere - much of which represents anthropogenic releases accumulated over the decades. Also, the ocean currents are media for long-range mercury transport, and the oceans are important dynamic sinks of mercury in the global cycle.

82. The majority of atmospheric anthropogenic emissions are released as gaseous elemental mercury. This is capable of being transported over very long distances with the air masses. The remaining part of air emissions are in the form of gaseous divalent compounds (such as $HgCl_2$) or bound to particles present in the emission gas. These species have a shorter atmospheric lifetime than elemental vapour and will deposit via wet or dry processes within roughly 100 to 1000 kilometers. However, significant conversion

between mercury species may occur during atmospheric transport, which will affect the transport distance.

83. The atmospheric residence time of elemental mercury is in the range of months to roughly one year. This makes transport on a hemispherical scale possible and emissions in any continent can thus contribute to the deposition in other continents. For example, based on modelling of the intercontinental mercury transport performed by EMEP/MSC-E (Travnikov and Ryaboshapko, 2002), up to 50 percent of anthropogenic mercury deposited to North America is from external sources. Similarly, contributions of external sources to anthropogenic mercury depositions to Europe and Asia were estimated to be about 20 percent and 15 percent, respectively.

84. Furthermore, as mentioned, mercury is also capable of re-emissions from water and soil surfaces. This process greatly enhances the overall residence time of mercury in the environment. Recent findings by Lindberg *et al.* (2001) indicate re-emission rates of approximately 20 percent over a two-year period, based on stable mercury isotope measurements in north-western Ontario, Canada.

Anthropogenic sources of mercury releases

85. A large portion of the mercury present in the atmosphere today is the result of many years of releases due to anthropogenic activities. The natural component of the total atmospheric burden is difficult to estimate, although a recent study (Munthe *et al.*, 2001) has suggested that anthropogenic activities have increased the overall levels of mercury in the atmosphere by roughly a factor of 3.

86. While there are some natural emissions of mercury from the earth's crust, anthropogenic sources are the major contributors to releases of mercury to the atmosphere, water and soil.

Examples of important sources of anthropogenic releases of mercury

Releases from mobilisation of mercury impurities:

- Coal-fired power and heat production (largest single source to atmospheric emissions)
- Energy production from other fossil carbon fuels
- Cement production (mercury in lime)
- Mining and other metallurgic activities involving the extraction and processing of virgin and recycled mineral materials, for example production of:
  - iron and steel
  - ferromanganese
  - zinc
  - gold
  - other non-ferrous metals

Releases from intentional extraction and use of mercury:

- Mercury mining
- Small-scale gold and silver mining (amalgamation process)
- Chlor-alkali production
- Use of fluorescent lamps, various instruments and dental amalgam fillings.
- Manufacturing of products containing mercury, for example:
  - thermometers
  - manometers and other instruments
  - electrical and electronic switches

Releases from waste treatment, cremation etc. (originating from both impurities and intentional uses of mercury):

- Waste incineration (municipal, medical and hazardous wastes)
- Landfills
- Cremation
- Cemeteries (release to soil)

87. There are significant uncertainties in the available release inventories, not only by source, but also by country. The best available estimates of mercury emissions to air from various significant sources are shown in the table below.

Table *Estimates of global atmospheric releases of mercury from a number of major anthropogenic sources in 1995 (metric tons/year). Releases to other media are not accounted for here. *1.*

Continent	Stationary combustion	Non-ferrous metal production *5	Pig iron and steel production	Cement production	Waste disposal *2	Artisanal gold mining *4	Sum, quantified sources *3
Europe	186	15	10	26	12		250
Africa	197	7.9	0.5	5.2			210
Asia	860	87	12	82	33		1070
North America	105	25	4.6	13	66		210
South America	27	25	1.4	5.5			60
Australia and Oceania	100	4.4	0.3	0.8	0.1		100
Sum, quantified sources, 1995 *3,4	1470	170	30	130	110	300	1900 +300
Based on references:	Pirrone *et al.* (2001)	Pirrone *et al.* (2001)	Pirrone *et al.* (2001)	Pirrone *et al.* (2001)	Pirrone *et al.* (2001)	Lacerda (1997)	

1 Note that releases to aquatic and terrestrial environments - as well as atmospheric releases from a number of other sources - are not included in the table, because no recent global estimates have been made. See chapter 6 for description of this issue.
2 Considered underestimated by authors of the inventory, see notes to table 6.10.
3 Represents total of the sources mentioned in this table, not all known sources. Sums are rounded and may therefore not sum up precisely.
4 Estimated emissions from artisanal gold mining refer to late 1980's/early 1990's situation. A newer reference (MMSD, 2002) indicates that mercury consumption for artisanal gold mining - and thereby most likely also mercury releases - may be even higher than presented here.
5 Production of non-ferrous metals releasing mercury, including mercury, zinc, gold, lead, copper, nickel.

88. The emissions from stationary combustion of fossil fuels (especially coal) and incineration of waste materials accounts for approximately 70 percent of the total quantified atmospheric emissions from major anthropogenic sources. As combustion of fossil fuels is increasing in order to meet the growing energy demands of both developing and developed nations, mercury emissions can be expected to increase accordingly in the absence of the deployment of control technologies or the use of alternative energy sources. Control technologies have been developed for coal combustion plants and waste incinerators with the primary intention of addressing acidifying substances (especially SO_2 and NO_x), and particulate matter (PM). Such existing technologies may provide some level of mercury control, but when viewed at the global level, currently these controls result in only a small reduction of mercury from these sources. Many control technologies are significantly less effective at reducing emissions of elemental mercury compared to other forms. Optimised technologies for mercury control are being developed and demonstrated, but are not yet commercially deployed.

89. Available global estimates of atmospheric emissions from waste incineration, as well as other releases originating from intentional uses of mercury in processes and products, are deemed underestimated, and to some degree incomplete. However, recorded virgin mercury production has been decreasing from about 6000 to about 2000 metric tons per year during the last two decades, and consequently, related releases from mining and usage of mercury may also be declining.

90. Anthropogenic emissions from a number of major sources have decreased during the last decade in North America and Europe due to reduction efforts. Also, total anthropogenic emissions to air have been declining in some developed countries in the last decade. For example, Canadian emissions were reduced from about 33 metric tons to 6 metric tons between 1990 and 2000.

Natural sources of mercury releases

91. Natural sources include volcanoes, evaporation from soil and water surfaces, degradation of minerals and forest fires. The natural mercury emissions are beyond our control, and must be considered part of our local and global living environment. It is necessary to keep this source in mind, however, as it does contribute to the environmental mercury levels. In some areas of the world, the mercury concentrations in the Earth's crust are naturally elevated, and contribute to elevated local and regional mercury concentrations in those areas.

92. Today's emissions of mercury from soil and water surfaces are composed of both natural sources and re-emission of previous deposition of mercury from both anthropogenic and natural sources. This makes it very difficult to determine the actual natural mercury emissions.

93. Published estimates of natural versus anthropogenic mercury emissions show significant variation, although more recent efforts have emphasized the importance of human contributions. Attempts to directly measure natural emissions are ongoing. Nonetheless, available information indicates that natural sources account for less than 50 percent of the total releases.

94. On average around the globe, there are indications that anthropogenic emissions of mercury have resulted in deposition rates today that are 1.5 to 3 times higher than those during pre-industrial times. In and around industrial areas the deposition rates have increased by 2 to 10 times during the last 200 years.

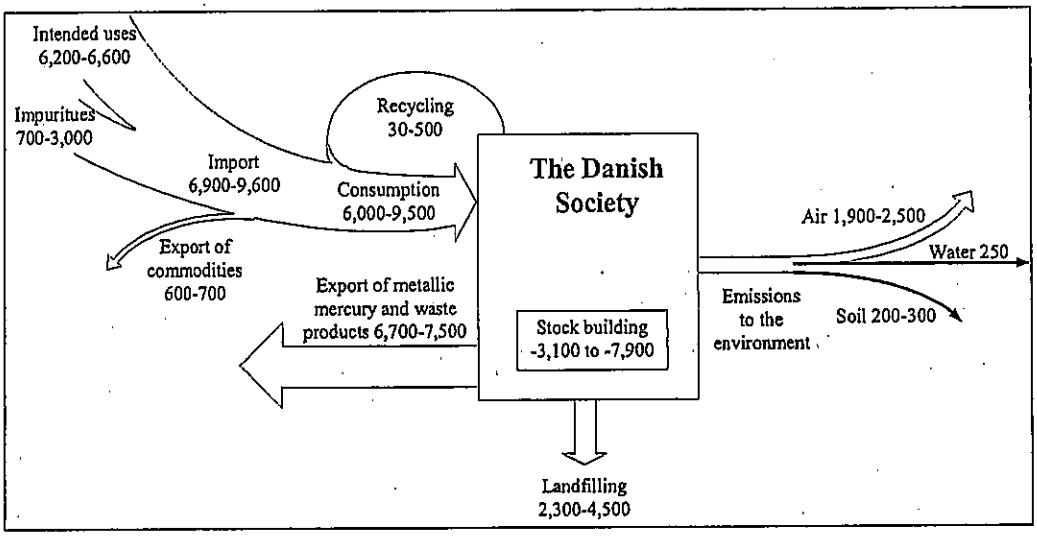
Contributions from intentional uses versus impurities in high volume materials

95. Regarding anthropogenic releases, the relative importance of intentional uses versus mobilisation of mercury impurities varies between countries and regions, particularly depending on:

- State of substitution of intentional uses (products and processes);
- Reliance on fossil fuels for energy production, particularly coal, and the presence of controls for other pollutants, which also reduce mercury emissions;
- Extent of mining and mineral extraction industry;
- Waste disposal pattern – incineration/landfilling;
- State of implementation of release control technologies in power production, waste incineration and various industrial processes.

96. For a number of countries, estimated contributions of intentional uses vary between 10 and 80 percent of the total domestic emissions to air, depending on the influence of the factors listed above. Rough estimates of distribution by main anthropogenic source types in each of these countries are shown in the chapter.

97. As an illustration, the figure below shows the overall turnover of mercury in the Danish society in 1992/93 in kilograms mercury/year (based on Maag *et al.*, 1996). (Note that inputs and outputs in the figure do not balance because outputs reflect higher inputs from previous years. Net change in stocks was negative.)



98. Denmark is a quite small country with relatively accurate monitoring of the flows of products and waste in the economy and the environment. Therefore, it has been possible to perform rather detailed balances, so-called substance flow assessments for mercury, which provide useful information on the contributions from different sectors to the total mercury burden in society and the environment. As shown in the figure, the majority of the input – more than two thirds – originated from intentional uses (chlor-alkali production and products), and the contributions from intentional uses to releases to air in 1992/93 could roughly be estimated at 50-80 percent of the total releases to air from Denmark. It should be noted that primary mineral extraction and processing is not as large a sector in Denmark, as in many other countries.

99. Examples of national distributions of anthropogenic mercury releases from different individual source types are given in the chapter. In countries where mercury mining or intentional use of mercury for small-scale gold mining is taking place, these sources can be significant.

CHAPTER 7 – Current production and use of mercury

Origin of mercury

100. Mercury is a natural component of the earth, with an average abundance of approximately 0.05 mg/kg in the earth's crust, with significant local variations. Mercury ores that are mined generally contain about one percent mercury, although the strata mined in Spain typically contain up to 12-14 percent mercury. While about 25 principal mercury minerals are known, virtually the only deposits that have been harvested for the extraction of mercury are cinnabar. Mercury is also present at very low levels throughout the biosphere. Its absorption by plants may account for the presence of mercury within fossil fuels like coal, oil, and gas, since these fuels are conventionally thought to be formed from geologic transformation of organic residues.

Sources of mercury to the market

101. The mercury available on the world market is supplied from a number of different sources, including (not listed in order of importance):

- Mine production of primary mercury (meaning extracted from ores within the earth's crust):
  - either as the main product of the mining activity,
  - or as by-product of mining or refining of other metals (such as zinc, gold, silver) or minerals;
- Recovered primary mercury from refining of natural gas (actually a by-product, when marketed, however, is not marketed in all countries);
- Reprocessing or secondary mining of historic mine tailings containing mercury;
- Recycled mercury recovered from spent products and waste from industrial production processes. Large amounts ("reservoirs") of mercury are "stored" in society within products still in use and "on the users' shelves";

- Mercury from government reserve stocks, or inventories;
- Private stocks (such as mercury in use in chlor-alkali and other industries), some of which may later be returned to the market.

102. The mining and other mineral extraction of primary mercury constitute the human mobilisation of mercury for intentional use in products and processes. Recycled mercury and mercury from stocks can be regarded as an anthropogenic re-mobilisation of mercury previously extracted from the Earth.

Continued mining of primary mercury

103. Despite a decline in global mercury consumption (global demand is less than half of 1980 levels), supply from competing sources and low prices, production of mercury from mining is still occurring in a number of countries. Spain, China, Kyrgyzstan and Algeria have dominated this activity in recent years, and several of the mines are state-owned. The table below gives information on recorded global primary production of mercury since 1981. There are also reports of small-scale, artisanal mining of mercury in China, Russia (Siberia), Outer Mongolia, Peru, and Mexico. It is likely that this production serves robust local demand for mercury, often for artisanal mining of gold – whether legal or illegal. Such mercury production would require both accessible mercury ores and low-cost labor in order for it to occur despite low-priced mercury available in the global commodity market.

Period	1981-1985	1986-1989	1990-1995	1996	1997	1998	1999	2000
Recorded annual, global primary production (in metric tons)	5500-7100	4900-6700	3300-6100	2600-2800	2500-2900	2000-2800	2100-2200	1800

Sources: See section 7.2.1.

Large supplies of recycled mercury may be marketed

104. Large quantities of mercury have come onto the market as a result of ongoing substitution and closing of mercury-based chlor-alkali production in Europe and other regions. Market analysis indicates that 700 - 900 metric tons per year of recycled mercury (corresponding to about 30 percent of the recorded primary production) has been marketed globally since the mid-1990's, of which the majority originated from chlor-alkali production facilities. However, to the extent there remains a legitimate demand for mercury, the re-use and recycling of mercury replaces the mining and smelting of virgin mercury, which would involve additional releases and would result in mobilising new mercury into the market and the environment.

105. The preference for reuse and recycling of mercury over mining - especially in the context of large mercury inventories coming onto the market - is complicated by the generally accepted economic rule that an excess supply of mercury drives the market price lower, which in turn encourages additional use or waste of mercury. For this reason, certain precautions are being taken, as described below.

106. Within the current decade and beyond, vast supplies of mercury will become available from conversion or shutdown of chlor-alkali facilities using the mercury process, as many European countries press for a phase-out of this process before 2010. From the European Union alone, this may introduce up to 13,000 metric tons of additional mercury to the market (equal to some 6-12 years of primary mercury production). In response to this potential glut of mercury, Euro Chlor, which represents the European chlor-alkali industry, has signed a contractual agreement with Miñas de Almadén in Spain. The agreement provides that Miñas de Almadén will buy the surplus mercury from the West-European chlor-alkali plants and put it on the market in place of mercury Almadén would otherwise have mined. All EU members of Euro Chlor have agreed to sell their surplus mercury to Almadén according to this agreement, and Euro Chlor believes most of the central and eastern European chlorine producers will also commit to this agreement. While this agreement clearly represents an effort by all parties to responsibly address the problem of surplus mercury, some people have the view that there are not yet adequate controls on where this mercury would be sold or how it would be used.

107. Similarly, large reserve stocks of mercury held by various governments have become superfluous, and are subject to future sales on the world market if approved by the relevant national authorities. This is the case in the USA, for example, which holds a 4,435 metric ton inventory of mercury. The sale of this mercury has been suspended since 1994, awaiting a determination of its potential environmental and market impacts. Prior to that, however, the sale of some of these stocks contributed significantly to the supply of mercury on the domestic US-market, and to exports as well. US government sales were equivalent to 18 to 97 percent of the domestic US demand for mercury in the years 1990-94 (US EPA, 1997; Maxson and Vonkeman, 1996).

Uses of mercury

108. The element mercury has been known for thousands of years, fascinating as the only liquid metal, and applied in a large number of products and processes utilising its unique characteristics. Being liquid at room temperature, being a good electrical conductor, having very high density and high surface tension, expanding/contracting uniformly over its entire liquid range in response to changes in pressure and temperature, and being toxic to micro-organisms (including pathogenic organisms) and other pests, mercury is an excellent material for many purposes.

109. In the past, a number of organic mercury compounds were used quite broadly, for example in pesticides (extensive use in seed dressing among others) and biocides in some paints, pharmaceuticals and cosmetics. While many of these uses have diminished in some parts of the world, organic mercury compounds are still used for several purposes. Some examples are the use of seed dressing with mercury compounds in some countries, use of dimethylmercury in small amounts as a reference standard for some chemical tests, and thimerosal (which contains ethylmercury) used as a preservative in some vaccines and other medical and cosmetic products since the 1930's. As the awareness of mercury's potential adverse impacts on health and the environment has been rising, the number of applications (for inorganic and organic mercury) as well as the volume of mercury used have been reduced significantly in many of the industrialised countries, particularly during the last two decades.

Examples of uses of mercury

As the metal (among others):

- for extraction of gold and silver (for centuries)
- as a catalyst for chlor-alkali production
- in manometers for measuring and controlling pressure
- in thermometers
- in electrical and electronic switches
- in fluorescent lamps
- in dental amalgam fillings

As chemical compounds (among others):

- in batteries (as a dioxide)
- biocides in paper industry, paints and on seed grain
- as antiseptics in pharmaceuticals
- laboratory analyses reactants
- catalysts
- pigments and dyes (may be historical)
- detergents (may be historical)
- explosives (may be historical)

110. However, many of the uses discontinued in the OECD countries are still alive in other parts of the world. Several of these uses have been prohibited or severely restricted in a number of countries because of their adverse impacts on humans and the environment.

111. Furthermore, while there is a general understanding of mercury production and use around the world, it is crucial to gain an even better understanding of global mercury markets and flows in order to assess demand, to design appropriate pollution prevention and reduction measures, and to monitor progress towards specific objectives.

CHAPTER 8 – Prevention and control technologies and practises

112. As noted in chapter 6, the sources of releases of mercury to the biosphere can be grouped in four major categories. Two of these categories (releases due to natural mobilisation of mercury and re-mobilisation of anthropogenic mercury previously deposited in soils, sediments and water bodies) are not well understood and largely beyond human control.

113. The other two are current anthropogenic mercury releases. Reducing or eliminating these releases may require:

- Investments in controlling releases from and substituting the use of mercury-contaminated raw materials and feedstocks, the main source of mercury releases from "unintentional" uses; and
- Reducing or eliminating the use of mercury in products and processes, the main source of releases caused by the "intentional" use of mercury.

114. The specific methods for controlling mercury releases from these sources vary widely, depending upon local circumstances, but fall generally under the following four groups:

A. Reducing mercury mining and consumption of raw materials and products that generate mercury releases;

B. Substitution (or elimination) of products, processes and practices containing or using mercury with non-mercury alternatives;

C. Controlling mercury releases through end-of-pipe techniques;

D. Mercury waste management.

115. The first two of these are "preventive" measures – preventing some uses or releases of mercury from occurring at all. The latter two are "control" measures, which reduce (or delay) some releases from reaching the environment. Within these very general groupings are a large number of specific techniques and strategies for reducing mercury releases and exposures. Whether or not they are applied in different countries depends upon government and local priorities, information and education about possible risks, the legal framework, enforcement, implementation costs, perceived benefits and other factors.

A. Reducing consumption of raw materials and products that generate mercury releases

116. Reducing consumption of raw materials and products that generate mercury releases is a preventive measure that is most often targeted at mercury containing products and processes, but may also result from improved efficiencies in the use of raw materials or in the use of fuels for power generation. This group of measures could potentially include the choice of an alternative raw material such as using natural gas for power generation instead of coal, or possibly by using a coal type with special constituents (such as more chlorine), because the mercury emissions from burning this type of coal might be easier to control than other coal types.

117. Another possible approach in some regions might be the use of coal with a lower trace mercury content (mercury concentrations appear to vary considerably in some regions depending on the origin of the raw materials). However, there are some limitations and potential problems with this approach. For example, as in the case of the utility preference for low-sulfur crude oil, it is likely that some utilities might be willing to pay more for low-mercury coal, which effectively lowers the market value of all high-mercury coal, which in turn might lead to higher consumption of high-mercury coal in regions where utilities have less rigorous emission controls. Moreover, data collected recently in the US indicate that coal supplies in the US do not vary significantly in mercury content.

118. Nonetheless, such preventive measures aimed at reducing mercury emissions are generally cost-effective, except in cases where an alternative raw material is significantly more expensive or where other problems limit this approach.

B. Substitution of products and processes containing or using mercury

119. Substitution of products and processes containing or using mercury with products and processes without mercury may be one of the most powerful preventive measures for influencing the entire flow of mercury through the economy and environment. It may substantially reduce mercury in households (and reduce accidental releases, as from a broken thermometer), the environment, the waste stream, incinerator emissions and landfills. Substitutions are mostly cost-effective, especially as they are demanded by a larger and larger market. This group of measures would also include the conversion of a fossil-fueled generating plant to a non-fossil technology.

120. At the same time, it would be a mistake to assume that substitution is always a clear winner. For example, in the case of energy-efficient fluorescent lamps, as long as there are no competitive substitutes that do not contain mercury, it is generally preferable from a product-life-cycle perspective to use a mercury-containing energy-efficient lamp rather than to use a less efficient standard incandescent lamp containing no mercury, as a result of current electricity production practises.

C. Controlling mercury emissions through end-of-pipe techniques

121. Controlling mercury emissions through end-of-pipe techniques, such as exhaust gas filtering, may be especially appropriate to raw materials with trace mercury contamination, including fossil-fueled power plants, cement production (in which the lime raw material often contains trace mercury), the extraction and processing of primary raw materials such as iron and steel, ferromanganese, zinc, gold and other non-ferrous metals and the processing of secondary raw materials such as iron and steel scrap. Existing control technologies that reduce SO_2, NO_x and PM for coal-fired boilers and incinerators, while not yet widely used in many countries, also yield some level of mercury control. For coal-fired boilers, reductions range from 0 to 96 percent, depending on coal type, boiler design, and emission control equipment. On average, the lower the coal rank, the lower the mercury reductions; however, reductions may also vary within a given coal rank. Technology for additional mercury control is under development and demonstration, but is not yet commercially deployed. In the long run, control strategies that target multiple pollutants, including SO_2, NO_x, PM and mercury, may be a cost-effective approach. However, end-of-pipe control technologies, while mitigating the problem of atmospheric mercury pollution, still result in mercury wastes that are potential sources of future emissions and must be disposed of or reused in an environmentally acceptable manner.

D. Mercury waste management

122. Mercury wastes, including those residues recovered by end-of-pipe technologies, constitute a special category of mercury releases, with the potential to affect populations far from the initial source of the mercury. Mercury waste management, the fourth "control" measure mentioned above, may consist of rendering inert the mercury content of waste, followed by controlled landfill, or it may not treat the waste prior to landfill. In Sweden, the only acceptable disposal of mercury waste now consists of "final storage" of the treated waste deep underground, although some technical aspects of this method are yet to be finalised.

123. Mercury waste management has become more complex as more mercury is collected from a greater variety of sources, including gas filtering products, sludges from the chlor-alkali industry, ashes, slags, and inert mineral residues, as well as used fluorescent tubes, batteries and other products that are often not recycled. Low concentrations of mercury in waste are generally permitted in normal landfills, while some nations only allow waste with higher mercury concentrations to be deposited in landfills that are designed with enhanced release control technologies to limit mercury leaching and evaporation. The cost of acceptable disposal of mercury waste in some countries is such that many producers now investigate whether alternatives exist in which they would not have to produce and deal with mercury waste. Mercury waste management, as it is most commonly done today, in accordance with national and local

regulations, increasingly requires long-term oversight and investment. Proper management of mercury wastes is important to reduce releases to the environment, such as those that occur due to spills (i.e. from broken thermometers and manometers) or releases that occur over time due to leakage from certain uses (e.g., auto switches, dental amalgams). In addition, given that there is a market demand for mercury, collection of mercury-containing products for recycling limits the need for new mercury mining.

Emission prevention and control measures

124. A well thought-out combination of emission prevention and control measures is an effective way to achieve optimal reduction of mercury releases. If one considers some of the more important sources of anthropogenic mercury releases, one may see how prevention and control measures might be combined and applied to these sources:

- Mercury emissions from **municipal and medical waste incinerators** may be reduced by separating the small fraction of mercury containing waste before it is combusted. For example, in the USA, free household mercury waste collections have been very successful in turning up significant quantities of mercury-containing products and even jars of elemental mercury. Also, separation programmes have proved successful in the hospital sector and a number of hospitals have pledged to avoid purchasing mercury-containing products through joint industry-NGO-Government programmes. However, separation programmes are sometimes difficult or costly to implement widely, especially when dealing with the general public. In such cases a better long-term solution may be to strongly encourage the substitution of non-mercury products for those containing mercury. As a medium term solution, separation programmes may be pursued, and mercury removed from the combustion stack gases. Mercury emissions from medical and municipal waste incineration can be controlled relatively well by addition of a carbon sorbent to existing PM and SO_2 control equipment, however, control is not 100% effective and mercury-containing wastes are generated from the process;
- Mercury emissions from **utility and non-utility boilers**, especially those burning coal, may be effectively addressed through pre-combustion coal cleaning, reducing the quantities of coal consumed through increased energy efficiency, end-of-pipe measures such as stack gas cleaning and/or switching to non-coal fuel sources, if possible. Another potential approach might be the use of coal with a lower mercury content. Coal cleaning and other pre-treatment options can certainly be used for reducing mercury emissions when they are viable and cost-effective. Also, additional mercury capture may be achieved by the introduction of a sorbent prior to existing SO_2 and PM control technologies. These technologies are under development and demonstration, but are not yet commercially deployed. Also, by-products of these processes are potential sources of future emissions and must be disposed of or reused in an environmentally acceptable manner;
- Mercury emissions due to **trace contamination of raw materials or feedstocks** such as in the cement, mining and metallurgical industries may be reduced by end-of-pipe controls, and sometimes by selecting a raw material or feedstock with lower trace contamination, if possible.
- Mercury emissions during **scrap steel production**, scrap yards, shredders and secondary steel production, result primarily from convenience light and anti-lock brake system (ABS) switches in motor vehicles; therefore a solution may include effective switch removal/collection programmes;
- Mercury releases and health hazards from **artisanal gold mining** activities may be reduced by educating the miners and their families about hazards, by promoting certain techniques that are safer and that use less or no mercury and, where feasible, by putting in place facilities where the miners can take concentrated ores for the final refining process. Some countries have tried banning the use of mercury by artisanal miners, which may serve to encourage their use of central processing facilities, for example, but enforcement of such a ban can be difficult;
- Mercury releases and occupational exposures during **chlor-alkali production** may be substantially reduced through strict mercury accounting procedures, "good housekeeping" measures to keep mercury from being dispersed, properly filtering exhaust air from the facility and careful handling and proper disposal of mercury wastes. There are a number of specific prevention methods to reduce mercury emissions to the atmosphere. The US chlor-alkali industry invented the use of ultraviolet lights to reveal mercury vapour leaks from production equipment, so that they could be plugged.

Equipment is allowed to cool before it is opened, reducing mercury emissions to the atmosphere. A continuous mercury vapour analyser can be employed to detect mercury vapour leaks and to alert workers so that they can take remedial measures. The generally accepted long-term solution is to encourage the orderly phase-out of chlor-alkali production processes that require mercury, and their substitution with technologies that are mercury free;

- Mercury releases and exposures related to mercury-containing **paints, soaps, various switch applications, thermostats, thermometers, manometers, and barometers**, as well as **contact lens solutions, pharmaceuticals and cosmetics** may be reduced by substituting these products with non-mercury products;
- Mercury releases from **dental practices** may be reduced by preparing mercury amalgams more efficiently, by substituting other materials for mercury amalgams, and by installing appropriate traps in the wastewater system;
- Mercury emissions from dental amalgams during **cremation** may only be reduced by removing the amalgams before cremation, which is not a common practice, or by filtering the gaseous emissions when the practice takes place in a crematorium. Since a flue gas cleaner is an expensive control technique for a crematorium, prevention by substituting other materials for mercury amalgams during normal dental care might be a preferred approach;
- In cases of **uncontrolled disposal of mercury-containing products or wastes**, possible reductions in releases from such practises might be obtained by making these practices illegal and adequately enforcing the law, by enhancing access to hazardous waste facilities, and, over the longer term, by reducing the quantities of mercury involved through a range of measures encouraging the substitution of non-mercury products and processes.

CHAPTER 9 - Initiatives for controlling releases and limiting use and exposures

National initiatives

125. The environmental authorities in a number of countries consider mercury to be a high-priority substance with recognised adverse effects. They are aware of the potential problems caused by use and release of mercury and mercury compounds, and therefore have implemented measures to limit or prevent certain uses and releases. Types of measures that have been implemented by various countries include:

- Environmental quality standards, specifying a maximum acceptable mercury concentration for different media such as drinking water, surface waters, air and soil and for foodstuffs such as fish;
- Environmental source actions and regulations that control mercury releases into the environment, including emission limits on air and water point sources and promoting use of best available technologies and waste treatment and disposal restrictions;
- Product control actions and regulations for mercury-containing products, such as batteries, cosmetics, dental amalgams, electrical switches, laboratory chemicals, lighting, paints/pigments, pesticides, pharmaceuticals, thermometers and measuring equipment;
- Other standards, actions and programmes, such as regulations on exposures to mercury in the workplace, requirements for information and reporting on use and releases of mercury in industry, fish consumption advisories and consumer safety measures.

126. Although legislation is the key components of most national initiatives, safe management of mercury also includes efforts to reduce the volume of mercury in use by developing and introducing safer alternatives and cleaner technology, the use of subsidies to support substitution efforts and voluntary agreements with industry or users of mercury. A number of countries have through implementation of this range of measures obtained significant reductions in mercury consumption, and corresponding reductions of uses and releases.

127. The table below gives a general overview of some of the types of implemented measures of importance to management and control of mercury, as related to its production and use life-cycle and an in-

dication of their status of implementation, based on information submitted for this report. More detailed descriptions of most of these measures are provided in chapter 9 and the separate Appendix to this report.

	TYPE AND AIM OF MEASURE	STATE OF IMPLEMENTATION
	Production and use phases of life cycle	
POINT SOURCES	Prevent or limit the intentional use of mercury in processes	General bans implemented in very few countries
	Prevent or limit mercury from industrial processes (such as chlor-alkali and metallurgic industry) from being released directly to the environment	Implemented in many countries, especially OECD countries
	Apply emission control technologies to limit emissions of mercury from combustion of fossil fuels and processing of mineral materials	Implemented in some OECD countries
	Prevent or limit the release of mercury from processes to the wastewater treatment system	Implemented in some OECD countries
	Prevent or limit use of obsolete technology and/or require use of best available technology to reduce or prevent mercury releases	Implemented in some countries, especially OECD countries
PRODUCTS	Prevent or limit products containing mercury from being marketed nationally	General bans implemented in a few countries only. Bans or limits on specific products are more widespread, such as batteries, lighting, clinical thermometers
	Prevent products containing mercury from being exported	Only implemented in a few countries
	Prevent or limit the use of already purchased mercury and mercury-containing products	Only implemented in a few countries
	Limit the allowable content of mercury present as impurities in high-volume materials	Only implemented in a few countries
	Limit the allowed contents of mercury in commercial foodstuffs, particularly fish, and provide guidance (based on same or other limits values) regarding consumption of contaminated fish	Implemented in some countries, especially OECD countries. WHO guidelines used by some countries.
	Disposal phase of life cycle	
	Prevent mercury in products and process waste from being released directly to the environment, by efficient waste collection	Implemented in many countries, especially OECD countries
	Prevent mercury in products and process waste from being mixed with less hazardous waste in the general waste stream, by separate collection and treatment	Implemented in many countries, especially OECD countries
	Prevent or limit mercury releases to the environment from incineration and other treatment of household waste, hazardous waste and medical waste by emission control technologies	Implemented or implementation ongoing in some countries, especially OECD countries.
	Set limit values for allowable mercury contents in sewage sludge spread on agricultural land	Implemented in a number of countries
	Restrict the use of solid incineration residues in road building, construction and other applications	Implemented in some OECD countries
	Prevent the re-marketing of used, recycled mercury	Only implemented in a few countries

Regional and international initiatives

128. It is also apparent that because of mercury's persistence in the environment and the fact that it is transported over long distances by air and water, crossing borders and often accumulating in the food chain far from it's original point of release, a number of countries have concluded that national measures are not sufficient. There are a number of examples where countries have initiated measures at regional, sub-regional and international levels to identify common reduction goals and ensure coordinated implementation among countries in the target area.

129. Three regional, legally binding instruments exist that contain binding commitments for parties with regards to reductions on use and releases of mercury and mercury compounds:

- LRTAP Convention on Long-Range Transboundary Air Pollution and its 1998 Aarhus Protocol on Heavy Metals (for Central and Eastern Europe and Canada and the USA);
- OSPAR Convention for Protection of the Marine Environment of the North-East Atlantic; and
- Helsinki Convention on the Protection of the Marine Environment of the Baltic Sea.

All these three instruments have successfully contributed to substantial reductions in use and releases of mercury within their target regions.

130. The regional and sub-regional cooperation is, however, not limited to legally binding agreements. Six initiatives exist at regional or sub-regional levels that inspire and promote cooperative efforts to reduce uses and releases of mercury within the target area without setting legally binding obligations on the countries/regions participating. The initiatives are: the Arctic Council Action Plan, the Canada-US Great Lakes Binational Toxics Strategy, the New England Governors/Eastern Canada Premiers Mercury Action Plan, the North American Regional Action Plan, the Nordic Environmental Action Programme and the North Sea Conferences. Important aspects of these initiatives are the discussion and agreement on concrete goals to be obtained through the cooperation, the development of strategies and work plans to obtain the set goals and the establishment of a forum to monitor and discuss progress. Although these initiatives are not binding on their participants, there is often a strong political commitment to ensure that the agreements reached within the initiative are implemented at national/regional level.

131. There are also a number of examples of national/regional initiatives being taken by the private sector in the form of voluntary commitments that can be seen as an adjunct to public sector initiatives and as having a good chance of success as they have, by definition, the support of the primary stakeholders. All these voluntary initiatives are valuable supplements to national regulatory measures and facilitate awareness raising, information exchange and the setting of reduction goals that benefit the target region.

132. At the international level, two multilateral environmental agreements (MEAs) exist that are of relevance to mercury and mercury compounds: the Basel Convention on Control of Transboundary Movements of Hazardous Wastes and their Disposal and the Rotterdam Convention on the Prior Informed Consent Procedure for Certain Chemicals and Pesticides in International Trade. These instruments regulate trade in unwanted chemicals/pesticides or hazardous wastes. However, they do not contain specific commitments to reduce uses and releases of mercury directly. The most recently negotiated agreement relevant to chemicals, the Stockholm Convention on POPs, does not cover mercury. In addition, a number of international organizations have ongoing activities addressing the adverse impacts of mercury on humans and the environment.

133. A more detailed compilation of national initiatives, including legislation, in each individual country is contained in an appendix to this report, entitled "Overview of existing and future national actions, including legislation, relevant to mercury". The Appendix is published in a separate document. The information compiled therein has been extracted from the national submissions received from countries under this project.

CHAPTER 10 – Data gaps

National research and information needs

134. A number of countries have in their submissions to UNEP expressed a need for establishing or improving their national "database" (i.e. knowledge of and information on uses and emissions, sources of releases, levels in the environment and prevention and control options) on mercury and mercury compounds. Although the situation varies from country to country, there seems to be a general need for information relevant to the various elements of an environmental management strategy for mercury. Also, countries with a longer tradition of environmental management of mercury have expressed the need to continue to expand their knowledge base on mercury to improve risk assessment and ensure effective risk management. Some of the needs include, among others:

- Inventories of national use, consumption and environmental releases of mercury;
- Monitoring of current levels of mercury in various media (such as air, air deposition, surface water) and biota (such as fish, wildlife and humans) and assessment of the impacts of mercury on humans and ecosystems, including impacts from cumulative exposures to different mercury forms;
- Information on transport, transformation, cycling, and fate of mercury in various compartments;
- Data and evaluation tools for human and ecological risk assessments;
- Knowledge and information on possible prevention and reduction measures relevant to the national situation;
- Public awareness-raising on the potential adverse impacts of mercury and proper handling and waste management practises;
- Appropriate tools and facilities for accessing existing information relevant to mercury and mercury compounds at national, regional and international levels;
- Capacity building and physical infrastructure for safe management of hazardous substances, including mercury and mercury compounds, as well as training of personnel handling such hazardous substances.
- Information on the commerce and trade of mercury and mercury-containing materials.

135. In principle, some parts of this information might be exchanged nationally, regionally or internationally, as its relevance is often universal, however, it might need to be "translated" into the context of the individual country's framework of traditions, economic and industrial activities and political reality. This, in itself, demands a substantial degree of priority, knowledge and funding. Other parts of the information are country specific and would require national efforts to research, collect and process the information.

Data gaps of a general, global character

136. Although mercury is probably among the best-studied environmental toxicants, there are data gaps in the basic understanding of a number of general, global issues relevant to mercury. Based on submitted information and the compilation and evaluation hereof, a possible division of current data gaps of global relevance on mercury could be as follows (not in order of priority):

- Understanding and quantification of the **natural mechanisms affecting the fate of mercury** in the environment, such as mobilisation, transformation, transports and intake. In other words, the pathways of mercury in the environment, and from the environment to humans.
- Understanding and quantification – in a global perspective – of the **human conduct in relation to mercury releases**, and the resulting human contributions to the local, regional and global mercury burden. In other words, the pathways of mercury from humans to the environment.
- Understanding of how and to what degree humans, ecosystems and wildlife are **adversely affected by the current mercury levels** found in the local, regional and global environment. In other words, the possible effects, number affected, and the magnitude and severeness in those affected.

137. A basic understanding has been established for all three categories mentioned above, based on about half a century's extensive research on the impacts and pathways of mercury. However, in a number of areas, further research is needed to provide new information to improve environmental modelling assessments and modern decision-making tools. Despite these gaps in information, a sufficient understanding has been developed of mercury (including knowledge of its fate and transport, health and environmental impacts, and the role of human activity) that international action to address the global adverse impacts of mercury should not be delayed.

CHAPTER 11 – Options for addressing any significant global adverse impacts of mercury

138. The UNEP Governing Council requested, as part of the global assessment on mercury, an outline of options for consideration by the Governing Council, addressing any significant global adverse impacts of mercury, inter alia, by reducing and or eliminating the use, emissions, discharges and losses of mercury and its compounds; improving international cooperation; and ways to enhance risk communication.

139. As part of the implementation of Governing Council decision 21/5, UNEP established a Working Group to assist it in preparing for the Governing Council's discussions on the issue at its session in February 2003. The Global Mercury Assessment Working Group, at its first meeting held from 9 to 13 September 2002, finalized this assessment report for presentation to the Governing Council at its 22nd session. At this meeting, the Working Group arrived at a number of conclusions of relevance to the Governing Council's considerations:

- Based on the key finding of this report, the Working Group concluded that, in its view, there was sufficient evidence of significant global adverse impacts to warrant international action to reduce the risks to human health and/or the environment arising from the release of mercury into the environment. While it was important to have a better understanding of the issue, the Working Group emphasized that it was not necessary to have full consensus or complete evidence in order to take action and therefore potentially significant global adverse impacts should also be addressed.

- The Working Group also agreed on an outline of options for recommendation on measures to address global adverse impacts of mercury at the global, regional, national and local levels. The options include measures such as reducing or eliminating the production, consumption and releases of mercury, substituting other products and processes, launching negotiations for a legally-binding treaty, establishing a non-binding global programme of action, and strengthening cooperation amongst governments on information-sharing, risk communication, assessment and related activities.

- Finally, the Working Group agreed to the need to submit to the Governing Council a range of possible immediate actions in light of their findings on the impacts of mercury, such as increasing protection of sensitive populations (through enhanced outreach to pregnant women and women planning to become pregnant), providing technical and financial support to developing countries and to countries with economies in transition, and supporting increased research, monitoring and data-collection on the health and environmental aspects of mercury and on environmentally friendly alternatives to mercury.

3 Toxicology

3.1 Overview

195. The toxicity of mercury depends on its chemical form, and thus symptoms and signs are rather different in exposure to elemental mercury, inorganic mercury compounds, or organic mercury compounds (notably alkylmercury compounds such as methylmercury and ethylmercury salts, and dimethylmercury). The sources of exposure are also markedly different for the different forms of mercury. For alkylmercury compounds, among which methylmercury is by far the most important, the major source of exposure is diet, especially fish and other seafood. For elemental mercury vapour, the most important source for the general population is dental amalgam, but exposure at work may in some situations exceed this by many times. For inorganic mercury compounds, diet is the most important source for the majority of people. However, for some segments of populations, use of skin-lightening creams and soaps that contain mercury and use of mercury for cultural/ritualistic purposes or in traditional medicine, can also result in substantial exposures to inorganic or elemental mercury.

196. While it is fully recognised that mercury and its compounds are highly toxic substances for which potential impacts should be considered carefully, there is ongoing debate on how toxic these substances, especially methylmercury, are. New findings during the last decade indicate that toxic effects may be taking place at lower concentrations than previously thought, and potentially larger parts of the global population may be affected. As the mechanisms of subtle toxic effects – and proving whether such effects are taking place – are extremely complex issues, a complete understanding has so far not been reached on this very important question.

Methylmercury

197. Of the organic mercury compounds, methylmercury occupies a special position in that large populations are exposed to it, and its toxicity is better characterized than that of other organic mercury compounds. Within the group of organic mercury compounds, alkylmercury compounds (especially ethylmercury and methylmercury) are thought to be rather similar as to toxicity (and also historical use as pesticides), while other organic mercury compounds, such as phenylmercury, resemble more inorganic mercury in their toxicity.

198. Methylmercury is a well-documented neurotoxicant, which may in particular cause adverse effects on the developing brain. Moreover, this compound readily passes both the placental barrier and the blood-brain barrier, therefore, exposures during pregnancy are of highest concern. Also, some studies suggest that even small increases in methylmercury exposures may cause adverse effects on the cardiovascular system, thereby leading to increased mortality. Given the importance of cardiovascular diseases worldwide, these findings, although yet to be confirmed, suggest that methylmercury exposures need close attention and additional follow-up. Moreover, methylmercury compounds are considered possibly carcinogenic to humans (group 2B) according to the International Agency for Research on Cancer (IARC, 1993), based on their overall evaluation.

Elemental mercury and inorganic mercury compounds

199. The main route of exposure for elemental mercury is by inhalation of the vapours. About 80 percent of inhaled vapours are absorbed by the lung tissues. This vapour also easily penetrates the blood-brain barrier and is a well-documented neurotoxicant. Intestinal absorption of elemental mercury is low. Elemental mercury can be oxidized in body tissues to the inorganic divalent form.

200. Neurological and behavioral disorders in humans have been observed following inhalation of elemental mercury vapour. Specific symptoms include tremors, emotional lability, insomnia, memory loss, neuromuscular changes, and headaches. In addition, there are effects on the kidney and thyroid.

High exposures have also resulted in death. With regard to carcinogenicity, the overall evaluation, according to IARC (1993), is that metallic mercury and inorganic mercury compounds are not classifiable as to carcinogenicity to humans (group 3). A critical effect on which risk assessment could be based is therefore the neurotoxic effects, for example the induction of tremor. The effects on the kidneys (the renal tubule) should also be considered; they are the key endpoint in exposure to inorganic mercury compounds. The effect may well be reversible, but as the exposure to the general population tends to be continuous, the effect may still be relevant.

Summary of effect levels

201. This chapter gives a brief presentation of the different adverse effects on human health from elemental (and inorganic) mercury, as well as methylmercury. To put the level of exposures for methylmercury in perspective, for the most widely accepted non-lethal adverse effect (neurodevelopmental effects), the United States (US) National Research Council (NRC, 2000) has estimated the benchmark dose (BMD) to be 58 µg/l total mercury in cord blood (or 10 µg/g total mercury in maternal hair) using data from the Faroe Islands study of human mercury exposures (Grandjean *et al.*, 1997). This BMD level is the lower 95% confidence limit for the exposure level that causes a doubling of a 5% prevalence of abnormal neurological performance (developmental delays in attention, verbal memory and language) in children exposed *in-utero* in the Faroe Islands study. These are the tissue levels estimated to result from an average daily intake of about 1 µg methylmercury per kg body weight per day (1 µg/kg body weight per day).

202. Other adverse effects have been seen in humans with less reliability or at much higher exposures. For methylmercury, effects have been seen on the adult nervous system, on cardiovascular disease, on cancer incidence and on genotoxicity. Also, effects have been reported on heart rate variability and blood pressure in 7 year-old children exposed prenatally, and on cardiovascular mortality in adults. For elemental mercury and inorganic mercury compounds, effects have been seen on: the excretion of low molecular weight proteins; on enzymes associated with thyroid function; on spontaneous abortion rates; genotoxicity; respiratory system; gastrointestinal (digestion) system; liver; immune system; and the skin. Several detailed evaluations of response as a function of exposure that have been conducted are reviewed in Chapter 4. As this report presents the toxicity of mercury in summary only, the reviews, which the presentation was based on, have not been checked in the original references for correct quoting during the preparation of this report.

Dietary considerations

203. Fish are an extremely important component of the human diet in many parts of the world and provide nutrients (such as protein, omega-3 fatty acids and others) that are not easily replaced. Mercury is a major threat to this food supply. Certainly, fish with low methylmercury levels are intrinsically more healthful for consumers than fish with higher levels of methylmercury, if all other factors are equal.

204. There is limited laboratory evidence suggesting that several dietary components might reduce (e.g. selenium, vitamin E, omega-3 fatty acids) or enhance (e.g. alcohol) mercury's toxicity for some endpoints. However, conclusions cannot be drawn from these data at this time.

Explanation of some of the medical terms used in this chapter

Albuminuria: Albuminuria is a form of proteinuria.

Anaemia: Condition in which the number of red blood cells per unit volume of blood is decreased from normal, resulting in decreased oxygen-carrying capacity of the blood.

Ataxia: Wobbliness. Incoordination and unsteadiness due to the brain's failure to regulate the body's posture and regulate the strength and direction of limb movements.

Atrophy of the brain: Shrinkage/loss/waste of the brain.

Cardiovascular effect: Effect on the circulatory system, comprising the heart and blood vessels.

Cerebellar ataxia: Ataxia (see above) due to disease of the cerebellum.

Cerebrovascular: Related to blood vessels of the brain.

Creatinine: A chemical waste molecule that is generated from muscle metabolism and excreted in the urine. The concentration of creatinine in serum is used as a measure for the function of the kidneys. Mercury concentrations measured in urine samples are sometimes presented on the basis of the creatinine contents in the same urine sample (µg mercury/g creatinine) – rather than per volume of urine (µg mercury/l) – in order to eliminate the variation in water contents in urine.

Cystic cavities and spongy foci: Tissue abnormality with holes and spongy areas.

Diastolic and systolic blood pressures: Diastolic blood pressure is the pressure when the heart is extending (dilating) and filled with blood. Systolic blood pressure when the heart is contracting. (A blood pressure of 140/90 means that the systolic blood pressure is 140 and the diastolic blood pressure 90).

Dysarthria: Speech that is characteristically slurred, slow, and difficult to produce (and understand). The person with dysarthria may also have problems controlling the pitch, loudness, rhythm and voice qualities of their speech.

Glomerular proteinuria: Proteinuria (see below) due to dysfunction of the renal glomerulus (unit of the kidney).

Glomerulonephritis: A variety of nephritis (inflammation of the kidney) characterised by inflammation of the capillary loops in the glomeruli of the kidney. (The glomerulus is a functional unit of the kidney).

Interstitial pneumonitis: A form of pneumonia which involves the interstitial tissues (connective tissue) of the lung.

Ischemia: Local anaemia due to obstruction of the blood supply (e.g., narrowing of the arteries).

Ischemic heart disease: Heart disease because of local anaemia.

Micronuclei in peripheral lymphocytes: Small cell nucleus in the peripheral white blood cells.

Neoplastic effect: Has the effect of creating new cells that grow autonomously. A neoplasm is new and abnormal growth of tissue, which can be benign or malign (cancerous).

Nephritic/nephrotic syndrome: A disease of the kidneys that results in inflammation of the glomerulus (the portion of the kidney that filters the blood). A type of nephritis that is characterised by low serum albumin, large amount of protein in the urine and swelling (oedema).

Nephritis: Inflammation of the kidneys.

Nephrosis: Non-inflammatory, non-neoplastic disease of the kidney.

Paresthesia: An abnormal sensation, such as burning, pricking, tingling, or numbness that appears to have no objective cause.

Peripheral neuropathy: Degeneration of peripheral nerves (peripheral nerves are all nerves except the brain and the spinal cord).

Pneumonitis: Inflammation of the lungs secondary to viral or bacterial infection.

Proteinuria: More protein in the urine than normal (normal excretion is 150mg protein daily).

Renal tubule: Small structures in the kidney that filter the blood and produce the urine.

Stomatitis: Infection of the mucous membrane (the inside) of the mouth.

Tachycardia: A rapid heart rate, usually defined as greater than 100 beats per minute.

Tubular proteinuria: More protein in the urine than normal due to dysfunction of the renal tubules.

3.2 Methylmercury

205. While mainly focusing on methylmercury, this section also gives a few remarks on other organic mercury substances.

206. The compound dealt with most extensively in toxicological research in recent years is methylmercury. Like other alkylmercury compounds, the toxicity of methylmercury is much higher than that of inorganic mercury. Methylmercury is a potent neuro-toxin, hence human exposure to methylmercury is clearly unwelcome and should be regarded with concern. It is present worldwide in fish and marine mammals consumed by humans. Methylmercury is formed naturally (from anthropogenic and naturally released mercury) by biological activity in aquatic environments, and it is bio-magnified in the food chain, resulting in much higher concentrations in higher predatory fish and mammals than in water and lower organisms. Most of the total mercury concentrations in fish are in the form of methylmercury (close to 100 percent for older fish). Methylmercury has also been used deliberately as a pesticide/biocide (e.g. seed grain treatment), and this use gave rise to severe historical poisoning incidents in Iraq before 1960 and again in the early 1970's (US EPA, 1997).

207. Consumption of contaminated fish and marine mammals is the most important source of human exposure to methylmercury (WHO/IPCS, 1990; US EPA, 1997). The highest concentrations are found in large predatory fish like shark, king mackeral, swordfish and some large tuna (as opposed to the smaller tuna usually used for canned tuna), as well as in some freshwater fish like pike, walleye, bass, perch, and eels, and in mammals like seals and whales. Due to long-range atmospheric emission transport and ocean currents, methylmercury is also present in the environment far away from local or regional mercury sources. This implies that population groups particularly dependent on – or accustomed to – marine diets, such as the Inuits of the Arctic, as well as marine and freshwater fish-dependent populations anywhere else on the globe, are particularly at risk due to methylmercury exposure.

208. Methylmercury is highly toxic, and the nervous system is its principal target tissue. In adults, the earliest effects are non-specific symptoms such as paresthesia, malaise, and blurred vision; with increasing exposure, signs appear such as concentric constriction of the visual field, deafness, dysarthria, ataxia, and ultimately coma and death (Harada, 1995). The developing central nervous system is more sensitive to methylmercury than the adult. In infants exposed to high levels of methylmercury during pregnancy, the clinical picture may be indistinguishable from cerebral palsy caused by other factors, the main pattern being microcephaly, hyperreflexia, and gross motor and mental impairment, sometimes associated with blindness or deafness (Harada, 1995; Takeuchi and Eto, 1999). In milder cases, the effects may only become apparent later during the development as psychomotor and mental impairment and persistent pathological reflexes (WHO/IPCS, 1990; NRC, 2000). Studies from one population exposed to methylmercury from fish also suggest an association with increased incidence of cardiovascular system diseases (Salonen *et al.*, 1995, Rissanen *et al.*, 2000). From research on animals there is evidence of genotoxicity and effects on the immune system and the reproductive system.

209. Substantial parts of the descriptive text in this section were based on Pirrone *et al.* (2001) and to a lesser extent the submission from the Nordic Council of Ministers (sub84gov). Pirrone *et al.* (2001), mention that their presentation was largely based on previous reviews by WHO (WHO/IPCS 1990; 1991), IARC (IARC, 1993) and the US EPA (US EPA 1997; 2001b).

3.2.1 Neurological effects

210. In the most recent authorative evaluations of the toxicological effects of methylmercury (WHO/IPCS, 1990; NRC, 2000) it was concluded that the effects on the developing nervous system in unborn and newborn children are the most sensitive, well-documented effects judged from the evidence from human and animal studies. Such effects can take place even at exposure levels where the mother (through whom the children receive the mercury) remains healthy or suffers only minor symptoms due to mercury exposure (WHO/IPCS, 1990; Davis *et al.*, 1994, as cited by Pirrone *et al.*, 2001).

211. Methylmercury in our food is rapidly absorbed in the gastrointestinal tract and readily enters the brain. From the methylmercury poisoning episodes in Japan and Iraq it was known that the most severe effects take place in the development of the brain and nervous system of the unborn child (the fetus), but also severe effects on adults were observed. A series of large epidemiological studies have recently provided evidence that methylmercury in pregnant women's marine diets – even at low mercury concentrations (about 1/10 - 1/5 of observed effect levels on adults) – appears to have subtle, persistent effects on the children's mental development as observed at about the start of the school age (so-called cognitive deficits; NRC, 2000).

212. The Faroe Islands population was exposed to methylmercury mainly from pilot whale meat with relatively high concentration of methylmercury, around 2 mg/kg (US EPA, 2001b). The study of about 900 Faroese children showed that prenatal exposure to methylmercury resulted in neuropsychological deficits at 7 years of age (Grandjean *et al.*, 1997). The brain functions most vulnerable seem to be attention, memory, and language, while motor speed, visiospatial function, and executive function showed less robust decrements at increased mercury exposures. The mercury concentration in cord blood appeared to be the best risk indicator for the adverse effects, which were apparently only slightly affected by a large number of covariates examined. Special concern was expressed with respect to the impact of PCBs, which was present in the diet (in whale blubber) of these Faroese mothers. The results were roughly unchanged, however, when PCB levels were taken into account, and increased prenatal exposure to methylmercury appeared to enhance PCB toxicity (Grandjean et al., 2001). Developmental delays were significantly associated the methylmercury exposures, even if excluding the children whose mothers had hair mercury concentrations above 10 µg/g. Within the low exposure range, each doubling of the prenatal methylmercury exposure level was associated with a developmental delay of 1-2 months. On an individual basis the effects at these dose levels may not seem severe, but they may have severe implications on a population basis.

213. To put the level of exposures for methylmercury in perspective, for the most widely accepted non-lethal effect (neurodevelopmental effects), the benchmark dose (BMD) level is calculated to be 58 µg/l total mercury in cord blood (or 10 µg/g total mercury in maternal hair) using data from the Faroe Islands study of human mercury exposures (NRC, 2000; Budtz-Jorgensen *et al.*, 2000). This BMD level is the lower 95 percent confidence limit for the exposure level that causes a doubling of a 5 percent prevalence of abnormal neurological performance (developmental delays in attention, verbal memory and language) in children exposed *in-utero* in the Faroe Islands study. This dose level is estimated from actual test observations and analysis hereof, involving a number of scientifically based choices including statistic model and specific effect/test of effect used for evaluation. The 58 µg/l total mercury in cord blood and 10 µg/g total mercury in maternal hair are the tissue levels estimated to result from an average daily intake of about 1 microgram methylmercury per kilogram body weight per day (1 µg/kg body weight per day). By using an uncertainty factor of 10, this BMD level has been used to estimate safe exposure levels for humans (US EPA, 2001b; NRC, 2000; Pirrone *et al.*, 2001).

214. Another prospective study is ongoing in the Seychelles islands, where the methylmercury exposures are of similar extent. The fish consumption of pregnant women in the Seychelles is high, typically 10-15 meals per week (Shamlaye, 1995), while the mercury concentrations in the ocean fish consumed is lower (than the mercury concentrations in the pilot whale meat consumed by the Faroe Islands population), with a mean of 0.2-0.3 mg/kg (Cernichiari *et al.*, 1995). No effects on developmental tests up to 5.5 years of age were found to be associated with methylmercury exposure, as measured by hair-mercury in the pregnant mothers (Davidson *et al.*, 1998; Crump *et al.*, 2000; Myers *et al.*, 2000; Axtell *et al.*, 2000; Palumbo *et al.*, 2000). The main longitudinal study was started in 1989-1990 and comprised about 700 mother-child pairs. Maternal hair (mean about 7 µg/g) and child hair, but not cord-blood levels were used as markers of methylmercury exposure in this study. A reanalysis using raw scores rather than age standardized scores showed similar results. (Davidson *et al.*, 2001)

215. In addition, there is a study from New Zealand, suggesting an effect on the mental development of children at the age of 4 and 6-7 years. In a high-exposure group the average maternal hair-mercury was about 9 µg/g, and control groups were selected with lower exposure levels. In total, about 200 chil-

dren were examined at 6-7 years of age and a negative association was found between maternal hair-mercury and neuropsychological development of the children. Although carried out a decade earlier than the Seychelles and Faroe Islands studies (published as reports from the Swedish Environmental Protection Agency (Kjellstrom *et al.*, 1986; 1989)), inclusion of the findings from this study was considered appropriate by the US EPA in their recent assessment (US EPA, 2001b) given the similarities in study design and endpoints considered, and following a later analysis of data by Crump using a "benchmark dose" approach (Crump *et al.*, 1998).

216. Some cross-sectional studies using neuropsychological testing of older children in different settings (such as in the Amazonas and on the Madeira island), also found significant associations with mercury exposure (for a review, see US EPA, 2001b). As the relationship between mercury concentrations found in maternal hair, as well as in umbilical cord blood, and mercury concentrations in human diet is relatively well described (with some biological variation), it is possible to estimate corresponding levels of methylmercury doses in human diet, deemed to be safe. See section 4.2.1 on the use of such a risk evaluation tool.

217. The original epidemiological report of methylmercury poisoning involved 628 human cases that occurred in Minamata, Japan, between 1953 and 1960. The overall prevalence rate for the Minamata region for neurologic and mental disorders was 59 percent. Among this group 78 deaths occurred, and hair concentrations of mercury ranged from 50–700 μg/g. The most common clinical signs observed in adults were paresthesia, ataxia, sensory disturbances, tremors, impairment of hearing and difficulty in walking. Examination of the brains of severely affected patients that died revealed marked atrophy of the brain (55 percent normal volume and weight) with cystic cavities and spongy foci. Microscopically, entire regions were devoid of neurons, granular cells in the cerebellum, Golgi cells and Purkinje cells. Extensive investigations of congenital Minamata disease (children of exposed women) were undertaken, and 20 cases that occurred over a 4-year period were documented. In all instances the congenital cases showed a higher incidence of symptoms than did the cases wherein exposure occurred as an adult. Severe disturbances of nervous function were described, and the affected offspring were very late in reaching developmental milestones. Hair concentrations of mercury in affected infants ranged from 10 to 100 μg/g (Harada, 1995; 1997; Tsubaki and Takahashi, 1986; WHO/IPCS, 1990). In addition, later studies of patients with Minamata disease reported increased pain thresholds (an adverse effect) in the body and distal extremities (Yoshida *et al.*, 1992).

Symptoms and health effects of Minamata disease

The symptoms of Minamata disease include:

- sensory disorders in the four extremities (loss of sensation in the hands and feet);
- ataxia (difficulty in coordinating movement of hands and feet);
- narrowing of the field of vision;
- hearing impairment;
- impairment of faculties for maintaining balance;
- speech impediments;
- trembling of hands and feet; and
- disorders of the ocular movement.

In very severe cases, victims fall into a state of madness, lose consciousness and may even die. In relatively mild cases, the condition is barely distinguishable from other ailments such as headache, chronic fatigue and generalized inability to distinguish taste and smell.

When the first outbreaks of Minamata disease occurred, most patients exhibited a full set of severe symptoms. In 16 cases, the patient died within 6 months of the onset of symptoms, and in 1965 the mortality was 44.3 percent. Since then a large number of incomplete or mild cases, displaying an incomplete set of symptoms, have also been identified. (Minamata City, 2000)

Methylmercury poisoning in Minamata Bay, Japan

During the 1960/70's, the Minamata Bay mercury pollution problem received world-wide media attention, opening the world's eyes to the negative health effects of methylmercury and contributing to raising public awareness of the importance of environmental protection.

More than forty years ago, Minamata Bay in Japan was seriously polluted by wastewater containing methylmercury, formed as a by-product in the acetaldehyde synthesizing process of the local acetaldehyde chemical plant; 70-150 metric tons or more of mercury, mixed in the effluents from the factory, were discharged over a number of years into the Bay. The pollution affected the people of Minamata in the form of methylmercury poisoning, referred to as "Minamata disease", causing damage to the central nervous system in people eating large quantities of contaminated fish and shellfish from Minamata Bay. In addition, Congenital Minamata disease occurred, in which victims were born with a condition resembling cerebral palsy, caused by methylmercury poisoning of the fetus via the placenta when the mother consumed contaminated seafood during pregnancy. The disease, which was officially recognized on 1 May 1956, severely affected the local community and was a great burden to the city. Many people lost their lives or suffered from physical deformities and have had to live with the physical and emotional pain of "Minamata Disease" since.

After the cause of the disease was finally confirmed, a number of measures were gradually implemented to deal with the problems arising from the mercury pollution, ranging from regulation of the factory effluent, voluntary restrictions on harvesting of fish and shellfish from the Bay, installation of dividing nets in order to enclose the mouth of the Bay and prevent the spread of contaminated fish, to dredging of mercury-containing sediments in the Bay and appropriate deposit to contain the mercury-contaminated sludge. Finally, in October 1997, the dividing nets that had closed off the bay for 23 years were removed. After several studies confirming that mercury levels in fish were below regulatory levels and had remained so for three years, Minamata Bay was reopened as a general fishing zone and the Minamata Fisheries Co-op recommenced harvesting for the fish market (Minamata City, 2000).

The National Institute for Minamata Disease was formed to investigate the impacts of mercury contamination, and has contributed substantially to the knowledge of mercury toxicology and exposure both nationally and in other regions of the world since then.

The Ministry of Environment of Japan, in its report "Our Intensive Efforts to Overcome the Tragic History of Minamata Disease (JME, 1997)" concludes:
"From the incidence of Minamata Disease, Japan has learned a very important lesson on how activities that place priority on the economy, but lack consideration for the environment can cause grave damage to health and environment, and how it is difficult to recover from this damage later on. From the purely economic standpoint, too, a large amount of cost and a great deal of time are required to deal with such damages, and, when we compare these costs incurred vs. the cost of the measures that could have prevented the pollution, allowing such pollution is certainly not an economically advisable option. In our country, with the experience of suffering from disastrous damage by pollution including the Minamata Disease as a turning point, measures to protect the environment have made dramatic progress. But the sacrifices incurred on the way were truly huge, indeed. We sincerely hope that Japan's experience can be utilized as a vital lesson by other countries, that consideration is paid to the importance of the environment, and that pollution will be prevented without ever undergoing this kind of tragic pollution-related damage."

218. Several neurological signs and symptoms are among the cardinal features of high-dose exposures to methylmercury in adults. As no specific medical test is available to confirm the diagnosis of Minamata disease, cases were identified on the basis of a characteristic combination of symptoms (Harada, 1997; Uchino *et al.*, 1995). These included peripheral neuropathy, dysarthria, tremor, cerebellar ataxia, gait disturbance, visual-field constriction and disturbed ocular movements, hearing loss, disturbance of equilibrium, and subjective symptoms such as headache, muscle and joint pain, forgetfulness, and fatigue. Based on the assessment conducted by WHO/IPCS (1990), paresthesias in five percent of the adult population were judged to occur at hair mercury concentrations above 50 μg/g or blood mercury concentrations above 200 μg/l (WHO/IPCS, 1990). Later research provides some evidence of effects at lower concentrations on adults, see Lebel *et al.* (1998) below.

219. The predominant symptom noted in adults in the 1971 Iraqi poisoning incident was paresthesia, and it usually occurred after a latent period of from 16 to 38 days. In adults symptoms were dose-dependent, and among the more severely affected individuals ataxia, blurred vision, slurred speech and hearing difficulties were observed (Bakir *et al.*, 1973). Signs noted in the infants exposed during fetal development included cerebral palsy, altered muscle tone and deep tendon reflexes, as well as delayed developmental milestones. The mothers experienced paresthesia and other sensory disturbances but at higher doses than those associated with their children exposed *in utero* (during mothers pregnancy; Bakir *et al.*, 1973; WHO/IPCS, 1990; Al-Mufti *et al.*, 1976).

Mercury poisoning incidents in Iraq

Methyl- and ethylmercury poisonings occurred in Iraq following consumption of seed grain that had been treated with fungicides containing these alkylmercury compounds. The first outbreaks were caused by ethylmercury, and occurred in 1956 and 1959-1960, and about 1000 people were adversely affected. The second outbreak was caused by methylmercury and occurred in 1972. The number of people admitted to the hospital from the second outbreak with symptoms of poisoning has been estimated to be approximately 6,500, with 459 fatalities reported. Imported mercury-treated seed grains arrived after the planting season and were subsequently used as grain to make into flour that was baked into bread. Unlike the long-term exposures in Japan, the epidemic of methylmercury poisoning in Iraq was short in duration, but the magnitude of the exposure was high. Because many of the people exposed to methylmercury in this way lived in small villages in very rural areas (and some were nomads), the total number of people exposed to these mercury-contaminated seed grains is not known.

220. Lebel *et al.* (1998) found that abnormal performance on the Branches Alternate Movement Task (BAMT) was significantly associated with all measures of mercury exposure in adults from an Amazonian village, and abnormal visual fields were associated with mean and peak hair mercury concentrations. The authors state that the dose-related decrements in visual and motor functions were associated with hair mercury concentrations below 50 µg/g, a range in which clinical signs of mercury intoxication are not apparent. [1]

3.2.2 Cancer (neoplastic effects)

221. Studies were conducted on causes of death in populations in Minamata, Japan, with high exposures to methylmercury. The only clear indication of an increased cancer risk was in the most informative of these studies, in which excess mortality from cancer of the liver and of the oesophagus was found in the area with the highest exposure, together with an increased risk for chronic liver disease and cirrhosis. Consumption of alcoholic beverages was known to be higher than average in the area (IARC, 1993).

222. A cohort study of individuals in Sweden with a licence for seed disinfection with mercury compounds and other agents found no excess of brain cancer. Of the three Swedish case-control studies on exposure to mercury seed dressings and soft-tissue sarcomas, only one showed an odds ratio above unity. In all three studies the confidence intervals included unity. For malignant lymphomas, there was a slightly but nonsignificantly elevated odds ratio for exposure to mercury seed dressings, but other exposures had higher odds ratios and consequently, potential confounding factors (IARC, 1993).

223. Methylmercury chloride caused renal tumours in several studies in mice exposed through the diet, but not in rats. IARC judged that there is sufficient evidence for carcinogenicity of methylmercury

[1] The USA, in their comments to the first draft of this report (comm-24-gov), comment that in the Amazonian population, concurrent or previous exposure to metallic mercury vapour could not be entirely ruled-out, and there were other problems with nutrition, parasitism, and possible nutritional deficiencies in that population. Therefore, according to the US comments, other factors may have contributed to the neurological deficits reported; and the hair mercury concentration may thus be an inappropriate index for full attribution of the observed neurotoxicity.

chloride in experimental animals. In its overall evaluation for methylmercury compounds, where other relevant data were taken into consideration when making the overall evaluation, it concluded that methylmercury compounds are possibly carcinogenic to humans (group 2B) (IARC, 1993).

3.2.3 Renal effects (kidneys)

224. Renal toxicity has rarely been reported following human exposure to organic forms of mercury. The only evidence of a renal effect following ingestion of mercury-contaminated fish comes from a death-certificate review conducted by Tamashiro *et al.* (1986). They evaluated causes of death among residents of a small area of Minamata City that had the highest prevalence of Minamata disease using age-specific rates for the entire city as a standard. Between 1970 and 1981, the number of deaths attributed to nephritic diseases was higher than expected among women who resided in that region (mortality rate "SMR", 2.77; 95% CI, 1.02 – 6.02), but was within the expected range (mortality rate "SMR", 0.80; 95% CI, 0.17 – 2.36) among men who resided in this region.

3.2.4 Cardiovascular effects (heart and blood system)

225. Jalili and Abbasi (1961) described ECG (heart function) abnormalities in severely poisoned patients hospitalized during the Iraqi grain ethylmercury poisoning epidemic, and similar findings were reported in four family members who consumed ethylmercury-contaminated pork (Cinca *et al.*, 1979). Salonen *et al.* (1995) compared dietary intake of fish and mercury concentrations in hair and urine with the prevalence of acute myocardial infarction (AMI) and death from coronary heart disease or cardiovascular disease in a cohort of 1,833 Finnish men. Dietary mercury intake ranged from 1.1 to 95.3 µg per day (mean 7.6 µg per day). Over a 7-year observation period, men in the highest tertile (at or more than 2 µg/g) of hair mercury content had a two-fold higher risk (1.2 – 3.1) of AMI than men in the two lowest tertiles. A later follow-up (Rissanen *et al.*, 2000) showed a protective effect of omega-3 fatty acids with respect to acute coronary disease, which was, however, less evident in those with hair mercury at or above 2 µg/g. The authors concluded that a high mercury content in fish could reduce the protective effect of these fatty acids. A recent study by Sørensen *et al.* (1999) showed an association between prenatal exposure to methylmercury and cardiovascular function at age 7 in the children from the Faroe Islands, though this study was based on a single measurement per subject of blood pressure, with accompanying high uncertainty. Diastolic and systolic blood pressures increased by 13.9 and 14.6 mmHg, respectively, as cord-blood mercury concentrations rose from 1 to 10 µg/l. In boys, heart-rate variability, a marker of cardiac autonomic control, decreased by 47 percent as cord-blood mercury concentrations increased from 1 to 10 µg/l.

226. These studies suggest that even small increases in methylmercury exposures may cause adverse effects on the cardiovascular system, thereby leading to increased mortality. Given the importance of cardiovascular diseases worldwide, these findings need close attention and additional follow-up.

3.2.5 Genotoxicity

227. Skervfing (1974) found limited support for an association between chromosomal aberations and mercury in red blood cells in subjects consuming large amounts of contaminated freshwater fish. Wulf *et al.* (1986) reported an increased prevalence of sister chromatid exchange in humans who ate mercury-contaminated seal meat. However, information on smoking status and exposure to other heavy metals was not provided for those individuals, making interpretation of the study difficult. No increase in the frequency of sister chromatid exchange or numerical chromosomal alterations was detected in 16 subjects who ate fish caught from a methylmercury contaminated area in Colombia as compared to 14 controls (Monsalve and Chiappe, 1987). More recently, Franchi *et al.* (1994) reported a correlation between the prevalence of micronuclei in peripheral lymphocytes and blood mercury concentrations in a population of fishermen who had eaten mercury-contaminated seafood.

3.3 Elemental and inorganic mercury

228. While many sources of elemental mercury exist, a major exposure route of elemental mercury is dental amalgam. Other exposures to this mercury species are considered in general decline in Europe and most likely also in many other OECD countries. In these regions, methylmercury is considered the remaining exposure of most importance to humans. The national submissions to UNEP for this assessment indicate however that the exposures to elemental and inorganic mercury from local pollution, occupational exposure, certain cultural and ritualistic practices, and some traditional medicines may vary considerably between countries and regions in the world, and that these exposures are significant in some areas.

229. The following presentation of toxic effects of elemental and inorganic mercury is based on a presentation prepared by Pirrone *et al.* (2001), and was edited slightly for this report. Pirrone *et al.* (2001), mention that their presentation was largely based on previous reviews by WHO (WHO/IPCS, 1990; 1991), IARC (IARC, 1993), and US EPA (US EPA, 1997; 2001b). Also, some information was obtained from the recent IPCS report (WHO/IPCS, 2002).

230. Signs and symptoms observed in mercury vapour poisoning differ depending on the level and duration of exposure. Most studies have been performed in occupationally exposed subjects, but there are also some data from accidents in the general population, and on low-level exposure from dental amalgams. The latter subject has been widely discussed and reviewed (US Public Health Service, 1993; Clarkson, 2002; WHO/IPCS, 2002).

3.3.1 Neurological effects

231. As reviewed by the US EPA (1997), the reports from accidental exposures to high concentrations of mercury vapours (Aronow *et al.*, 1990; Fagala and Wigg, 1992; Taueg *et al.*, 1992), as well as studies of populations chronically exposed to potentially high concentrations (Ehrenberg *et al.*, 1991; Roels *et al.*, 1982; Sexton *et al.*, 1978) have shown effects on a wide variety of cognitive, sensory, personality and motor functions. In general, symptoms have been observed to subside after removal from exposure. However, persistent effects (tremor, cognitive deficits) have been observed in occupationally exposed subjects 10-30 years after cessation of exposure (Albers *et al.*, 1998; Kishi *et al.*, 1993; Mathiesen *et al.*, 1999; Letz *et al.*, 2000).

232. Studies of workers exposed to elemental mercury vapour have reported a clear increase in symptoms of disfunction of the central nervous system at exposure levels greater than 0.1 mg/m^3 (Smith *et al.*, 1970) and clear symptoms of mercury poisoning at levels resulting in urinary mercury greater than 300 µg in a 24-hour urine sample (Bidstrup *et al.*, 1951). Several studies, however, have shown evidence of neurotoxicity at approximately 2- to 4-fold lower concentrations. Self-reported memory disturbances, sleep disorders, anger, fatigue, and/or hand tremors were increased in workers chronically exposed to an estimated air concentration of 0.025 mg/m^3 (approximately equal to urinary and blood mercury levels of about 25 µg/g and 10 µg/l) (Langworth *et al.*, 1992), but not in a recent study with somewhat lower exposure levels, urinary mercury 10-15 µg/g (Ellingsen *et al.*, 2001).

233. Objective measures of cognitive and/or motor function in exposed populations have shown significant differences from unexposed controls (Ehrenberg *et al.*, 1991; Liang *et al.*, 1993; Roels *et al.*, 1982). In the study by Langworth *et al.* (1992), there were, however, no objective findings in neuropsychological tests or tremor recordings. This was also mainly the case in the study by Ellingsen *et al.* (2001), although there were possibly some exposure-related effects. Tremor was reported at long-term exposure to relatively low concentrations of mercury vapour (Fawer *et al.*, 1983; Chapman *et al.*, 1990), and mild tremor may constitute an early adverse effect (Biernat *et al.*, 1999; Netterstrøm *et al.*, 1996). Several studies failed, however, to show an increase of tremor at low-level exposure (Roels *et al.*, 1989; Langworth *et al.*, 1992; Ellingsen *et al.*, 2001).

234. In a recent assessment of all studies on the exposure-response relationship between inhaled mercury vapour and adverse health effects, IPCS concluded that several studies consistently demon-

strate subtle effects on the central nervous system in long-term occupational exposures to mercury vapour at exposure levels of approximately 20 $\mu g/m^3$ or higher (WHO/IPCS, 2002).

3.3.2 Renal effects (kidneys)

235. The kidney is, together with the central nervous system, a critical organ for exposure to mercury vapour. Elemental mercury can be oxidized in body tissues to the inorganic divalent form. The kidney accumulates this inorganic mercury to a larger extent than most other tissue with concentrations in occupationally unexposed groups typically of 0.1 – 0.3 µg/g (Drasch *et al.*, 1996; Barregard *et al.*, 1999; Hac *et al.*, 2000; Falnoga *et al.*, 2000). The critical kidney mercury concentration is not known, but levels in subjects with ongoing occupationally exposure may be about 25 µg/g (Kazantzis *et al.*, 1962; Borjesson *et al.*, 1995; Barregard *et al.*, 1999).

236. High exposure may cause (immune-complex mediated) glomerulonephritis with proteinuria and nephritic syndrome. This has been shown at occupational exposures (Kazantzis, 1962; Tubbs *et al.*, 1982), as well as after use of mercury-containing ointment or skin-lightening creams (Becker *et al.*, 1962; Kibukamusoke *et al.*, 1974), but the reported cases are relatively few. Therefore, a specific genetic susceptibility is probably needed for a frank nephritis to develop. For a review, see Enestrom and Hultman (1995).

237. More common at high exposure is proteinuria, glomerular (albumin) as well as tubular (low molecular weight proteins). Albuminuria is, however, generally not seen at exposure levels resulting in urinary mercury below 100 µg/g creatinine (Buchet *et al.*, 1980; Roels *et al.*, 1982; 1989; Langworth *et al.*, 1992; Barregard *et al.*, 1997; Ellingsen *et al.*, 2000).

238. Effects on the renal tubules, as demonstrated by increased excretion of low molecular proteins, have been shown at low-level exposure, and may constitute the earliest biological effect. This effect was previously shown at occupational exposure with urinary mercury of about 35 µg/g creatinine, equivalent to long-term exposure to air levels of 25-30 $\mu g/m^3$ (Barregard *et al.*, 1988; Langworth *et al.*, 1992; Cardenas *et al.*, 1993). In a recent report by Ellingsen *et al.* (2000), such an effect was also shown in workers with urinary mercury of about 10 µg/g creatinine. Ongoing research (Wastensson G, personal communication, 2001, as quoted by Pirrone *et al.*, 2001) appears to support the finding of low-level effects in Swedish chlor-alkali workers at levels in the range of 5 µg/g creatinine, which is only slightly higher than that found in the general population. On the other hand, the possible long-term implications of tubular proteinuria are still unclear (Jarup *et al.*, 1998). For example, Ellingsen *et al.* (1993a) have suggested that some renal effects may be reversible after a long enough period of time, and Frumkin *et al.* (2001) have concluded from their research that "no strong associations were demonstrated with neurological or renal function or with porphyrin excretion."

239. Among male European mercury miners an increased mortality was observed from nephritis and nephrosis (mortality rate "SMR" 1.55, 95 % CI 1.13-2.06) (Boffetta *et al.*, 2001), whereas this was not shown among chlor-alkali workers (Barregard *et al.*, 1990; Ellingsen *et al.*, 1993).

240. The IPCS recently concluded (WHO/IPCS, 2002), based on existing studies, that adverse effects on the kidney usually occur at exposures higher than those inducing neurophysiological effects. Also, although a large number of serious and even fatal intoxications (often suicides or suicide attempts) have been described after ingestion of inorganic mercury compounds, data from humans does not allow identification of lowest harmful or non-adverse exposure levels, especially in long-term exposure. From studies on experimental animals, a No-Adverse-Effect Level (NOAEL) of 0.23 mg/kg per day was identified (US ATSDR, 1999; WHO/IPCS, 2002).

3.3.3 Cancer (neoplastic effects)

241. Data on the carcinogenicity of metallic mercury and its inorganic compounds mainly come from studies on cancer occurrence in occupational populations, including dentists, nuclear weapon

manufacturers, chlor-alkali workers and miners. Previous data are summarized in reviews (IARC, 1993; Boffetta *et al.*, 1993).

242. In 1993, IARC evaluated metallic mercury and inorganic mercury compounds and found that there was inadequate evidence in experimental animals for carcinogenicity of metallic mercury and limited evidence in experimental animals for carcinogenicity of mercuric chloride. In its overall evaluation, it concluded that metallic mercury and inorganic mercury compounds are not classifiable (group 3) with respect to carcinogenicity in humans (IARC, 1993).

243. Citing a number of studies of occupational mercury exposure, including studies done after the IARC evaluation in 1993, Pirrone *et al.* (2001) concludes that lung cancer is the only cancer form, which seems to be consistently increased among various groups of workers exposed to metallic and inorganic mercury. The main difficulty in the interpretation of the data on lung cancer is the possible co-exposure to other lung carcinogens, in particular arsenic (in the fur industry), radon and silica (among miners). An additional limitation is the almost universal lack of data on tobacco smoking. The fact that no increase was found in a large group of European mercury miners not exposed to quartz (Boffetta *et al.*, 1998) argues against the hypothesis that mercury vapour may cause lung cancer. There is no suggestion of a consistent increase of any other neoplasm, including brain and kidney cancers, in these populations.

3.3.4 Respiratory effects

244. Respiratory toxicity in humans following exposure to elemental mercury vapours has been characterized by pulmonary edema and congestion, coughing, interstitial pneumonitis and respiratory failure (Bluhm *et al.*, 1992; Taueg *et al.*, 1992; WHO/IPCS, 1991). Barregard *et al.* (1990) and Ellingsen *et al.* (1993) found no associations between mortality from respiratory disease and mercury exposure among workers exposed to mercury in the chlor-alkali industry, although the power of the studies were low. Merler *et al.* (1994) found no excess mortality of respiratory disease in men (mortality rate "SMR", 0.67; 95% CI, 0.35 – 1.14) exposed to mercury in the fur hat industry. This was also true for mercury miners, except for pneumoconiosis (Boffetta *et al.*, 2001).

3.3.5 Cardiovascular effects (heart and blood system)

245. Signs of cardiovascular toxicity in humans after acute exposure to elemental mercury include tachycardia, elevated blood pressure and heart palpitations (Bluhm *et al.*, 1992; Snodgrass *et al.*, 1981; Soni *et al.*, 1992, Wossmann *et al.*, 1999). Intermediate-duration exposure to elemental mercury vapours produced similar effects (i.e., tachycardia and elevated blood pressure) (Fagala and Wigg, 1992; Foulds *et al.*, 1987). Piikivi (1989) demonstrated a positive correlation between heart palpitations and urinary mercury concentrations in workers from a chlor-alkali plant but also "found only a tendency for a subtle reduction of cardiovascular reflex responses and a slight increase of subjective symptoms, but no significant autonomic dysfunction associated with the low levels of exposure." Nevertheless, it is unclear from the available scientific literature whether the effects on cardiovascular function are due to direct cardiac toxicity or to indirect toxicity (e.g., due to effects on neural control of cardiac function) of elemental mercury. Barregard *et al.* (1990) showed that Swedish chlor-alkali workers had increased mortality due to ischemic heart disease and cerebrovascular disease. However, there were no such findings in Norwegian chlor-alkali workers (Ellingsen *et al.*, 1993a). Nonetheless, the IPCS (2003) and US ATSDR (1999) have recently reported that acute inhalation exposure to high concentrations of elemental mercury vapour from the heating of elemental/inorganic mercury resulted in increased blood pressure and palpitations. Exposures of longer durations due to spills or occupational exposures have also been reported to result in increased blood pressure and increased heart rate (WHO/IPCS, 2002; US ATSDR, 1999).

246. Among European mercury miners, increased mortality from hypertension (SR 1.46, 95 % CI 1.08-1.93) and from heart diseases (other than ischemic disease) have been reported (mortality rate "SMR", 1.36, 95 % CI 1.20-1.53), and these effects increased with time since first employment and

with estimated cumulative mercury exposure. But, findings were not consistent among countries. Also, no increase was shown for ischemic heart disease or cerebrovascular diseases (Boffetta *et al.*, 2001).

247. Statistically significant increases of approximately 5 mmHg in both systolic and diastolic blood pressure were found in 50 volunteers with dental amalgam when compared to an age- and sex-matched control group (average age approximately 22 years) without mercury amalgam fillings. Potential confounding differences between the two groups, such as life-style and body mass, were not discussed. Significantly decreased hemoglobin and hematocrit, and increased mean corpuscular hemoglobin concentration were also found compared to controls without dental amalgams (Siblerud, 1990, as cited in WHO/IPCS, 2002).

3.3.6 Gastrointestinal (digestive system) and hepatic (liver) effects

248. The most common sign of frank mercury poisoning is stomatitis, which is usually reported following acute, high concentration exposure to elemental mercury vapours (Bluhm *et al.*, 1992; Snodgrass *et al.*, 1981). Other commonly reported gastrointestinal effects include nausea, vomiting, diarrhea and abdominal cramps (Bluhm *et al.*, 1992; Lilis *et al.*, 1985; Sexton *et al.*, 1978; Snodgrass *et al.*, 1981; Vroom and Greer, 1972). However, no increased mortality from the digestive system was observed in European mercury miners (Boffetta *et al.*, 2001).

3.3.7 Effects on the thyroid gland

249. The thyroid may accumulate mercury with continued exposure to elemental mercury (Kosta *et al.*, 1975; WHO/IPCS, 1991; Falnoga *et al.*, 2000). It has been shown that moderate occupational exposure affects a particular enzyme system in the thyroid at urinary mercury levels of 15-30 µg/g creatinine – the same levels as those associated with reports of minor effects on the central nervous system and the kidneys (Barregard *et al.*, 1994; Ellingsen *et al.*, 2000). A recent study (Ellingsen *et al.*, 2000) compared thyroid function in 47 chlor-alkali workers exposed to mercury vapours for an average of 13.3 years with 47 "referents." The median serum concentration of reverse triiodothyronine (T3) was statistically significantly higher in the exposed group compared to the referents. Also, the free thyroxine (T4)/free T3 ratio was higher in the highest exposed subgroups compared with referents. The enzyme deiodinase responsible for the deiodination of thyroxine (T4) to triiodothyronine (T3), a seleno-enzyme, seems to be affected. However, Ellingsen *et al.* (2000) also reported that the "overall function of the thyroid gland as assessed by measuring TSH and the thyroid hormones appears to be maintained in the workers exposed to low levels of elemental mercury."

3.3.8 Effects on the immune system

250. The ability of mercury to induce immune-mediated disease has been thoroughly investigated in mice and rats experimentally exposed to inorganic mercury compounds, in most studies divalent mercury, but also mercury vapour. The type of response depends on the strains, some of them being susceptible to autoimmune disease and some being resistant. It is therefore assumed that the genotype is probably important also for the potential immunological effects in humans. For a review, see Enestrom and Hultman (1995) and Sweet and Zelikoff (2000). Some studies in humans occupationally exposed to moderate levels of elemental mercury reported changes in biochemistry of the immune response system (see Pirrone *et al.*, 2001).

3.3.9 Effects on the skin (dermal)

251. Exposure to elemental mercury vapours for acute or intermediate duration may result in a response known as acrodynia or "pink disease", which is characterized by peeling palms of hands and soles of feet, excessive perspiration, itching, rash, joint pain and weakness, elevated blood pressure and tachycardia (Fagala and Wigg, 1992; Karpathios *et al.*, 1991; Schwartz *et al.*, 1992). Also, rash and stomatitis have been reported after high inhalation exposures (Bluhm *et al.*, 1992; Barregard *et al.*, 1996).

3.3.10 Reproductive and developmental effects

252. A study of the pregnancies of Polish dental professionals showed a high frequency of malformations of a nonspecified nature (Sikorski *et al.*, 1987). In contrast, a study of Swedish dental professionals found no increases in malformations, abortions, or stillbirths (Ericsson and Källén, 1989). An increase in low birth weight infants was noted in the offspring of female dental nurses (Ericsson and Källén, 1989); however, in this same study similar effects were not observed for either dentists or dental technicians, and socioeconomic factors may have contributed to the effects observed.

253. Studies of occupational exposure indicate that exposure to elemental mercury may affect human reproduction. Possible effects are increased spontaneous abortions, congenital anomalies, and reduced fertility among women.

254. In occupational exposure studies, paternal exposure to metallic mercury does not appear to cause infertility or malformations (Alcser *et al.*, 1989; Lauwerys *et al.*, 1985). However, a study of pregnancy outcomes among the wives of 152 mercury-exposed men revealed an increased incidence of spontaneous abortions (Cordier *et al.*, 1991). Preconception paternal urinary mercury concentrations above 50 µg/l were associated with a doubling of the spontaneous abortion risk. Elghancy *et al.* (1997) compared the pregnancy outcomes of 46 mercury-exposed workers to those of 19 women who worked in nonproduction areas of the same factory. Women exposed to inorganic mercury had a higher rate of births with congenital anomalies. Concentrations were up 0.6 mg/m^3.

255. However, no significant differences in stillbirths or miscarriage rates were noted between the two groups of women. Also, no increase in spontaneous abortions was observed among dental assistants (potentially exposed to mercury vapour) in a historical prospective study of pregnancy outcomes among women in 12 occupations (Heidam, 1984). Similarly, no relationship between the amalgam fillings prepared per week and rate of spontaneous abortions or congenital abnormalities was observed in a postal survey in California (Brodsky *et al.*, 1985). No excess in the rate of still births or congenital malformations was observed among 8,157 infants born to dentists, dental assistants, or technicians, nor were the rates of spontaneous abortions different from the expected values (Ericsson and Källén, 1989). Rowland *et al.* (1994), however, found that the probability of conception among female dental hygienists who prepared more than 30 amalgams per week and had at least five poor hygiene practices when handling mercury was only 63 percent of that among unexposed controls. Women with lower exposures, however, were more fertile than unexposed controls. A large study conducted in Norway compared reproductive success rates among 558 female dental surgeons with those of 450 high-school teachers (Dahl *et al.*, 1999). They concluded that exposure to mercury, benzene, and chloroform was not associated with decreased fertility except for a possible mercury effect on the last pregnancy of multiparous dental surgeons.

3.3.11 Genotoxicity

256. Two occupational studies (Anwar and Gabal, 1991; Popescu *et al.*, 1979) reported on workers inhaling inorganic mercury; the data were inconclusive regarding the clastogenic activity of inorganic mercury. Workers involved in the manufacture of mercury fulminate ($Hg[OCN]_2$) had a significant increase in the incidence of chromosomal aberrations and micronuclei in peripheral lymphocytes when compared to unexposed controls (Anwar and Gabal, 1991). There was no correlation between urinary mercury levels or duration of exposure to the increased frequency of effects; the study authors concluded that mercury may not have been the clastogen in the manufacturing process. In a study by Popescu *et al.* (1979), 18 workers exposed to a mixture of mercuric chloride, methylmercuric chloride and ethylmercuric chloride had significant increases in the frequency of acentric fragments. Barregard *et al.* (1991) demonstrated a correlation between cumulative mercury exposure and induction of micronuclei among a group of chlor-alkali workers, suggesting a possible genotoxic effect. Other studies did not observe genotoxic effects among workers exposed to mercury vapour (Vershaeve *et al.*, 1976, 1979; Mabille *et al.*, 1984).

3.4 Interactions – possible confounding effects of certain nutrients

257. The evidence is inconclusive and uncertain on the possible effects of various nutrients in relation to mercury toxicity. Nonetheless, limited evidence suggests that diet and nutrition may potentially reduce or enhance the toxicity of mercury, depending on dietary patterns and specific substances in the diet. Thus, nutritional status and dietary interactions might potentially affect the outcome of mercury studies, either by influencing the toxicity of mercury or by having effects on the endpoints measures. Some limited evidence suggests that protective effects of some nutrients (such as selenium, vitamin E, omega-3 fatty acids) might possibly reduce potentially harmful effects of mercury. Other components of the diet (such as ethanol) might possibly enhance toxicity of mercury. Also, mal-nourishment might possibly affect study results either by directly reducing the sensitivity of an endpoint tested or by exacerbating the effects of mercury and thereby increasing the sensitivity to mercury toxicity. Other nutritional factors such as iron or folate deficiencies that disrupt neuronal development might also possibly influence the impact of mercury.

258. Moreover, in studies of mercury toxicity to humans, other pollutants in the diet (such as PCBs) may prevent obtaining clear information on mercury toxicity. This is particularly the case when investigating more subtle toxic effects at low exposure levels, and much effort has been given to eliminating the misinterpretation of results due to such so-called "confounders." More information on possible interactions of nutrients and other components of food can be found, among others, in the following references: Block, 1985; Bulat *et al.*, 1998; Chalon *et al.*, 1998; Chapman and Chan, 2000; Drasch *et al.*, 1996; Falnoga *et al.*, 2000; Goyer, 1997; Kling *et al.*, 1987; McNeil *et al.*, 1988; NRC, 2000; Petridou *et al.*, 1998; Rowland *et al.*, 1986; Rumbeiha *et al.*, 1992; Turner *et al.*, 1981 and WHO/IPCS, 1990.

4 Current mercury exposures and risk evaluations for humans

4.1 Overview

259. As mentioned earlier, the general population is primarily exposed to methylmercury through the diet (especially fish) and to elemental mercury vapours due to dental amalgams. Depending on local mercury pollution load, substantial additional contributions to the intake of total mercury can occur through air and water. Also, personal use of skin-lightening creams and soaps, mercury use for religious, cultural and ritualistic purposes, the presence of mercury in some traditional medicines (such as certain Traditional Asian remedies) and mercury in the home or working environment can result in substantial elevations of human mercury exposure. For example, elevated air levels in homes have resulted from mercury spills from some old gas meters and other types of spills. Also, elevated mercury levels in the working environment have been reported for example in chlor-alkali plants, mercury mines, thermometer factories, refineries and dental clinics (WHO/IPCS, 1991), as well as in mining and manufacturing of gold extracted with mercury. Additional exposures result from the use of Thimerosal or Thiomersal (ethylmercury thiosalicylate) as a preservative in some vaccines and other pharmaceuticals. The national submissions to UNEP for this assessment indicate that the relative impacts of mercury from local pollution, occupational exposure, certain cultural and ritualistic practices, and some traditional medicines may today vary considerably between countries and regions in the world, and are significant in some regions.

260. Examples of data on total mercury and methylmercury exposures primarily from fish diets, but also other sources in different parts of the world, including Sweden, Finland, the USA, the Arctic, Japan, China, Indonesia, Papua New Guinea, Thailand, Republic of Korea, Philippines, the Amazonas and French Guyana are provided in section 4.4. For example, in a study of a representative group of about 1700 women in the USA (aged 16-49 years) for years 1999-2000, about 8 percent of the women had mercury concentrations in blood and hair exceeding the levels corresponding to the US EPA's reference dose (an estimate of a safe dose, see section 4.2.1). As shown in the chapter, data indicate exposures are generally higher in Greenland, Japan and some other areas compared to the USA. Other examples of human exposures exist and have been submitted for use in this report. Unfortunately, it has not been possible to present all submitted examples here.

261. In some of these countries and areas, local and regional mercury depositions have affected the mercury contamination levels over the years and countermeasures have been taken during the last decades to reduce national emissions. Mercury emissions are, however, distributed over long distances in the atmosphere and oceans. This means that even countries with minimal mercury emissions, and other areas situated remotely from dense human activity, may be adversely affected. For example, high mercury exposures have been observed in the Arctic, far distances from any significant sources of releases.

262. Data on mercury concentrations in fish have been submitted from a number of nations and international organisations. Additionally, many investigations of mercury levels in fish are reported in the literature. Submitted data, giving examples of mercury concentrations in fish from various locations in the world, are summarised for illustrative purposes in table 4.5. The mercury concentrations in various fish species are generally from about 0.05 to 1.4 mg/kg depending on factors such as pH and redox potential of the water, and species, age and size of the fish. Since mercury biomagnifies in the aquatic food web, fish higher on the food chain (or of higher trophic level) tend to have higher levels of mercury. Hence, large predatory fish, such as king mackeral, pike, shark, swordfish, walleye, barracuda, large tuna (as opposed to the small tuna usually used for canned tuna), scabbard and marlin, as well as seals and toothed whales, contain the highest concentrations. The available data indicate that mercury is present all over the globe (especially in fish) in concentrations that adversely affect human beings and wildlife. These levels have led to consumption advisories in a number of countries (for fish, and some-

times marine mammals), warning people, especially sensitive subgroups (such as pregnant women and young children), to limit or avoid consumption of certain types of fish from various waterbodies. Moderate consumption of fish (with low mercury levels) is not likely to result in exposures of concern. However, people who consume higher amounts of contaminated fish or marine mammals may be highly exposed to mercury and are therefore at risk.

4.2 Evaluations of exposure levels causing risks

4.2.1 Methylmercury

263. As mentioned, intake of methylmercury in fish and other aquatic foods is considered the most serious general impact on humans. Based on risk assessments and other societal considerations, several countries and international organisations have established risk evaluation tools such as levels of daily or weekly methylmercury or mercury intakes considered safe (Reference Dose and Provisional Tolerable Weekly Intake), limits/guidelines for maximum concentrations in fish and fish consumption advisories.

264. Table 4.1 gives an overview of examples of maximum allowed or recommended levels of mercury in fish in various countries (based on submissions to UNEP, unless otherwise noted). Also, examples of tolerable intake levels of mercury or methylmercury are mentioned.

Table 4.1 Examples of maximum allowed or recommended levels of mercury (Hg) in fish in various countries and by WHO/FAO (based on submissions to UNEP, unless otherwise noted).

Country/ Organization	Fish type	Maximum allowed/recommend levels in fish *1	Type of measure	Tolerable intake levels *1
Australia	Fish known to contain high levels of mercury, such as swordfish, southern bluefin tuna, barramundi, ling, orange roughy, rays, shark All other species of fish and crustaceans and molluscs	1.0 mg Hg/kg 0.5 mg Hg/kg	The Australian Food Standards Code	Tolerable Weekly Intake: 2.8 µg Hg/kg body weight per week for pregnant women.
Canada	All fish except shark, swordfish or fresh or frozen tuna (expressed as total mercury in the edible portion of fish) Maximum allowable limit for those who consume large amounts of fish, such as Aboriginal people	0.5 ppm total Hg 0.2 ppm total Hg	Guidelines/ Tolerances of Various Chemical Contaminants in Canada	Provisional Tolerable Daily Intake: 0.47 µg Hg/kg body weight per day for most of the population and 0.2 µg Hg/kg body weight per day for women of childbearing age and young children
China	Freshwater fish	0.30 mg/kg	Sanitation standards for food	
Croatia	*Fresh fish* Predatory fish (tuna, swordfish, molluscs, crustaceans) All other species of fish *Canned fish (tin package)* Predatory fish (tuna, swordfish, molluscs, crustaceans) All other species of fish	1.0 mg Hg/kg 0.8 mg methylHg/kg 0.5 mg Hg/kg 0.4 mg methylHg/kg 1.5 mg Hg/kg 1.0 mg methylHg/kg 0.8 mg Hg/kg 0.5 mg methylHg/kg	Rules on quantities of pesticides, toxins, mycotoxins, metals and histamines and similar substances that can be found in the food	
European Community *2	Fishery products, with the exception of those listed below. Anglerfish, atlantic catfish, bass, blue ling, bonito, eel, halibut, little tuna, marlin, pike, plain bonito, portuguese dogfish, rays, redfish, sail fish, scabbard fish, shark (all species), snake mackerel, sturgeon, swordfish and tuna.	0.5 mg Hg/kg wet weight 1 mg Hg/kg wet weight	Various Commission decisions, regulations and Directives	

Country/ Organization	Fish type	Maximum allowed/recommend levels in fish *1	Type of measure	Tolerable intake levels *1
Georgia	Fish (freshwater) and fishery products Fish (Black Sea) Caviar	0.3 mg Hg/kg 0.5 mg Hg/kg 0.2 mg Hg/kg	Georgian Food Quality Standards 2001	
India	Fish	0.5 ppm total Hg	Tolerance Guidelines	
Japan	Fish	0.4 ppm total Hg/kg 0.3 ppm methylHg (as a reference)	Food Sanitation Law - Provisional regulatory standard for fish and shellfish	Provisional Tolerable Weekly Intake: 0.17 mg methylHg (0.4 µg/kg body weight per day) (Nakagawa *et al.*, 1997).
Korea, Republic of	Fish	0.5 mg Hg/kg	Food Act 2000	
Mauritius	Fish	1 ppm Hg	Food Act 2000	
Philippines	Fish (except for predatory) Predatory fish (shark, tuna, swordfish)	0.5 mg methylHg /kg 1 mg methylHg/kg	Codex Alimentarius	
Slovak Republic	Freshwater non-predatory fish and products thereof Freshwater predatory fish Marine non-predatory fish and products thereof Marine predatory fish	0.1 mg total Hg/kg 0.5 mg total Hg/kg 0.5 mg total Hg/kg 1.0 mg total Hg/kg	Slovak Food Code	
Thailand	Seafood Other food	0.5 µg Hg/g 0.02 µg Hg/g	Food Containing Contaminant Standard	
United Kingdom	Fish	0.3 mg Hg/kg (wet flesh)	European Statutory Standard	
United States	Fish, shellfish and other aquatic animals (FDA) States, tribes and territories are responsible for issuing fish consumption advise for locally-caught fish; Trigger level for many state health departments:	1 ppm methylHg 0.5 ppm methylHg	FDA action level Local trigger level	US EPA reference dose: 0.1 µg methylHg/kg body weight per day
WHO/FAO	All fish except predatory fish Predatory fish (such as shark, swordfish, tuna, pike and others)	0.5 mg methylHg/kg 1 mg methylHg/kg	FAO/WHO Codex Alimentarius guideline level	JECFA provisional tolerable weekly intake: 3.3 µg methylHg/kg body weight per week.

Note: 1 Units as used in references. "mg/kg" equals "µg/g" and ppm (parts per million). It is assumed here that fish limit values not mentioned as "wet weight" or "wet flesh" are most likely also based on wet weight, as this is normally the case for analysis on fish for consumers.

2 The European Commission has recently (February 2002) revised the previous maximum limit values for mercury in a small number of specific fish species for consumption (Commission Regulation No 221/2002 of 6 February 2002). These changes are not reflected in the table.

Recent risk evaluation process in USA

265. Three comprehensive risk evaluations on methylmercury were recently completed in the USA by the Environmental Protection Agency (EPA), the Agency for Toxics Substances and Disease Registry (ATSDR) and the National Research Council (NRC). All three are summarized here with greater detail given for the EPA evaluation, as it is a very recent comprehensive evaluation and presents one example of a scientific approach to estimate a safe exposure level.

266. The earlier-mentioned NRC evaluation was initiated by the EPA upon the request of the US Congress, and it is has been part of a major effort by the EPA to review the available toxicological findings on methylmercury as a basis for a re-evaluation of the EPA reference dose (RfD). The RfD is generally defined as an "estimate (with uncertainty spanning perhaps an order of magnitude) of a daily exposure to the human population (including sensitive subgroups) that is likely to be without an appreciable risk of deleterious effects during a lifetime." The methylmercury RfD is used by the EPA to evaluate the potential for adverse health effects from exposure to methylmercury for humans as well as establishing guidance for fish consumption advisories (NRC, 2000; NIEHS, 1998; US EPA, 1997).

267. The RfD is a daily intake of methylmercury for which "exposures" (intake) at or below the RfD are expected to be safe. The risks following exposures above the RfD are uncertain, but risk increases as exposure to methylmercury increases above the RfD (US EPA, 1997). In 1995, an RfD was set by the EPA on the basis of neurological effects observed on children exposed prenatally (in the mothers womb) to methylmercury in the poisoning incidence in Iraq (epidemiological data transformed by calculations from observed mercury concentrations in maternal hair to daily intakes – divided by a safety factor of 10 due to biological variability and insufficient data on reproductive effects on adults). The NRC evaluation committee concluded in 2000 that the value of the US EPA's RfD for methylmercury, 0.1 micrograms of methylmercury per kilogram body weight per day, "is a scientifically justifiable level for the protection of public health". However, the committee recommended that the above-mentioned results from the Faroe Islands study should be used for the US EPA's determination of a new RfD instead of the Iraq study (NRC, 2000). The NRC recommended an uncertainty factor (UF) of not less than 10 to account for variability in human kinetics (i.e., pharmacokinetics) and sensitivity of the fetus' brain to methylmercury. The NRC review and the studies were again reviewed by an external expert panel, and then the US EPA evaluation was presented in 2001 (US EPA, 2001b), as part of a water quality criterion.

268. The US EPA evaluation includes a thorough analysis of the relevant studies, especially those conducted on children from the Faroe Islands and the Seychelles islands. Since the results from these two studies disagree, the merits and weaknesses of the studies were discussed, as well as possible reasons for the conflicting results. Both studies were considered being of high quality, and no serious flaws could be detected. In this situation, the US EPA decided to use data from the Faroe Islands study (which showed a negative effect on neurological development related to methylmercury exposures) as the starting point to derive the RfD. Similar results from the smaller New Zealand study as well as some later cross-sectional studies from other parts of the world, contributed to this conclusion.

269. The current RfD was derived from a benchmark dose (BMD) divided by an uncertainty factor of 10. The BMD analysis used was based on the lower 95 percent confidence limit for a 5 percent effect level (above background) applying a linear model to dose-response data based on cord blood mercury. The cord blood data were converted to maternal intakes. Several of the neuropsychological tests used, and also an integrated analysis gave similar results with respect to benchmark doses. Most of these endpoints yielded RfDs of about 0.1 µg/kg body weight per day (comm-24-gov). Overall, the EPA RfD was primarily based on a number of neurological endpoints and the weight of evidence from the Faroe Islands and the New Zealand study, plus an integrated analysis of those two studies plus the Seychelles study. Other models for the benchmark analyses are possible (Budtz-Jørgensen *et al.*, 2000) and resulted in lower benchmark dose limits, but the linear model was considered the most appropriate one (Pirrone *et al.*, 2001). The US EPA chose an uncertainty factor of 10 accounting for pharmacokinetic inter-individual variability, gaps of knowledge on possible long term effects, and uncertainty concerning the relationships between cord and maternal blood mercury concentration, and as mentioned, the US EPA's current RfD was set at 0.1 µg/kg body weight per day (US EPA, 2001b, and Pirrone *et al.*, 2001). A daily average methylmercury intake of 0.1 µg/kg body weight per day by an adult woman is estimated to result in hair mercury concentrations of about 1 µg/g, cord blood levels of about 5 to 6 µg/l and blood mercury concentrations of about 4-5 µg/l. However, there are limitations, uncertainties and variability in these estimates. These estimates were derived from data and methods presented in US ATSDR, 1999; NRC, 2000; US EPA, 2001b and US EPA, 1997.

270. Based on an average daily intake of 17.5 gram of fish, the US EPA also calculated a Tissue Residue Criterion of 0.3 mg methylmercury per kg of fish (0.3 mg/kg). This limit is weighted on all fish and shellfish consumed. For higher intakes, a lower limit would be needed. Additionally, US EPA calculated a set of recommendations for fish consumption limits based on the above mentioned risk assessment, see table 4.2 (US EPA, 2001b).

271. Consumption limits have been calculated as the number of allowable fish meals per month based on the ranges of methylmercury in the consumed fish tissue. For example, when methylmercury

levels in fish tissue are 0.4 mg/kg, then two 0.23 kg meals per month can safely be consumed. The following assumptions were used to calculate the consumption limits:

- Consumer adult body weight of 72 kg (less meals recommended if lower body weight);
- Average fish meal size of 0.23 kg;
- Time-averaging period of 1 month (30.44 d);
- EPA's reference dose for methylmercury (0.1 µg/kg body weight per day) from EPA's Water Quality Criterion for the Protection of Human Health: Methylmercury (US EPA, 2001b).

Table 4.2 US EPA's monthly fish consumption limits for methylmercury (US EPA, 2001b).

Max. number of fish meals/month	Fish tissue concentrations (ppm = mg/kg, wet weight)
16	> 0.03–0.06
12	> 0.06–0.08
8	> 0.08–0.12
4	> 0.12–0.24
3	> 0.24–0.32
2	> 0.32–0.48
1	> 0.48–0.97
0.5	> 0.97–1.9
None (<0.5)*	> 1.9

* None = No consumption recommended.

> means "above" (example "> 0.06–0.08" means: "above 0.06 to 0.08")

272. Using an alternative approach, the US ATSDR developed its current Minimal Risk Level (MRL) of 0.3 µg/kg body weight per day for methylmercury using the Seychelles Child Development Data (US ATSDR, 1999). The MRL is an estimate of the level of human exposure to a chemical that does not entail appreciable risk of adverse non-cancer health effects. They are intended for use by the public health officials as screening tools to determine when further evaluation of potential human exposure at hazardous waste sites is warranted.

Europe

273. Guidelines for maximum mercury concentrations in fish and consumption advice vary somewhat among the European countries. In 2001, a group of European scientists evaluated the risks from mercury exposure in Europe and presented their view in this regard in their "Position Paper on Mercury" (Pirrone *et al.*, 2001). Regarding methylmercury, they recommended that the US EPA reference dose should apply in Europe also, stating that:

> "We share the view of the recent evaluations by the US EPA and NRC. No new information has emerged that would change the risk assessment. Moreover, the considerations made for the USA will be valid also for the European population. We therefore consider the US EPA RfD of 0.1 µg per kg body weight (and day) to be appropriate for Europe. It should be noted that it is mainly relevant for fertile women, and that it includes an uncertainty factor.
>
> The reference dose will be exceeded if a substantial amount of fish, contaminated with mercury, is ingested. As an example, if the weekly intake is about 100 g (one typical fish meal per week) of fish with > 0.4 mg/kg, the RfD will be exceeded. This suggests that fish mercury levels should be kept below this limit.
>
> Fish is, however, a valuable part of the diet, in adults as well as in children, and a source of e.g. protein, vitamin E, selenium, and omega 3 fatty acids. At high consumption of fish with low levels of mercury, like in the Seychelles Islands, the advantages and disadvantages may counterbalance each other. Because of the beneficial effects of fish consumption, the long-term aim is not to replace fish in the diet by other foods, but to reduce the methylmercury concentrations in fish. If this

is not possible, dietary restrictions with respect to fish with high levels of methylmercury should be advised for pregnant women."

274. An additional overview of some toxicological reference values (and briefs on their background) from a number of countries, and covering a few more mercury compounds, is given in the document "Compilation of toxicological and environmental data on chemicals – mercury and its derivates" (INERIS, 2000) submitted by France (can be viewed from UNEP's GMA home page, link: http://www.chem.unep.ch/mercury/gov-sub/Sub49govatt18.pdf).

275. The current EU limits for mercury in fish can be tightened for health reasons in individual member countries. Thus, some EU member states have lower limits than required by the directive. Because of high mercury concentrations in fish, certain lakes and rivers are closed to sports fishing, e.g., in Sweden. In addition, EU member states such as Denmark, Finland, Sweden and the United Kingdom, address specific advisories to sensitive populations. These can include women who are pregnant, plan to become pregnant, or who breast-feed, and children, in regard to avoiding or limiting the intake of fish species where the EU limit of 1 mg/kg applies (Finnish National Authority for Foodstuff, 2002)

UN Organizations

276. The Joint FAO/WHO Expert Committee on Food Additives (JECFA) established a provisional tolerable weekly intake (PTWI) of 200 µg (equivalent to 3.3 µg/kg body weight) for methylmercury in 1978, which was confirmed in 1988. In 1999, the Committee evaluated the Faroe Islands and Seychelles studies available at that time, as well as new neurodevelopmental toxicity studies in animals, and concluded that the studies did not provide consistent evidence of neurodevelopmental effects in children of mothers whose intake of methylmercury yielded hair burdens of 20 µg/g or less. The Committee could not evaluate the risks for the complex and subtle neurological end-points used in these studies that would be associated with lower intakes. In the absence of any clear indication of a consistent risk in these recent studies, the Committee recommended that methylmercury be re-evaluated when the 96-month evaluation of the Seychelles cohort and other relevant data that may become available can be considered. The Committee thus did not revise the PTWI of 3.3 µg/kg body weight.

4.2.2 Elemental mercury vapour and inorganic mercury compounds

277. For mercury vapour, studies of occupationally exposed humans have shown slight adverse effects on the central nervous system and kidneys at long-term air levels of 25-30 µg/m^3 or equivalent urinary mercury levels of 30-35 µg/g creatinine. Based on the LOAEL for effect on the central nervous system, the US EPA determined a reference concentration (RfC) for mercury vapour of 0.3 µg/m^3 for the general population (US EPA, 1997). The RfC took into account a conversion from occupational exposure to continuous exposure for the general population, lack of data on reproductive effects, the use of a LOAEL instead of a NOAEL, and susceptible subgroups. The US ATSDR established a minimum risk level (MRL) of 0.2 µg/m^3, also based on the occupational data. [2] Using the ATSDR document as the source document, and complementing the information with further studies on adverse effects observed among workers exposed to mercury vapour, and on studies on the relationship between concentrations of mercury in urine/blood of exposed workers and in the breathing zone air, IPCS identified 0.2 µg/m^3 as a guidance value for long-term inhalation exposure of the general public to metallic mercury vapour (WHO/IPCS, 2002).

278. In the European Position Paper on mercury (Pirrone *et al.*, 2001) it was concluded that – under European conditions – human exposure to elemental mercury in ambient air is generally negligible. As mentioned elsewhere, the case may be different in regions with higher direct air pollution loads. The following risk evaluation was presented:

[2] The USA, in their comments to this report (comm-24-gov), has stated the following as a remark to the risk evaluation presented by Pirrone *et al.* (2001): "The United States Government has used the best available data to determine safe exposure levels. These estimates are significantly above the 0.05 µg/m^3 value discussed in this paragraph (eds.: Quote of Pirrone *et al.*'s risk evaluation), but are nonetheless believed to be protective of health."

"For mercury vapour, studies of occupationally exposed humans have shown slight adverse effects on the central nervous system and kidneys, and probably also on the thyroid, at long-term air levels of 25-30 μg/m^3 or equivalent urinary mercury levels of 30-35 μg/g creatinine. The US EPA determined a reference concentration (RfC) for mercury vapour of 0.3 μg/m^3 for the general population (US EPA, 1997). Recent studies suggested that the limit for adverse effects (LOAEL) in occupationally exposed subjects may be lower than indicated above. There is no universal agreement on which uncertainty factors to use. In ongoing work on a EU position paper on arsenic, cadmium, and nickel, factors of 5-10 were used for similar conversion from occupational exposure to continuous exposure, factors of 5-10 for the use of a LOAEL, and a factor of 10 for variation of susceptibility. The total factor was 500. A similar procedure would result in a limit value for elemental mercury of 0.05 μg/m^3. We propose the use of 25 μg/m^3 as starting point, a factor of 10 for continuous exposure of the general population during a whole life-time, and uncertainty factors of 5 for the use of a LOAEL and 10 for individual susceptibility. The proposed limit value will then be 0.05 μg/m^3, as an annual average. This air level is rarely exceeded in ambient air in Europe, however. A typical daily absorbed dose would be 0.6-0.8 μg of mercury for adults. Exposure to elemental mercury from dental amalgam in most cases represents a much higher daily uptake than this level would give rise to (WHO/IPCS, 1991)."

279. Studies on exposed humans do not provide sufficient information to derive acceptable intakes for inorganic mercury compounds; therefore, based on No adverse effects and lowest adverse effects in medium- and long-term animal experiments, ATSDR and IPCS derived a guidance value of 0.2 μg/kg body weight per day for inorganic mercury compounds (US ATSDR, 1999; WHO/IPCS, 2002).

4.3 Routes of mercury exposure – a general overview

280. As mentioned above, the general population is primarily exposed to methylmercury through the diet (especially fish) and to elemental mercury vapours due to dental amalgams.

281. Human exposure to the three major forms of mercury present in the environment is summarised in table 4.3 in section 4.3.1. Although the choice of values given is somewhat arbitrary, this table nevertheless provides a perspective on the relative magnitude of the contributions from various media. Humans may be exposed to additional quantities of mercury occupationally and in heavily polluted areas, and to additional forms of mercury, e.g. to aryl and alkoxyaryl compounds, which are still used as fungicides in some countries. The following paragraphs present general contributions to human mercury exposure in a bit more detail, as reviewed by Pirrone *et al.* (2001), except for the text on occupational exposure.

Elemental mercury vapour from ambient air and dental fillings

282. Regarding vapour of metallic mercury, dental fillings, and to a lesser extent, the ambient air, represent the two major sources of human exposure for the general population. From the atmosphere the daily amount absorbed as a result of respiratory exposure into the bloodstream in adults is about 32 ng mercury in rural areas and about 160 ng mercury in urban areas, assuming rural concentrations of 2 ng/m^3 and urban concentrations of 10 ng/m^3 (absorption rate 80 percent).

283. Local contributions from airborne mercury may vary greatly depending on emissions from local sources. For example, the Indian submission (sub71govatt1) reports observed elevated mercury exposure in an area influenced heavily by emissions from thermal power plants. Another example is the submission of the Slovak Republic reporting ambient air concentration in urban areas in Slovakia in the range of 1.7 – 20 ng/m^3 (geometric mean 4.57 ng/m^3) and in industrial areas in the range of 1.5–40 ng/m^3 (geometric mean 5.28 ng/m^3), with the highest levels in areas with metallurgic industry and coal combustion (Hladiková *et al.*, 2001, as presented in sub10gov). Elevated air levels may also occur downwind from some types of emissions sources such as chlor-alkali plants.

284. Release of mercury from amalgam fillings has been reviewed by Clarkson et al. (1988). It was concluded that amalgam surfaces release mercury vapour into the mouth, and this is the predominant

source of human exposure to elemental mercury in the general population. Depending upon the number of amalgam fillings, the estimated average daily absorption of mercury vapour from dental fillings vary between 3 and 17 µg mercury (WHO/IPCS, 1991; Clarkson et al., 1988; Skare and Engqvist, 1994). In rare cases the blood mercury levels due to dental amalgam may be as high as 20 µg/l (Barregard et al. 1995, as quoted by Pirrone et al., 2001). Effects of exposure from dental amalgam has been widely discussed and reviewed (US Public Health Service, 1993, as quoted by Pirrone et al., 2001; and others). However, the Working Group for this Global Mercury Assessment, in line with its mandate, focused on environmental exposures to mercury and their adverse effects on health, and did not review or assess the potential effects of exposures to elemental mercury vapour from dental amalgams or the possible conversion to other mercury forms in the body. Moreover, the Working Group did not reach any conclusions about whether or not dental amalgams cause adverse effects.

Indoor non-occupational air exposure

285. Very little data are available on non-occupational indoor human exposure due to mercury vapour. However, fatalities and severe poisonings have resulted from heating metallic mercury and mercury-containing objects in the home. Also, incubators used to house premature infants have been found to contain mercury vapour at levels approaching occupational threshold limit values; the source was mercury droplets from broken mercury thermostats. In addition, significant exposures can occur due to use of metallic mercury in religious, ethnic, or ritualistic practices. Exposures can occur during the practice and afterwards from contaminated indoor air. A few of the activities reported that result in human mercury exposures include sprinkling elemental mercury in homes or cars, mixing mercury in bath water or perfume or placing mercury in candles (US ATSDR, 1999).

286. Indoor air mercury levels can also become elevated due to leaks from central-heating thermostats and by the use of vacuum cleaners after thermometer breakage and other spills. Another source of exposure to mercury vapor has been the release of mercury from paint containing mercury compounds used to prolong shelf-life of interior latex paint, in which levels of 0.3-1.5 µg Hg/m^3 (Beusterien *et al.*, 1991) have been reported. However, as explained in other sections of this report, the use of mercury in paints has decreased substantially in many nations of the world, therefore this source of exposure may be less common today than it was 10-30 years ago.

Drinking water

287. Mercury in drinking water is usually in the range of 0.5-100 nanograms of mercury per litre of water (ng Hg/l), the average value being about 25 ng Hg/l. The forms of mercury in drinking water are not well studied, but Hg(II) is probably the predominant species present as complexes and chelates with ligands. The resulting intake from drinking water is about 50 ng mercury per day, mainly as Hg(II); only a small fraction is absorbed. There are reports of methylmercury in drinking water under some conditions. It is, however, considered to be quite unusual (USA; comm-24-gov).

Intake from foods

288. Concentrations of mercury in most foodstuffs are often below the detection limit (usually 20 ng Hg per gram fresh weight) (US EPA, 1997). Fish and marine mammals are the dominant sources, mainly in the form of methylmercury compounds (70-90 percent or more of the total). The normal mercury concentrations in edible tissues of various species of fish cover a wide range, generally from 0.05 to 1.400 mg/kg fresh wet weight depending on factors such as pH and redox potential of the water, species, age and size of the fish (see sections 4.4 and 4.5). Large predatory fish, such as king mackeral, pike, shark, swordfish, walleye, barracuda, scabbard and marlin, as well as seals and toothed whales, contain the highest average concentrations. While large tuna typically have levels of mercury that are similar to other large predatory fish, data indicate that the levels usually seen in canned tuna are substantially lower. This results from the fact that the tuna currently used for canned tuna are those of smaller size.

289. The intake of mercury depends not only on the level of mercury in fish, but also the amount consumed. Thus, many governments have provided dietary advice to consumers to limit consumption

where levels are elevated. Fish consumption advisories typically take into account suspected concentrations, amount of fish - or canned fish - consumed and patterns of consumption.

290. Intake of fish and fish products, averaged over months or weeks, results in an average daily absorption of methylmercury variously estimated (in the 1970's) to be between 2 and 4.7 µg mercury (WHO/IPCS, 1976). The absorption of inorganic mercury from foodstuffs is difficult to estimate because levels of total mercury are close to the limit of detection in many food items and the chemical species and ligand binding of mercury have not usually been identified. The average daily intake of total dietary mercury has been measured over a number of years for various age groups. The intake of total dietary mercury (µg/day) measured during a market basket survey (1984-1986) of the Food and Drug Administration (FDA) in the USA (WHO/IPCS, 1990), according to age group was: 0.31 µg (6-11 months); 0.9 µg (2 years) and 2-3 µg in adults. In Belgium, two surveys estimated the total mercury intake from all foodstuffs to vary between 6.5 µg and 13 µg mercury (Fouasuin and Fondu, 1978; Buchet *et al.*, 1983).

Occupational exposure

291. Mercury in the working environment can lead to elevated exposures. As described in chapter 3 on human toxicology, a significant amount of the knowledge on the toxic effects of mercury and its compounds has been attained through the investigation of occupational exposures. Depending on the types of occupational activity and extent of implemented protective measures, the severity of effects may range from the subtlest disturbances to serious damages and death. Occupational exposures can happen in virtually all working environments where mercury is produced, used in processes or incorporated in products. Occupational exposure has been reported from – among others – chlor-alkali plants, mercury mines, mercury-based gold extraction, processing and sales, thermometer factories, dental clinics with poor mercury handling practices and production of mercury-based chemicals (US ATSDR, 1999).

292. In many countries a general improvement of protection against occupational exposure has taken place during the last decades by introduction of a range of working environment improvements including more closed manufacturing systems, better ventilation, safe handling procedures, personal protection equipment and through substitution of mercury-based technologies. This does, however, not seem to be a universal development, and many workers may still be exposed to mercury levels causing risks.

293. An example of the potential for improvements through implementation of such improvements and substitutions is that reported by Zavaris (1994) concerning mercury concentrations in employees exposed to mercury in specific industries: chlor-alkali, electric light bulbs, batteries and control instruments. Initially about 17 percent of the workers exceeded the legal limits for mercury in urine. After subsequent improvement in the working environment, and in some cases substitution of the mercury-based technology, in the industries involved, more than 98 percent of urinary levels had returned to the range of normal levels (abstracts of occupational exposure and industrial protection/substitution studies submitted by Brazil, sub66govatt6).

294. A UNIDO study has reported on the effects of mercury intoxication in the gold-mining area of Diwalwal, dominated by Mount Diwata (also known as Mt. Diwalwal), on the island of Mindanao - one of the major islands of the Philippines. At the time of the study, more than 70 percent (73 of 102) of the occupationally exposed population suffered from chronic mercury intoxication. Among the occupational sub-group of amalgam smelter workers the percentage was even higher – 85.4 percent. Of the non-occupationally exposed population in the area of Mt. Diwata and downstream, approximately one-third (55 of 163) showed signs of chronic mercury intoxication, including such classical symptoms as memory problems, restlessness, loss of weight, fatigue, tremor, sensory disturbances, and bluish discolouration of the gums (Böse-O'Reilly *et al.*, 2000).

Other exposures

295. Exposure to organic mercury, inorganic mercury or elemental mercury might occur through the use of mercury-containing skin-lightning creams, some traditional medicines, ritualistic uses, and cer-

tain pharmaceuticals (US ATSDR, 1999; Pelclova *et al.*, 2002). For example, thimerosal (ethylmercury thiosalicylate), also known as thiomersal, is used for preservation of some types of vaccines and immunoglobulins in parts of the world. Significant exposures can also occur from use of some Traditional Chinese Medicines or Traditional Asian Medicines (Ernst and Coon 2001; Koh and Woo, 2000; Garvey *et al.*, 2001).

4.3.1 Estimated Average Exposures

296. The WHO (1990) estimated the daily intake of each form of mercury as shown in table 4.3. For details on the methodology and assumptions used, see original reference. This table presents average estimated intakes for the different routes of exposure. However, exposures vary considerably across populations. For example, people who consume greater amounts of mercury-contaminated fish will obviously have greater exposures to methylmercury than those shown in the table.

Table 4.3 Estimated average daily intake and retention in the body (retention given in brackets) of different mercury forms in a scenario relevant for the general population not occupationally exposed to mercury, values in µg/day (WHO/IPCS, 1991; for more details, consult reference).

Exposure	Elemental Hg vapour	Inorganic Hg compounds	Methylmercury
Air	0.03 (0.024)*	0.002 (0.001)	0.008 (0.0069)
Dental amalgams	3.8-21 (3-17)	0	0
Food			
- fish	0	0.60 (0.042)	2.4 (2.3)**
- non-fish	0	3.6 (0.25)	0
Drinking water	0	0.050 (0.0035)	0
Total	3.9-21 (3.1-17)	4.3 (0.3)	2.41 (2.31)

Note: The data in brackets represent retained part of mercury input in the body of an adult.

* If the concentration is assumed to be 15 ng/m^3 in an urban area, the figure would be 0.3 (0.24) µg/day.

** Assumes 100 g of fish per week with the mercury concentration of 0.2 mg/kg.

297. When relating the intakes of the different mercury species in table 4.3, it should be remembered that their toxic impacts varies. [3] Therefore, it is not contradictory that the methylmercury intakes are lower than other mercury intakes, but still generally constitute the major adverse impact on humans from mercury compounds.

4.3.2 General aspects of dietary mercury intake

298. Daily intakes and retention of mercury from food is difficult to estimate accurately. In most food stuff mercury concentration is below 20 µg/kg. Mercury is known to bioconcentrate in aquatic organisms and it is biomagnified in aquatic food webs. For example, the concentration of mercury in small fish at low food web level (such as anchovies) is below 0.085 mg/kg, while in swordfish, shark and tuna values above 1.2 mg/kg are frequently reported (WHO/IPCS, 1991). In Scandinavian predatory fresh-water fish (perch and pike) average levels are about 0.5 mg/kg.

299. The use of fishmeal as the feed for poultry and other animals used for human consumption may result in increased levels of mercury. In Germany, the poultry contains 0.03 - 0.04 mg/kg. Cattle are able to demethylate mercury in the rumen, and therefore, beef meat and milk contain very low concentrations of mercury.

300. One of the major problems to accurately estimate daily intakes of various mercury forms from diet is that national survey programmes mainly report total mercury concentrations and the percentage of mercury as methylmercury is not known. Total mercury daily intakes reported in various countries

[3] Some conversion of elemental mercury takes place in the body, and therefore the species humans are exposed to may not necessarily be the species actually inflicting the specific toxicological mechanisms.

are given in table 4.4. In some national surveys the percentage of mercury originating from fish is provided. It is assumed that in this foodstuff (fish) the percentage of methylmercury is from 60 to 90 percent. Therefore fish and fish products represent the major source of methylmercury. It may be concluded that in those areas where fish consumption represent a considerable part of diet, exposures could be considerably higher than the value of the US EPA RfD.

Table 4.4 Selected estimates of the typical daily intake of mercury from dietary sources in a selection of countries (as presented by Pirrone et al., *2001).*

Country	Intake (µg/day)	References
Belgium	All food: 13 of which 2.9 is from fish All foodstuff: 6.5	Fouassin and Fondu, 1978 Buchet *et al.*, 1983
Poland	5.08 (age group 1-6 years) 5.43 (age group 6-18 years) 15.8 in adults From fish: 7% of total dietary intake	Szprengier-Juszkiewicz, 1988 Nabrzyski and Gajewska, 1984
Germany	0.8 from fish 0.2 from food (except fish and vegetables)	LAI, 1996
Croatia	From fish: 27.7 (total Hg) 20.8 (MeHg form)	Buzina *et al.*, 1995
Spain	4-8 (60-90 % from seafood) in Valencia only 27% is from the seafood 18 of which about 10 is from fish (Basque country)	Moreiras *et al.*, 1996 Urieta *et al.*, 1996
Sweden	1.8 (market-basket)	Becker and Kumpulainen, 1991
United Kingdom	2	MAFF, 1994
Finland	2	Kumpulainen and Tahvonen, 1989
The Netherlands	0.7	Van Dokkum *et al.*, 1989
Czech Rep.	0.7	Ruprich, 1995
Brazil	315 – 448 (Amazon, Medeira river)	Boishio and Henshel, 2000
Japan	10 6.9 –11.0 24 (18 as MeHg)	Tsuda *et al.*, 1995 Ikarashi *et al.*, 1996 Nakagawa *et al.*, 1997

301. Pirrone *et al.* (2001) give the following conclusion regarding the general exposure pattern in Europe:

> "Mercury vapour is a risk of decreasing importance in Europe, as mercury-containing thermometers and other instruments are being phased-out, and the emissions from the chlor-alkali industry have decreased. In addition, only one mercury mine remains in operation in Europe today. New developments in dental technology have resulted in filling materials that can substitute amalgam for many purposes.
>
> The methylmercury risk will depend on the dietary habits and local sources of contaminated fish and seafood. The substantial exposures documented in the Faroe Islands, Greenland and other northern populations are mainly due to ingestion of marine mammals. The extent of this problem within Europe is therefore limited. However, a study from the island of Madeira showed that the consumption of local black scabbard resulted in average methylmercury exposures that were even higher than on the Faroe Islands. Similarly, evidence on mercury in seafood from the Tyrrhenian Sea have shown concentration levels which overlap with those present in pilot whale meat. Thus, excess exposures occur in Europe and may reach or even exceed levels observed in populations in which adverse effects on brain development have been documented. "

302. This conclusion may possibly apply to large parts of the western world.

4.4 Exposure through diets of fish and marine mammals

303. In the following sections, examples of data on methylmercury exposure from fish diets in different parts of the world are presented: Sweden, Finland, USA, the Arctic, Japan, China, Indonesia, Papua New Guinea, Thailand, Republic of Korea, the Amazonas and French Guyana. In some of these countries or areas mercury depositions have affected mercury contamination levels over years, and countermeasures have been set in during the last decades to reduce national emissions. Mercury emissions are, however, distributed over long distances in the atmosphere and by the oceans. This means that even countries with minimal local and national mercury emissions, and other areas situated remotely from dense human activity, may very well be similarly affected. For example, high mercury exposures have been observed in the Arctic, far distances from any significant sources of releases.

304. Data on mercury concentrations in fish have been submitted from a number of nations and international organisations. Additionally, many investigations of mercury levels in fish are reported in the literature. Submitted data giving examples of mercury concentrations in fish from various locations in the world are summarised in this chapter. The overview illustrates that mercury is present all over the globe in concentrations that may affect human beings and wildlife.

4.4.1 Exposure from fish diet in Sweden and Finland

305. According to von Rein and Hylander (2000), fish has traditionally been an important part of the diet in Sweden thanks to a long coastline and many lakes and rivers. Today, because of mercury contents in the fish, detailed recommendations for the consumption are given for fresh water fish such as pike, perch, pike-perch, burbot and eel. Women of childbearing age are recommended not to eat these fish from Swedish lakes at all, and the rest of the population should not eat them more than once a week. Based on comprehensive data sets, it has been estimated that in about 50 percent of the approximately 100,000 Swedish lakes, pike (1 kg size) contain mercury levels above the international WHO/FAO limit of 0.5 mg mercury/kg wet weight, and in 10 percent of the lakes pike contains over 1 mg/kg wet weight (Lindquist *et al.*, 1991). It has been calculated that the mercury deposition in Sweden must decrease by 80 percent from the level of the late 1980's in order to reduce the mercury content in Swedish fish to below 0.5 mg mercury/kg wet weight. The emissions to air from point sources in Sweden itself have decreased to about 1 metric ton/year from peak values in the 1960's of around 30 metric tons/year, and releases to water have been reduced similarly (Naturvårdsverket, 1991). Most of the present mercury deposition in Sweden originates from long-range atmospheric transport from other countries (Håkansson and Andersson, 1990; Iverfeldt *et al.*, 1995). This means that in order to meet the 80 percent reduction goal, emissions from Europe and other parts of the Northern hemisphere must also be reduced further. There are indications of recent reductions in deposition, and during the last few decades a general decrease of about 20 percent has been observed in mercury concentrations in fish in Sweden (Johansson *et al.*, 2001).

306. Also in Finland, the accumulation of mercury in fish has been studied during several decades (Louekari *et al.*, 1994). In the late 1960's about 10-15 percent of the lakes and coastal waters in Finland were affected by elevated mercury concentrations mainly caused by direct aqueous releases from pulp and paper industry and (related) mercury-based chlor-alkali production. Average concentrations of mercury in northern pike in these freshwaters and brackish coastal waters averaged as much as 1.52 mg/kg wet weight at that time. Since the abandonment of the use of mercury compounds for slimicides in paper production in Finland in 1968 and decreasing demand for chlorine in the same industry, releases of mercury have been reduced significantly. In 1990 average concentrations in pike in these waters had decreased to 0.60 mg mercury/kg wet weight (concentrations in pikes in freshwaters were generally higher than in brackish waters). Louekari *et al.* (1994) combined these findings with dietary surveys and calculated estimated daily intakes of mercury in different consumer segments, and the relative influence of pike/fish consumption. In 1967/68, mercury intakes of the farmer segment known to be most depending on locally caught fish were estimated at 22 µg mercury/day in the areas with elevated mercury contamination. Similar intakes in 1990 were estimated at 15 µg mercury/day. For office employees, who consume less locally caught fish, corresponding intakes were 13 and 8 µg mercury/day.

307. The mercury concentration limit of 0.5 mg/kg in fish, recommended by WHO/FAO, is exceeded for one-kilo pike (*Esox lucius*) in 85 per cent of the lakes in southern and central Finland (22,000 lakes), (Lindquist *et al.*, 1991; Verta 1990; all in Pirrone *et al.*, 2001).

4.4.2 Exposure from fish diet in the USA

308. In the mid-1990's the US EPA estimated from comprehensive national dietary surveys that up to 5 percent of women in the child bearing age (ages 15-44 years) in the USA consumed 100 grams of fish and shellfish per day or more. WHO recommends "special considerations" regarding mercury exposure for persons eating more than 100 g/day. Furthermore, the US EPA calculated from the same dietary surveys combined with average total mercury concentrations in the species of fish consumed, that 7 percent of US women in the child-bearing age may exceed the exposure of the US EPA RfD (see section 4.2.1). A recent study (by the US Centers for Disease Control and Prevention) of mercury concentrations measured in blood and hair in a representative group of women aged 16-49 in the USA (about 1700 women) confirmed these calculations, as approximately 8 percent of the women had hair and blood mercury levels exceeding the levels corresponding to the US EPA RfD (CDC, 2001; Schober *et al.*, 2003). The CDC also collected hair and blood samples for year 2002, but these results are not yet available. Moreover, the CDC plans to continue the blood measurements in future years, but the hair samples are not planned after year 2002.

309. The US EPA noted that the calculated results reflected the average choice of fish species, and that "consumption of fish with mercury levels higher than average may pose a significant source of methylmercury exposure to consumers of such fish" (elevated mercury concentrations have been measured in fish in quite a number of freshwater bodies in the USA). The US EPA concluded in their risk characterisation that "most USA consumers need not be concerned about their exposure to mercury", but the exposure of "those who regularly and frequently consume large amounts of fish" (especially species with high mercury concentrations), may be of concern (US EPA, 1997).

310. In the USA, fish advisories (consumption recommendations) have been issued for mercury in one or more freshwater bodies in 41 states, and 13 states have issued statewide mercury fish advisories. Mercury is the most frequent basis for fish advisories in the USA, representing 79 percent of all advisories (as of December 2000; US EPA, 2001a). The US EPA has presented a set of general recommendations for fish consumption. For example, fish with mercury concentrations ranging from 0.48 -0.97 mg methylmercury/kg wet weight should be eaten no more than once a month and with 0.97 - 1.9 mg/kg wet weight only every second month, whereas fish containing more than 1.9 mg/kg wet weight should not be eaten at all (US EPA, 2001a); see table 4.2 in section 4.2.1 above.

311. Fish sold in commerce in the USA are under the jurisdiction of the Food and Drug Administration (FDA), which issues action levels for concentration of mercury in fish and shellfish. The current FDA action level (as per 1998) is 1 ppm (1 mg/kg) total mercury based on a consideration of health impacts. As illustrated in table 4.5 in section 4.5, US freshwater fish can have mercury levels which exceed the FDA action limit of 1 ppm. The levels in some marine species such as shark, swordfish, and king mackeral are also typically this high. The concentration of methylmercury in commercially important marine species is on average close to ten times lower than the FDA action level in the USA. Mercury levels in marine fish have been monitored by the National Marine Fisheries Service for at least 20 years. The data in marine fish have shown mercury levels over this time to be relatively constant in various species. Comparable trends data for freshwater fish do not exist, although there are data for coastal and estuarine sites (US EPA, 1997).

312. See also the description of Canadian experiences related to mercury in aquatic ecosystems, including a map showing national fish mercury concentrations, in section 5.3.

4.4.3 Exposure from marine diet in the Arctic

313. The comprehensive AMAP (1998) assessment report on arctic pollution issues describes the high exposures of the Arctic population. AMAP and other Arctic Council activities relevant to mercury

cover the whole of the Arctic region, and mercury is a priority substance for assessment and abatement initiatives for the Council. Here, examples of mercury exposure in Greenland are given.

314. As for much of the population in the region, the diet in Greenland is to a high degree composed of marine mammals and also fish. The traditional Greenlandic diet is also a very important part of the Greenlandic culture and identity.

315. The concentration and distribution of mercury in humans in Greenland have been thoroughly studied in the last 15 years. Surveys have been performed in adults, pregnant women and newborn babies in most parts of Greenland including both hunting districts and more densely populated areas. In all regions studied, the determining factors for mercury exposure were the daily intake of meat from marine mammals. At a regional level, the blood mercury concentrations were directly proportional to the registered number of seals caught (and consumed), indicating that mercury concentration in meat is probably similar in all regions of Greenland (Hansen, 1990). In adults, whole blood concentrations of mercury are lowest in the Southwest and increasing towards the North where the intake of marine mammals is higher – see figure 4.1.

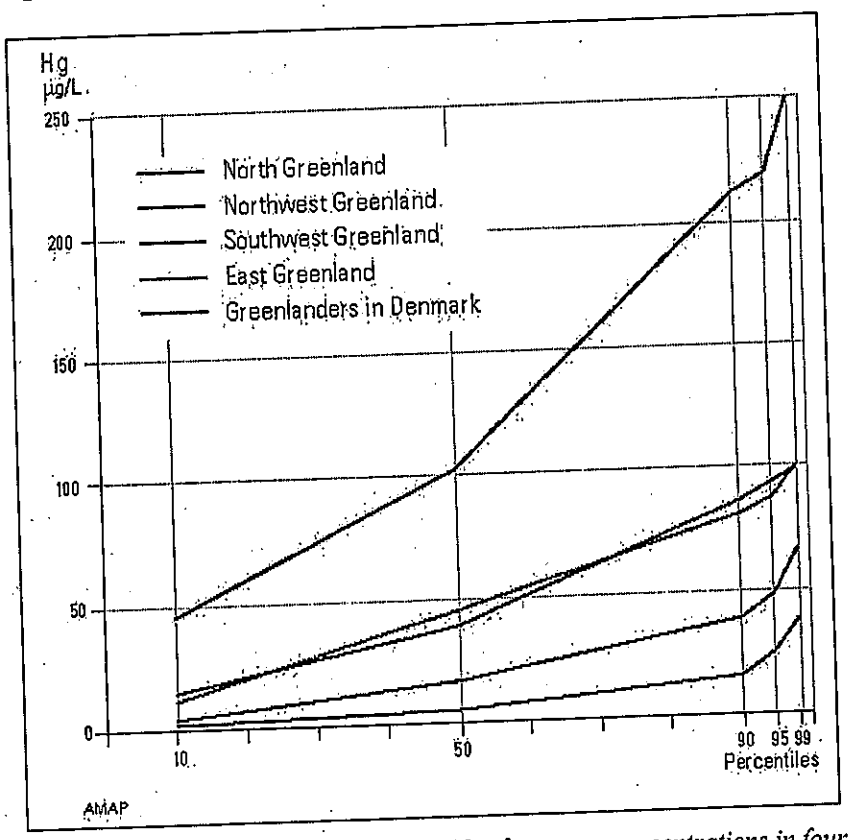


Figure 4.1 *Distribution (in percentiles) of whole blood mercury concentrations in four regions in Greenland and in Greenlanders living in Denmark (AMAP, 1998, based on 1988 measurements). Original figure presented courtesy of AMAP, Norway.*

316. In North Greenland, 16 percent of the adult population studied had blood mercury concentrations exceeding 200 µg/l, which is the level regarded by WHO as the minimum toxic blood concentration in non-pregnant adults (AMAP, 1998). More than 80 percent of the population in North Greenland exceeded 50 µg/l blood (Hansen and Pedersen, 1986), which almost corresponds to the benchmark dose level from the US NRC report (2000). Blood levels of 200 µg/l are approximately the level expected to occur following a daily average intake of about 4 µg methylmercury per kg body weight per day. Likewise, a daily intake of about 1 µg methylmercury per kg body weight per day is expected to result in blood mercury levels of about 50 µg/l and hair mercury levels of about 10 µg/g (US EPA, 1997; US ATSDR, 1999).

317. In a small set of 20 paired samples of maternal and umbilical cord blood taken under the AMAP programme, the mean concentrations were 24.2 and 53.8 µg/l, respectively. This level is very close to the NRC (2000) benchmark dose level (58 µg/l) based on the NRC evaluation of the Faroe Islands studies (see section 3.2.1).

318. As of 1997, no disease or symptoms had been registered which could be unequivocally related to environmental contaminant exposure in Greenland (AMAP, 1998). However, it should be noted that this can generally not be done for environmental contaminants because of its complexity, except in cases of extreme acute or sub-acute exposure. Furthermore, at that time measurements of more subtle neurological and reproductive effects had not yet taken place in Greenland. A recent study suggested exposure-related neurobehavioral deficits in Inuit children in Qaanaaq, Greenland, but the study was too small to provide solid statistical significance of the associations (Weihe *et al.*, 2002).

319. The traditional marine diet on Greenland and in parts of Arctic Canada has very positive nutritional qualities and is not readily replaced with other foods. Dietary advice from the Canadian Government states that the positive health benefits of a traditional northern marine diet outweigh the known risks associated with consumption of these foods. However, it is clear that the risks associated with this diet increase with increasing levels of methylmercury contamination. It is further important to note that, beyond the physical benefits associated with the traditional diet, it also plays an important role in the social and cultural life of indigenous communities in the North.

320. As mentioned above, the investigation of mercury exposure and effects on the Faroe Islands on the border of the Arctic area has been extensive, and subtle neurological effects have been shown on children at low prenatal exposure levels, see description in section 3.2.1 above.

321. The Arctic Council and the substantial coverage of mercury in its monitoring and assessment programme (AMAP) and its current action plan (ACAP) are described in section 9.5.1.

4.4.4 Examples from Asia

China, Japan and Indonesia

322. Feng *et al.* (1998) investigated total mercury and methylmercury concentrations in scalp hair of 243 male persons in three areas of the Tokushima Prefecture, Japan as well as in 64 males of the Chinese city Harbin and 55 males in the Indonesian city Medan (all subjects were randomly chosen males aged 40-49 years). They found the highest concentrations in subjects living in a seaside area reported to be without local direct anthropogenic contamination. Total mercury concentrations here ranged from 1.7-24 µg/g hair (mean 6.2 µg/g, 78 subjects), thus close to and exceeding the adverse effect benchmark level of about 10 µg/g maternal hair derived from the Faroe Islands studies (see section 3.2). The mean concentration for all three investigated areas in Japan was only slightly lower: 4.6 µg/g hair (243 subjects).

323. In Japan, where the diet is relatively high in fish and shellfish, methylmercury constituted large parts of the total mercury measured, and there was a high correlation between concentrations of methylmercury and total mercury, underlining that a marine diet was the major contributor to mercury exposure. Feng *et al.* (1998) quote the Japan General Affairs Department for 1996 dietary surveys estimating average national consumption of fish and shellfish at 107 g/day per person, being the third highest consumption rates among 23 countries investigated.

324. In the industrial cities of Harbin, China, and Medan, Indonesia, Feng *et al.* (1998) found lower mean total mercury concentrations (means 1.7 µg/g and 3.1 µg/g hair respectively). In both of these places methylmercury concentrations were lower – even for subjects with high total mercury concentrations - and correlation between methylmercury and total mercury concentrations was low, indicating that these subjects were mainly exposed to elemental or inorganic mercury from other sources.

Papua New Guinea

325. Feng *et al.* (1998) quotes Suzuki (1991) for mercury hair concentration levels found in residents of three villages in Papua New Guinea not influenced by local direct anthropogenic contamination. The highest concentrations were found in the seaside village Dorogi with means at 4.1 and 4.4 µg/g hair for males and females respectively, while concentrations were slightly lower in a riverside village 6 kilometres from the coast and lowest in a village 25 kilometres from the coast.

Thailand

326. For Thailand, the national submission (sub53gov) quotes Menasveta (1993) for an average national fish consumption rate of 61 g/day per person for Thai people (with average weight 60 kg). There is no study on hazards from methylmercury exposure of the Thai population.

Philippines

327. The average estimated national fish consumption rate is 75 g/person per day, and the average person weighs 60 kg. Also, the exposures described in the study by UNIDO (described in section 4.3 above) on mercury intoxication on the island of Mindanao (a gold-mining area) are probably partially due to exposures through the diet, especially for the non-occupationally burdened part of the population downstream from Mt. Divalwal, where approximately a third (55 of 163) are intoxicated (Global Mercury Assessment Working Group - Philippines delegation, 2002).

Republic of Korea

328. According to the national submission from the Republic of Korea, the supply of fish amounted to between 74 and 94 g fish/day per person in this country in the years 1996-1999 (Republic of Korea submission, sub76govatt2).

4.4.5 Exposure from fish diet in the Amazonas and French Guyana, South America

329. Several studies in the Amazonas have reported elevated exposures to methylmercury and total mercury in fish dependent populations in and around areas affected by mercury-based gold extraction.

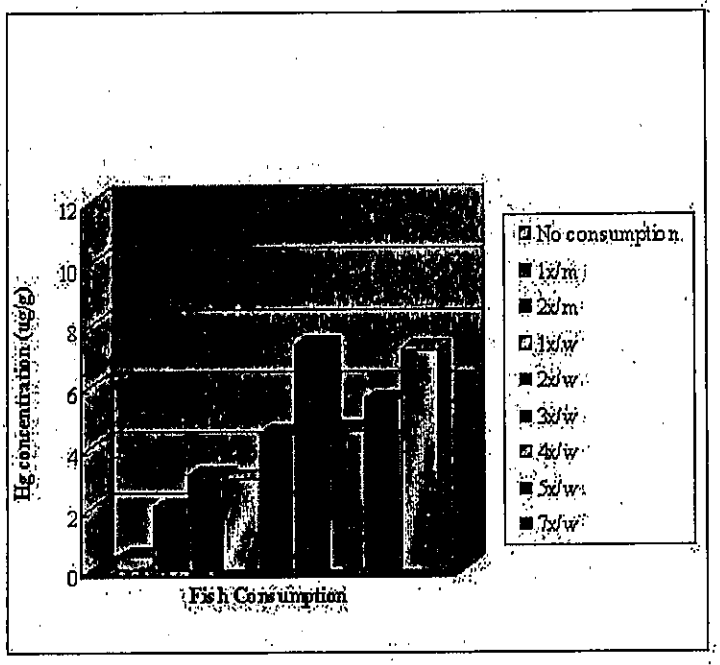
330. Some studies in the Amazonas have shown adverse effects from mercury exposure on humans. For example, in the Tapajós river community of Brazil, cognitive deficits have recently been reported in 7-year children who were exposed, in uterus, to mercury levels corresponding to maternal hair mercury levels below 10 µg/g hair (Malm *et al.*, 1999, as quoted the in Brazilian submission sub66govatt2A). Quite a number of studies have investigated exposures and toxic impacts from mercury in individual areas affected by gold mining activities in the Amazonas. The Ministry of Health, Brazil, reports to be in the process of reviewing the available exposure data from the Amazon area with fish consumption and mercury concentration in fish as focal points (sub66govatt2A). The Ministry has also submitted a list of a large number of references relevant to the impacts of mercury in the Amazon (sub66govatt2B).

331. Akagi and Naganuma (2000) used separate measurements of methylmercury and total mercury to distinguish between exposures through an aquatic diet and direct exposures of elemental mercury from gold extraction activities. They found methylmercury concentrations exceeding the adverse effects level for adults of 50 µg/g in hair in 3.2 percent of the 559 inhabitants surveyed, with the highest individual level being 132 µg/g. These values are substantially higher than the adverse effect benchmark level of 10 µg/g maternal hair derived from the Faroe Islands studies (see section 3.2.1).

332. Vasconcellos *et al.* (1998) determined total mercury concentrations in scalp hair in 13 of the 17 tribes of Indians inhabiting the Xingu Park in the Brazilian Amazon. In six of the investigated groups methylmercury concentrations in hair were also measured. Geometrical means for total mercury concentrations varied among the tribes in the range of 3.2-21 µg/g hair, but most group means were between 10 and 20 µg/g. In the tribes where methylmercury was also measured, methylmercury comprised nearly all of the mercury found in the hair samples. In the same study, three groups of inhabitants in the Brazilian State of Amapá were also investigated. Total mercury in hair versus numbers of fish

meals per week are shown in figure 4.2 - first for a region not affected directly by gold extraction (figure 4.2 a) and then for another region which is affected by gold extraction (figure 4.2 b).

a) Total mercury concentrations in hair versus fish consumption – region of Serra do Navio, State of Amapá, Brazil (not directly affected by gold extraction)



b) Total mercury concentrations in hair versus fish consumption – region of Vila Nova, State of Amapá, Brazil (directly affected by gold extraction)

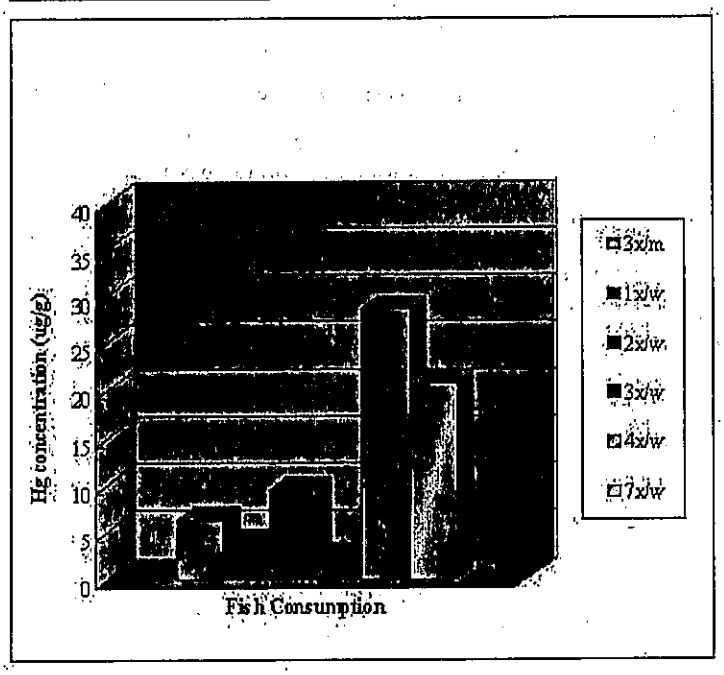


Figure 4.2 Total mercury concentrations in hair vs fish consumption in two regions of the State of Amapá, Brazil (from Vasconcellos et al., *1998, submitted by Brazil, sub68govatt1)*

333. Some researchers have considered if gold extraction alone could explain the observed mercury contamination levels in the Amazonas area. Other mercury sources mentioned are volcanic contributions and increased mobilisation due to deforestation and other sources of soil erosion (based on USA, comm-24-gov, 2002).

French Guyana

334. A study undertaken by Fréry *et al.* (1999) among the Wayana people in the higher area of the Maroni River, French Guyana, whose diet is based mainly on fish, confirmed mercury exposure due to consumption of river fish contaminated by mercury from gold extraction activities. Of 242 fish samples analysed, 14.5 percent had mercury levels over 0.5 mg/kg (with a high of 1.62 mg/kg). Based on the Wayana's fish consumption patterns, adults were found to consume between 40 and 60 µg total mercury per day, nursing infants approximately 3 µg per day, children between 1 and 3 years of age 7 µg per day, between 3 and 6 years approximately 15 µg per day and between 10 and 15 years between 28 and 40 µg per day. Over half of the population had hair mercury levels over the WHO recommended level of 10 µg total mercury/g, with an average of 11.4 µg/g. (Mercury levels in the population of Guyana are approximately 3 µg/g and 1.7 µg/g in people from urban areas.)

4.5 Submitted data on mercury concentrations in fish

335. Information on mercury concentrations in fish in different parts of the world has been chosen in this report as an indicator illustrating the presence of mercury in the global environment. Data on mercury concentrations in fish have been submitted from a number of nations and international organisations. Additionally, many investigations of mercury levels in fish are reported in the literature. Submitted data giving examples of mercury concentrations in fish from various locations in the world are summarised in table 4.5. The available data illustrate that mercury is present all over the globe in concentrations that may affect human beings and wildlife.

336. As an illustration of how the observed concentration levels are related to potential adverse effect levels, **concentrations at or exceeding 0.3 mg/kg wet weight** – the US EPA Tissue Residue Criterion (at 17.5 gram fish intake/day) and the Japanese guideline value (see section 4.2.1) – have been **marked in bold text in the table**. These values represent the most recent comprehensive risk assessments regarding mercury exposure from fish diets. As mentioned in table 4.1, FAO/WHO Codex Alimentarius guideline levels for fish are 0.5 mg/kg wet weight for non-predators and 1 mg/kg wet weight for predators (such as shark, swordfish, tuna, pike and others).

Table 4.5 Examples of mercury concentrations in fish/shellfish in different regions of the world, as reported in submissions to the Global Mercury Assessment. Sample collection, treatment, and analysis methodology may vary and may have affected results. Consult references for details.

Geographic location	Fish and shellfish species	Concentration (-level) *3 ww: Wet weight *4 dw: dry weight *5	Year of sampling	Trophic level *1	Contamination level in habitat *2	References
Arctic area	Marine fish	0.01 - 0.1 mg/kg ww Peaks: 0.1 - 0.9 mg/kg ww	Various			AMAP, 1998
	Marine mussels	<0.009 - 0.033 mg/kg ww	Various			
Australia (southwest Tasmania)	Australian eel (Lake Gordon)	**0.86 – 2.15 mg/kg** **(mean 1.40 mg/kg, 9 samples)**	1994			Bowles, 1998, in National submission from Australia, sub63gov
	Brown trout (Lake Pedder)	0.06 – 0.3 mg/kg (mean 0.16 mg/kg, 20 samples)	1993			
	Brown trout (Lake Gordon)	0.1 – 1.4 mg/kg (mean 0.35 mg/kg, 20 samples)	1994			
	Brown trout (Gordon River)	0.3 – 2.35 mg/kg (mean 1.09 mg/kg, 25 samples)	1993			
	Redfin perch (Lake Gordon)	0.12 – 1.3 mg/kg (mean 0.52 mg/kg, 20 samples)	1993			
Baltic Sea	Round fish	0.010-0.050 mg/kg ww	1994-1998		Back	ICES, 1997, in Helcom, 2001
	Marine fish	0.016 - 0.091 mg/kg ww (muscle, all investigated species).			Gen	
	Blue mussel	0.005 - 0.010 mg/kg ww		Non	Back	
	Blue mussel	Slightly exceeding 0.01 mg/kg ww			Gen	

Geographic location	Fish and shellfish species	Concentration (-level) *3 ww: Wet weight *4 dw: dry weight *5	Year of sampling	Trophic level *1	Contamination level in habitat *2	References
Brazil	46 species from six trophic levels: Herbivore/Denitrivore Planktophagus/Omnivore I Omnivore II/Piscuvore	 0.10/0.15 mg/kg (ww) 0.36/0.21 mg/kg (ww) 0.55/0.64 mg/kg (ww)	1991-1993			Boischio and Henshel, 2000
Brazil (Amazonas)	River fish from pristine areas Predatory fish from contaminated areas (main mined Amazonas river basin)	Lower than 0.2 mg/kg ww of Hg Can reach levels of 2 – 6 mg/kg or more, Average values above 0.5 mg/kg	1990's	 Pre	Back Con	Malm, as contained in NIMD Forum, 2001, in national submission from Japan (sub6govatt1)
Côte d'Ivoire	Tuna species, "Thon Albacore" (Thunnus Albacares) Large individuals (80-91 kg): Sole, "sole" Herring, "hareng"	0.30 - 0,36 mg/kg ww 0.8 mg/kg ww (muscle) 0.064 - 0,090 mg/kg ww 0.037 - 0,047 mg/kg ww	1991	Pre Non Non	Gen Gen Gen	National submission from Côte d'Ivoire (sub72gov)
Cyprus	Sword fish Sea bream Red mullet Common dentex (dentex dentex)	0.20 - 2.00 mg/kg ww (mean 0.54 of 21 samples) 0.00 - 2.00 mg/kg ww (mean 0.38 of 42 samples) 0.00 - 0.70 mg/kg ww (mean 0.11 of 15 samples) 0.00 - 2.00 mg/kg ww (mean 0.51 of 20 samples)	1993-1997	Pre Non	Gen Gen Gen Gen	National submission from Cyprus (about 15 species reported in all)
Fiji	Shellfish (*Crassostrea mordax*) Shellfish (*Crassostrea mordax*) Shellfish (*Grafarium tumidum*) Shellfish (*Anadara spp.*) Canned tuna	<0.001-0.061 mg/kg ww 0.55-0.95 mg/kg dw 0.05-0.20 mg/kg dw 0.037-0.099 mg/kg dw 0.01-0.97 mg/kg ww	1987/88 1988 1985/86 1992/93 1990/92		Back Con Back Back ?	Naidu *et al.*, 1991 Naidu and Morrison, 1994 Gangaiya *et al.*, 1988 Morrison *et al.*, 2001 IAS, 1992
Finland	Northern pike in freshwater and brackish coastal waters	1.52 mg/kg ww of Hg (average concentration) 0.60 mg/kg ww of Hg (average concentration)	1960's 1990			Submission from the Nordic Council of Ministers, sub84gov
France	Mussels (369 samples from 96 sampling stations along the coast of France	0.008 – 0.238 mg methylHg/kg dry weight (mean 0.064 mg/kg dry weight)	1996			Claisse *et al.*, 2001, in national submission from France, sub49gov
	Fish, Atlantic Sea: Conger Merlu Rousette	 1.2 +/- 0.3 mg/kg dw 0.4 +/- 0.1 mg/kg dw 2.0 +/- 0.6 mg/kg dw				Cossa, 1994 in national submission from France (sub49gov).
	Fish, Mediterranean Sea: Conger Merlu Rousette	 4.5 +/- 2.8 mg/kg dw 3.2 +/- 2.1 mg/kg dw 9.4 +/- 5.2 mg/kg dw				
	Fish caught in Baltic and North Sea, English Channel, Atlantic Ocean) Swordfish (*Xiphias gladius*) Shark (*Lamna sp.*) Red tuna (*Thunnus thynnus*)	 Mean 0.780 mg/kg ww (41 samples) Mean 0.692 mg/kg ww (497 samples) Mean 0.470 mg/kg ww (344 samples)	1971 – 1980			Thibaud, 1992 in national submission from France (sub49gov)

Geographic location	Fish and shellfish species	Concentration (-level) *3 ww: Wet weight *4 dw: dry weight *5	Year of sampling	Trophic level *1	Contamination level in habitat *2	References
Ghana	River species: Mostly "tilapia" (tilapia guineensis) and "catfish" (heterobranchus spp.)	General: **0,55 - 1,59** mg/kg ww Tilapia, mean: **1,17** mg/kg ww (of 8 fish)	2000		Con	National submission from Ghana and UNIDO report sub2igoatt6part2
Guam	Fish	0.009-0.045 mg/kg ww			Back	Denton *et al.*, 2001
Hong Kong	Mud carp (*Cirrhinus molitorella*) Freshwater grouper (*Micropterus sp.*) Golden thread (*Nemipterus virgatus*) Hair tail (*Trichiurus haumela*)	0.025 mg/kg ww 0.195 mg/kg ww 0.219 mg/kg ww 0.146 mg/kg ww	1995			Dickman and Leung, 1998
India	18 groups of fish and other seafood in the Bay of Bengal, Arabian Sea and Indian Ocean	0.005-0.065 mg total Hg/kg (mean average values)			Back	Ramamurthy, 1979, in comments from India (comm.-13-gov)
	Bombay, west coast Fish Bivalves Gastropods Crabs *Madras, southeast coast* Fish Fish *Sagar Island, east coast* Bivalves	 0.03-**0.82** mg total Hg/kg dw 0.13-**10.82** mg total Hg/kg dw **1.05-3.60** mg total Hg/kg dw **1.42-4.94** mg total Hg/kg dw Below detection limit (100 ng/g) 0.08-0.14 mg total Hg/kg ww 0.06-**2.24** mg total Hg/kg dw				Bhattacharya and Sarkar, 1996
Italy	Bluefin tuna (Thunnusthynnus thynnus)	0-4 mg total Hg/kg ww		pre	gen	Renzoni *et al.*, 1998
Japan	Scorpionfish, inside Minamta Bay Scorpionfish, outside Minamata Bay	**0.655** mg/kg ± 0.162 **0.511** mg/kg ± 0.241 **0.603** mg/kg ± 0.216 **0.531** mg/kg ± 0.194 **0.431** mg/kg ± 0.163	1978 1993 1983 1990 1999			Yasuda *et al*, in national submission from Japan, sub6gov
Kiribati	Shellfish (*Anadara spp.*)	<0.0001-0.006 mg/kg ww	1987		Back	Naidu *et al.*, 1991
Korea, Republic of	Unspecified freshwater fish species from 12 places each in Keum and Nakdong River Basins, respectively	Mean 0.126 mg/kg total Hg (10 species, 90 samples) Mean 0.196 mg/kg total Hg (6 species, 124 samples.	1989 1985			National submission from Korea (sub76govatt1)
	7 freshwater fish species(Givel, Carp, Grey mullet, Cat fish, Shake head, Eel, Mandarin fish) from Kangkyung area in Keum River	Mean **0.351** mg/kg (muscle, 7species, 57 samples)	1980			National submission from Korea (sub76govatt1)
	Freshwater fish species from 24 streams in South eastern area in Korea (Carassius auratus, Zacco temmincki, plecoglossus altivelis, Moroco lagowskii, Chaenogobius urotaenia urotaenia etc.)	0.02 – 0.12 mg/kg mean 0.07 mg/kg	1979			National submission from Korea (sub76govatt1)
Kuwait	Shrimp, various species	Not detected – 1.57 mg/kg (average less than 0.4 mg/kg)	1980's			Khordagui and Dhari, 1991, in UNESCWA submission, sub1igo

Geographic location	Fish and shellfish species	Concentration (-level) *3 ww: Wet weight *4 dw: dry weight *5	Year of sampling	Trophic level *1	Contamination level in habitat *2	References
Mauritius	Shark (unspecified)	0.13 - 0.60 mg/kg of Hg (52 samples of fresh shark)	?	Pre	Gen	National submission from Mauritius, sub56gov
	Marlin	1.20 – 3.00 mg/kg of Hg (in 8 samples), 0.10-0.90 mg/kg of Hg (in 18 other samples)				
	Tuna	0.10 – 0.70 mg/kg of Hg (16 samples of fresh tuna)				
	Swordfish	0.22 – 0.65 mg/kg of Hg (in 17 samples of swordfish)				
North East Atlantic (OSPAR waters)	Marine fish	0.01-0.2 mg/kg ww (general) Up to 0.9 mg/kg ww (peak areas)	1993-1996		Gen	OSPAR, 2000b and 2000, in submission from the Nordic Council of Ministers, sub84gov)
	Marine mussels	0.01-0.1 mg/kg ww(general) Up to 0.9 mg/kg ww (peak areas)		Non	Gen	
Norway	Pike	0.1 – 2.5 mg/kg	1988-1994			National submission from Norway, sub70gov
	Perch	0.1 – 2.5 mg/kg				
Philippines	Fish in river systems	0.00107 – 0.439 mg/kg totalHg 0.00071 – 0.377 mg/kg methylHg	1996-1999		Con (artisinal gold mining area)	National submission from Philippines, sub1gov
	Taiwan clam	0.233 -1.208 mg/kg total Hg	1997-1999	Non		
	Tilapia	0.109-0.494 mg/kg total Hg	1996-1999			
Seycelles	Various ocean species	Mean of 0.2-0.3 mg/kg				Cernichiari *et al.*, 1995, as quoted by Pirrone *et al.*, 2001
Slovak Republic	Some river and lake species:		1995-2000			Comments from Slovak Republic (Comm-14-gov)
	Barbel (*Barbus barbus*)	0.053-7.329 mg/kg ww (mean 0.728 mg/kg, 29 samples)				
	European perch (*Perca fluviazlis*)	0.009-1.964 mg/kg ww (mean 0.212 mg/kg, 34 samples)	1995-2000			
	Grayling (*Thymallus thymallus*)	0.032-0.110 mg/kg ww (mean 0.064 mg/kg, 6 samples)	1995-1997			
	Rainbow trout (*Salmo gairdnerii*)	0.001-0.970 mg/kg ww (mean 0.038 mg/kg, 56 samples)	1995-2001			
	Eel (*Anguilla anguilla*)	0.007-0.220 mg/kg ww (mean 0.093 mg/kg, 8 samples)	1995-1996			
Solomon Islands	Fish flesh (spp. Unknown)	0.0002-0.0014 mg/kg ww			Back	Kannan *et al.*, 1995
	Fish liver (spp. Unknown)	0.089-0.120 mg/kg ww				
Sweden	Northern pike of one kilogram in inland waters	0.1-2.0 mg/kg ww				Comments from Sweden (Comm-12-gov)
Taiwan	Blue marlin (*Makaira mazara*)	10.3 mg/kg dw	1995-1996			Han *et al.*, 1998
	Tuna (*Thunnus albacores*)	9.75 mg/kg dw				
	Grass shrimp (*Penaeus mondon*)	2.19 mg/kg dw				
	Oyster (*Crassostrea gigas*)	0.180 mg/kg dw				
Thailand	Unspecified fish, shrimp and shellfish species at 15 different river mouths (caught with "artisanal gear")	0.041-0.32 mg/kg (dw)	1998		Gen	National submissions from Thailand, sub53gov
		0.01-0.6 mg/kg (dw)	1999			
	Snapper, Grouper, Threadfin bream, Lizard fish, Cobia	0.049 – 0.694 mg/kg (ww)	1997			Windom and Cranmer, 1998

Geographic location	Fish and shellfish species	Concentration (-level) *3 ww: Wet weight *4 dw: dry weight *5	Year of sampling	Trophic level *1	Contamination level in habitat *2	References
Tonga	Shellfish (*Grafarium tumidum*)	0.022-0.191 mg/kg ww	1987		Back	Naidu *et al.*, 1991
United Kingdom (Irish Sea)	Flounder (*Platichthys flesus*) caught close to Ireland, Wales, Isle of Man Flounder caught close to Liverpool Bay Plaice (*Pleuronectes platessa*) Dab (*Limanda limanda*) Lesser spotted dogfish (*Scyliorhinus canicula*)	0.008 – 0.331 mg/kg ww Up to 1.96 mg/kg ww Less than 0.5 mg/kg ww Less than 1.1 mg/kg ww Less than 2.5 mg/kg ww	?			Leah *et al*, 1992 in national submission from United Kingdom, sub39govatt1
United Kingdom	Eels (*Anguilla anguilla*) Caught in various East Anglia locations	0.001 – 0.082 µg/kg (mean 20) 0.014 – 0.788 µg/kg (mean 170) 0.022—0.168 µg/kg (mean 82)	?			Downs *et al.*, 1999 in national submission from United Kingdom, sub39govatt1
United Kingdom	Survey of 336 fresh/frozen/processed sea fish and shellfish - Halibut Marlin Shark Swordfish Tuna	0.038-0.617 mg/kg (mean 0.290, 2 samples) 0.409-2.204 mg/kg (mean 1.091, 4 samples) 1.006-2.200 mg/kg (mean 1.521, 5 samples) 0.153-2.706 mg/kg (mean 1.355, 17 samples) 0.141-1.500 mg/kg (mean 0.401, 34 samples)				University of Bristol Survey - Mercury in imported fish and shellfish and UK farmed fish and their products, unpublished, posted at www.food.gov.uk/multimedia/pdfs/Mercury_in_Fish_table.pdf
United States of America	Bottom feeders – Carp Channel catfish White sucker Predators – Smallmouth bass Brown trout Largemouth bass Walleye Northern pike	 0.061 –0.250 mg/kg 0.010 - 0.890 mg/kg 0.042 - 0.456 mg/kg 0.094 - 0.766 mg/kg 0.037 - 0.418 mg/kg 0.101 - 1.369 mg/kg 0.040 - 1.383 mg/kg 0.084 - 0.531 mg/kg	1990-1995	Non Pre		US EPA, 1997
Vanuatu	Shellfish (*Anadara spp.*) Shellfish (*Crassostrea mordax*)	0.02-0.04 mg/kg ww 0.01-0.04 mg/kg ww	1987 1987		Back	Naidu *et al.*, 1991

Notes:

1 Indication of trophic level: **Pre** - predator/higher level; **Non** - non-predator/lower level;

2 Indication of contamination level in habitat: **Gen** - general/unspecified; **Back** - background level; **Con** – contaminated.

3 Unless otherwise mentioned, it is assumed that the results refer to measured content of total mercury (and not methylmercury).

4 Mercury concentration may be assumed to be wet weight (ww) unless otherwise indicated.

5 Dry weight results will by definition be higher than wet weight result (because of the water content in fish and seafood), and is therefore not directly comparable to wet weight results and guideline values based on wet weight.