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# Mercury

Mercury is a toxic element and serves no beneficial physiological function in man; a maximum acceptable concentration of  $0.001\text{ mg/L}$  ( $1\text{ }\mu\text{g/L}$ ) in drinking water has therefore been established. The presence of mercury in water has become a source of concern because of the finding that organic mercury is bioconcentrated by fish. Elevated mercury levels have been found in all freshwater fish taken from areas with suspected mercury contamination and frequently render the fish unacceptable for human consumption. Long-term daily ingestion of approximately  $0.25\text{ mg}$  of mercury as methyl mercury has caused the onset of neurological symptoms; however, even in heavily polluted Canadian waters, mercury concentrations rarely exceed  $0.03\text{ mg/L}$ . The maximum acceptable concentration for mercury therefore provides a considerable margin of safety. Mercury levels in both surface water and tap water are generally well below the maximum acceptable concentration.

## General

Mercury is a dense, silver-white metal that melts at  $-38.9^\circ\text{C}$ . Mercury is present in the Earth's crust at an average concentration of  $0.08\text{ mg/kg}$ ; cinnabar (mercury[II] sulphide,  $\text{HgS}$ ) is the most common mercury ore.<sup>(1,2)</sup> Canadian soils contain mercury at an average concentration of  $0.1\text{ mg/kg}$ , but concentrations up to  $10\text{ mg/kg}$  have been found in soils near cinnabar deposits in British Columbia.<sup>(3)</sup> Igneous, metamorphic, and sedimentary rocks contain mercury at concentrations up to  $0.25$ ,  $0.40$ , and  $3.25\text{ mg/kg}$ , respectively.<sup>(1)</sup>

Mercury and its compounds are used in dental preparations, thermometers, fluorescent and ultraviolet lamps, and pharmaceuticals, and as fungicides in paints, industrial process waters, and seed dressings. The most important consumer of mercury in Canada is the chlor-alkali industry, which produces chlorine and caustic soda. The pulp and paper industry also consumes mercury in significant amounts in the form of phenyl mercuric acetate, a fungicide, and in caustic soda, which may contain up to  $5\text{ mg/kg}$  as an impurity.<sup>(3)</sup>

Mercury has not been mined in Canada since 1975. In 1980, Canada imported  $50\text{ tonnes}$  of metallic mercury and consumed  $36\text{ tonnes}$ .<sup>(4)</sup> Imports of metallic mercury

and mercury oxide, chlorides, and sulphide totalled  $303\text{ tonnes}$  in 1981 and  $71\text{ tonnes}$  in 1982.<sup>(5)</sup> Annual world mercury production in 1981 was  $7100\text{ tonnes}$ ;<sup>(4)</sup> world reserves have been estimated at  $200\,000\text{ tonnes}$ .<sup>(2)</sup>

## Occurrence

Many mercury compounds are volatile, and most decompose to form mercury vapour, although some sublime without decomposition.<sup>(6)</sup> Elemental mercury has a substantial vapour pressure even at ambient temperatures but, except at elevated temperatures, does not react readily with oxygen in air. Mercury can exist as univalent and divalent ions. Mercury(I) is always in the dimeric form,  $\text{Hg}_2^{2+}$ , and all of its compounds are ionized in solution. Mercury(II),  $\text{Hg}^{2+}$ , forms both covalent and ionic bonds;  $\text{HgCl}_2$ , for example, is covalent. This causes a relatively low solubility of  $\text{HgCl}_2$  in water and higher solubility in organic solvents. Mercury(II) can also form complexes by accepting pairs of electrons from ligands. The covalent property of mercury(II) allows a stable mercury-carbon bond and the formation of organometallic compounds. No organomercury(I) compound has been isolated as yet. The organomercury salts are soluble in organic solvents, and compounds such as dimethyl mercury,  $(\text{CH}_3)_2\text{Hg}$ , can easily be separated from inorganic salts and even from  $\text{HgCl}_2$ , as  $\text{HgCl}_2$  can first be complexed to form water-soluble  $\text{HgCl}_4^{2-}$  with excess chloride.<sup>(7)</sup>

Mercury can enter the atmosphere by simple transport as metallic mercury vapour or as volatilized organic mercury compounds. The formation of volatile organomercurials may occur through microbial, animal, or plant metabolic activity. These natural processes result in the constant circulation of significant quantities of mercury in the atmospheric environment. An estimated  $30$  to  $50\text{ tonnes}$  of mercury are released in Canada annually due to natural processes.<sup>(6)</sup> Atmospheric emissions of mercury due to human activities in Canada in 1978 totalled  $40\text{ tonnes}$ . Of this,  $41\text{ percent}$  came from the recovery of base metals,  $13\text{ percent}$  from coal combustion,  $12\text{ percent}$  from paint application, and  $6\text{ percent}$  from the chlor-alkali industry.<sup>(8,9)</sup>

Mercury levels at ground surface are significantly higher than concentrations at higher altitudes.<sup>(2)</sup> Background concentrations are probably 0.000001 mg/m<sup>3</sup> or less.<sup>(8)</sup> Higher levels are found in urban or industrial areas and near mercury deposits and active volcanoes. The U.S. Environmental Protection Agency has estimated rural concentrations of mercury in air to be 0.000005 mg/m<sup>3</sup>, urban concentrations 0.00003 mg/m<sup>3</sup>, and indoor concentrations 0.0001 to 0.0002 mg/m<sup>3</sup>; the average atmospheric concentration was estimated at 0.00002 mg/m<sup>3</sup>, and it was stated that atmospheric concentrations are unlikely to exceed an average value of 0.00005 mg/m<sup>3</sup>.<sup>(10-12)</sup> In 1972, four chlor-alkali plants located in Quebec and New Brunswick were studied, and the average downwind mercury concentration was found to be 0.0025 to 0.25 mg/m<sup>3</sup>.<sup>(13)</sup> Mercury concentrations in random air sample taken in an industrial area of Quebec were found to range from 0.0000379 mg/m<sup>3</sup> in Noranda to 0.000835 mg/m<sup>3</sup> at Lebel.<sup>(14)</sup>

Mercury in air can be washed out by rain. In industrial areas, mercury concentrations as high as 0.0002 mg/L have been reported in rain. An estimated 0.06 to 0.4 mg mercury is deposited on each square metre of soil from precipitation.<sup>(15)</sup>

In general, an equilibrium is established between Hg<sup>0</sup>, Hg<sup>2+</sup>, and Hg<sup>2+</sup> in aqueous solution. The distribution of mercury between the three oxidation states is determined by the redox potential, pH, and the anions present. Under laboratory conditions, metallic mercury is slightly soluble in water (about 0.025 mg/L) at 20°C. In oxygenated water, the overall solubility increases as Hg(OH)<sub>2</sub> forms. Chloride-rich, acidic water favours the formation of undissociated HgCl<sub>2</sub> (slightly soluble in water), and the total mercury load in solution is thus increased. In most surface waters, Hg(OH)<sub>2</sub> and HgCl<sub>2</sub> are the predominant mercury species. In reducing sediments, however, most of the mercury is immobilized as the sulphide.<sup>(7,16)</sup> Concentrations of mercury in surface and drinking waters are generally below 0.001 mg/L.<sup>(17)</sup> The presence of higher levels of mercury in water is due to effluents from the chlor-alkali industry, the pulp and paper industry, mining, gold, and other ore-recovering processes, and irrigation or drainage of areas in which agricultural pesticides are used. Prior to 1960, as much as 90 kg/day was lost to water by a chlor-alkali plant in Sarnia, Ontario.<sup>(18)</sup> In the pulp and paper industry in Canada, an estimated 5 to 20 percent of mercurial fungicides reach the waterways.<sup>(3)</sup>

In the United States, 95.5 percent of 273 water samples were found to have mercury concentrations below 0.001 mg/L;<sup>(12)</sup> in polluted waters such as the St. Clair River near Windsor, Ontario, up to 0.03 mg/L has been reported.<sup>(19)</sup> Concentrations in Canadian surface water vary from area to area but are usually below

0.00025 mg/L and often near 0.00005 mg/L, although streams and rivers near mercury deposits may contain up to 0.1 mg/L.<sup>(20,21)</sup> In the Great Lakes, mean mercury concentrations were 0.017 mg/L for lakes Erie and Huron, 0.013 mg/L for Lake Ontario, and 0.018 mg/L for Lake Superior. Concentrations ranged from non-detectable to 0.040 mg/L; high concentrations were usually detected near industrialized areas.<sup>(22)</sup> In a later study, the mercury concentration in Lake Michigan was found to be 0.003 mg/L.<sup>(23)</sup> In 1974, only six water quality stations in western Canada reported mean mercury concentrations above 0.02 mg/L. It has been reported that from 1971 to 1976 there was a decline in the mercury content of the waters of Alberta, Saskatchewan, and British Columbia. This has been attributed to the various regulations governing mercury emissions in Canada.<sup>(24)</sup> In the Atlantic provinces, surface water samples at 20 locations contained mercury in excess of 0.02 mg/L, and a special study on the status of mercury in these provinces is now under way.<sup>(25)</sup>

Some drinking water analyses for 1971 were reported in NAQUADAT.<sup>(17)</sup> Concentrations of extractable mercury ranged from non-detectable to 0.6 mg/L; the high value was most unusual and probably due to some error. Median concentrations in Alberta, Newfoundland, and Quebec were 0.0002, 0.00015, and 0.00029 mg/L, respectively.

Inorganic mercury in sediments, under anaerobic conditions, can be transformed by micro-organisms into organic mercury compounds, the most common of which is methyl mercury.<sup>(26,27)</sup> These compounds can readily associate with suspended and organic matter and be taken up by aquatic organisms.<sup>(28)</sup> Methyl mercury has high affinity for lipids and is distributed to the fatty tissues of living organisms. Although methyl mercury is estimated to constitute only 1 percent of the total mercury content of water, more than 90 percent of the mercury in biota is in the form of methyl mercury.<sup>(28)</sup> All organisms in water may be exposed to dissolved or suspended mercury, but methyl mercury is bioconcentrated via the limnic food chain, and the tissues of the top predators can contain mercury levels that render their flesh unacceptable for human consumption.<sup>(26,28)</sup>

Elevated mercury levels have been found in all freshwater fish taken from areas with suspected mercury contamination; the levels were highest in fish from Pinchi Lake, the St. Clair River, and Lake St. Clair, where the maximum concentrations in the muscles were 10.5, 7.09, and 5.01 mg/kg, respectively. In the Ottawa River, the muscle tissue of fish, collected 3 to 8 km downstream from a pulp mill, contained mercury at 2.73 mg/kg of tissue.<sup>(3)</sup> In one study of mercury in fish from Lake Erie, even the lowest reported tissue concentrations exceeded 0.5 mg/kg, the Canadian maximum acceptable level for mercury in fish;<sup>(29)</sup> however, in another study,

only two (white bass) of 78 fish samples contained more than 0.5 mg/kg.<sup>(30)</sup> In 1974, 30 species of Atlantic fish and shellfish were surveyed, and tissue mercury concentrations in the range of 0.005 to 1.53 mg/kg were found;<sup>(31)</sup> two species had mercury concentrations above 0.5 mg/kg: the American lobster (*Homarus americanus*) and the red crab (*Geryon quinquedens*). Methyl mercury concentrations in cod were reported to be in the 0.068 to 0.074 mg/kg range.<sup>(32)</sup>

In a Canadian study<sup>(33)</sup> of mercury residues in foods, fish, and wildlife, samples not suspected of contamination had mercury concentrations of 0.005 to 0.075 mg/kg; some specimens from areas of known mercury contamination had concentrations in excess of 1 mg/kg. Samples of fish, meat, grain, flour, and milk products that contained more than 0.15 mg/kg were suspected of mercury contamination.<sup>(33)</sup> In another study, only two of 545 food samples analysed had mercury concentrations above 0.10 mg/kg.<sup>(34)</sup> In 1973, samples of Canadian cured meat were found to have an average mercury concentration of 0.006 mg/kg.<sup>(35)</sup>

In 1975, a survey of fish from lakes in northwest Ontario found concentrations of 0.4 to 1.3 mg/kg in walleye and pike and 0.04 to 0.28 mg/kg in whitefish.<sup>(36)</sup> A more recent review of reported data from Quebec gave average concentrations in fish of 0.6 to 0.9 mg/kg, with upper concentrations of about 5 mg/kg.<sup>(37,38)</sup>

## Canadian Exposure

The average daily intake of mercury from food in Canada was estimated to be 0.02 mg per person in 1964<sup>(39)</sup> and 0.013 mg per person in 1974.<sup>(34)</sup> Analysis of representative diets in Vancouver and Halifax gave estimated daily mercury intakes of 0.02 and 0.01 mg per person; meat and fish contributed over 80 percent of the mercury intake.<sup>(40)</sup> These values agree well with the 0.01 mg daily intake figure (range, 0 to 0.019 mg) reported in 1973<sup>(41)</sup> and the 0.007 to 0.009 mg/day level found in Great Britain.<sup>(42)</sup> The daily mercury intake from food for Canadians is considered to be 0.013 mg. It is recognized, however, that higher levels may occur with diets containing a large proportion of fish or seafood. Mercury intake from food may be significantly higher for the residents of White Dog and Grassy Narrows communities, for example, because their diet contains large amounts of fish; fish caught in the Wabigoon–English River system had mercury concentrations up to 15 mg/kg.<sup>(43)</sup> Consequently, in Canada, a regulatory guideline of 0.5 mg/kg (wet weight) of mercury has been set for the edible portion of fish. From the foregoing considerations, the average daily mercury intake from air, food, and water is estimated to be less than 0.015 mg per person.

“Food basket” analyses have estimated the U.S. mean intake of mercury in diet (including drinking water) to be in the range 0.003 to 0.005 mg/day over the period 1978 to 1982.<sup>(44)</sup> An independent estimate in 1979 suggested that the mean daily intake in Canada was 0.005 to 0.01 mg, and in the United States 0.02 mg.<sup>(45)</sup>

## Treatment Technology

Iron coagulation, alum coagulation, and lime softening are only moderately effective in removing inorganic mercury from drinking water and quite ineffective in removing organic mercury. Ferric sulphate coagulation was found to be the most effective of the three processes, removing 97 percent of an initial inorganic mercury concentration of 0.05 mg/L at pH 8, but only 66 percent at pH 7. At the same pHs, alum coagulation removed 38 and 48 percent, respectively. Both methods were more effective with turbid water than with clear water. Lime softening removed 60 to 80 percent in the pH range 10.7 to 11.4, but only about 30 percent at pH 9.4. None of the three methods removed more than about 40 percent of organic mercury at the same initial concentration.<sup>(46)</sup>

Treatment with granular activated carbon has been found to be much more effective in removing both organic and inorganic mercury. The effectiveness of the method depends on the contact time, but removal of over 80 percent has been achieved with initial organic mercury concentrations of 0.02 to 0.03 mg/L and a contact time of 3 to 5 minutes. The method appears to be capable of removing over 95 percent of both organic and inorganic mercury.<sup>(46)</sup>

In preliminary studies, about 98 percent of organic and inorganic mercury has been removed from water by ion exchange.<sup>(46)</sup> Reverse osmosis may be capable of removing 80 percent of mercury in drinking water.<sup>(47)</sup>

## Health Considerations

### Absorption

Absorption of metallic mercury following ingestion is negligible;<sup>(48)</sup> less than 0.01 percent of an administered dose of metallic mercury was absorbed in animals, for example. In humans, accidental ingestion of several grams of metallic mercury increased blood mercury levels,<sup>(49)</sup> but only rarely did doses of 100 to 500 g cause clinical illness (stomatitis and diarrhoea).<sup>(50)</sup> Soluble inorganic mercury(II) salts are absorbed to a limited extent, 7 to 15 percent in humans,<sup>(51,52)</sup> and sparingly water-soluble mercury(I) salts are absorbed to an even lesser degree. The mercury(I) ion can be biotransformed to the mercury(II) ion *in vivo*, however.<sup>(53)</sup> Ingested organic mercury, on the other hand, is readily absorbed;<sup>(54)</sup> 95 percent or more is

absorbed in humans. Experiments with mice have shown that the amount of mercuric chloride absorbed is 38 percent at age one week and 7 percent in adults on a milk diet, compared with about 1 percent in adults on a normal diet.<sup>(55)</sup>

It is estimated that 80 percent of inhaled mercury is absorbed.<sup>(11)</sup> Absorption depends on particle size, solubility, and rate of decomposition of the salts in biological fluids. A fraction of inhaled mercury salts will be cleared to the alimentary tract and absorbed by ingestion. Generally, aerosols of inorganic mercury compounds are absorbed to a lesser degree than is mercury vapour.

Metallic mercury, inorganic mercury compounds, and alkyl mercurials are known to cross the skin barrier, but to what extent is unknown. As much as 5 percent of a 2 percent solution of mercuric chloride was absorbed through the intact skin of guinea pigs over a 5-hour period.<sup>(56,57)</sup> Mercury may also be absorbed through the cornea.<sup>(50)</sup>

### Distribution and Metabolism

Inorganic mercury compounds are rapidly accumulated by the kidney, the main target organ for these compounds. Mercury in the kidneys is in the form of a metallothionein-like complex. Binding of the mercury by the protein, metallothionein, is enhanced in the presence of cadmium. Phenyl and methoxyethyl mercuric salts rapidly degrade to mercuric salts and distribute as such in the bodies of men and animals. The toxicity of these organomercurials is dependent on the rate of their conversion (biotransformation) to inorganic mercury; because this conversion is rapid, the toxicity of these compounds in cases of chronic exposure is similar to that seen after inorganic mercury exposure.<sup>(58)</sup> Elemental mercury vapour that is inhaled rapidly diffuses through the alveolar membrane; in the body, it is oxidized to mercuric ions, which produce the toxic effects.

Absorption of methyl mercury from food (bound to protein) or water (as chloride salt) is almost complete both in animals and in man.<sup>(59)</sup> Methyl mercury has considerable stability in the body and circulates for a time unchanged in the blood. It is distributed in high concentrations to the kidney and somewhat less to liver. In the kidney, 40 percent is present in the inorganic form. The "critical" organ, however, is the brain, especially the calcarine cortical portion. Other brain structures, the spinal cord, and peripheral nerves are also affected. In human tracer experiments, 10 percent of the total body burden was found in the head, probably mainly in the brain, and about 5 to 10 percent in the blood<sup>(54)</sup> as unaltered methyl mercury.<sup>(60)</sup> In man, methyl mercury has a ratio of 20:1 between red blood cells and plasma in contrast to the 1:1 ratio after

exposure to inorganic or phenyl mercury.<sup>(12)</sup> The most reliable index of exposure to methyl mercury and of retention in the central nervous system is the finding of methyl mercury in red blood cells. Hair mercury levels reflect past exposure and are dependent on the rate of hair growth. There is an almost linear relationship between the amount of methyl mercury in blood and that in the hair that was formed during exposure; the ratio of hair to blood levels has been consistently found in the range 230 to 300:1.<sup>(12)</sup>

At steady state, the level of mercury in blood is proportional to the daily intake of methyl mercury; the constant of proportionality for a 70-kg adult has been estimated to be between 0.3 and 1.0 (units of days per litre).<sup>(38)</sup> It has been estimated that a concentration of 0.2 mg/L in blood corresponds to an intake of 0.3 mg mercury per day.<sup>(61)</sup>

### Excretion

Mercuric salts are excreted from the kidney, the liver, the intestinal mucosa, the sweat glands, and the salivary glands, and through milk; the most important routes of excretion are via the urine and faeces. The faecal route is dominant soon after an exposure, especially when the dose is large; about 50 percent of the mercury is excreted by this route. In rats, urinary excretion predominates two weeks after exposure and accounts for 70 percent of the total then being excreted. Whole-body measurements of mercuric salts in human subjects have shown a biological half-life of 30 to 60 days.<sup>(54)</sup>

Experimental clinical data have shown a close correlation between plasma concentration of mercury and urine excretion; the plasma concentration is dependent on the release of mercury from the body compartment as well as on recent absorption of mercury.<sup>(60)</sup> The ratio of mercury in red blood cells to that in plasma was found to be 0.4, with whole blood containing less than 1 percent of the whole-body burden 24 hours after the administration of labelled inorganic mercury salt.<sup>(62)</sup>

About 7 percent of inhaled mercury is exhaled. In workers exposed to mercury vapour, the output of mercury in urine slightly exceeded that in the faeces.<sup>(63)</sup> Urinary excretion may be used to evaluate recent exposure to the vapour. The urinary excretion of 0.1 to 0.3 mg of mercury generally corresponds to inhalation of air containing mercury at a concentration of 0.1 mg/m<sup>3</sup>.

Although mercury concentrations in the urine are of limited diagnostic value in individual cases, mercury concentrations above the normal value of 0.01 mg/L may serve as a supporting criterion for a mercury etiology in clinical diagnosis of the "asthenic-vegetative syndrome".<sup>(64)</sup>

Methyl mercury is excreted slowly and unevenly into the bile but is immediately reabsorbed across the intestinal wall back into the bloodstream.<sup>(65)</sup> Some methyl mercury is converted to mercury(II) ions in the intestine. Excretion in the faeces accounts for approximately 80 percent of the total excretion from the body, but enterohepatic recirculation is large compared with faecal excretion. Ten percent of methyl mercury is excreted in urine, and the remaining 10 percent is eliminated mainly in the hair and lungs. The total daily excretion amounts to about 1 percent of the total body burden. The biological half-life determined by human experiments using a single exposure is roughly 50 days; measurement in which a long-term exposure was interrupted indicated a half-life of roughly 70 days; in both cases there was a large spread in values. The half-life in the head (brain) may be slightly longer than that in the rest of the body.<sup>(12,38)</sup> No sex difference in body burden was noted.

### Toxicity

The appearance, character, and extent of the toxic effects of mercury depend on a number of factors: the chemical form of the mercury; the mercury compound and its ionization potential; the dose, duration of exposure, and the route of administration; and the dietary levels of interacting elements, especially selenium.<sup>(54)</sup>

The primary biochemical lesions associated with mercury poisoning have not yet been established. It is known, however, that mercury reacts with sulfhydryl groups in proteins; because almost all cell proteins contain sulfhydryl groups that are metal-reactive, mercury compounds have the potential to damage virtually every cell in the body. When given in acute massive doses, mercury, in whatever chemical form, will denature proteins, inactivate enzymes, and cause severe disruption of any tissue with which it comes into contact in sufficient concentration.

The two major responses to mercury poisoning involve neurological and renal disturbances. The former is characteristic of poisoning by methyl and ethyl mercuric salts, in which liver and renal damage are of relatively little significance. The latter is characteristic of inorganic mercurial poisoning.<sup>(58)</sup> In general, however, acute lethal toxic doses by ingestion of any form of mercury will result in the same terminal signs and symptoms, which consist of shock, cardiovascular collapse, acute renal failure, and severe gastrointestinal damage.

After acute administration of ionizable inorganic salts of mercury to animals or man, the highest levels of mercury are found in the kidney; although acute oral poisoning results primarily in haemorrhagic gastritis and colitis, the ultimate damage is to the kidney.<sup>(66)</sup> Clinical

symptoms of acute intoxication include pharyngitis, dysphagia, abdominal pain, nausea and vomiting, bloody diarrhoea, and shock. Later, swelling of the salivary glands, stomatitis, loosening of the teeth, nephritis, anuria, and hepatitis occur.<sup>(66)</sup> Ingestion of 500 mg mercuric chloride causes severe poisoning and sometimes death in man.<sup>(50)</sup> Acute exposure results from inhalation of air containing mercury vapour in the range 0.05 to 0.35 mg/m<sup>3</sup>.<sup>(42,67)</sup> Exposure for a few hours to a concentration of between 1 and 3 mg/m<sup>3</sup> may give rise to pulmonary irritation and destruction of lung tissue and occasionally central nervous system disorders.<sup>(54)</sup>

Chronic exposure occurs in persons occupationally exposed to large amounts of mercury on occasion and as a result of prolonged therapeutic use. Excessive long-term use of calomel (mercury[I] chloride) has caused systemic poisoning with symptoms of stomatitis and salivation in non-fatal cases,<sup>(54)</sup> and dementia, erethism, colitis, and renal failure in ultimately fatal incidents.<sup>(53)</sup> It is not clear whether mercury levels in the brain or testes reach toxic concentrations before renal damage (marked proximal tubular necrosis and calcinosis) occurs. Signs and symptoms of exposure to mercury vapour include objective tremors, mental disturbances (erethism), and gingivitis. The "asthenic-vegetative syndrome" or "micromercurialism" has been attributed to airborne concentrations below 0.1 mg/m<sup>3</sup>.<sup>(64)</sup>

Alkyl compounds of mercury are the most toxic to man, producing illness, irreversible neurological damage, or death from the ingestion of milligram quantities.<sup>(68)</sup> Outbreaks of poisonings by these organic derivatives have been the result of accidents or of environmental contamination in a number of countries — Iraq, Guatemala, Pakistan, Japan (Minamata and Niigata),<sup>(69)</sup> and the United States.<sup>(68)</sup> Persons who had consumed contaminated fish (Japan) or grain treated with alkyl mercurials as seed dressings (Guatemala, Iraq, Pakistan, and the United States) were poisoned; some died.

Symptoms may occur weeks or months after exposure to toxic concentrations of either methyl mercury or ethyl mercury. Therefore, no clear distinction between acute and chronic symptomatology can be made.

In animals, subacute doses of alkyl mercurials (which cause no neurological symptoms) cause reversible damage to liver and kidney. In animals and in man, larger doses cause irreversible damage to the central nervous system. Morphological damage precedes clinical symptoms. Dermal exposure to alkyl mercurials may give rise to acute toxic dermatitis and eczematous changes.<sup>(60)</sup>

In cases of severe poisoning, pronounced weight loss can occur with or without intestinal symptoms. Neurological symptoms include mental deterioration, rigidity and hyperkinesia, and salivation and sweating.<sup>(60)</sup> From epidemiological evidence in humans, the onset of the first detectable adverse health effects (neurological symptoms) due to methyl mercury is estimated to occur at a concentration in blood of 0.2 mg/L. The concentration in hair associated with this blood level is 0.05 mg/g.<sup>(38)</sup> The corresponding daily intake for a 70-kg adult is approximately 0.3 mg, and the total body burden 25 mg.

Blood levels in the foetus can be as much as a factor of 2 greater than maternal levels, and the foetus is more sensitive to methyl mercury toxicity than the adult. For these reasons, the threshold for adverse health effects in the foetus has been estimated to be a concentration of 0.05 mg/L in maternal blood,<sup>(38)</sup> corresponding to a daily intake of about 0.08 mg methyl mercury.

On the basis of animal experiments and a safety factor of 1000, the U.S. Environmental Protection Agency has calculated an acceptable daily intake of 0.01 mg inorganic mercury for a 70-kg adult.<sup>(12)</sup>

In cases of lethal poisoning, the concentration of mercury in the brain is 0.005 mg/g or more at onset; in the liver and kidney, it is 0.02 mg/g. Normal concentrations of mercury in blood are considered to be 0.000005 mg/g whole blood, and in hair, 0.01 mg/g. The maximum safe concentration limit for mercury in blood has been set at 0.0001 mg/g, but concentrations of 0.0005 mg/g and higher have been encountered without any evident clinical symptoms.

In animals, oestrogenic hormones and spironolactone<sup>(61)</sup> protect the kidney from methoxyethyl mercury.<sup>(70)</sup> Spironolactone also protects against mercuric chloride.<sup>(71)</sup> The presence of zinc, manganese, or cadmium in the diet may influence the gastrointestinal absorption of mercury.<sup>(72,73)</sup> In rats, the oral administration of zinc and mercuric chloride suppresses the toxicity of the mercuric ion.<sup>(74)</sup> Toxicity is also decreased by pretreatment with selenite ion;<sup>(75,76)</sup> however, this is not due to decreased mercury absorption or increased excretion.<sup>(77)</sup>

### Teratogenicity and Mutagenicity

Alkyl mercury readily crosses the placenta unchanged and concentrates in foetal tissues. As a result, infants born to exposed mothers may suffer from mental retardation, cerebral palsy, and convulsions. The foetus is far more sensitive to methyl mercury poisoning than is the child, and children under 10 years of age are more susceptible than adults.<sup>(78)</sup>

Alkyl mercurials are embryotoxic and teratogenic in laboratory animals; chromosome breakage has occurred in the lymphocytes of humans exposed to methyl

mercury.<sup>(78)</sup> Phenyl mercury has been shown to induce non-specific damage to the central nervous system of the mouse foetus; a similar effect has not been observed in man. Although methyl mercury acts on basic genetic systems like the spindle fibre mechanism and DNA, its mutagenic potential appears to be small.

No evidence for genetic, teratogenic, or carcinogenic effects has yet been described for inorganic mercury.

### Rationale

1. Mercury is a toxic element and serves no beneficial physiological function in man. As a result of industrial and agricultural applications, high levels of mercury may occur in localized areas of the environment. Alkyl derivatives of mercury are of the greatest concern because of their toxicity and because they are bioconcentrated. Mercury levels in freshwater fish, taken from areas with suspected mercury contamination, frequently render the fish unacceptable for human consumption.

2. From epidemiological data, it has been estimated that the onset of irreversible neurological symptoms is associated with a mercury concentration of 0.2 mg/L in blood. The corresponding daily intake for a 70-kg adult has been estimated at 0.3 mg. Application of a safety factor of 10 would give a tolerable daily intake of 0.03 mg of mercury as methyl mercury. Daily consumption of 1.5 L of water<sup>(61)</sup> containing mercury at a concentration of 0.001 mg/L would contribute approximately 5 percent of the tolerable intake.

3. The maximum acceptable concentration for mercury in drinking water is therefore 0.001 mg/L. This value applies to all possible forms of mercury in water.

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