IPCS International Programme on Chemical Safety

Environmental Health Criteria 86

Mercury — Environmental Aspects

Published under the joint sponsorship of the United Nations Environment Programme, the International Labour Organisation, and the World Health Organization

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The **International Programme on Chemical Safety (IPCS)** is a joint venture of the United Nations Environment Programme, the International Labour Organisation, and the World Health Organization. The main objective of the IPCS is to carry out and disseminate evaluations of the effects of chemicals on human health and the quality of the environment. Supporting activities include the development of epidemiological, experimental laboratory, and risk-assessment methods that could produce internationally comparable results, and the development of manpower in the field of toxicology. Other activities carried out by the IPCS include the development of know-how for coping with chemical accidents, coordination of laboratory testing and epidemiological studies, and promotion of research on the mechanisms of the biological action of chemicals.

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NOTE TO READERS OF THE CRITERIA DOCUMENTS

Every effort has been made to present information in the criteria documents as accurately as possible without unduly delaying their publication. In the interest of all users of the environmental health criteria documents, readers are kindly requested to communicate any errors that may have occurred to the Manager of the International Programme on Chemical Safety, World Health Organization, Geneva, Switzerland, in order that they may be included in corrigenda, which will appear in subsequent volumes.

* * *

A detailed data profile and a legal file can be obtained from the International Register of Potentially Toxic Chemicals, Palais des Nations, 1211 Geneva 10, Switzerland (Telephone no. 988400 - 985850).

ENVIRONMENTAL HEALTH CRITERIA FOR MERCURY - ENVIRONMENTAL ASPECTS

A WHO Task Group on Environmental Health Criteria for Mercury - Environmental Aspects met at the Institute of Terrestrial Ecology, Monks Wood, UK, from 7 to 11 December 1987. Dr B.N.K. Davis welcomed the participants on behalf of the host Institution, and Dr M. Gilbert opened the meeting on behalf of the three co-sponsoring organizations of the IPCS (ILO/UNEP/WHO). The Task Group reviewed and revised the draft criteria document and made an evaluation of the risks for the environment from exposure to mercury.

The first draft of this document was prepared by Dr S. Dobson and Mr P.D. Howe, Institute of Terrestrial Ecology. Dr M. Gilbert and Dr P.G. Jenkins, both members of the IPCS Central Unit, were responsible for the overall scientific content and editing, respectively.

* * *

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INTRODUCTION

There is a fundamental difference in approach between the toxicologist and the ecotoxicologist concerning the appraisal of the potential threat posed by chemicals. The toxicologist, because his concern is with human health and welfare, is preoccupied with any adverse effects on individuals, whether or not they have ultimate effects on performance or survival. The ecotoxicologist, in contrast, is concerned primarily with the maintenance of population levels of organisms in the environment. In toxicity tests, he is interested in effects on the performance of individuals - in their reproduction and survival - only insofar as these might ultimately affect the population size. To him, minor biochemical and physiological effects of toxicants are irrelevant if they do not, in turn, affect reproduction, growth, or survival.

It is the aim of this document to take the ecotoxicologist's point of view and consider effects on populations of organisms in the environment. No attempt has been made to link the conclusions reached in this document with possible effects on human health. This will only be feasible when Environmental Health Criteria 1: Mercury (WHO, 1976), which considered the effects of mercury on human health, has been updated. Due attention has been given to the persistence in the environment and the bioaccumulation and transport of mercury in aquatic food chains. These will have implications for human consumption of the metal.

This document, although based on a thorough survey of the literature, is not intended to be exhaustive in the material included. In order to keep the document concise, only those data which were considered to be essential in the evaluation of the risk posed by mercury to the environment have been included. Concentration figures for mercury in the environment, or in particular species of organism, have not been included unless they illustrate specific toxicological points. "Snap shot" concentration data, where a causal relationship between the presence of the metal and an observed effect is not clearly demonstrated, have been excluded.

The term bioaccumulation indicates that organisms take-up chemicals to a greater concentration than that found in their environment or their food. 'Bioconcentration factor' is a quantitative way of expressing bioaccumulation: the ratio of the concentration of the chemical in the organism to the concentration of the chemical in the environment or food. Biomagnification refers, in this document, to the progressive accumulation of chemicals along a food chain.

1. SUMMARY AND CONCLUSIONS

1.1 Physical and chemical Properties

Mercury is a metal which is liquid at normal temperatures and pressures. It forms salts in two ionic states mercury(I) and mercury(II). Mercury(II), or mercuric, salts are very much more common than mercury(I) salts, and hence it is mercuric salts which will be mainly considered here. Mercury also forms organometallic compounds, some of which have found industrial and agricultural use. "Organometallic" is used here to indicate a covalently-bonded compound, and does not include mercury bound to proteins nor salts formed with organic acids. These organometallic compounds are stable, though some are readily broken down by living organisms, while others are not readily biodegraded. Elemental mercury gives rise to a vapour which dissolves only slightly in water.

1.2 Sources in the Environment

Natural mercury arises from the degassing of the earth's crust through volcanic gases and, probably, by evaporation from the oceans. Local levels in water derived from mercury ores may also be high (up to 80 μg/litre). Atmospheric pollution from industrial production is probably low, but pollution of water by mine tailings is significant. The burning of fossil fuels is a source of mercury. The chloralkali industry and, previously, the wood pulping industry also released significant amounts of mercury. Although the use of mercury is reducing, high concentrations of the metal are still present in sediments associated with the industrial applications of mercury. Some mercury compounds have been used in agriculture, principally as fungicides.

1.3 Uptake, Elimination, and Accumulation in Organisms

Mercuric salts, and, to a much greater extent, organic mercury, are readily taken up by organisms in water. Aquatic invertebrates, and most particularly aquatic insects, accumulate mercury to high concentrations. Fish also take up the metal and retain it in tissues, principally as methylmercury, although most of the environmental mercury to which they are exposed is inorganic. The source of the methylation is uncertain, but there is strong indication that bacterial action leads to methylation in aquatic systems. Environmental levels of methylmercury depend upon the balance between bacterial methylation and demethylation. The indications are that methylmercury in fish arises from this bacterial methylation of inorganic mercury, either in the environment or in bacteria associated with fish gills, surface, or gut. There is little indication that fish themselves either methylate or demethylate mercury. Elimination of

methylmercury is slow from fish (with half times in the order of months or years) and from other aquatic organisms. Loss of inorganic mercury is more rapid and so most of the mercury in fish is retained in the form of methylmercury. Terrestrial organisms are also contaminated by mercury, with birds being the best studied. Sea birds and those feeding in estuaries are most contaminated. The form of retained mercury in birds is more variable and depends on species, organ, and geographical site.

1.4 Toxicity to Microorganisms

The metal is toxic to microorganisms. Inorganic mercury has been reported to have effects at concentrations of the metal in the culture medium of 5 μg/litre, and organomercury compounds at concentrations at least 10 times lower than this. Organomercury compounds have been used as fungicides. One factor affecting the toxicity of the organometal is the rate of uptake of the metal by cells. Mercury is bound to the cell walls or cell membranes of microorganisms, apparently to a limited number of binding sites. This means that effects are related to cell density as well as to the concentration of mercury in the substrate. These effects are often irreversible, and mercury at low concentrations represents a major hazard to microorganisms.

1.5 Toxicity to Aquatic Organisms

The organic forms of mercury are generally more toxic to aquatic organisms than the inorganic forms. Aquatic plants are affected by mercury in the water at concentrations approaching 1 mg/litre for inorganic mercury but at much lower concentrations of organic mercury. Aquatic invertebrates vary greatly in their susceptibility to mercury. Generally, larval stages are more sensitive than adults. The 96-h LC_{50}s vary between 33 and 400 μg/litre for freshwater fish and are higher for sea-water fish. However, organic mercury compounds are more toxic. Toxicity is affected by temperature, salinity, dissolved oxygen, and water hardness. A wide variety of physiological and biochemical abnormalities has been reported after fish have been exposed to sublethal concentrations of mercury, although the environmental significance of these effects is difficult to assess. Reproduction is also affected adversely by mercury.

1.6 Toxicity to Terrestrial Organisms

Plants are generally insensitive to the toxic effects of mercury compounds. Birds fed inorganic mercury show a reduction in food intake and consequent poor growth. Other, more subtle, effects on enzyme systems, cardiovascular function, blood parameters, the immune response, kidney function and structure, and behaviour have been reported. Organomercury compounds are more toxic for birds than are inorganic.

1.7 Effects of Mercury in the Field

Pollution of the sea with organomercury led to the death of fish and fish-eating birds in Japan. Except for this incident at Minamata, few follow-up studies of the effects of localised release have been conducted. The use of organomercury fungicides as seed dressings in Europe led to the deaths of large numbers of granivorous birds, together with birds of prey feeding on the corpses. Residues of mercury in birds' eggs have been associated with deaths of embryos in shell. The presence of organochlorine residues in the same birds and their eggs makes an accurate assessment of the effects of mercury difficult. It is, however, thought to be a contributing factor in the population decline of some species of raptors.

2. PHYSICAL AND CHEMICAL PROPERTIES

The physical and chemical properties of mercury have been detailed in Environmental Health Criteria 1: Mercury (WHO, 1976). The relevant chapter is summarized here.

Mercury can exist in a wide variety of physical and chemical states. The different chemical and physical forms of this element all have their intrinsic toxic properties and different applications in industry and agriculture, and require a separate assessment of risk.

Mercury, along with cadmium and zinc, falls into Group IIb of the Periodic Table. In addition to its elemental state, mercury exists in the mercury (I) and mercury (II) states in which the mercury atom has lost one and two electrons, respectively. The chemical compounds of mercury (II) are much more numerous than those of mercury (I).

In addition to simple salts, such as chloride, nitrate and sulfate, mercury (II) forms an important class of organometallic compounds. These are characterized by the attachment of mercury to either one or two carbon atoms to form compounds of the type RHgX and RHgR′ where are R and R′ represent the organic moiety. The most numerous are those of the type RHgX. X may be one of a variety of anions. The carbon-mercury bond is chemically stable. It is not split in water nor by weak acids or bases. The stability is not due to the high strength of the carbon-mercury bond but to the very low affinity of mercury for oxygen. The organic moiety, R, takes a variety of forms, some of the most common being the alkyl, the phenyl, and the methoxyethyl radicals. If the anion X is nitrate or sulfate, the compound tends to be "salt-like" having appreciable solubility in water; however, the chlorides are covalent, non-polar compounds that are more soluble in organic solvents than in water. From the toxicological standpoint, the most important of these organometallic compounds is the subclass of short-chain alkyl mercurials in which mercury is attached to the carbon atom of a methyl, ethyl, or propyl group.

3. SOURCES OF MERCURY IN THE ENVIRONMENT

The sources of mercury have been detailed in Environmental Health Criteria 1: Mercury (WHO, 1976). Relevant data are summarized here.

3.1 Natural and Anthropogenic Sources and Cycling

The major source of mercury is the natural degassing of the earth's crust and amounts to between 25 000 and 125 000 tonnes per year. Anthropogenic sources are probably less than natural sources. World production of mercury by mining and smelting was estimated at 10 000 tonnes per year in 1973 and has been increasing at an annual rate of about 2%. The chloralkali, electrical equipment, and paint industries are the largest consumers of mercury, accounting for about 55% of the total consumption. Mercury has a wide variety of other uses in industry, agriculture, military applications, medicine, and dentistry.

Several of man's activities, not directly related to mercury, account for substantial releases into the environment. These include the burning of fossil fuel, the production of steel, cement, and phosphate, and the smelting of metals from their sulfide ores.

Alkylmercury fungicides used as seed dressings are important original sources of mercury in terrestrial food chains, although the use of these materials has decreased considerably.

Two cycles are believed to be involved in the environmental transport and distribution of mercury. One is global in scope and involves the atmospheric circulation of elemental mercury vapour from sources on land to the oceans. However, the mercury content of the oceans is so large, at least 70 million tonnes, that the yearly increases in concentration due to deposition from the global cycle are not detectable.

The other cycle is local in scope and depends upon the methylation of inorganic mercury mainly from anthropogenic sources. Many steps in this cycle are still poorly understood, but it is believed to involve the atmospheric circulation of dimethylmercury formed by bacterial action.

3.2 Speciation

The following speciation among mercury compounds has been proposed by Lindquist et al. (1984), where V stands for volatile, R for water-soluble or particle-borne reactive species, and NR for non-reactive species (Hg^0 is elemental mercury):

V: Hg^0, $(CH_3)_2Hg$

R: Hg^{2+}, HgX_2, HgX_3^-, and HgX_4^{2-},
with X = OH^-, Cl^- and Br^-.
HgO on aerosol particles. Hg^{2+} complexes with organic acids.

NR: CH_3Hg^+, CH_3HgCl, CH_3HgOH and other organomercuric compounds, $Hg(CN)_2$. HgS and Hg^{2+} bound to sulfur in fragments of humic matter.

The main volatile form in air is elemental mercury but dimethylmercury may also occur (Slemr et al., 1981).

Uncharged complexes, such as $HgCl_2$, CH_3HgOH etc., occur in the gaseous phase, but are also relatively stable in fresh water (snow and rain as well as standing or flowing water). $HgCl_4^{2-}$ is the dominant form in sea water.

3.3 Levels in the Environment

The following data have been extracted from Lindquist et al. (1984) and are included here to indicate background levels of mercury in the environment. Considerable local variations can occur and local levels close to anthropogenic sources of mercury would be much higher.

Reliable data on mercury concentrations in the *air* are scarce. Recent information suggests a background level at about 2 ng/m^3 in the lower troposphere of the northern hemisphere and about 1 ng/m^3 in the southern hemisphere, at least over oceanic areas. In European areas remote from industrial sources, such as the rural parts of southern Sweden and Italy, concentrations most often lie in the range from 2 to 3 ng/m^3 in summer and from 3 to 4 ng/m^3 in winter (Brosset 1983, Ferrara et al., 1982). In urban air the concentrations could be higher.

Deposition with *precipitation* is a major factor in removing mercury from the atmosphere. The lowest concentrations of mercury in rain water, around 1 ng/litre, have been reported from a coastal site in Japan and from the islands of Samoa. Most other values reported lie in the range between 5 and 100 ng/litre.

Recent measurements of mercury in *aquatic systems* have given the following concentration ranges, which may be considered representative for dissolved mercury:

Open ocean	0.5-3 ng/litre
Coastal sea water	2-15 ng/litre
Rivers and lakes	1-3 ng/litre

Local variations from these values are considerable, especially in coastal sea water and in lakes and rivers where mercury associated with suspended material may also contribute to the total load.

The mercury content in minerals forming ordinary rock and *soils* is usually very low. The normal level in igneous rocks and minerals seems to be less than 50 µg/kg, and in many cases is less than 10 µg/ kg. Due to the strong binding of mercury to soil particles, including

organic matter, only small amounts of the metal are present in soil solution; reported averages range between 20 and 625 μg/kg soil.

Background levels in *sediments* are approximately the same as levels in unpolluted surface soils. Average concentrations in ocean sediments probably lie in the range between 20 and 100 μg/kg.

3.4 Methylation of Mercury

The methylation of inorganic mercury in the sediment of lakes, rivers and other waterways, as well as in the oceans, is a key step in the transport of mercury in aquatic food chains.

It was first demonstrated by Jensen & Jernelov (1967) that microorganisms in lake sediments could methylate mercury. They later showed that the degree of methylation correlated well with the overall microbial activity in the sediment (Jensen & Jernelov, 1969). Detailed mechanisms of methylation in microorganisms have been proposed by Wood (1971) and Landner (1971). Some soil organisms capable of methylating mercury have also been isolated (Kitamura et al., 1969; Yamada & Tonamura, 1972).

The following general conclusions have been drawn by Bisogni & Lawrence (1973) concerning methylation by microorganisms:

(a) mono-methylmercury is the predominant product of biological methylation near neutral pH,
(b) the rate of methylation is greater under oxidising conditions than under anaerobic conditions,
(c) the output of methylmercury doubles for a ten-fold increase in inorganic mercury,
(d) temperature affects methylation as a result of its effect on overall microbial activity,
(e) higher microbial growth rate increases mercury methylation,
(f) methylation rates are inhibited by the addition of sulfide to anaerobic systems.

The formation of new or enlarged artificial lakes considerably increases the production of methylmercury, although this increase was found to be short-lived in new lakes in Finland (Simola & Lodenius, 1982; Alfthan et al., 1983). A similar problem of increased mercury in new lakes, which was taken up by fish and fish-eating mammals, occurred in the scheme to divert the Churchill River in Manitoba, Canada (Canada-Manitoba, 1987). Methylation rates in one lake, which had been flooded 20 years previously, had returned to normal. Methylation rates in the new lake, which had flooded arboreal forest, were high and were expected to remain high for decades. The source of mercury in all of these artificial lakes appeared to be natural rather than anthropogenic in origin. Anaerobic conditions after the flooding of large amounts of organic material and the subsequent increase in microbial activity are thought to be the causes of the increased availability of mercury through methylation.

4. UPTAKE, LOSS, AND ACCUMULATION IN ORGANISMS

Background levels of naturally-occuring mercury in the environment are generally low, except in the immediate vicinity of mining sites and chloralkali plants for the industrial extraction of mercury. The majority of mercury in the environment is natural rather than the result of human activities. Inorganic mercury can be methylated in the environment, and the resultant methylmercury is taken up into organisms readily; more readily than inorganic mercury. Although environmental levels are low, the high capacity of organisms to accumulate mercury means that the metal is found widely in both aquatic and terrestrial animals and plants. Methylmercury is released more slowly by aquatic organisms than inorganic mercury. Aquatic invertebrates, and particularly aquatic insects, accumulate mercury to a greater extent than fish.

Speciation of mercury is of great importance in determining the uptake of the metal from water and soil. Much of the mercury in natural waters and in soil is strongly bound to sediment or organic material and is unavailable to organisms.

Mercury has been found in many terrestrial organisms, birds being the subjects of most of the monitoring.

In many experimental studies, the concentrations of mercury quoted are nominal rather than measured. Few attempts have been made to estimate available mercury in experimental studies.

Because of the very extensive literature on the uptake of metals into organisms, this section contains illustrative examples and is not exhaustive.

Bioconcentration factors for mercury, determined in laboratory experiments, are summarized in Tables 1 and 2.

Bioconcentration factors are simple ratios between the concentration of mercury in an organism and the concentration in the medium to which the organism was exposed. This means that results should be treated with caution. A relatively low body burden resulting from exposure to very low levels of mercury in the medium can give a high bioconcentration factor. Conversely, exposure to very high mercury levels in the medium can lead to a low bioconcentration factor. Exposure to mercury under static test conditions will lead to the removal of mercury during the course of the test, whereas flow-through conditions maintain a constant level of exposure. Since mercury is strongly bound to sediment in the field, it is unclear which of these two exposure regimes is the most realistic. It is probable that static exposure underestimates and flow-through exposure overestimates mercury uptake. Most studies have failed to distinguish between mercury taken into the tissues of the organism and mercury adsorbed on external surfaces. This should also be taken into account when interpreting results.

Table 1. Accumulation of mercury into aquatic organisms

Organism	Life-stage[c]	Stat/flow[d]	Organ[b]	Temperature (°C)	Compound[e]	Duration (days)	Exposure (µg/litre)	Bioconcentration factor[g]	Reference
Alga *(Croomonas salina)*					mercuric chloride	2	164	8537[h]	Parrish & Carr (1976)
Filamentous algae *(Oedogonium sp.)*		stat			phenyl mercuric acetate	35	10	1200[f]	Hannerz (1968)
		flow		16.5	methoxyethylmercuric OH	18	0.58	2610[f]	Hannerz (1968)
		flow		15.2	mercuric chloride	54	0.05	871[f]	Hannerz (1968)
Duckweed *(Lemna minor)*		flow			methylmercuric OH	32	3	2950[f]	Hannerz (1968)
		flow		16.5	methoxyethylmercuric OH	24	0.58	480[f]	Hannerz (1968)
		flow		15.2	mercuric chloride	41	0.05	70[f]	Hannerz (1968)
Water hyacinth *(Eichhornia crassipes)*	mature	stat	roots	23-27	mercuric chloride	16	1000	580	Muramoto & Oki (1983)
Reed *(Phragmites communis)*		stat	emergent		phenyl mercuric acetate	35	10	0[f]	Hannerz (1968)
		stat	submerged		phenyl mercuric acetate	35	10	850[f]	Hannerz (1968)
		flow	emergent		methylmercuric OH	32	3	25[f]	Hannerz (1968)
		flow	submerged		methylmercuric OH	32	3	530[f]	Hannerz (1968)
		flow	emergent	16.5	methoxyethylmercuric OH	24	0.58	74[f]	Hannerz (1968)
		flow	submerged	16.5	methoxyethylmercuric OH	24	0.58	139[f]	Hannerz (1968)
		flow	emergent	15.2	mercuric chloride	41	0.05	56[f]	Hannerz (1968)
		flow	submerged	15.2	mercuric chloride	14	0.05	149[f]	Hannerz (1968)
Bulrush *(Scirpus lucustris)*		stat	emergent		phenyl mercuric acetate	35	10	90[f]	Hannerz (1968)
		stat	submerged		phenyl mercuric acetate	35	10	790[f]	Hannerz (1968)
		flow	emergent		methylmercuric OH	32	3	8[f]	Hannerz (1968)
		flow	submerged		methylmercuric OH	32	3	1250[f]	Hannerz (1968)
		flow	emergent	16.5	methoxyethylmercuric OH	24	0.58	39[f]	Hannerz (1968)
		flow	submerged	16.5	methoxyethylmercuric OH	24	0.58	190[f]	Hannerz (1968)
		flow	emergent	15.2	mercuric chloride	21	0.05	77[f]	Hannerz (1968)
		flow	submerged	15.2	mercuric chloride	41	0.05	70[f]	Hannerz (1968)

Table 1 (Contd).

Organism	Life-stage[c]	Stat/flow[a]	Organ[b]	Temperature (°C)	Compound[e]	Duration (days)	Exposure (µg/litre)	Bioconcentration factor[g]	Reference
Yellow iris		stat	emergent		phenyl mercuric acetate	35	10	20[f]	Hannerz (1968)
(Iris pseudacorus)		stat	submerged		phenyl mercuric acetate	35	10	40[f]	Hannerz (1968)
		flow	emergent		methylmercuric OH	32	3	18[f]	Hannerz (1968)
		flow	submerged		methylmercuric OH	32	3	34[f]	Hannerz (1968)
		flow	emergent	16.5	methoxyethylmercuric OH	18	0.58	31[f]	Hannerz (1968)
		flow	submerged	16.5	methoxyethylmercuric OH	18	0.58	90[f]	Hannerz (1968)
		flow	emergent	15.2	mercuric chloride	49	0.05	18[f]	Hannerz (1968)
		flow	submerged	15.2	mercuric chloride	49	0.05	23[f]	Hannerz (1968)
Bloodworm		stat			phenyl mercuric acetate	35	10	12 700[f]	Hannerz (1968)
(Chironomidae)		flow			methylmercuric OH	32	3	3070[f]	Hannerz (1968)
		flow		16.5	methoxyethylmercuric OH	89	0.58	988[f]	Hannerz (1968)
Annelid		stat			phenyl mercuric acetate	35	10	2030[f]	Hannerz (1968)
(Haemopis sanguisuga)		flow			methylmercuric OH	32	3	450[f]	Hannerz (1968)
		flow		16.5	methoxyethylmercuric OH	89	0.58	1148[f]	Hannerz (1968)
Annelid		flow			methylmercuric OH	32	3	110[f]	Hannerz (1968)
(Glossosiphonia complanata)		flow		16.5	methoxyethylmercuric OH	89	0.58	640[f]	Hannerz (1968)
		flow		15.2	mercuric chloride	65	0.05	670[f]	Hannerz (1968)
Worm		flow			methylmercuric OH	32	3	1780[f]	Hannerz (1968)
(Oligochaeta)		flow		16.5	methoxyethylmercuric OH	65	0.58	690[f]	Hannerz (1968)
		flow		15.2	mercuric chloride	65	0.05	517[f]	Hannerz (1968)
Freshwater leech *(Herpobdella octoculata)*		flow		15.2	mercuric chloride	65	0.05	534[f]	Hannerz (1968)

Table 1 (Contd).

Mussel		WB		mercuric chloride	4	50	664	Tsuruga (1963)
(Mytilus edulis)	flow	WB			4	0.06	236[f]	Hannerz (1968)
Short-necked clam		WB		mercuric chloride	8	50	190	Tsuruga (1963)
(Venerupis philippinarum)								
Pond snail	stat			phenyl mercuric acetate	35	10	1280[f]	Hannerz (1968)
(Planorbis sp.)	flow			methylmercuric OH	32	3	3570[f]	Hannerz (1968)
	flow		16.5	methoxyethylmercuric OH	31	0.58	1970[f]	Hannerz (1968)
	flow		15.2	mercuric chloride	49	0.05	795[f]	Hannerz (1968)
Giant pond snail	stat			phenyl mercuric acetate	35	10	1800[f]	Hannerz (1968)
(Lymnaea stagnalis)	flow			methylmercuric OH	32	3	3480[f]	Hannerz (1968)
	flow		16.5	methoxyethylmercuric OH	31	0.58	1178[f]	Hannerz (1968)
	flow		15.2	mercuric chloride	14	0.05	297[f]	Hannerz (1968)
Snail	flow		16.5	methoxyethylmercuric OH	24	0.58	4266[f]	Hannerz (1968)
(Physa fontinalis)	flow		15.2	mercuric chloride	14	0.05	637[f]	Hannerz (1968)
Water flea	stat			phenyl mercuric acetate	35	10	3570[f]	Hannerz (1968)
(Daphnia sp.)								
Cladoceran	flow		16.5	methoxyethylmercuric OH	89	0.58	286[f]	Hannerz (1968)
(Eurycerus)								
Copepod	stat	WB	21-26	mercuric chloride	1	0.1	7600	Hirota et al. (1983)
(Acartia clausi)								
	stat	WB	21-26	methyl mercuric chloride	1	0.1	249 000	Hirota et al. (1983)
Grass shrimp		WB		mercuric chloride	3	1.5	333	Ray & Tripp (1976)
(Palaemonetes pugio)								

Table 1 (Contd).

Organism	Life-stage[c]	Stat/flow[a]	Organ[b]	Temperature (°C)	Compound[e]	Duration (days)	Exposure (µg/litre)	Bioconcentration factor[g]	Reference
Mayfly	naiad	stat			phenyl mercuric acetate	35	10	900[f]	Hannerz (1968)
(*Ephemeridae*)	naiad	flow			methylmercuric OH	32	3	3290[f]	Hannerz (1968)
	naiad	flow		16.5	methoxyethylmercuric OH	24	0.58	680[f]	Hannerz (1968)
	larva	flow		15.2	mercuric chloride	65	0.05	138[f]	Hannerz (1968)
Lesser water boatman		stat			phenyl mercuric acetate	35	10	4200[f]	Hannerz (1968)
(*Corixa sp.*)		flow			methylmercuric OH	32	3	8470[f]	Hannerz (1968)
		flow		16.5	methoxyethylmercuric OH	89	0.58	740[f]	Hannerz (1968)
		flow		15.2	mercuric chloride	65	0.05	414[f]	Hannerz (1968)
Water boatman		flow			methylmercuric OH	32	3	2460[f]	Hannerz (1968)
(*Notonecta glaaca*)		flow		16.5	methoxyethylmercuric OH	89	0.58	674[f]	Hannerz (1968)
		flow		15.2	mercuric chloride	65	0.05	483[f]	Hannerz (1968)
Midge (*Chironomus riparius*)	larva	flow	WB		mercuric chloride	30	5.5	19 600	Rossaro et al. (1986)
	pupa	flow	WB		mercuric chloride	30	5.5	15 600	Rossaro et al. (1986)
	adult	flow	WB		mercuric chloride	30	5.5	7500	Rossaro et al. (1986)
Caddisfly	larva	flow		16.5	methoxyethylmercuric OH	89	0.58	710[f]	Hannerz (1968)
(*Trichoptera sp.*)	larva	flow		15.2	mercuric chloride	49	0.05	513[f]	Hannerz (1968)
Dragonfly (*Odonata sp.*)	nymph	flow		16.5	methoxyethylmercuric OH	89	0.58	1296[f]	Hannerz (1968)
Damselfly	nymph	flow		16.5	methoxyethylmercuric OH	89	0.58	1186[f]	Hannerz (1968)
(*Odonata sp.*)	nymph	flow		15.2	mercuric chloride	65	0.05	655[f]	Hannerz (1968)

Table 1 (Contd).

Alderfly (*Sialis lutaria*)	larva	flow		16.5	methoxyethylmercuric OH	89	0.58	1270[f]	Hannerz (1968)
Cranefly (*Tipula sp.*)	larva	flow		16.5	methoxyethylmercuric OH	18	0.58	625[f]	Hannerz (1968)
	larva	flow		15.2	mercuric chloride	41	0.05	840[f]	Hannerz (1968)
Great diving beetle (*Dytiscus marginalis*)	imago	flow		16.5	methoxyethylmercuric OH	89	0.58	800[f]	Hannerz (1968)
	larva	flow		16.5	methoxyethylmercuric OH	89	0.58	3134[f]	Hannerz (1968)
	larva	flow		15.2	mercuric chloride	65	0.05	603[f]	Hannerz (1968)
	imago	flow		15.2	mercuric chloride	65	0.05	862[f]	Hannerz (1968)
Pond skater (*Gerris najas*)		flow		16.5	methoxyethylmercuric OH	89	0.58	754[f]	Hannerz (1968)
		flow		15.2	mercuric chloride	65	0.05	431[f]	Hannerz (1968)
Aquatic saw bug (*Asellus aquaticus*)		flow		16.5	methoxyethylmercuric OH	89	0.58	954[f]	Hannerz (1968)
Water spiders (*Hydracnidae*)		flow		16.5	methoxyethylmercuric OH	89	0.58	624[f]	Hannerz (1968)
Pike (*Esox lucius*)		stat	liver	17.2	methoxyethylmercuric OH	10	0.4	7673[f]	Hannerz (1968)
		stat	kidney	17.2	methoxyethylmercuric OH	10	0.4	7230[f]	Hannerz (1968)
		stat	liver		methylmercuric OH	10	0.3	2002[f]	Hannerz (1968)
		stat	kidney		methylmercuric OH	10	0.3	2198[f]	Hannerz (1968)

Table 1 (Contd).

Organism	Life-stage[c]	Stat/flow[a]	Organ[b]	Temperature (°C)	Compound[e]	Duration (days)	Exposure (µg/litre)	Bioconcentration factor[g]	Reference
Rainbow trout	juv	flow	WB	5	methyl mercuric chloride	84	0.263	4525	Reinert et al. (1974)
(Salmo gairdneri)	juv	flow	WB	10	methyl mercuric chloride	84	0.258	6628	Reinert et al. (1974)
	juv	flow	WB	15	methyl mercuric chloride	84	0.244	8033	Reinert et al. (1974)
			WB[d]	5	mercuric chloride	4	50	5	MacLeod & Pessah (1973)
			WB[d]	10	mercuric chloride	4	50	12	MacLeod & Pessah (1973)
			WB[d]	20	mercuric chloride	4	50	26	MacLeod & Pessah (1973)
Bluegill sunfish		stat	WB	9	methyl mercuric chloride	28.6	0.5	222[f]	Cember et al. (1978)
(Lepomis macrochirus)		stat	WB	21	methyl mercuric chloride	28.6	0.5	1138[f]	Cember et al. (1978)
		stat	WB	33	methyl mercuric chloride	28.6	0.5	2454[f]	Cember et al. (1978)

a stat = static conditions (water unchanged for duration of experiment); flow = flow-through conditions (mercury concentration in water continuously maintained).
b WB = whole body.
c juv = juvenile.
d muscle, skin & bone.
e OH = hydroxide.
f radiometrically calculated.
g bioconcentration factor = concentration in organism/concentration in medium.
h dry weight.

Table 2. Accumulation of mercury into terrestrial organisms

Organism	Route	Organ	Compound	Duration (days)	Exposure[b] (mg/kg)	Bioconcentration factor[c]	Reference
Broccoli	soil	leaves	mercuric chloride	60	20	0.002[d]	John (1972)
(Brassica oleracea)	soil	roots	mercuric chloride	60	20	0.09[d]	John (1972)
Pea	soil	roots	mercuric chloride	95	20	0.07[d]	John (1972)
(Pisum sativum)							
Cauliflower	soil	leaves	mercuric chloride	70	20	0.003[d]	John (1972)
(Brassica oleracea)	soil	roots	mercuric chloride	70	20	0.12[d]	John (1972)
Spinach	soil	leaves	mercuric chloride	55	20	0.03[d]	John (1972)
(Spinacia oleracea)	soil	roots	mercuric chloride	55	20	0.05[d]	John (1972)
Chicken	diet	muscle	methyl mercury dicyandiamide	35-42	8	1.25	Borg et al. (1970)
	diet	liver	methyl mercury dicyandiamide	35-42	8	5	Borg et al. (1970)
Mallard	diet	liver	methyl mercury dicyandiamide	14	8	2.1	Stickel et al. (1977)
(Anas platyrhynchos)	diet	kidney	methyl mercury dicyandiamide	14	8	2.2	Stickel et al. (1977)

Table 2 (Contd).

Organism	Route	Organ	Compound	Duration (days)	Exposure[b] (mg/kg)	Bioconcentration factor[c]	Reference
Redwinged blackbird	diet	liver	methyl mercury dicyandiamide	11	40	2.3	Finley et al. (1979)
(Agelaius phoeniceus	diet	kidney	methyl mercury dicyandiamide	11	40	2.1	Finley et al. (1979)
Cowbird	diet	liver	methyl mercury dicyandiamide	11	40	1.7	Finley et al. (1979)
(Molothrus ater)	diet	kidney	methyl mercury dicyandiamide	11	40	1.5	Finley et al. (1979)
Grackle	diet	liver	methyl mercury dicyandiamide	11	40	1.3	Finley et al. (1979)
(Quiscalus quiscula)	diet	kidney	methyl mercury dicyandiamide	11	40	1.1	Finley et al. (1979)
Mink	diet	liver	ceresan L[a]	5	32	11.1	Aulerich et al.(1974)
(Mustela vision)	diet	kidney	ceresan L[a]	5	32	7.4	Aulerich et al.(1974)
(adult)	diet	liver	mercuric chloride	10	135	0.3	Aulerich et al.(1974)
	diet	kidney	mercuric chloride	10	135	3.2	Aulerich et al.(1974)

[a] ceresan L = methylmercury 2,3-di-hydroxy propyl mercaptide + methylmercury acetate.
[b] exposure as mg/kg of mercury soil or diet according to route.
[c] concentration factors calculated on a wet weight basis unless otherwise stated; bioconcentration factor = concentration in organism/concentration in medium.
[d] dry weight.

Taking these factors into account, it is still clear that organisms take up both inorganic and organic forms of mercury from the medium. This uptake can result in high concentration factors. Under identical conditions, organic mercury is taken up by organisms to a greater degree than inorganic mercury, although the latter may often be strongly adsorbed to the outer surfaces.

4.1 Speciation of Mercury

Appraisal

Different species of mercury differ greatly in their physico-chemical properties: in their solubility, rates of accumulation by organisms, and behaviour in ecosystems. It is in its methyl form that mercury is most hazardous. Although not all sites of methylation in the environment are fully known, several have been identified in the aquatic environment.

Mercury accumulated in the tissues of fish is usually in the form of methylmercury, while the source is usually inorganic mercury (Huckabee et al., 1979). Several hypotheses of how and where methylation occurs have been proposed. The main hypotheses are:

(a) biological methylation, bacterial in origin, which produces methylmercury in the environment (methylmercury is taken up by fish more readily than inorganic mercury),
(b) methylation by microorganisms associated with branchial mucus of the fish or in the fish gut, and
(c) methylation in the fish's liver (Thellen et al, 1981).

It is generally agreed that methylation by fish, other than by bacteria associated with the fish, either does not occur or accounts for only an insignificant amount of the methylmercury produced. There is good evidence for methylation by bacteria in aquatic systems.

Jernelov (1968) suggested that fish could not methylate mercury themselves and this is generally accepted (Huckabee et al., 1979), though not universally. Jernelov & Lann (1971) showed that 60% of the mercury content of predator fish (northern pike) arose from prey fish. This mercury was already methylated in the prey. The concentration of mercury in predator species was similar to that in their prey. They also measured the mercury content of organisms that were the food of the prey fish. Mercury levels in benthic fauna were very low and contributed less than 25% of the mercury in bottom-feeding fish. Most of the mercury accumulated by non-predator species was, therefore, accumulated directly from water. This conclusion was also reached by Fagerstrom & Asell (1973). The question of where the methylation, which gives rise to methylmercury residues in fish, occurs is still unresolved. It is also generally accepted that fish do not demethylate mercury either.

4.2 Uptake and Loss in Aquatic Organisms

Appraisal

The data presented on uptake by aquatic invertebrates are difficult to interpret because most studies do not differentiate between external adsorption and actual uptake into the organism. This is especially important for methylmercury compounds for which uptake seems to be correlated with surface adsorption capacity, as expressed by the relative size of the organism.

The extrapolation of data on uptake to other organisms appears risky because of a lack of knowledge regarding the mechanisms of uptake. This is even true for phenomena that are apparently fairly universal, e.g., the facilitating influence of chelators upon uptake.

Most data on uptake by fish support the notion that uptake correlates positively with available concentration, with exposure time, and with temperature, although hardly any investigation differentiates between nominal and available concentrations. The importance of this distinction seems to be illustrated by the positive influence of lowered pH upon uptake.

None of the studies address the problem of distinguishing between adsorption to gills and slime on the one hand and real uptake into the body on the other. Studies of mercury distribution between organs are valuable for the potential effects of the total body burden, but they give no reliable insight into the time-dependent process of accumulation.

Data consistently show a higher uptake of methylmercury than of inorganic mercury. However, other organic mercury compounds exhibit a lower uptake, since they are adsorbed to a lesser extent.

4.2.1 Microorganisms, plants, and invertebrates

When Glooschenko (1969) exposed the marine diatom *Chaetoceros costatum* to labelled mercury, he found no difference between uptake in the light or the dark in non-dividing cells. Dead cells took up twice as much mercury as living cells, presumably by surface adsorption. As dividing cells in the light accumulated the labelled mercury for longer than non-dividing cells, the author suggested the possibility of some active uptake.

Hannerz (1968) demonstrated that there was no appreciable assimilation of mercury into the tissues of aquatic plants. Although concentrations were 10-20 times higher in submerged parts compared to emergent parts, this was attributed to surface adsorption differences. De et al. (1985) grew the plant *Pistia stratiotes* in nutrient solution to which mercuric chloride had been added at concentrations ranging from 0.05 to 20 mg/litre. They found that uptake gradually increased with an increase in the mercury concentration. Maximum accumulation occurred within one day. Maximum removal (approximately 90%) was recorded at 6 mg/litre or less, only 20% being lost from plants

receiving the highest concentration. Mercury accumulation into the roots was about 4 times higher than into the shoots at lower concentrations and about twice as high at 20 mg/litre.

Zubarik & O'Connor (1977) studied the accumulation of mercury in aquatic organisms from the Hudson River, USA. The organisms were maintained in filtered river water that contained mercury concentrations of < 0.1 µg/litre (less than levels normally found in the Hudson River). Planktonic organisms were exposed to various forms of labelled mercury, and the concentration factors after 24 h ranged from 10^2 to 10^6. Mercury uptake was greater in microplankton and algae than in macroplankton and fish larvae. An amphipod *(Gammarus sp.)* was exposed for one day to each of four types of mercury, two organic (phenylmercuric acetate and methylmercury chloride) and two inorganic (mercurous nitrate and mercuric chloride). No differences in uptake were found, but when the amphipod was exposed for a week the organic forms were accumulated to 3 times the concentration of the inorganic forms.

Riisgard et al. (1985) transferred mussels *(Mytilus edulis)* from clean water to an area chronically polluted with mercury. The mussels accumulated mercury readily during 3 months of exposure. They were then transferred to clean water in the laboratory and the elimination of the mercury was measured. The biological half-life was 293 days, but was only 53 days in the case of mussels contaminated by a temporary massive mercury contamination. In both cases, 75% of the mercury in the mussels was inorganic, but both inorganic and organic species were immobilized in the mussels from the chronically polluted area. In another study, only 6% of the total mercury in *Macoma balthica*, a sediment-feeding bivalve, was methylated, a much lower percentage than in *Mytilus* from the same area.

Hirota et al. (1983) exposed the copepod *Acartia clausi* to inorganic (mercuric chloride) and organic (methylmercury chloride) mercury at concentrations of 0.05-0.5 µg/litre for 24 h. The bioconcentration factor for inorganic mercury was nearly constant (approximately 7500), regardless of the mercury concentration in the water or the density of the copepods. In contrast, the concentration factor of methylmercury fluctuated, showing an inverse relationship with density but no relationship with the mercury concentration in the water.

DeFreitas et al. (1981) found a net assimilation of 70%-80% for methylmercury and 38% for inorganic mercury when fed in the diet to the shrimp *Hyalella azteca*. From water, inorganic mercury was assimilated 2 to 3 times more slowly than methylmercury. Khayrallah (1985) found that the accumulation of ethylmercuric chloride was almost twice as rapid as that of mercuric chloride in the amphipod *Bathyporeia pilosa*, although death occurred at similar levels of mercury.

Ray & Tripp (1976) exposed the grass shrimp *(Palaemonetes pugio)* to radioactively labelled methylmercury chloride and mercuric chloride for 24 and 72 h. After 24 h, the methylated form was mostly concentrated in the ventral nerve cord and to a lesser extent in the

gills. The reverse was true for mercuric chloride. The concentrations of mercury accumulated in the other tissues (exoskeleton, foregut, and remainder) were similar for both compounds, and were in decreasing order of the above list. After 72 h the tissue distribution had changed, and there was no consistent order of the relative tissue concentrations. There was an increase in the mercury levels of the exoskeleton, foregut, and remainder tissues, while that in the gills remained about the same and that in the ventral nerve cord decreased.

Vernberg & O'Hara (1972) measured the uptake of labelled mercury into the gills and hepatopancreas of fiddler crabs *(Uca pugilator)* maintained in a solution containing 0.18 mg mercury/litre (as mercuric chloride) for 72 h. Uptake was determined under various temperature (5 °C to 33 °C) and salinity (5 and 30 g/litre) regimes. The total mercury taken up by the gills and hepatopancreas pooled together was unaffected by the different regimes. However, the ratio of uptake into the two tissues was affected. At higher temperatures, the crabs seem able to transport mercury from gill tissue to the hepatopancreas more effectively than at low temperatures.

When Rossaro et al. (1986) exposed various life stages of the midge *Chironomus riparius* to mercuric chloride for a period of 30 days, the levels were still increasing at the end of the experiment. Both larvae and pupae accumulated mercury to about the same levels, some accumulation being due to passive adsorption. In a small experiment to illustrate this, larvae kept in a solution of 5 μg/litre for only 1 min accumulated 9.32 mg mercury/kg. The adults accumulated only 40% of the levels found in the larval stage. The authors suggested that this is because the adults have some means for eliminating the mercury.

Getsova & Volkova (1964) reported concentration factors for the accumulation of radioactively labelled mercury in four insect species. A midge, *Glyphotaelius punctatolineatus*, accumulated 5240 times the water concentration within 16 days, while a dragonfly, *Leucorrhinia rubicunda*, accumulated 8310 times the concentration over 16 days. Another dragonfly, *Aeschna grandis*, accumulated 4000 times the waterborne mercury in 8 days, while a waste-water inhabiting fly, *Eristalis tenax*, accumulated only 640 times the water concentration after 4 days and the concentration factor had fallen to just 266 after 8 days. The authors stated that the concentration factors that they found were in agreement with other Russian work on mercury accumulation.

4.2.2 Fish

When Birge et al. (1979) exposed rainbow trout eggs to an inorganic mercury concentration of 0.1 μg/litre in a flow-through system, the eggs accumulated 42.4, 68.2, and 96.8 μg mercury/kg after 1, 4, and 7.5 days, respectively. Control eggs contained 18.6 μg mercury/kg. The bioconcentration factor over 7.5 days was 782, taking into account the degree of contamination of controls. This represented a daily

uptake rate of about 20 μg/kg. There was no evidence to suggest that the mercury penetrated the outer covering of the eggs and there was a high probability that most of the "uptake" was surface adsorption.

Backstrom (1969) found that the uptake by fish of various mercury compounds was similar to that observed with birds (where methylmercury is rapidly absorbed compared with phenylmercury, methoxyethylmercury, and inorganic mercury), but the difference in uptake between methylmercury and the other mercury compounds was less pronounced. Mercury uptake into the spleen and the thyroids was greater than for birds. Phenylmercury was also retained in the wall of the gall bladder. In general the uptake of mercury into fish was far more localized than in birds. The levels of methylmercury steadily increased in the muscles and in the brain, whereas the other compounds accumulated primarily in the kidneys, spleen, and liver. More mercury accumulated in red flesh than white. There was also a high uptake of mercury into the gills and pseudobranch.

Kramer & Neidhart (1975) demonstrated that methylmercury was taken up from water by guppies *(Lebistes reticulatus)* 17 times faster than inorganic mercury. Organic mercury was also eliminated more slowly than inorganic. The authors suggested that some methylation of mercury occurred in the fish.

Ribeyre & Boudou (1984) examined the uptake of mercury over time into specific organs of the rainbow trout. The uptake was sigmoid with a linear phase and a plateau. The majority (55% for inorganic and 60% for methylmercury) of the metal was found in muscle and gills, while blood contained 3%-12%, liver 2%-5%, and kidneys 2%-7%. Brain, posterior intestine, and spleen together accounted for only 2% of total mercury. Those organs which would eventually contain most mercury accumulated their mercury exponentially. After the exposure, some organs lost their mercury while others (the ones with most mercury) continued to increase their mercury content. The organs which lost mercury in clean water had accumulated the metal with a flatter sigmoid curve.

Schindler & Alberts (1977) found that the mosquitofish *(Gambusia affinis)* readily accumulated metallic mercury during short-term continuous exposure. Within 24 h, 20 mg/kg wet weight had been taken up from a solution containing 0.1 mg total mercury/litre. The uptake curves for metallic mercury and mercuric chloride were very similar. The authors suggested that uptake in the short-term is largely the result of physical adsorption. This rate of uptake closely agrees with that found by McKone et al. (1971) in goldfish *(Carassius auratus)* where 22 mg mercuric chloride/kg was accumulated from a solution containing 0.25 mg/litre over a period of 24 h.

When Schindler & Alberts (1977) periodically exposed (2 h/day for 10 days) mosquitofish to metallic mercury and mercuric chloride (in separate experiments) at 100 μg/litre, the uptake of metallic mercury was 5 times greater than that of the chloride. The authors suggested that the metallic mercury remained unchanged and that its high lipid solubility enabled it to penetrate the gill membrane, whereas the salt

bound more tightly to the mucoproteins of the gills and penetration was restricted. The rate of elimination in mercury-free water was about the same for both, with the half-time calculated to be about 45 days.

McKim et al. (1976) exposed 3 generations of brook trout *(Salvelinus fontinalis)* to methylmercury at concentrations measured at < 0.01–2.93 µg/litre. The uptake was rapid and 2-week concentration factors ranged from 1000 to 12 000, depending on the tissue. There was a tendency for the uptake to reach a steady state (that is the tissue content reached a constant level) over 20-28 weeks. There was no significant elimination over this period.

In studies by Pentreath (1976), the thornback ray *(Raja clavata)* readily absorbed both inorganic mercuric chloride and organic methylmercuric chloride from sea water. Methylmercury, in contrast to inorganic mercury, was readily absorbed from food and slowly eliminated. The half-lives of elimination of mercury taken up from food were 61.6 days for inorganic and 323 days for organic components.

Thellen et al. (1981) found that methylmercuric chloride rapidly accumulated in the organs and muscular tissue of rainbow trout exposed to 1 mg/kg diet. However, mercuric chloride, at the same concentration, did not accumulate. During exposure to a continuous sublethal concentration of 0.25 µg mercury/litre, both organic and inorganic mercury accumulated, primarily in the internal organs and to a lesser extent the muscle tissue. Mercuric chloride was detected in the muscle at half of the concentration of organic mercury. Wobeser (1975b) fed rainbow trout fingerlings a diet containing methylmercuric chloride (at 4, 8, 16, or 24 mg mercury/kg) over a 15-week period. The total accumulation of mercury in muscle tissue was directly related to the concentration of mercury in the food, as was the rate of accumulation. Mercury was accumulated in muscle to a higher concentration than there had been in the diet.

When Amend (1970) exposed juvenile sockeye salmon (1 h per day for 12 to 15 days) to 1 mg/litre of lignasan (6.25% ethylmercury phosphate), the fish contained highest levels in the kidneys and liver. One week after the cessation of treatment, these levels were 36.5 and 20.4 mg/kg for the kidney and liver, respectively. Three years later, the fish having migrated, levels were still higher than normal but had returned to normal after 4 years. Similar studies using coho and chinook salmon yielded similar results. When Kendall (1975) injected channel catfish intraperitoneally with methylmercury chloride at 15 mg/litre, the mean concentration of mercury in the kidneys was 51.03 µg/g after 24 h and fell to 14.24 mg/kg after 96 h.

4.2.2.1 Effects of environmental variables on uptake by fish

Appraisal

Environmental variables such as temperature and pH increase the uptake of mercury, particularly methylmercury, by fish. This is of potentially considerable importance in the field.

Reinert et al. (1974) found that yearling rainbow trout *(Salmo gairdneri)* exposed to methylmercury chloride for 12 weeks accumulate more mercury at 15 °C than at 5 °C (Table 1). When Cember et al. (1978) exposed bluegill sunfish *(Lepomis macrochirus)* to methylmercury chloride at concentrations ranging from 0.2 to 50 µg/litre for up to 688 h, mercury accumulation was not affected by the different mercury concentrations. It did, however, increase when the temperature was increased from 9 °C to 33 °C (Table 1). MacLeod & Pessah (1973) found an increase in mercury accumulation, in response to an increase in temperature (from 5 to 20 °C), in rainbow trout exposed to concentrations of between 50 and 200 µg/litre for 4 days. The authors also interpolated (from 7-day data) a 4-day bioconcentration factor for phenylmercuric acetate of 100, when the fish were exposed to 5 µg/litre mercury at 10 °C. Tsai et al. (1975) studied the effect of pH on the accumulation of inorganic mercury (mercuric chloride) at a concentration in water of 1500 µg mercury/litre for 15 min. The accumulation increased as pH decreased. At pHs of 5, 6.5, and 7.5, fathead minnow accumulated whole body residues of 2.7, 1.8, and 0.4 mg mercury/kg, calculated on a wet weight basis, respectively. A similar result was found for the emerald shiner *(Nicropterus atherinoides)*.

Rodgers & Beamish (1981) found that the uptake of methylmercury by rainbow trout was increased when the hardness of the water was decreased from 385 mg/litre to 30 mg/litre. The addition of inorganic mercuric chloride increased the uptake of methylmercury in both hard and soft water. Kudo & Mortimer (1979) exposed guppies to mercury in a double chambered system, with an exchange of water. Only in one chamber did the fish have access to sediment. After being exposed for 20 days to a sediment mercury concentration of 1.023 mg/kg, the fish without direct access to the sediment showed a concentration factor of 57 and those with access a factor of 570.

4.2.3 Studies on more than one type of organism

Cultures of the alga *Croomonas salina*, grown for 48 h in the presence of mercuric chloride (164 µg mercury/litre), retained about half of the mercury (1400 mg/kg dry weight) (Parrish & Carr 1976). When the alga was fed to the copepod *Acartia tonsa* for 5 days, neither the copepods nor their eggs or faeces retained mercury in detectable amounts.

Boudou et al. (1979) exposed mosquitofish *(Gambusia affinis)* to methylmercury directly from the water and via food organisms and water in a simple model ecosystem. More mercury was taken up at higher temperatures. The authors calculated mercury uptake from water as a percentage of the "global" uptake from both water and food. This percentage varied with temperature, being 83% at 10 °C, 40% at 18 °C, and 11% at 26 °C.

In studies by Boudou & Ribeyre (1984), alevins of rainbow trout *(Salmo gairdneri)* were exposed to a constant water concentration of

methylmercuric or mercuric chloride at 1 μg/litre for 83 days. Mercury uptake was faster with organic than inorganic and both were initially linear. A plateau was eventually achieved in both cases. Uptake was negatively related to fish weight, although the authors pointed out that in the field there is usually a positive relationship.

Fang (1973) maintained the pond weed *Elodea canadensis*, snail *Helisoma campanulata*, coontail plant *Ceratophyllum demersum*, and guppy *Lebistes reticulatus* in solutions containing labelled phenylmercuric acetate (PMA) at concentrations between 5×10^{-8} and 5×10^{-7}mol/litre. All of the organisms readily accumulated PMA and the uptake was related to the length of exposure and the concentration. The absorbed PMA was largely converted to inorganic mercury. Although the uptake curves were very similar, pond weed and coontail both accumulated much more PMA than guppy or snail. The half-life of Hg^{203} residues ranged from 43 to 58 days. When Fang (1974) exposed *Lebistes reticulatus* and *Ceratophyllum demersum* to labelled ethylmercuric chloride (EMC), the uptake was positively related to the time of exposure over 200 h and the concentration up to 5×10^{-7}mol/litre. Highest concentrations were accumulated in the internal organs. The half-life of EMC was 20-23 days. Both organisms converted EMC to inorganic mercury, 34% being converted by the coontail and 29% by the guppy over a 7-day period. When the same organisms were exposed to methylmercury chloride, little or no breakdown to inorganic mercury occurred.

4.3 Uptake and Loss in Terrestrial Organisms

Appraisal

The accumulation of mercury in plants increases with increasing soil mercury concentration. Soil type has a considerable influence on this process, a high organic matter content decreasing the uptake. Generally, the highest concentrations of mercury are found at the roots, but translocation to other organs (e.g., leaves) occurs. In contrast to higher plants, mosses take up mercury via the atmosphere.

In exposed birds, the highest mercury levels are generally found in liver and kidneys. Methylmercury is more readily absorbed than inorganic mercury and it exhibits a longer biological half-time. Depending on speciation, mercury occurs in different compartments of birds' eggs; methylmercury tends to concentrate in the white and inorganic mercury in the yolk.

Huckabee & Janzen (1975) found that the mat-forming moss *Dicramum scoparium* did not take up radioactively labelled mercury from substrate. The authors concluded that the uptake of mercury into this point was mostly from the atmosphere. This is commonly true for mosses, which have been used extensively as monitor organisms for atmospheric pollutants in the field. Weaver et al. (1984) maintained bermuda grass *(Cynodon dactylon)* in three types of soil (clay, silt

loam, and fine sand) treated with mercuric chloride (1-50 mg/kg). Mercury was accumulated into the roots from silt loam, clay, and sand in increasing order. The accumulation increased with increasing mercury concentration. At 50 mg/kg the concentration of mercury in (and on the surface of) the roots was 800 mg/kg, when the grass was grown in sand.

John (1972) grew eight types of food crop in soil treated with mercuric chloride at 4 or 20 mg mercury/kg, and uptake was measured after 35 to 130 days, depending on the plant species. Higher concentrations of mercury were found in the roots compared to the above-ground samples. At the highest treatment level the mercury content of the roots, calculated on a dry weight basis, ranged from 0.387 mg/kg for lettuce to 2.447 mg/kg for cauliflower. Of the edible plant parts, spinach leaves and radish tubers contained the highest concentrations (0.695 and 0.663 mg/kg mercury, respectively).

Siegel & Siegel (1985) found that the seed-pods of several leguminous species exposed to soil mercury concentrations of 10-69 μg/kg lost 75-85% of their tissue water during maturation but showed no loss of mercury content. However, the seeds not only lost most of their water but also at least 75% of their mercury. The authors suggested that the elimination was by "bio-volatilisation", i.e., loss of elemental mercury as vapour rather than by translocation.

Nuorteva et al. (1980) reared blowfly *Lucilia illustris)* on trout flesh contaminated with mercury (0.66 mg/kg). Levels rose from 0.14 to 1.18 mg/kg during the larval feeding period, whereas pupae and freshly emerged adults contained 0.99 and 1.01 mg/kg, respectively. When adults were then fed honey, mercury levels were reduced to a third within 2 days. The authors found that it was easier for the flies to eliminate inorganic mercury than methylmercury. Nuorteva & Nuorteva (1982), after rearing blowfly larvae on mercury-contaminated fish flesh and obtaining mercury levels of 2, 6.3, and 13.3 mg/kg in different groups, fed the flies to staphylinid beetles *(Creophilus maxillosus)* for a 1-week period. This gave residues of 6.9, 17.4, and 33.4 mg/kg, respectively, in the beetles.

Kiwimae et al. (1969) fed white leghorn hens for 140 days on a diet containing 400 or 1600 μg of mercury per day as either mercury nitrate, phenylmercury hydroxide, or methoxyethylmercury hydroxide. The total mercury accumulated in the egg-whites of eggs laid was 0.31, 0.53, and 0.46 mg/kg, respectively, for the lower dose and 0.44, 0.85, and 0.88 mg/kg for the higher dose. At the higher dose, the mercury residue in the egg yolks was 2.12, 4.53, and 2.89 mg/kg, for the three mercury compounds, respectively.

Backstrom (1969) administered labelled mercury compounds, either parenterally or perorally, to Japanese quail and studied the tissue uptake and elimination. The route of administration did not affect the final uptake or subsequent elimination. Methylmercury was readily absorbed and was stable, while the other compounds, phenylmercury, methoxyethylmercury, and inorganic mercury, were less well absorbed,

and the phenylmercury was rapidly decomposed to inorganic mercury. Methylmercury was characterized by an even tissue distribution and a slow excretion, which was enhanced in egg-laying hens. The author attributed this to an increased concentration of methylmercury in the egg-white. Little of the other compounds were taken up into the brain, but methylmercury slowly reached a high concentration. The other mercury compounds were accumulated in the *yolks* of the eggs laid, and also in the liver and kidneys of the adult birds, and were rapidly excreted. The plumage and other keratinised structures strongly concentrated mercury, irrespective of the compound. These structures seem to be an important excretion route, especially for methylmercury.

Nicholson & Osborn (1984) fed juvenile starlings *(Sturnus vulgaris)* on a mercury-contaminated synthetic diet (1.1 mg mercury/kg) and analysed the birds after 8 weeks. The highest mercury levels were found in the kidneys and the liver (36.3 and 6.55 mg/kg dry weight, respectively).

In studies by Finley & Stendell (1978), black ducks *(Anas rubripes)* were fed a diet containing 3 mg mercury/kg (as methylmercury dicyandiamide) for periods of 28 weeks over two consecutive breeding seasons, during which time any ducklings that hatched were also fed the dosed diet. Mercury levels were highest in the feathers of the adult birds (61 mg/kg wet weight), followed by the liver and kidneys (22 and 14 mg/kg, respectively). Similarly the highest levels were also found in the feathers, liver, and kidneys of first-year ducklings. Eggs and embryos analysed during the first year revealed mercury levels of 6.14 and 9.62 mg/kg, respectively. Mercury residues in eggs, embryos, and ducklings were, on average, about 30% lower during the second year. Stickel et al. (1977) dosed mallard *(Anas platyrhynchos)* with 8 mg mercury/kg for 2 weeks, and found that the highest levels of mercury were accumulated in the liver (16.5 mg/kg wet weight) and the kidney (17.6 mg/kg wet weight). One week later the liver and kidney had retained 64 and 66%, of the mercury, respectively. No significant additional loss was noted during the next 8 weeks.

Adams & Prince (1976) showed that ring-necked pheasants *(Phasianus colchicus)* accumulated more mercury in the tissues after consuming methylmercury dicyandiamide than after consuming the corresponding mass of phenylmercuric acetate. This reflects the greater toxicity of alkyl mercury compounds than aryl ones.

When Borg et al. (1970) fed goshawks *(Accipiter gentilis)* liver and muscle from chickens dosed with methylmercury (average dietary mercury content 13 mg/kg), the hawks died within 6-7 weeks. The highest residues of mercury were found in the liver at 113 mg/kg wet weight (102 mg methylmercury/kg), and the kidneys at 129 mg/kg (98 mg methylmercury/kg). Substantially higher levels of mercury were found in the skeletal muscle and brain of treated birds than in those of controls. The reproductive organs also showed an ability to accumulate mercury.

4.4 Accumulation in the Field

Appraisal

Observations on given species of marine and freshwater fish indicate that all tissue concentrations of mercury increase with increasing age (as inferred from length) of the fish. In certain species males have been found to have higher levels than females.

In aquatic systems, fish-eating birds tend to have higher mercury levels than non-fishing birds. In terrestrial systems, seed-eating birds, small mammals, and their predators can have high levels in areas where methylmercury fungicides are used.

Bird feathers are useful for biological monitoring for methylmercury exposure. Analysis of feathers, especially using neutron activation, can allow recapitulation of past exposure. In general liver and kidney have higher levels than other bird tissues.

Sea mammals are reported to have a wide range of total mercury concentrations in liver (0.4 to over 300 mg/kg), only a small fraction (2-17%) being in the methylated form. Selenium and mercury have been found in seal livers in a consistent 1:1 atomic ratio. A number of studies have indicated that selenium plays a protecting role.

Point sources of mercury pollution often lead to elevated mercury levels in organisms living in the affected area. There are some circumstances where toxic effects have been produced. These effects should be taken into account in various countries during the process of industrialization.

4.4.1 General exposure

Gilmartin & Revelante (1975) analysed Northern Adriatic anchovy *(Engraulis encrasicholus)* and sardine *(Sardina pilchardus)* for mercury content. Seasonal distribution of mercury in various tissues of both anchovy and sardine ranged between 5 and 610 ng/g wet weight, the highest concentrations of mercury being in the liver and kidney. Perttila et al. (1982) found that mercury levels in the Baltic herring *(Clupea harengus)* increased significantly with age. Bache et al. (1971) observed that concentrations of both total mercury and methylmercury increased with the age of lake trout *(Salvelinus namaycush)*, the proportion of methylmercury to total mercury increasing with age. However, Westoo (1973) did not find that the proportion of methylmercury to total mercury in salmon *(Salmo salar)* and sea trout *(Salmo ocla)* was dependant on age.

Forrester et al. (1972) found a correlation between length and mercury concentration in *Squalus acanthias* (the spurdog, an elasmobranch fish). Olsson (1976) analysed northern pike *(Esox lucius)* in 1968 and 1972 and found a correlation between mercury levels and length of fish, and that males contained significantly more mercury than females. It was considered that, during a general decrease of mercury levels within pike population, the age of the fish is not a

suitable parameter for estimating mercury levels. This is because uptake and retention of mercury is dependant on body size but loss of accumulated mercury is less dependent on fish size. May & Mckinney (1981) sampled freshwater fish, in 1976 and 1977, from selected sites throughout the United States, and found mercury levels of 0.01-0.84 mg/kg wet weight.

Berg et al. (1966) analysed feathers from Swedish birds collected over a period of 100 years, and found roughly constant levels of mercury during the period 1840 to 1940. However, a well documented increase of 10 - 20 times appeared in the 1940s and 1950s, which the authors concluded was due to the use of alkylmercury seed dressings. Martin (1972) and Martin & Nickerson (1973) sampled starlings throughout the United States in 1970 and 1971 and found that most of the birds had mercury levels of < 0.5 mg/kg (76% of the birds analysed in 1971 contained levels of < 0.05 mg/kg). Lindsay & Dimmick (1983) found mercury in the liver, breast muscle, and body fat of wood duck taken from the area of the Holston River, Tennessee, USA. The highest levels were in juveniles (0.42, 0.15, and 0.1 mg/kg, for the three tissues, respectively. Local sediment contained 0.76 mg mercury/kg, black fly larvae and aquatic plants < 0.1 mg/kg.

Osborn & Nicholson (1984) sampled puffin from the islands of St. Kilda and May, off the British coast, and found liver and kidney mercury levels of approximately 1.25 mg/kg dry weight (in both tissues) for the Isle of May, and 3.75 and 5 mg/kg dry weight, respectively, for St. Kilda. Braune (1987) analysed tissues of nine species of sea birds sampled in New Brunswick, Canada, for total mercury content, and found highest levels in the liver (0.046 to 0.606 mg/kg) and kidney (0.242 to 5.345 mg/kg). Birds which fed on benthic invertebrates or fish had the highest levels, while those feeding mainly on pelagic invertebrates had the lowest.

Fimreite et al. (1982) sampled eggs from a Norwegian gannet colony for mercury in 1972, 1978, and 1979, and obtained values of 0.58, 0.8, and 0.36 mg/kg, respectively. Ohlendorf (1986) analysed eggs from three Hawaiian seabird species in 1980, and found mercury in all eggs, ranging from 0.122 to 0.359 mg/kg wet weight. Koeman et al. (1975) analysed oiled seabirds (guillemot and razorbill) from the Dutch coast for mercury residues and reported levels ranging from 1.8 to 2.4 mg/kg wet weight. Hoffman & Curnow (1979) analysed the levels of mercury in the tissues of herons, egrets, and their food collected from two sites near Lake Erie, USA. One population fed on Lake Erie (food items, 0.02-0.81 mg/kg wet weight; bird livers, 3.0-16.5 mg/kg wet weight). The other population fed predominantly on bordering marshland (food items, up to 0.24 mg/kg; bird livers, 1.03-8.22 mg/kg).

Honda et al. (1986) sampled striped dolphin *(Stenella coeruleoalba)*, and found that the accumulation of total mercury in bone correlated significantly with age. Levels rose to 1.44 and 1.55 mg/kg for adult male and female, respectively, and similar trends were seen for methylmercury, levels reaching 0.27 mg/kg in adults. Falconer et al. (1983) found that in common porpoise *(Phocoena*

phocoena) highest mercury levels were in the liver, where mean levels for females were 6.03 mg/kg and for males 3.42 mg/kg. Heppleston & French (1973) analysed tissues of common and grey seals, from the British coast for mercury and found highest levels in the livers (4.9-113 mg/kg). Koeman et al. (1975) determined mercury levels of 0.37-326 mg/kg in the livers of marine mammals (seals, dolphins, and porpoises) and also reported an almost perfect correlation between mercury and selenium content of these mammals (1:1 ratio between mercury and selenium concentrations). The authors suggested that selenium uptake may protect marine mammals from the toxic effects of mercury. Gaskin et al. (1974) found liver total mercury levels ranging from 13 to 157 mg/kg in short-finned pilot whales and long-snouted dolphins from the Lesser Antilles. Between 2% and 17% of the total mercury was methylated.

4.4.2 Mercury manufacturing and general industrial areas

Yeaple (1972) analysed bryophytes from various localities of eastern USA for mercury content and found that highest levels (1.45 mg/kg) were in plants from a large city. Levels in cities and industrial areas were higher than those in rural areas (e.g., < 0.05 mg/kg in a high, isolated mountain area). Kraus et al. (1986) collected leaves of the salt marsh cordgrass *(Spartina alterniflora)* from two sites in the USA, one site near a heavily industrialized area and the other in a non-industrialized area. The mean soil concentrations of mercury for the two sites were 18.17 and 0.22 mg/kg, respectively, while the residues in the leaves were 0.16 and 0.02 mg/kg, respectively. Salts collected from the surface of plants in the contaminated area contained 0.11 mg mercury/kg; laboratory studies have shown the plant capable of mercury excretion.

Nuorteva et al. (1980) analysed trout *(Salmo trutta)* from the Idrijca River, Yugoslavia, about 3 km downstream from a mercury distillation plant. The fish had a mercury content of 0.66 mg/kg in the flesh, and highest levels were found in the spleen and kidney (17.5 and 24 mg/kg, respectively). Three samples of ephemerids, taken 6 km from the plant, contained 0.27, 0.36, and 0.56 mg/kg wet weight, and a sample containing 4.28 mg/kg was found 1 km from the plant. These were lower levels than those reported previously, presumably because of six months inactivity at the plant. The same authors analysed blow flies from various polluted and non-polluted localities. From an unpolluted area mercury levels were < 0.1 mg/kg, near a Finnish pulp factory, 0.2 mg/kg, and near a caustic soda factory, 0.3 mg/kg. Higher levels (0.8 mg/kg) were found close to a mercury mine and distillation plant in Yugoslavia, whereas levels were near normal 1 km upstream or downstream from the mine.

Doi et al. (1984) analysed feathers from birds collected over a period of 25 years from the mercury-polluted shores of the Shiranui Sea, Japan. Relatively high levels were found until the late 1970s even though the draining of water containing methylmercury from a local

factory was stopped in 1968. Mean mercury levels were: fish-eating birds, 7.1 mg/kg; omnivorous waterfowl, 5.5 mg/kg; predatory birds, 3.6 mg/kg; omnivorous terrestrial birds, 1.5 mg/kg; and herbivorous waterfowl, 0.9 mg/kg.

Fimreite et al. (1971) analysed 156 fish and 48 bird livers from the Great Lakes area of Canada in 1968 and 1969. Elevated mercury levels were found in all fish samples, highest levels occurring in lake trout, pumpkinseed sunfish, and walleye (10.5, 7.09, and 5.01 mg/kg, respectively). Levels were generally highest in fish collected downstream from suspected sources. The highest mercury level in a fish-eating bird was found in a red-necked grebe, where the liver level was 17.4 mg/kg. Three grebes sampled showed a range of 0.45-17.4 mg/kg. Lower concentrations were found in cormorants, herons, murrelets, terns, kingfisher, and other fish-eating birds, but mean mercury liver burden was greater in these birds than in non fish-eating species.

4.4.3 Mining activity

Huckabee et al. (1983) monitored levels of mercury in vegetation in the vicinity of the mercury mine at Almaden in Spain. Mean concentrations of total mercury in vegetation ranged from > 100 mg/kg within 0.5 km of the mine to 0.20 mg/kg 20 km from the mine. There was still a significantly higher mercury content in vegetation 25 km upwind from the mine (about 10 times the background level). Mosses were found to contain the greatest concentration of mercury (7.58 mg/kg), and woody plants accumulated less of the metal (0.72 mg/kg) than herbaceous plants (2.25 mg/kg). The figures given are for samples collected in spring. There was a correlation between distance from the mine and plant mercury content for woody plants and mosses but not for herbaceous plants. No methylmercury, at quantifiable levels, was found in any of the plants analysed, although traces were seen in several samples indicating a methylmercury content of less than 10 pg per sample.

When Phillips & Buhler (1980) analysed rainbow trout (*Salmo gairdneri*), stocked in a reservoir contaminated by a disused mine, for mercury, they found that lateral muscle tissue levels increased linearly during the first five months that the fish were in the reservoir. Trout sampled 7, 19, or 31 months after being introduced showed levels that did not differ significantly (mean level = 1.25 mg mercury/kg). Matsunaga (1975) analysed crucian carp, dace, and zacco temmincku from two rivers receiving discharge from mercury mines in Japan. Total mercury levels in the fish were approximately 0.2-4.5 mg/kg and reflected the levels of mercury in the water (4-50 ng/kg).

Hesse et al. (1975) determined total mercury concentrations in the muscle, liver, and kidney of 22 species of birds collected from a western South Dakota watershed contaminated by mining activity. Elevated mercury levels were found in fish-eating birds, especially double-crested cormorants. Levels in non fish-eating birds were lower

but still significantly higher than background. In general, greater accumulations occurred in the livers of fish-eating birds (0.89 to 30.9 mg/kg) and in the kidneys of non-fish-eating birds (0.27 to 0.60 mg/kg).

4.4.4 Chloralkali plants

Gardner et al. (1978) analysed sediment, plants, and animals from a salt marsh contaminated by a chloralkali plant in Brunswick, Georgia, USA. Chloralkali plants produce metallic mercury from salts. Sediment levels ranged from 0.27 to 1.7 mg/kg dry weight for the top 5 cm and they varied according to distance from plant and depth of sample. The roots of *Spartina alterniflora*, the marsh grass, contained the highest levels (0.07-1.47 mg/kg dry weight) within the plant. Of the animals analysed from the contaminated marsh and nearby river, the invertebrates contained 0.3-9.4 mg/kg dry weight, the fish 0.3-1.9 mg/kg dry weight, the birds 2.4-37.0 mg/kg dry weight (liver) and the mammals 3.8-15 mg/kg dry weight (liver). Methylmercury levels were low (< 0.002 mg/kg) in sediment and plants but accounted for most of the mercury found in the tissues of higher organisms.

Hildebrand et al. (1980) sampled fish and invertebrates from the Holston River, USA, above and below an inactive chloralkali plant. Rock bass and hog sucker contained total mercury levels at less than 1 mg/kg above the plant, and 1-3 mg/kg immediately below it. Benthic invertebrates gave a similar pattern, lower levels being found above the plant and the higher levels below it. Total mercury concentrations in the individual taxonomic groups of the invertebrates ranged from a maximum of 3.75 mg/kg (*Hydropsychidae*. 3.7 km below the plant) to a minimum of 0.016 mg/kg (*Psepheridae*. 5.5 km above the plant). Total mercury concentrations in fish and invertebrates decreased with distance down stream of the plant. Mercury in the methyl form comprised 91.7% of total mercury in the fish and 50% in the invertebrates.

Wallin (1976) reported that samples of the carpet-forming moss *Hypnum cupressiforme* from sites around six Swedish chloralkali plants all contained similar mercury levels. Levels were highest (1-15 mg/kg) close to the plants and decreased with increasing distance from each plant. Background levels for the region (90-150 µg/kg) were reached at distances of 9-15 km from the plants. The author calculated that only a small part of the annual fallout (< 10%) was deposited locally. Shaw & Panigrahi (1986) analysed soil and five species of dwarf plants, from an area adjacent to a chloralkali factory, for mercury content. Soil from around the roots of the plants was analysed, and the mercury content was found to be very variable (2.13-893 mg/kg dry weight). Uptake into the roots, stem, leaf, and fruit of all plants in the area was significant. Leaves contained the highest levels of mercury, ranging between 2.32 and 38.8 mg/kg dry weight. Greater accumulation of mercury was found in the stem than roots of *Croton sp.* and *Jatropha sp.*; similar amounts in both stem and

roots of *Argemone sp.*, and more mercury in the roots than the stem of *Ipomoea sp.* and *Calotropis sp.* No correlation was found between the soil mercury level and plant uptake. Bull et al. (1977) measured mercury in soil, grass, earthworms, and small mammals near a chloralkali factory. At a distance of < 0.5 km from the factory, mean mercury levels in surface soil (3.81 mg/kg dry weight), grass (4.01 mg/kg dry weight), earthworms (1.29 mg/kg wet weight) and moss bags (63 ng/dm^2 per day) were significantly higher than levels found 10 to 30 km from the works. Levels of mercury at this distance were comparable with those found at sites not associated with mercury sources. Mercury levels in all tissues analysed, except muscle of bank voles *(Clethrionomys glareolus)* and woodmice *(Apodemus sylvaticus,)* were significantly higher in the study area than control areas. The authors also found elevated levels of methylmercury in small mammals and earthworms in the study area, suggesting methylation of the inorganic mercury fall-out.

4.4.5 Mercurial fungicides

Fimreite et al. (1970) found that seed-eating birds, and their avian predators, had higher liver mercury levels in areas where treated grain (mercurial fungicide) had been sown compared with areas using untreated grain. Jefferies & French (1976) analysed specimens of the long-tailed field mouse *(Apodemus sylvaticus)* taken from a wheat field that had been drilled two months previously with wheat dressed with dieldrin and mercury. Whole body mercury concentrations were much higher (0.83 ± 0.44 mg/kg wet weight) than those found immediately after drilling (0.39 ± 0.04 mg/kg wet weight).

5. TOXICITY TO MICROORGANISMS

Mercury in an inorganic form is toxic to microorganisms. It is much more toxic in an organic form, owing to increased availability of the metal to cells. The following are illustrative examples, rather than an exhaustive cover, of research into the effects of mercury on microorganisms.

Wood (1984) discussed six protective mechanisms available to microorganisms (and certain higher organisms) that increase their resistance to metal ions in general, and specifically to mercury. These mechanisms are biochemical in nature and, generally, render the mercury ion ineffective in disturbing the normal biochemical processes of the cell. The mechanisms are: (a) efflux pumps that remove the ion from the cell, a process which requires energy; (b) enzymatic reduction to the less toxic elemental form; (c) chelation by intracellular polymers (not firmly established for mercury); (d) binding of mercury to cell surfaces; (e) precipitation of insoluble inorganic complexes, usually sulfides and oxides, at the cell surface; and (f) biomethylation with subsequent transport through the cell membrane by simple diffusion. It is this last mechanism, biomethylation, which renders the mercury more toxic to higher life-forms.

5.1 Toxicity of Inorganic Mercury

Appraisal

Inorganic mercury is toxic to microorganisms over a wide range of concentrations. Its effects on development and survival are modified by environmental factors such as temperature, light intensity, pH, and chemical composition of the medium, and by cell-related factors such as genetic variation. Through selective effects on particular species, it can change the composition of a plankton community. The mechanism of action is not fully understood.

5.1.1 Single species cultures

Kamp-Nielson (1971) demonstrated a time-dependent effect of mercuric chloride, added at 300 µg/litre, on the photosynthesis of *Chlorella pyrenoidosa*. There was little effect in the first hour of incubation, a pronounced drop in photosynthetic rate in the second hour, and a period of little further effect between 2 and 5 h. An overall rate reduction of about 50% occurred after 5 h with a cell density of 6.5×10^7 cells/litre. There was a greater effect on photosynthesis at lower cell densities. It was also found that photosynthesis had to occur for the effect to develop, since exposure to mercuric chloride for 2 h in the light had the same effect as exposure to the same concentration of mercury for 2 h in the dark followed by 2 h in the light. Similar results were found after 1-h

exposures in light and darkness followed by light. There was an effect of light intensity; in short-term experiments mercury had a deleterious effect on photosynthesis only at high light intensities. Mercury also affected photosynthesis at low light intensity, but only after 20-h exposures. Mercury affected photosynthesis adversely at concentrations between about 50 and 300 µg/litre, but had no greater effect at concentrations up to 1000 µg/litre (the highest tested). The effect was dependant on cell density, pH, light intensity, and duration of exposure. Potassium and sodium in the growth medium had no effect on mercury toxicity to *Chlorella*. Increasing the concentration of mercuric chloride in the medium increased the "leakage" of potassium from the cells of *Chlorella*. This was maximal at a mercury concentration of about 300 µg/litre and was considered to be the main toxic effect of mercury. The effect on potassium leakage occurred equally in darkness and light and was, therefore, independent of the photosynthetic effect. Mercury increased the length of the lag-phase during the growth of *Chlorella pyrenoidosa* cultures. A greater effect was seen at 660 than at 330 µg/litre, the only two doses tested. This effect was also demonstrated by Osokina et al. (1984) in the green alga *Scenedesmus quadricauda*. The effect was highly dependant on the cell density of the original inoculum.

Rai et al. (1981) exposed *Chlorella vulgaris* to mercuric chloride concentrations between 100 and 1000 µg/litre for 3 weeks, and monitored growth and survival. LC_{50} for survival was at 400 µg/litre of mercuric chloride. The growth rate was 92% of the control value at 100 µg/litre and 31% at 800 µg/litre, and there was no growth at 1000 µg mercuric chloride/litre. The chlorophyll content of the cells was reduced throughout the dose range. There was a greater toxic effect of mercuric chloride at low pH, with the greatest amelioration of toxicity at pH 9. There was also a protective effect of calcium and phosphate in the medium and, to a lesser extent, of magnesium. Both calcium and phosphate increased the yield of algae, in the presence of sublethal concentrations of mercury, when added at concentrations up to 20 mg/litre. At higher concentrations of both calcium and phosphate, the protection was less marked. Den Dooren de Jong (1965) determined the no-observed-effect-level (NOEL) for mercuric chloride on *Chlorella vulgaris* to be 50 µg/litre. Hannon & Patouillet (1972) emphasized the irreversibility of the effects of mercuric chloride on *Chlorella pyrenoidosa*. If mercury was present in sufficient concentration to affect growth of the alga, then no recovery was found following transfer in clean medium. Similar effects were reported for three species of marine unicellular algae. Mercury toxicity was dependant on cell numbers in the initial inoculum (Kuiper, 1981). In studies with unialgal cultures of *Chlamydomonas sp.*, there was a relationship between cell concentration and mercury toxicity. The author attributed this to a surface area effect, the metal is being adsorbed onto cell walls to cause its effect on the unicellular algae.

Huisman et al. (1980) investigated the effect of temperature on the toxicity of mercuric salts to the green alga *Scenedesmus acutus*. Mercury concentration in the cultures was kept constant by a mercury(II) buffer system, and the growth and photosynthesis of the alga were monitored. Toxicity increased with increasing temperature over the range 15-30 °C. There was no effect observed in this study on the lag phase, no later increase in growth, and no effect of initial cell numbers. This was attributed to the buffer system which prevented changes in free mercury concentrations over time. The authors also examined the binding of mercury to algal cells. Metal bound to the cell wall consists of two fractions: one which can be washed off with cysteine solution and one which cannot. The amount of mercury which can be washed off the cell wall increase with increasing temperature. The mercury bound to cell walls, but washable with cysteine, appears to be the toxic fraction. The total mercury content of algal cells does not correlate with effect. A total mercury content not lethal at 15 °C causes complete inhibition of growth and photosynthesis at 30 °C. Recovery occurs under circumstances where the cells retain the non-washable mercury, indicating that the washable fraction is the toxic component. The authors suggested that the reversibility of the action of cysteine-washable mercury indicates that the metal is bound to carboxyl or phosphate groups and not to sulfhydryl groups. These mercury ions can be readily exchanged for other metal ions, leading to a decreased inhibition by mercury. Therefore, in media with a high concentration of dissolved salts, mercury appears to be less toxic. The authors postulated another mechanism by which mercury might be toxic to algal cells. Interference with potassium-sodium-dependent ATPase in the cell membrane influences the active transport of nutrients. This would give rise to disturbances of nitrogen metabolism and also of photosynthesis. The delayed action of mercury on cultures could be ascribed to their being initially rich in nitrogen, and, therefore, less susceptible to nitrogen starvation.

Nuzzi (1972) exposed *Phaeodactylum tricornutum*, *Chlorella sp.*, and *Chlamydomonas sp.*, isolated from the lower Hudson River, New York, USA, to mercuric chloride. The growth of all three organisms was severely inhibited by mercury at 7.5 µg/litre (to between 50% and 75% of control growth). The growth of *Chlamydomonas sp.* was completely inhibited by 15 µg mercuric chloride/litre and the other two species by 22 µg/litre.

Gray & Ventilla (1971) found no effect of mercuric chloride on growth of the marine ciliate *Cristigera spp.* at a concentration of 100 µg/litre, but growth was affected after exposure to 200 or 500 µg/litre. There was a synergistic interaction between mercury and lead on this ciliate. Gray & Ventilla (1973) reported reductions in growth rate of between 8% and 12% after exposing *Cristigera* to mercuric chloride at 25 or 50 µg/litre. Persoone & Uyttersprot (1975) found no effects of mercuric chloride, at concentrations up to 100 µg/litre, on the survival or reproduction of the marine ciliate *Euplotes vannus*. However, all cells died after exposure to 1000 µg/litre.

5.1.2 Mixed cultures and communities

Singleton & Guthrie (1977) investigated the effects of inorganic mercury, added as mercuric chloride at 40 μg/litre, on populations of bacteria from fresh and brackish water. Water was taken from the two sources and kept for 1 week in the laboratory before the metal salt was added. Results were assessed by measuring total colony-forming units (viable bacteria), percentage of chromagenic organisms, and numbers of different colony types (species diversity). Control systems maintained constant numbers of viable bacteria throughout the 14-day test period. When mercury was added, the numbers of viable bacteria from test samples increased and remained elevated throughout the test. The effect was greater in brackish than in fresh water. Diversity declined at the same time as total numbers increased. Some genera of bacteria disappeared from the community, notably *Flavobacterium* and *Brevibacterium*. Other organisms which disappeared or were greatly reduced included *Sarcina sp., Enterobacter sp., Achromobacter sp.,* and *Escherichia sp.* After mercury treatment, the percentage of chromagenic species decreased in the population. Controls maintained chromagens at a steady 20-25% of total bacteria. Chromagen percentage declined most markedly after 9-10 days of mercury exposure.

Kuiper (1981) exposed a mixed community of marine plankton to mercuric chloride (at 0.5, 5.0, or 50 μg mercury/litre) in 1400-litre plastic bags suspended from a raft in a Netherlands harbour. The addition of 50 μg mercury/litre resulted in complete inhibition of phytoplankton activity. There was a decrease in phytoplankton biomass because of settling of cells to the bottom of the bag. Phytoplankton growth resumed after about 20 days when mercury concentrations were still at 18 μg/litre. There was evidence for two possible mechanisms for this: either mercury-resistant species were growing or mercury was being adsorbed to inanimate particles or removed by chelation. Addition of 5.0 μg/litre reduced phytoplankton growth rate. Biomass decreased initially but began to increase again when the mercury concentration decreased to about 1.5 μg/litre. Mercury at 5.0 μg/litre delayed the phytoplankton peak by 9 days but relative carbon assimilation by only 1 day. One possible explanation is that mercury affected cell division more than carbon assimilation. Both 5.0 and 50 μg/litre altered the species composition of the growth peak; higher mercury concentrations favoured the selection of larger species. The first stage in the uptake and toxicity of mercury in phytoplankton is adsorption on cell surfaces (e.g., cell walls); the smaller surface to volume ratio of larger cells may explain why larger cells are more resistant to higher mercury concentrations. Another possible explanation involves predation; reduced numbers of predatory zooplankton might favour larger phytoplankton cells which might be preferred by predators. There was some evidence to support both hypotheses. Zooplankton were also affected by mercury. There was

immediate death of most copepods after the addition of mercuric chloride at 50 µg mercury/litre. Development of the copepods *Temorus longicornis* and *Pseudocalanus elongatus* was delayed by 5.0 µg/litre. The results suggest that the major effect on these zooplankton is a retardation of development rather than an increase in mortality. Laboratory experiments simulating conditions in the bags suggested that zooplankton grazing on phytoplankton was an important factor in the productivity of the bags during the second, but not the first, half of the experimental period. On day 10 of the experiment, viable bacteria numbers were higher in bags with 5.0 and 50 µg mercury/litre than in controls. This was probably due to the high mortality of phytoplankton increasing the food source for bacteria. Conversion rate of organic matter into ammonia was reduced. The author concluded that the toxicity of mercury to plankton depends on mercury concentration, total surface area for adsorption of mercury (and, therefore, on the ratio between living and non-living particles present and on absolute cell size), and on the metal species present (Kuiper, 1981).

Hongve et al. (1980) added mercuric salt, alone or in combination with humus or sediment, to cultures of a natural phytoplankton community in lake water, and monitored photosynthetic carbon fixation using a radiolabelled tracer. Mercury reduced carbon fixation by 50% at the lowest dose tested (5×10^{-9} mol/litre) and to less than 10% of control levels at the highest dose tested (2×10^{-7} mol/litre). Addition of either humus or sediment to the cultures reduced mercury toxicity presumably by binding the metal to surfaces.

Zelles et al. (1986) conducted a complex and comprehensive experiment to compare different methods for assessing the overall ecotoxicological effects of chemicals on soil microorganisms. Three soil types were used in an 18-week experiment which investigated ATP, heat production, respiration (as measured by carbon dioxide output), and iron reduction in the soils under dry and moist conditions. Two different dose levels of mercuric chloride were added to the soils (2 and 20 mg/kg). Averaging the results obtained in the different tests, adverse effects on microorganisms were least in peat soil and greatest in sandy soil. Some stimulation of microbial activity occurred in peat soil with both low and high concentrations of mercuric chloride. At both 2 and 20 mg/kg mercury there was inhibition of microbial activity in sandy soil. Effects were generally inhibitory in clay soil at both concentrations of mercuric chloride. The authors pointed out that it is not possible to assess the ecotoxicological effects of mercury on soil by using a single method to assess soil function.

5.2 Toxicity of Organic Mercury

Appraisal

Methylmercury is more toxic to microorganisms than are inorganic mercury salts. This is probably because greater surface adsorption

enhances the availability and subsequent uptake of methylmercury. This may explain why the toxicity of organomercury is inversely correlated with cell density. As the surface area of the total cells in the culture increases, so less mercury is available for uptake per cell. In organomercury compounds, it is the mercury-containing moiety, as opposed to the dissociable anion, which determines the toxicity. A common toxic effect in phytoplankton is the inhibition of growth, which may in turn often be due to reduced photosynthesis.

Methylmercury in water at 1 μg/litre has adverse effects on microorganisms.

Ukeles (1962) tested the effect of Lignasan (ethylmercuric phosphate 6.25%) on a variety of algae in pure culture. The cultures were exposed to Lignasan at 0.6, 6.0, and 60 μg/litre for 10 days. The highest dose of 60 μg/litre prevented all growth of cultures, and at the end of the exposure, all cells were killed by the treatment. Three out of the five algae tested were also killed by Lignasan at 6.0 μg/litre: *Protococcus sp.*, *Chlorella sp.*, and *Monochrysis lutheri*. Growth of the other two species was reduced; *Dunaliella euchlora* showed 31% of the growth of controls and *Phaeodactylum tricornutum* 17% of control growth. At 0.6 μg/litre, Lignasan reduced growth of four of the five cultures to between 55% and 86% of control levels, *Monochrysis* alone being unaffected.

Nuzzi (1972) exposed *Phaeodactylum tricornutum*, *Chlorella sp.* and *Chlamydomonas sp.* to phenylmercuric acetate (PMA) at concentrations of 0.06-15.0 μg mercury/litre. *P. tricornutum* was also tested against phenylacetate equivalent to the phenylacetate content of the PMA, but this had no effect. All three organisms were adversely affected by the mercury in PMA, growth being inhibited even at the lowest dose tested. *Chlamydomonas* was totally inhibited by 3 μg mercury/litre. *Chlorella sp.* showed a steep decline in growth as exposure increased from 0.06 to 3 μg/litre, where growth was about 25% of the control value. *Phaeodactylum* growth declined rapidly as dose increased to 9 μg/litre, where growth was minimal.

Holderness et al. (1975) cultured the green alga *Coelastrum microporum* with methylmercuric chloride (MMC) at 0.8, 3, 6, 12.6, and 250 μg/litre. There was no significant effect on cell concentration, as determined by transmittance, at 0.8 μg/litre, but higher concentrations were inhibitory. There was a steady reduction in cell concentration between 0 and 3 μg MMC/litre and a marked decline between 3 and 6 μg/litre, with cell concentration changing from 125 μlitre/litre, at 3 μg MMC/litre to 31 μlitre/litre at 6 μg MMC/litre. It was noted, in three series of experiments, that MMC caused increased storage of starch in the cells. A slight increase in photosynthesis was found after exposure to 0.6 μg MMC/litre.

Delcourt & Mestre (1978) exposed cultures of *Chlamydomonas variabilis* to concentrations of phenylmercuric acetate (PMA) between 10^{-9} and 7.5×10^{-8} mol/litre. Growth curves of the control cultures were linear, with no evident lag phase, irrespective of the

cell concentration (which varied between 2000 and 100 000 cells/ml) in the initial inoculum. The effect of mercury as PMA was initially tested with cell concentration at 20 000 cells/ml. Under these conditions, cultures exposed to PMA at 10^{-9} or 2.5 x 10^{-9} mol/litre grew exactly the same as controls. However, at PMA concentrations of 5 x 10^{-9} mol/litre or more, there was a dose-related lag phase. When exponential growth did start, the curves were parallel to those of the control. Final cell numbers were not affected, only the time taken to reach maximum growth. Changing the initial cell concentration in the cultures changed the toxic threshold of the PMA, PMA toxicity being higher at lower algal cell concentrations. The authors considered that there are a limited number of binding sites for mercury on the cell surface and that this was the reason for the effect of cell concentration on toxic threshold. Whilst the toxic threshold was higher than likely exposure levels in natural waters at high algal cell concentrations, the authors pointed out that the threshold would be exceeded at low, spring algal concentrations.

Harriss et al. (1970) exposed a pure culture of the marine diatom *Nitzschia delicatissima* and a natural phytoplankton community from a freshwater lake to four organomercurial compounds at concentrations between 0 and 50 µg/litre. The four compounds (PMA, methylmercury dicyandiamide [Panogen], *N*-methylmercuric-1,2,3,6-tetra hydro-3,6-methano-3,4,5,6,7,8-hexachlorophthalimide [MEMMI], and diphenylmercury) showed broadly similar effects on photosynthesis at the same concentrations expressed in terms of mercury content. The diphenylmercury was slightly less toxic than the other compounds. The diatom was exposed to the mercurials for 24 h, and the phytoplankton community was exposed for 24, 72, or 120 h, before estimating the photosynthetic uptake of labelled hydrogen carbonate over 5 h. At concentrations of 1 µg/litre, all four mercurials inhibited photosynthesis of the natural phytoplankton. Photosynthetic uptake of labelled carbon was between 35% and 55% of control levels for the four compounds. At 50 µg/litre, all uptake of carbon stopped and cell counts indicated cessation of growth in the case of all compounds except diphenylmercury. Photosynthetic carbon uptake was about 40% of control levels after exposure for 120 h to 50 µg diphenylmercury/litre. The authors stated that the toxicity of diphenylmercury to the natural phytoplankton was similar to that of mercuric chloride, but no details of studies with inorganic mercury were given. *Nitzschia* was similarly inhibited by all mercurials tested, except diphenylmercury, at 1 µg/litre. The diatom showed virtually no carbon, at assimilation in the presence of PMA, methylmercury dicyandiamide, or MEMMI 10 µg/litre. At 1 µg/litre, the carbon assimilation was 95% of the control value with diphenylmercury, 60% with PMA, 23% with methylmercury dicyandiamide, and < 10% with MEMMI. The authors noted that the toxicity of mercurials to the natural phytoplankton community decreased with increasing cell numbers, but no details were given.

6. TOXICITY TO AQUATIC ORGANISMS

Mercury is toxic to aquatic organisms, organic forms of the metal being generally more toxic than inorganic forms. Effects are more likely to be observed in soft freshwater, since the toxicity of the metal is reduced in the presence of high salt concentrations. The concentration of mercury that produces effects varies considerably from one species to another.

6.1 Toxicity to Aquatic Plants

Appraisal

As in the case of microorganisms, mercury, at a wide range of concentrations, has effects on various aspects of performance, including development and survival. These are partly the result of adverse effects on photosynthesis.

The presence of sediment or humic material reduces the availability of mercury to aquatic plants because of adsorption. In studies involving a dual medium, such as soil-water, actual exposures are more difficult to determine than in studies with a single medium, such as water alone.

Organic forms of mercury, such as methyl- or butylmercury chloride are more toxic to aquatic plants than inorganic forms.

Boney (1971) exposed 2-day-old sporelings of the red alga *Plumaria elegans* to mercuric chloride in solution, and found that 50% growth inhibition occurred after 6 h, approximately 12 h, and approximately 24 h at concentrations of 1.0, 0.5, and 0.25 mg/litre, respectively. Organic forms of mercury (methyl, butyl, and propylmercuric chlorides) were also investigated, and found to be much more toxic than inorganic mercury. Methylmercury gave 50% inhibition after 17.5 and 25 min of exposure to 0.08 and 0.04 mg/litre, respectively. Propylmercury, at 0.5 mg/litre, produced 50% growth inhibition after 2.5 min of exposure and 70% inhibition after 5 min. Butylmercury produced more marked inhibition than propylmercury (no detailed results given). Hopkin & Kain (1978) found that the survival of germinating gametophytes of the macroalga *Laminaria hyperborea*, in culture, was reduced by 0.01 mg mercury/litre. The lowest effective toxic level of mercury for the sporophyte culture was 0.05 mg/litre.

Stanley (1974) determined EC_{50}s, in the presence of a mercuric salt, for various growth parameters of Eurasian watermilfoil *(Myriophyllum spicatum)* grown in soil with water above. EC_{50}s (in mg/litre) were 3.4 for root weight, 4.4 for shoot weight, 12 for root length, 1.2 for shoot length. The author added mercury to the water, to the soil, or to the water in a system containing ferric silicate instead of soil. Comparison of the tissue concentrations of mercury when the metal salt was added in these different ways indicated a very

strong tendency for mercury to be adsorbed onto soil. There was no indication that the presence of soil affected mercury uptake in any way other than by simple adsorption, i.e., no soil component interacted with the mercury ions.

De et al. (1985) exposed the floating plant water cabbage *(Pistia stratiotes)* for 2 days to mercuric chloride at concentrations between 0.05 and 20.0 mg/litre. The highest dose of mercury promoted plant senescence by decreasing chlorophyll content, protein, RNA, dry weight, and catalase and protease activities, and by increasing free amino acid content. Lesser, mostly non-significant, effects on these parameters were recorded at lower doses. In studies by Brown & Rattigan (1979), the aquatic macrophyte *Elodea canadensis* (Canadian pond weed) and the free-floating duckweed *Lemna minor* were exposed for 28 days and 14 days, respectively, to a range of concentrations of mercuric chloride. Damage to the plants was assessed visually on a coded scale ranging from 0 (no damage) to 10 (plant killed). Water concentrations of 7.4 and 1.0 mg/litre produced 50% damage to the two plants, respectively. In a separate study, *Elodea* was exposed to mercury for 24 h in the dark and then oxygen evolution in the light was measured. Levels of 0.8 and 1.69 mg mercury/litre reduced photosynthetic oxygen evolution by 50% and 90%, respectively. Czuba & Mortimer (1980, 1982) exposed plants of *Elodea densa*, growing in flowing water, to concentrations of methylmercuric chloride at 7.5×10^{-10}, 7.5×10^{-9}, or 7.5×10^{-8}mol/litre, for 25 days. Toxicity was assessed by gross morphological examination and from the examination of histological sections embedded in paraffin wax. There was a difference in toxic effect between tissues. Apical cells were most sensitive to the mercury and developed aberrant nuclear and mitotic characteristics at lower concentrations than did roots. Root meristems showed total inhibition of mitotic activity at the middle concentration but no effect at the lowest concentration used. Mitotic activity in bud meristems was absent in controls, but increased in the presence of methylmercury; divisions were abnormal. Higher concentrations of methylmercury chloride, up to 2.5×10^{-6}mol/litre, stimulated the development of additional buds. The development of root and bud initials was inhibited by methylmercury at 7.5×10^{-8} and 2.5×10^{-6}mol/litre, respectively.

6.2 Toxicity to Aquatic Invertebrates

Appraisal

Factors which affect the toxicity of mercury to aquatic invertebrates include the concentration and species of mercury, the developmental stage of the organisms, and the temperature, salinity, water hardness, and flow rate. Methylmercury is more toxic than aryl or inorganic mercury. The larval stage is apparently the most sensitive stage of the organism's life cycle. Mercury toxicity increases with temperature and decreases with water hardness.

Toxicity, appears to be higher in flow-through systems than in static systems. This effect is probably due mostly to the actual concentration of mercury available to the organism, which is lower in static systems. The fact that lower salinity seems to increase toxicity may be due more to the stress that is placed on the organism.

Levels of 1 to 10 µg/litre normally causes acute toxicity for the most sensitive developmental stage of many different species of aquatic invertebrates.

The acute toxicity of mercury to aquatic invertebrates is summarized in Tables 3 and 4.

6.2.1 Acute and short-term toxicity to invertebrates

Wisely & Blick (1967) determined the concentration of mercury in water required to kill 50% of larvae for some species of bryozoans *(Watersipora cucullata* and *Bugula neritina)*, tubeworms *(Spirorbis lamellosa* and *Galeolaria caespitosa)*, bivalve molluscs *(Mytilus edulis* and *Crassostrea commercialis)*, and the brine shrimp *(Artemia salina)*. The 2-h LC_{50}s for the larvae of these species were 5×10^{-7}, 1×10^{-6}, 7×10^{-7}, 6×10^{-6}, 6.5×10^{-5}, 9×10^{-4}, and 9×10^{-3} mol mercuric chloride/litre, respectively.

Howell (1984) exposed two species of marine nematodes, one euryhaline[a] *(Enoplus brevis)* and one stenohaline[b] *(Enoplus communis)* to mercuric chloride. *E. brevis* was collected from two sites, one nonpolluted and one polluted with heavy metals. The stenohaline species was more sensitive to mercuric chloride than the related euryhaline species. At a concentration of 0.01 mg mercuric chloride/litre, *E. communis* showed an LT_{50} of approximately 65 h, whereas 50% *E. brevis* collected from the nonpolluted site survived for approximately 415 h at the same concentration. *E. brevis* from the polluted area was even less sensitive, with an LT_{50} of more than 600 h, suggesting the selection of resistant strains.

When Best et al. (1981) exposed the planarian *Dugesia dorotocephala* to concentrations of methylmercury chloride of between 0 and 2 mg/litre, 100% deaths were reported at 0.5, 1, and 2 mg/litre within 5 days, 1 day, and 5 h, respectively. No deaths occurred at 0.2 mg/litre over a 10-day-period, but other, non-lethal toxic responses, including varying degrees of head resorption, were observed within 1 day. This was followed by some head regeneration within 10 days. After some animals were decapitated, regeneration was retarded at 0.1 and 0.2 mg methylmercury chloride/litre. Although no deaths, malformations, visible lesions, or gross behavioural abnormalities were seen at 20 µg/litre or less, significant changes in fissioning

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[a] tolerant of a wide range of salinity
[b] tolerant of only a narrow range of salinity

Table 3. Toxicity of inorganic mercury (as mercuric chloride) to marine invertebrates

Organism	Life-stage	Stat/flow[a]	Temperature (°C)	pH	Salinity (‰)	Dissolved oxygen (mg/litre)	Parameter	Water concentration (µg/litre)	Reference
Starfish	adult	stat	20	7.8	20	> 4	24-h LC_{50}	1800	Eisler & Hennekey (1977)
(Asterias forbesi)	adult	stat	20	7.8	20	> 4	96-h LC_{50}	60	Eisler & Hennekey (1977)
	adult	stat	20	7.8	20	> 4	168-h LC_{50}	20	Eisler & Hennekey (1977)
Hard clam *(Mercenaria merceneria)*	embryo	stat	25-27	7-8.5	25		48-h LC_{50}	4.8 (3.8-5.6)	Calabrese & Nelson (1974)
Softshell clam	adult	stat	20	7.8	20	> 4	24-h LC_{50}	5200	Eisler & Hennekey (1977)
(Mya arenaria)	adult	stat	20	7.8	20	> 4	96-h LC_{50}	400	Eisler & Hennekey (1977)
	adult	stat	20	7.8	20	> 4	168-h LC_{50}	4	Eisler & Hennekey (1977)
American oyster *(Crassostrea virginica)*	embryo	stat	25-27	7-8.5	25		48-h LC_{50}	5.6 (4.2-6.8)	Calabrese et al. (1973)
Pacific oyster *(Crassostrea gigas)*	embryo	stat	19-21	7.9-8.3	33.7-33.8	6.5-8.0	48-h EC_{50}[c]	5.7	Glickstein (1978)
Oyster	larvae	stat	15				48-h LC_{50}	1.0-3.3	Connor (1972)
(Ostrea edulis)	adult	stat	15				48-h LC_{50}	4200	Portmann & Wilson (1971)
Cockle *(Cardium edule)*	adult	stat	15				48-h LC_{50}	9000	Portmann & Wilson (1971)

Table 3 (Contd).

Organism	Life-stage	Stat/flow[a]	Temperature (°C)	pH	Salinity (‰)	Dissolved oxygen (mg/litre)	Parameter	Water concentration (µg/litre)	Reference
Mud snail	adult	stat	20	7.8	20	> 4	24-h LC_{50}	32 000	Eisler & Hennekey (1977)
(Nassarius obsoletus	adult	stat	20	7.8	20	> 4	96-h LC_{50}	32 000	Eisler & Hennekey (1977)
	adult	stat	20	7.8	20	> 4	168-h LC_{50}	700	Eisler & Hennekey (1977)
American lobster *(Homarus americanus)*	stage I larvae	stat	18-22		29.5-31.5	7.6-8.6	96-h LC_{50}	20	Johnson & Gentile (1979)
European lobster *(Homarus gammarus)*	larvae	stat	15				48-h LC_{50}	33-100	Connor (1972)
Pink shrimp *(Pandalus montagui)*	adult	stat	15				48-h LC_{50}	75	Portmann & Wilson (1971)
White shrimp *(Penaeus setiferus)*	post-larval	stat	21-24		25		96-h LC_{50}	17 (13-21)	Green et al.(1976)
Brown shrimp *(Crangon crangon)*	larvae	stat	15				48-h LC_{50}	10	Connor (1972)
	adult	stat	15				48-h LC_{50}	3300-10 000	Portmann & Wilson (1971)
	adult	stat[b]	15				96-h LC_{50}	100-330	Portmann & Wilson (1971)
Grass shrimp *(Palaemonetes vulgaris)*	stage I larvae		26.5-27	6.3-6.9	32.73-33.29	5.6	48-h LC_{50}	10 (7.8-12.7) unfed	Shealy & Sandifer (1975)
	stage I larvae		27	6.4-6.7	33.99	5.8-7.6	48-h LC_{50}	15.6 (12.7-19.3) fed	Shealy & Sandifer (1975)

Table 3 (Contd).

Dungeness crab (*Cancer magister*)	1st stage zoeae		14-16	7.9-8.3	33.72-33.86	6.5-8.0	48-h LC_{50}	21.1 (19.7-22.5)	Glickstein (1978)
	1st stage zoeae		14-16	7.9-8.3	33.72-33.86	6.5-8.0	96-h LC_{50}	6.6 (5.6-4.6)	Glickstein (1978)
Shore crab (*Carcinus maenus*)	larvae	stat	15				48-h LC_{50}	14	Connor (1972)
	adult	stat	15				48-h LC_{50}	1200	Portmann & Wilson (1971)
Hermit crab	adult	stat	20	7.8	20	> 4	24-h LC_{50}	2200	Eisler & Hennekey (1977)
(*Pagurus longicarpus*)	adult	stat	20	7.8	20	> 4	96-h LC_{50}	50	Eisler & Hennekey (1977)
	adult	stat	20	7.8	20	> 4	168-h LC_{50}	50	Eisler & Hennekey (1977)
Crab	adult	stat	26.5-29.5	7-7.2			24-h LC_{50}	930	Krishnaja et al. (1987)
(*Scylla serrata*)	adult	stat	26.5-29.5	7-7.2			48-h LC_{50}	800 (740-860)	Krishnaja et al. (1987)
	adult	stat	26.5-29.5	7-7.2			72-h LC_{50}	680	Krishnaja et al. (1987)
	adult	stat	26.5-29.5	7-7.2			96-h LC_{50}	680 (600-760)	Krishnaja et al. (1987)
Polychaete	juv	stat		7.8			96-h LC_{50}	100[d]	Reish et al.(1976)
(*Neanthes*	adult	stat		7.8			96-h LC_{50}	22[d]	Reish et al.(1976)
arenaceodentata)	juv	stat		7.8			28-day LC_{50}	90[d]	Reish et al.(1976)
	adult	stat		7.8			28-day LC_{50}	17[d]	Reish et al.(1976)
Polychaete	larva	stat		7.8			96-h LC_{50}	14[d]	Reish et al.(1976)
(*Capitella capitata*)	adult	stat		7.8			96-h LC_{50}	>100[d]	Reish et al.(1976)
	adult	stat		7.8			28-day LC_{50}	100[d]	Reish et al.(1976)

Table 3 (Contd).

Organism	Life-stage	Stat/flow[a]	Temperature (°C)	pH	Salinity (‰)	Dissolved oxygen (mg/litre)	Parameter	Water concentration (µg/litre)	Reference
Sandworm	adult	stat	20	7.8	20	> 4	24-h LC_{50}	3100	Eisler & Hennekey (1977)
(Nereis virens)	adult	stat	20	7.8	20	> 4	96-h LC_{50}	70	Eisler & Hennekey (1977)
	adult	stat	20	7.8	20	> 4	168-h LC_{50}	60	Eisler & Hennekey (1977)

[a] stat = static conditions (water unchanged for duration of test).
[b] static conditions but test water changed every 24 h.
[c] abnormal development.
[d] with food.

Table 4. Toxicity of inorganic mercury to freshwater invertebrates[c]

Organism/ weight (g)	Stat/ flow[a]	Temperature (°C)	Alkalinity[f]	Hardness[f]	pH	Dissolved oxygen (mg/litre)	Parameter	Water concentration (µg/litre)	Reference
Mussel	stat	28-32	9.5-9.9	32-38	7-7.3	5.38-6.2	24-h LC_{50}	7390	Ramamurthi
(*Lamellidens*	stat	28-32	9.5-9.9	32-38	7-7.3	5.38-6.2	48-h LC_{50}	5910	et al.
maginalis)	stat	28-32	9.5-9.9	32-38	7-7.3	5.38-6.2	72-h LC_{50}	3690	(1982)
32-35									
Snail (egg)	stat	17		50	7.6	6.2	24-h LC_{50}	6300	Rehwoldt
(*Amnicola sp.*)	stat	17		50	7.6	6.2	96-h LC_{50}	2100	et al.
(adult)	stat	17		50	7.6	6.2	24-h LC_{50}	1100	(1973)
	stat	17		50	7.6	6.2	96-h LC_{50}	80	Rehwoldt et al. (1973)
Snail	stat	28-32	9.5-9.9	32-38	7-7.3	5.38-6.2	24-h LC_{50}	1108	Ramamurthi
(*Pila globosa*)	stat	28-32	9.5-9.9	32-38	7-7.3	5.38-6.2	48-h LC_{50}	369	et al.
20-25	stat	28-32	9.5-9.9	32-38	7-7.3	5.38-6.2	72-h LC_{50}	296	(1982)
Pulmonate snail		25.5-29.5	240-278	290-335	7.4-8.1	6.0-8.1	24-h LC_{50}	330 (297-360)	Mathur et al. (1981)
(*Lymnaea luteola*)		25.5-29.5	240-278	290-335	7.4-8.1	6.0-8.1	48-h LC_{50}	188 (163-210)	Mathur et al. (1981)
0.46-0.72		25.5-29.5	240-278	290-335	7.4-8.1	6.0-8.1	96-h LC_{50}	135 (112-186)	Mathur et al. (1981)
Crab	stat	28-32	9.5-9.9	32-38	7-7.3	5.38-6.2	24-h LC_{50}	739	Ramamurthi
(*Oziotelphusa*	stat	28-32	9.5-9.9	32-38	7-7.3	5.38-6.2	48-h LC_{50}	591	et al.
senex senex)	stat	28-32	9.5-9.9	32-38	7-7.3	5.38-6.2	72-h LC_{50}	443	(1982)
35-88									
Crayfish	flow[b]	15-17			7.0		96-h LC_{50}	20	Boutet &
(*Austropotamobius*	flow[b]	15-17			7.0		30-day LC_{50}	2	Chaisemartin
pallipes pallipes)	flow[b]	15-17			7.0		30-day LC_{50}	< 2[d]	(1973)

Table 4 (Contd).

Organism/ weight (g)	Stat/ flow[a]	Temperature (°C)	Alkalinity[f]	Hardness[f]	pH	Dissolved oxygen (mg/litre)	Parameter	Water concentration (µg/litre)	Reference
Crayfish (*Orconectes limosus*)	flow[b]	15-17			7.0		96-h LC_{50}	50	Boutet & Chaisemartin (1973)
	flow[b]	15-17			7.0		30-day LC_{50}	2	
	flow[b]	15-17			7.0		30-day LC_{50}	< 2[d]	
Scud (*Gammarus sp.*)	stat	17		50	7.6	6.2	24-h LC_{50}	90	Rehwoldt et al. (1973)
	stat	17		50	7.6	6.2	96-h LC_{50}	10	
Copepod (*Cyclops abyssorum*)	stat	10	0.58 meq/ litre		7.2		48-h LC_{50}	2200 (1500-3300)	Baudouin & Scoppa (1974)
Water flea (*Daphnia hyalina*)	stat	10	0.58 meq/ litre		7.2		48-h LC_{50}	5.5 (3.1-9.8)	Baudouin & Scoppa (1974)
Water flea	stat						48-h LC_{50}	1.8-4.3	Canton & Adema (1978)
(*Daphnia magna*)	stat	17-19	41-50	44-53	7.4-8.2		48-h LC_{50}	5	Biesinger & Christensen (1972)
	stat	17-19	41-50	44-53	7.4-8.2		21-day LC_{50}	13[e] (9-19)	
	stat	11.5-14.5	390-415	235-260	7.4-7.8	5.2-6.5	24-h LC_{50}	4890 (4190-5890)	Khangarot & Ray (1987)
	stat	11.5-14.5	390-415	235-260	7.4-7.8	5.2-6.5	48-h LC_{50}	3610 (2830-4400)	Khangarot & Ray (1987)
Water flea (*Daphnia pulex*)	stat						48-h LC_{50}	3.0	Canton & Adema (1978)
Water flea (*Daphnia cucullata*)	stat						48-h LC_{50}	3.2	Canton & Adema (1978)

Table 4 (Contd).

Copepod *(Eudiaptomus padanus)*	stat	10	0.58 meq/ litre		7.2		48-h LC_{50}	850 (710-1020)	Baudouin & Scoppa (1974)
Bristle worm *(Nais sp.)*	stat	17		50	7.6	6.2	24-h LC_{50}	1900	Rehwoldt et al. (1973)
	stat	17		50	7.6	6.2	96-h LC_{50}	1000	
Stone fly *(Acroneuria lycorias)*	stat	16-20	40	44	7.25	9.2	96-h LC_{50}	2000	Warnick & Bell (1969)
May fly *(Ephemerella subvaria)*	stat	16-20	40	44	7.25	9.2	96-h LC_{50}	2000	Warnick & Bell (1969)
Caddis fly *(Hydropsyche betteni)*	stat	16-20	40	44	7.25	9.2	96-h LC_{50}	2000	Warnick & Bell (1969)
Caddis fly *(unidentified sp.)*	stat	17		50	7.6	6.2	24-h LC_{50}	5600	Rehwoldt et al. (1973)
	stat	17		50	7.6	6.2	96-h LC_{50}	1200	
Damsel fly *(unidentified sp.)*	stat	17		50	7.6	6.2	24-h LC_{50}	3200	Rehwoldt et al. (1973)
	stat	17		50	7.6	6.2	96-h LC_{50}	1200	
Midge *(Chironomus sp.)*	stat	17		50	7.6	6.2	24-h LC_{50}	60	Rehwoldt et al. (1973)
	stat	17		50	7.6	6.2	96-h LC_{50}	20	

Table 4 (Contd).

Organism/ weight (g)	Stat/ flow[a]	Tem- perature (°C)	Alkali- nity[f]	Hard- ness[f]	pH	Dissolved oxygen (mg/litre)	Parameter	Water concentration (µg/litre)	Reference
Midge *(Chironomus riparius)*	flow[b]	20		50	6.8		24-h LC_{50}	1074 (760-1520)	Rossaro et al. (1986)
4th instar larvae	flow[b]	20		50	6.8		48-h LC_{50}	316 (230-440)	Rossaro et al. (1986)
	flow[b]	20		50	6.8		96-h LC_{50}	100 (50-180)	Rossaro et al. (1986)
	stat	20		50	6.8		24-h LC_{50}	1028 (880-1200)	Rossaro et al. (1986)
	stat	20		50	6.8		48-h LC_{50}	750 (660-850)	Rossaro et al. (1986)
	stat	20		50	6.8		96-h LC_{50}	547 (480-630)	Rossaro et al. (1986)

a stat = static conditions (water unchanged for duration of test).
b intermittent flow-through conditions.
c mercuric chloride was used except in the studies of Rehwoldt et al. (1973) and Khangarot & Ray (1987) (salt used unspecified).
d with food.
e extrapolated value from three concentrations less than the LC_{50}, daphnids were fed at the beginning of each week.
f alkalinity & hardness expressed as mg $CaCO_3$/litre unless otherwise stated.

were noted, even at the lowest mercury concentration tested (0.03 µg/litre). Fissioning was almost completely suppressed after 3 days in 0.1 µg/litre.

When Dorn (1974) exposed the bivalve mollusc *Congeria leucophaeata* for 48 h to mercuric chloride at concentrations of 0, 0.001, 0.01, 0.1, and 1.0 mg/litre, there was a significant increase, compared with controls, in respiration rate at all dose levels. The effect was dose related over the entire range. Stromgren (1982) exposed the mussel *Mytilus edulis* to mercuric chloride and found after 5 days a significant reduction in growth rate at 0.3 µg mercury/litre. At concentrations > 1.6 µg/litre, growth almost ceased within 3 to 4 days of exposure, while at 25 µg/litre acute lethal effects were observed within 24 h. Breittmayer et al. (1981) investigated the effects of metal concentration, size of organism, and seasonal differences on the toxicity of mercury to *Mytilus edulis*. The most important factor for mercury toxicity was season, though all factors interacted. MacInnes (1981) studied the effect of mercury on embryos of the American oyster *Crassostrea virginica*. The test was initiated 2 h after fertilization and continued for 48 h, the embryos then being checked for abnormal development (they did not undergo embryogenesis). The percentage of abnormal development for the test concentrations of 5 and 10 µg /litre were 6 and 15.7% for the chloride salt, and 2.9 and 9.8% for the nitrate. Dillon (1977) found that the 96-h LC_{50} for the estuarine marsh clam *Rangia cuneata* exposed to mercuric chloride was reduced from 0.122 to 0.058 mg/litre with an increase in salinity from 2 to 15‰. The pre-exposure of clams to 8.56 µg mercury/litre, followed by a period in clean water, significantly enhanced the survival of *Rangia* experimentally exposed to 0.87 mg mercury/litre. Results showed an LT_{50} of 135 h for unexposed clams compared to an LT_{50} of 210 h for pre-exposed clams.

Biesinger & Christensen (1972) found that in waterfleas *(Daphnia magna)* reproductive impairment was a more sensitive measure of the toxicity of mercuric chloride than survival. EC_{16} and EC_{100} values were 3.4 and 6.7 µg mercury/litre, respectively, for a 3-week exposure. Biesinger et al. (1982) exposed *Daphnia magna* to mercury (as mercuric chloride, methylmercuric chloride, or phenylmercuric acetate) in a chronic experiment over 3 weeks. The lowest concentrations of the three compounds to affect survival were 1.92, 0.2-0.98, and 2.25 µg/litre, respectively. Lowest concentrations affecting reproduction were 0.72, 0.04, and 1.90 µg/litre, respectively. All figures are in terms of mercury concentration in water.

Pyefinch & Mott (1948) studied the effect of mercuric chloride on the barnacles *Balanus balanoides* and *Balanus crenatus*. The toxicity of mercury to cyprids of *B. balanoides* was reduced by dilution of the sea water to reduce salinity. Older (11-12 day) larvae were less resistant than 1-day-old larvae. A mercury concentration of 0.01 mg/litre reduced the number of cyprids settling. Exposure of *B. balanoides* and *B. crenatus* after metamorphosis yielded median lethal concentrations, over 6 h, of 0.36 and 1.35 mg/litre, respectively.

Barnes & Stanbury (1948) found the median lethal concentration of mercuric chloride to the harpacticoid copepod *Nitocra spinipes* to be 0.6 mg mercury/litre. When the mercuric salt was added with copper sulfate, the chemicals acted synergistically. Lalande & Pinel-Alloul (1986) collected *Tropocyclops prasinus mexicanus* from three different Quebec lakes, two of low water hardness (10 mg $CaCO_3$/litre) and one of high (120 mg $CaCO_3$/litre). The lake with the high water hardness was polluted with human effluent. Animals from the two unpolluted lakes showed mean 48-h EC_{50}s (immobilization) of 0.015 and 0.045 mg/litre, whereas those from the polluted lake with a high water hardness showed an EC_{50} of 0.199 mg/litre.

When Sheally & Sandifer (1975) exposed newly-hatched grass shrimp *(Palaemonetes vulgaris)* larvae to mercury, a concentration of 56 µg/litre was lethal to all larvae within 24 h. No deaths occurred within 48 h when the shrimps were exposed to concentrations of 3.2 µg/litre or less. At 5.6 µg/litre, there were no deaths in fed larvae but some deaths occurred among unfed animals. The authors found that feeding slightly increased the resistance of *P. vulgaris* larvae to mercury. In surviving larvae some delayed effects of mercury were noted. Concentrations of 10 to 18 µg/litre caused a significant reduction in survival to the post-larval stage, a delayed moult, an extended development time, an increase in the number of larval instars, and an increase in the occurrence of deformities.

Portmann (1968) found that a reduction in temperature from 22 °C to 5 °C increased 5-fold the tolerance of brown shrimps to mercury (added as mercuric chloride) within 48 h. With cockles the effect was even more pronounced, increasing the 48-h LC_{50} by a factor of 130. It was also found that starving the animals reduced their tolerance to mercury. The 48-h LC_{50} for brown shrimps was halved (from 1.3 to 0.65 mg/litre) and reduced by a third in cockles (from 15.5 to 9.6 mg/litre). Larger shrimps were more resistant to mercury; the LC_{50} for the largest shrimps was 1.26 mg/litre, whereas that for the smallest was 0.58 mg/litre.

Brown & Ahsanullah (1971) studied the effects of mercuric chloride on the mortality of the adult brine shrimp *(Artemia salina)* and the worm *(Ophryotrocha labronica)*. After exposure to 1 mg mercury/litre, the LT_{50}s were 25 h for *Artemia* and 0.5 h for *Ophryotrocha*. Green et al. (1976) found that a 60-day exposure of post-larval white shrimp *(Penaeus setiferus)* to mercuric chloride (at either 0.5 or 1.0 µg mercury/litre) did not significantly affect respiratory rate, growth, or moulting rate.

In studies by Chinnayya (1971), mercuric chloride in freshwater (at 1 x 10^{-7}mol/litre) reduced oxygen consumption of the shrimp *Caridina rajadhari* from a control level of 0.485 ml/h per g wet weight of shrimps to 0.377 ml/h per g. This concentration of mercury caused no mortality over 10 days. The lowest concentration causing mortality in this species was 2.5 x 10^{-7}mol/litre.

Barthalamus (1977) found that concentrations of 2 and 5 mg mercuric chloride/litre killed 100% of grass shrimps *Palaemonetes pugio*, within

24 h, and 1 and 0.5 mg/litre over a period of 96 h. He calculated the 120 h LC_{50} to be 0.2 mg/litre, and found that 0.05 mg/litre significantly impaired the conditioned avoidance response.

Knapik (1969) studied the toxic effect of mercuric nitrate on four species of crustaceans, using concentrations of 10, 100, 200, and 500 mg mercuric nitrate/litre. The most sensitive species was *Neomysis vulgaris* (only 10% survived for 2 h at 10 mg/litre), followed by *Palaemonetes varians* and *Gammarus locusta. Rhithropanopeus harrisi tridentatus* was unaffected by a 3-h exposure to 100 mg/litre and 23% of animals survived 1 h at 500 mg/litre.

When Doyle et al. (1976) exposed crayfish *Orconectes limosus* to mercuric chloride, they observed 100% survival at 0.25 mg/litre over a period of 96 h. Survivors of a 96-h exposure to 1 mg/litre (the LC_{60}) showed a sluggish response to mechanical stimulation. Only occasional ventilative movements were observed in survivors of higher concentrations. All crayfish were dead within 96 h at 5 mg/litre.

Khayrallah (1985) studied the effect of both mercuric and methylmercuric chloride on the amphipod *Bathyporeia pilosa*. The toxicity of both inorganic and organic mercury was directly related to both concentration (0.04-0.75 mg mercury/litre) and temperature (1, 10, and 20 °C) and inversely related to salinity (10, 20, and $30^{o}/_{oo}$) and age (adult and juvenile).

Meadows & Erdem (1982) calculated LT_{50}s for *Corophium volutator* in 1 µg mercuric chloride/litre of about 30 days and in 1000 mg/litre of about 3 h. Krishnaja et al. (1987) studied the acute toxicity of phenylmercuric acetate to the intertidal crab *Scylla serrata* and calculated the 24-h, 48-h, 72-h, and 96-h LC_{50}s to be 700, 580, 540, and 540 µg/litre, respectively. DeCoursey & Vernberg (1972) exposed larval stages (zoea I, III, and V) of the fiddler crab *Uca pugilator* to mercuric chloride at concentrations of 0.018, 1.8, or 180 µg mercury/litre. No stage V, and only a few of stages I and III, survived 180 µg/litre for longer than 24 h. Vernberg et al. (1974) found that the adult fiddler crab *Uca pugilator* could survive prolonged periods of time in sea water (at 25 °C and a salinity of $30^{o}/_{oo}$) and at a mercuric chloride concentration of 0.18 mg mercury/litre. However, under temperature and salinity stress, survival periods were reduced. At 5 °C and $5^{o}/_{oo}$, LT_{50}s were 20 and 7 days, for females and males, respectively, and these were further reduced to 8 and 6 days, respectively, by the addition of 0.18 mg mercury/litre. When the temperature was increased to 35 °C, crabs survived to 28 days at low salinity, but the addition of mercury at 0.18 mg/litre again reduced survival, with LT_{50}s of 17 days for males and 26 days for females. Exposure of larvae revealed that 0.18 mg/litre was fatal to stage I zoeae, the LT_{50} being < 24-h. At 1.8 and 0.0018 mg/litre the 50% survival times were 8 days (stage II) and 11 days (stage III), respectively, compared to a control value of 18 days (stage IV).

McKenney & Costlow (1981) found that the survival of the megalopae stage of the blue crab *Callinectes sapidus* was highest at a salinity of $30^{o}/_{oo}$ and significantly reduced at $10^{o}/_{oo}$. Mercury at

10 µg/litre significantly increased the number of deaths of megalopae developing at 10‰ but not those at salinities of 20-40‰. At all salinities, fewer megalopae completed metamorphosis at 20 µg mercury/litre. Developmental times of the megalopae in the presence of 20 µg mercury/litre were increased to 8 to 10 days when the salinity was reduced to 10‰, and increased further, to nearly 13 days. Following metamorphosis, the crabs were found to be more resistant. There were no significant effects of salinity or mercury on survival or developmental duration at the first two adult crab stages.

Depledge (1984a) found that exposure of the shore crab *Carcinus maenus* to 0.05 mg mercuric sulfate/litre disrupted various endogenous rhythms. Locomotor activity increased and the mean heart rate rose from 32.1 beats/min to 44.7, although there was no change in the heart stroke volume (as indicated by a lack of change in the trace height of cardiograph readings). Exposure of crabs to 1 mg/litre suppressed cardiac activity and oxygen consumption. Alternating periods of bradycardia and tachycardia were observed together with marked changes in the heart stroke volume. There was an increase in the median perfusion index (volume of blood per unit volume of dissolved oxygen). All of the animals died within 24 to 48 h, this being associated with a loss of the ability to osmoregulate (Depledge, 1984b).

Weis (1980) exposed the fiddler crab *Uca pugilator* to a mixture of methylmercuric chloride (0.5 mg mercury/litre) and as zinc chloride (3 mg zinc/litre) and found the effect of the combination of metals on the retardation of limb regeneration to be additive. The effect was also additive at a reduced salinity (7-8‰).

6.2.2 Behavioural effects

Appraisal

Mercury appears to increase the probability of prey organisms being eaten by predators (at least in a single study). Prior exposure of prey organisms leads to the selection of a resistant strain and the effect of mercury, at the same concentration, disappears. The development of tolerance in invertebrates in the field must be taken into account when evaluating laboratory studies on test animals that have not experienced exposure to mercury before.

Kraus & Kraus (1986) tested predator avoidance in adult grass shrimps *(Palaemonetes pugio)* collected from two sites, one polluted with mercury (sediment mercury levels "as high as 10.3 mg/kg") and the other relatively pollution-free (sediment levels of 0.05 mg/kg). The shrimps were maintained in water containing either mercuric chloride or methylmercuric chloride (both at 0.01 mg/litre), for 96 h prior to testing. Killifish, collected only from the nonpolluted area, were then added to the tanks and the time between first and second captures of shrimp were noted. This was significantly reduced by both

inorganic and organic mercury in shrimp from the nonpolluted area. Control shrimp from the polluted area showed a reduced capture time compared to shrimp from the nonpolluted area, which was not reduced further by the mercury treatment. The overall survival of shrimps from the nonpolluted area, over 60 or 120 min of exposure to the predator, was not significantly affected by mercury treatment. In the shrimps from the polluted area, only the survival of shrimps in organic mercury, over the 60-min test period, showed a significant overall effect of the predator.

6.3 Toxicity to Fish

Appraisal

Inorganic mercury is toxic to fish at low concentrations. The 96-h LC_{50}s vary between 33 and 400 µg/litre for freshwater fish and are higher for sea water fish. Organic mercury compounds are more toxic. Toxicity is affected by temperature, salinity, dissolved oxygen, and water hardness. A wide variety of physiological and biochemical abnormalities have been reported after exposure of fish to sublethal concentrations of mercury. Reproduction is also adversely affected by mercury.

6.3.1 Acute and short term toxicity to fish

The acute toxicity of mercury to fish is summarized in Tables 5 and 6. Schweiger (1957) investigated the effects of mercury ions on fish and their food organisms and suggests a concentration of 0.03 mg mercury/litre as the toxic threshold for the various species tested.

Rodgers et al. (1951) investigated the toxicity of pyridyl mercuric acetate to three different species of trout. No deaths occurred in either brown trout or brook trout exposed to the compound at 10 mg/litre for 1 h. Rainbow trout were more susceptible with 99% mortality at 13 °C and 33% mortality at 8.5 °C. Deaths also occurred in rainbow trout exposed to 5 mg/litre (3% at 8.5 °C; 36% at 13 °C) but little mortality was noted at 2.5 mg/litre (0% at 8.5 °C; 2% at 13 °C). MacLeod & Pessah (1973) exposed rainbow trout *(Salmo gairdneri)* to mercuric chloride concentrations between 0 and 2 mg mercury/litre and calculated 96-h LC_{50}s of 0.4, 0.28, and 0.22 mg/litre at temperatures of 5, 10, and 20 °C, respectively. At 10 °C, the 24-h LC_{50} for mercuric chloride was approximately 30 times higher (in terms of mercury concentration) than for phenylmercuric acetate. Turnbull et al. (1954), using bluegill sunfish, calculated that the 24-h and 48-h LC_{50}s for pyridyl mercuric acetate were 12.5 and 11.3 mg/litre, respectively. Rehwoldt et al. (1972) measured the acute toxicity of inorganic mercury to six species of fish (Table 5), and found that it was less when tests were conducted at 15 °C than at 28 °C. Amend et al. (1969) exposed *Salmo gairdneri* to 125 µg ethylmercury phosphate/litre for 1 h, and found that increasing the temperature from 13 °C to

Table 5. Toxicity of inorganic mercury to fish[d]

Organism/ weight (g)	Stat/ flow[a]	Tem- perature (°C)	Alkali- nity[b]	Hard- ness[b]	pH	Dissolved oxygen (mg/litre)	Parameter	Water concentration (µg/litre)	Reference
Tilapia *(Tilapia mossambica)* 2.2-3.5	stat	28-30				> 4.8	48-h LC_{50}	1000 (792-1261)	Menezes & Qasim (1983)
10-13	stat	28-32	7.7-11.7	32-38	7-7.3	5.4-6.2	24-h LC_{50}	1256	Ramamurthi et al. (1982)
	stat	28-32	7.7-11.7	32-38	7-7.3	5.4-6.2	48-h LC_{50}	1108	
	stat	28-32	7.7-11.7	32-38	7-7.3	5.4-6.2	72-h LC_{50}	739	
Catfish *(Heteropneustes fossilis)* 25	stat	28					96-h LC_{50}	350	Das et al. (1980)
Catfish *(Sarotherodon mossambicus)* 25	stat	28-32	7.7-11.7	32-38	7-7.3	5.4-6.2	24-h LC_{50}	1700	Subbaiah et al. (1983)
	stat	28-32	7.7-11.7	32-38	7-7.3	5.4-6.2	48-h LC_{50}	1500	
	stat	28-32	7.7-11.7	32-38	7-7.3	5.4-6.2	72-h LC_{50}	1000	
	stat	28					96-h LC_{50}	75	Das et al. (1980)
Catfish *(Channa marulius)* 3-4.5		24-27.5	165-190	245-285	7.1-7.7	5.5-8.2	24-h LC_{50}	860 (801-916)	Khangarot (1981)
		24-27.5	165-190	245-285	7.1-7.7	5.5-8.2	96-h LC_{50}	314 (271-371)	Khangarot (1981)
		24-27.5	165-190	245-285	7.1-7.7	5.5-8.2	240-h LC_{50}	131 (103-158)	Khangarot (1981)

Table 5 (Contd).

Rainbow trout (*Salmo gairdneri*) 0.6-3.0	stat	9.3-10.7	70	101	8.55	> 8.0	24-h LC_{50}	903 (783-1023)	Wobeser (1975a)
9.1-15.5	stat	5		90	7.5-7.8		48-h LC_{50}	650	MacLeod &
	stat	5		90	7.5-7.8		96-h LC_{50}	400	Pessah
13.2-21.3	stat	10		90	7.5-7.8		48-h LC_{50}	450	(1973)
	stat	10		90	7.5-7.8		96-h LC_{50}	280	MacLeod &
18.5-27.8	stat	20		90	7.5-7.8		48-h LC_{50}	300	Pessah
	stat	20		90	7.5-7.8		96-h LC_{50}	220	(1973)
length: 51-76mm	flow		82-132		6.4-8.3	4.8-9.0	96-h LC_{50}	33[d]	Hale (1977)
Banded killifish (*Fundulus diaphanus*)	stat	28		55	8.0	6.9	24-h LC_{50}	270	Rehwoldt
	stat	28		55	8.0	6.9	48-h LC_{50}	160	et al.
	stat	28		55	8.0	6.9	96-h LC_{50}	110	(1972)
Striped bass (*Roccus saxatilis*)	stat	28		55	8.0	6.9	24-h LC_{50}	220	Rehwoldt
	stat	28		55	8.0	6.9	48-h LC_{50}	140	et al.
	stat	28		55	8.0	6.9	96-h LC_{50}	90	(1972)
Pumpkinseed (*Lepomis gibbosus*)	stat	28		55	8.0	6.9	24-h LC_{50}	410	Rehwoldt
	stat	28		55	8.0	6.9	48-h LC_{50}	390	et al.
	stat	28		55	8.0	6.9	96-h LC_{50}	300	(1972)
White perch (*Roccus americanus*)	stat	28		55	8.0	6.9	24-h LC_{50}	420	Rehwoldt
	stat	28		55	8.0	6.9	48-h LC_{50}	340	et al.
	stat	28		55	8.0	6.9	96-h LC_{50}	220	(1972)

Table 5 (Contd).

Organism/ weight (g)	Stat/ flow[a]	Tem- perature (°C)	Alkali- nity[b]	Hard- ness[b]	pH	Dissolved oxygen (mg/litre)	Parameter	Water concentration (µg/litre)	Reference
Carp	stat	28		55	8.0	6.9	24-h LC_{50}	330	Rehwoldt
(Cyprinus carpio)	stat	28		55	8.0	6.9	48-h LC_{50}	210	et al.
	stat	28		55	8.0	6.9	96-h LC_{50}	180	(1972)
American eel	stat	28		55	8.0	6.9	24-h LC_{50}	250	Rehwoldt
(Anguilla rostrata)	stat	28		55	8.0	6.9	48-h LC_{50}	190	et al.
	stat	28		55	8.0	6.9	96-h LC_{50}	140	(1972)
Mummichog *(Fundulus heteroclitus)*		20	20[c]		8.0		96-h LC_{50}	2000	Klaunig et al. (1975)
3.3-3.5									
2-6	stat	20	20[c]		7.8	< 4	24-h LC_{50}	23 000	Eisler &
	stat	20	20[c]		7.8	< 4	96-h LC_{50}	800	Henneky
	stat	20	20[c]		7.8	< 4	168-h LC_{50}	800	(1977)
Flounder (adult) *(Platichthys flesus)*	stat	15					48-h LC_{50}	3300	Portmann & Wilson (1971)

[a] stat = static conditions (water unchanged for duration of test); flow = flow-through conditions (mercury concentration in water continuously maintained).

[b] alkalinity & hardness expressed as mg $CaCO_3$/litre.

[c] These figures are values for salinity (expressed in $^{o}/_{oo}$), not alkalinity.

[d] Mercuric chloride was used, except in the studies of Hale (1977), where the salt used was mercurous nitrate.

Table 6. Toxicity of organic mercury to fish[c]

Organism/ weight (g)	Stat/ flow[a]	Tem- perature (°C)	Alkali- nity[b]	Hard- ness[b]	pH	Dissolved oxygen (mg/litre)	Parameter	Water concentration (µg/litre)	Reference
Blue gourami	stat	26-28			7.4	10	24-h LC_{50}	123 (115.62-130.38)	Roales & Perlmutter (1974)
(Trichogaster trichopterus) 1.5-2	stat	26-28			7.4	10	48-h LC_{50}	94.2 (85.5-102.9)	Roales & Perlmutter (1974)
	stat	26-28			7.4	10	96-h LC_{50}	89.5 (85.38-93.62)	
Rainbow trout (fry)	stat	9.3-10.7	70	101	8.55	> 8	24-h LC_{50}	84 (81-87)	Wobeser (1975a)
(Salmo gairdneri)	stat	9.3-10.7	70	101	8.55	> 8	48-h LC_{50}	45 (36-54)	
	stat	9.3-10.7	70	101	8.55	> 8	96-h LC_{50}	24 (22-26)	
(fingerling) 0.6-3	stat	9.3-10.7	70	101	8.55	> 8	24-h LC_{50}	125 (120-130)	Wobeser (1975a)
	stat	9.3-10.7	70	101	8.55	> 8	48-h LC_{50}	66 (63-69)	
	stat	9.3-10.7	70	101	8.55	> 8	96-h LC_{50}	42 (25-59)	
(juvenile) 22.9	flow	10		90	7.5-7.8		24-h LC_{50}	25[c]	MacLeod & Pessah (1973)
	stat	18		250			24-h LC_{50}	5[c]	Alabaster (1969)
	stat	18		250			48-h LC_{50}	4[c]	
Brook trout (juv.) *(Salvelinus fontinalis)*	flow	11-13	41-44	45-46	6.9-7.6	7.7	96-h LC_{50}	75	McKim et al. (1976)
Lamprey (larvae)	flow	12	150	146	8-8.5		24-h LC_{50}	> 166	Mallatt et al. (1986)
(Petromyzon marinus)	flow	12	150	146	8-8.5		48-h LC_{50}	88	
0.3-3	flow	12	150	146	8-8.5		96-h LC_{50}	48	

[a] stat = static conditions (water unchanged for duration of test); flow = flow-through conditions (mercury concentration in water continuously maintained).

[b] alkalinity & hardness expressed as mg $CaCO_3$/litre.

[c] methylmercuric chloride was used, except in the studies of MacLeod & Pessah (1973) and Alabster (1969), where phenyl mercuric acetate was used.

15 °C tended to increase the acute toxicity of the mercury solution. An increase in the water hardness from 23 to 120 mg $CaCO_3$/litre also decreased the toxicity. But the dissolved oxygen content of the water had the most pronounced effect. At saturation, no deaths occurred, even at the highest water hardness, but at a dissolved oxygen level of < 6 mg/litre substantial losses occurred (72-76%) and even at the lowest temperature 37% of the trout died.

Jones (1940) found that the mean survival time for the minnow *Phoxinus phoxinus* in mercuric chloride rose from 15 min for 10^{-3}mol/litre to 230 min at 5×10^{-6}mol/litre. The addition of enough sodium chloride to convert the whole of the mercuric chloride into a double-chloride sodium mercuric chloride, and even the addition of ten times this amount, did not affect the toxicity of the solution. The addition of a considerable excess of sodium chloride caused a marked prolongation of the survival time, the maximum effect being attained when the solution was approximately isotonic.

6.3.2 Reproductive effects and effects on early life stages

Appraisal

The data reveal an obvious difference between static and flow test concentrations, with LC_{50} values being up to 150 times lower under flow conditions. The increased LC_{50} in the static tests may be explained by a combination of adsorption of the compound to surfaces of the test vessels and to the gelatinous egg surface during embryo development. As a result, the larvae are exposed to much lower mercury concentrations at hatching time than are present at the beginning of the experiment. By contrast, the concentration is maintained throughout in a flow-through system.

Selenium may increase the toxicity of mercury to fish eggs at higher concentrations of mercury. At low water concentrations selenium effects are additive.

Table 7 summarizes the acute toxicity of mercury to embryolarval stages of fish.

When Ram & Sathyanesan (1983) exposed adults of the freshwater teleost *Channa punctatus* for 6 months to 0.01 mg mercuric chloride/litre, the mercury prevented oocytes development in the ovary and spermatogenesis in the testis. The number and activity of gonadotrophs in the pituitary were also reduced, giving the appearance of "resting phase" at a time when full reproductive development was expected. McIntyre (1973) exposed sperm from *Salmo gairdneri* to concentrations of methylmercuric chloride between 1 µg/litre and 10 mg/litre for 30 min. The sperm-containing solution was then added to eggs, and the percentage of fertilization was determined 17 days later. After exposure to 0.5 mg mercury/litre, there was an increase in the percentage of unfertilized eggs from 9.1% in controls to 12.5% in treated samples. This effect was enhanced with increasing mercury

Table 7. Toxicity of inorganic mercury to the embryo-larval stages of fish

Organism	Stat/ flow	LC_{50} (μg/litre)	95% confidence limits
Rainbow trout (*Salmo gairdneri*)	stat[a]	4.7	4.2-5.3
	flow[b]	< 0.1	
Channel catfish (*Ictalurus punctatus*)	stat[a]	30.0	26.9-33.2
	flow[b]	0.3	0.2-0.4
Bluegill sunfish (*Lepomis macrochirus*)	stat[a]	88.7	73.5-106.3
Goldfish (*Carassius auratus*)	stat[a]	121.9	112.3-132.1
	flow[b]	0.7	0.6-0.8
Redear sunfish (*Lepomis microlophus*)	stat[a]	137.2	115.0-162.8
Largemouth bass (*Micropterus salmoides*)	stat[a]	140.0	128.7-151.9
	flow[b]	5.3	5.0-5.6

[a] static conditions but water renewed every 12 h.
[b] flow-through conditions (mercury concentration in water continuously maintained).

Exposure was initiated 30 min to 2 h after spawning and continued through to 4 days post-hatching. Hatching times were 24 days for rainbow trout, 6 days for channel catfish, and 3 to 4 days for the other fish. Therefore, total exposure was as follows: rainbow trout 28 days, channel catfish 10 days, and the other fish 7 to 8 days. (Birge et al. 1979).

concentration, reaching 100% nonfertile eggs at 5 mg/litre or greater concentrations of mercury.

Kihlstrom & Hulth (1972) transferred eggs laid by mature zebrafishes *(Brachydanio rerio)* into solutions containing 10, 20, or 50 μg phenylmercuric acetate (PMA)/kg. The frequency of hatching was significantly higher in the 10 μg/kg group than in the controls and the same as the controls in the 20 μg/kg group. None of the eggs transferred to the solution containing 50 μg/kg hatched. Most eggs hatched 3 days after fertilization, the frequency of eggs hatching up to and including the third day being significantly higher in water containing 10 or 20 μg PMA/kg when compared to the controls.

Weis & Weis (1977) exposed early embryos of the killifish *Fundulus heteroclitus* to mercuric chloride at concentrations of 0.01, 0.03, 0.1, or 1.0 mg mercury/litre. Mercury was added to the water at the start of the experiment and the solution was not replaced. The authors cite Jackim et al. (1970) to indicate that the loss of mercury from the

solution would have amounted to about 26% over the course of the 96-h test period. Embryos treated at stage 12 of development (the early blastula stage) showed a reduction in axis formation in solutions of 0.01 and 0.03 mg mercury/litre and a severe reduction at 0.1 mg/litre. There were no forebrain defects at 0.01 mg, but 20% of embryos showed defects after exposure to 0.03 and 0.1 mg/litre. All the embryos exposed to 1 mg mercury/litre died before gastrulation. Embryos treated at stage 14, the late blastula stage of development, with concentrations of mercury of 0.01-0.1 mg/litre), showed no reduction in axis formation. Negligible defects were noted at 0.01 and 0.03, but 20% of embryos were affected at 0.1 mg mercury/litre.

When Sharp & Neff (1980) exposed embryos (4-8 cell stage) of *Fundulus heteroclitus* to mercuric chloride at concentrations of 0-100 µg mercury/litre for 1 to 32 days, survival was reduced at all concentrations above 40 µg/litre. The hatching success of embryos exposed for 32 days was significantly reduced at concentrations above 10 µg/litre. Reducing the duration of exposure from 5 days to 1 day significantly increased the total hatchability of the eleutheroembryos emerging after exposure for 32 days. Increases in the incidence of spinal curvature were also noted at concentrations exceeding 20 µg/litre, which were significantly reduced if the exposure was reduced to 5 days or less. The 24-h LC_{50} for the embryos was 89.6 µg/litre, the 24-h EC_{50} for spinal curvature was 61.45 µg/litre, and the 24-h EC_{50} for hatching success was 71.6 µg/litre.

McKim et al. (1976) exposed three generations of brook trout *(Salvelinus fontinalis)* to methylmercuric chloride concentrations of 0.03 to 2.93 µg/litre, over a 144-week period. At the highest dose, deformities were observed during the first 39 weeks and 88% of the first generation adults died. At 0.93 µg/litre, the second generation fish showed deformities and all but one female died during a 108-week exposure. No significant effects on survival, growth, or reproduction were observed in second generation trout at concentrations lower than 0.93 µg/litre, and no toxic symptoms were found in the third generation below 0.29 µg/litre. The authors established that the maximum acceptable toxicant concentration (MATC) for brook trout exposed to methylmercuric chloride (hardness = 45 mg $CaCO_3$/litre; pH = 7.5) was between 0.29 and 0.93 µg/litre.

Weis & Weis (1984) measured the tolerance of eggs of the killifish *Fundulus heteroclitus* to methylmercury in four successive years of sampling in the same pond. There was considerable variation in susceptibility between eggs from different females at the beginning of the sampling period, some females producing resistant and some susceptible eggs. After a period of heavy rainfall in the third year, when heavy metals and pesticides were washed into the ponds, the proportion of resistant eggs in the population increased. The authors noted an initial correlation between production of resistant eggs and numbers of fin rays in females. The same correlation indicated an increase in these females in the population after exposure to metals. Selection, rather than physiological adoption, had taken place.

Birge et al. (1979) investigated the effects of combinations of mercury and selenium on the hatchability of eggs of the rainbow trout, catfish, goldfish, and bass. Mercury and selenium were added to the test medium in a 1:1 ratio over a wide range of concentrations (from 1 to 2500 μg/litre). From separate tests with mercury and selenium alone, the author calculated additive values for the two materials and compared the results with those observed with the mixture. For each species, results were dependant on actual concentration. At lower concentrations the interaction between mercury and selenium was additive or antagonistic, whereas at higher concentrations interaction was synergistic, with the mixture leading to much greater inhibition of hatching than predicted. Calculated additive LC_{50}s for mercury and selenium were 0.09 mg/litre for trout, 0.1 mg/litre for catfish, 0.67 mg/litre for goldfish, and 0.35 mg/litre for bass. Actual LC_{50}s for the mixture of 1:1 mercury:selenium were 0.01 mg/litre for trout, 0.01 mg/litre for catfish, 0.16 mg/litre for goldfish, and 0.35 mg/litre for bass, in all cases substantially greater toxicities than predicted. For the two most sensitive species, trout and catfish, synergism became evident at water concentrations of 5 μg/litre and increased in parallel with increasing concentration. At water concentrations of 75 μg/litre, the predicted hatchability of eggs (assuming mercury and selenium effects to be additive) was 44% for trout and 57% for catfish. Actual observed hatchability at this concentration was 0% for trout and 2% for catfish.

6.3.3 Behavioural effects

Weir & Hine (1970) pretrained goldfish *(Carassius auratus)* to avoid electric shock with a light stimulus and then exposed them to solutions of mercuric chloride. The lowest concentration of mercuric chloride found to significantly impair the behavioural response was 3 μg/litre. The lowest concentration causing deaths, under the same conditions, was 360 μg/litre. Hartman (1978) fed rainbow trout *(Salmo gairdneri)* for a year on a diet containing ethylmercury (*p*-toluene sulfonanilide) "Ceresan" at 0.5-25 mg/kg diet each day, or 2.5 or 10 mg/kg delivered every fifth day of feeding. Fish receiving 10 mg/kg every 5 days or 5 mg/kg or more per day were unable, with few exceptions, to learn to avoid a shock preceded by a signal of light. However, there was no evidence of the impairment of general behaviour.

When Sharma (1984) exposed *Channa punctatus* to mercuric chloride (at concentrations of 0.034, 0.068, 0.102, or 0.136 mg/litre for 1, 7, 15, 30, or 45 days), hyperactive avoidance reaction was seen after exposure to the two highest doses within 24 h. Similar reactions occurred with the lower doses after 5 days (0.034 mg/litre) and 2 days (0.068 mg/litre). Acute distress symptoms were noted at the lowest two exposure levels during the last 5 days of the experiment. Feeding was normal up to 20 days of exposure at 0.034 mg mercury/litre, 12 days at 0.068 mg/litre, 6 days at 0.102 mg/litre, and only 3 days at

0.136 mg/litre. There were deaths in all treated groups within 45 days, ranging from 16% at 0.034 mg/litre to 100% at 0.102 mg mercury/ litre or more. Growth was inhibited by all treatments in proportion to the mercury dose. Blood glucose level showed an early elevation followed by a significant reduction, the timing of the effect varying according to dose. There was also a progressively significant depletion in liver and muscle glycogen which was similarly dose dependant.

6.3.4 Physiological and biochemical effects

Panigrahi & Misra (1978) found that concentrations of mercuric nitrate of 5 mg/litre or more killed all test fish *Anabas scandens* within 24 h. At 3 mg/litre, the fish survived but showed pathological and biochemical disorders. The major clinical disorders (lack of movement and reduced food consumption) showed themselves within 5 days of exposure. After 3 weeks, 29% of the fish were blind and their respiratory rate was greatly reduced; 71% were blind within 4 weeks. When the fish were transferred to clean water, partial recovery to normal respiratory rate occurred. Considerable reductions in blood haemaglobin content, erythrocyte count, body weight, and body protein content were recorded.

Lindahl & Hell (1970) exposed the roach *Leuciscus rutilus* to phenylmercuric hydroxide at 1 mg/litre for 40 min, then killed the fish and measured the respiration rate of isolated gill filaments. Filament respiration was reduced by about 30%, the cause being damage to secondary lamellae, and the oxygen content of blood was reduced by 82%. An *in vitro* study with erythrocytes showed that half the cells haemolyzed after exposure for 55 min to 0.5×10^{-4}mol phenylmercuric hydroxide/litre.

Hara et al. (1976) studied the effect of mercuric chloride on the olfactory response of the rainbow trout *Salmo gairdneri*. Mercury depressed the response, the lowest concentration to cause an appreciable effect within 2 h being 100 µg/litre. The depression increased with increases in mercury concentration and exposure time.

Hilmy et al. (1982) exposed the cyprinodont *Aphanius dispar* to acute concentrations of mercury of 1-12 mg/litre for 96 h or chronic concentrations of 1 mg/litre for up to 30 days. The acute treatment caused significant increases in plasma sodium, calcium, and potassium levels, which reached maxima of 3, 5, and 12 mg/litre, respectively. At the chronic exposure, the levels of sodium, calcium, and potassium initially rose, then fell to near normal levels by the end of the 30-day experiment.

Das et al. (1980) studied the acute and subacute toxicity of mercuric chloride to the air-breathing fish *Heteropneustes fossilis* and the non-air-breathing fish *Sarotherodon mossambica*. The air-breathing fish was more resistant to mercury, giving a 96-h LC_{50} value of 350 µg mercury/litre compared to 75 µg/litre for *Sarotherodon*. The effect on several enzymes of mercury at

50 µg/litre was also studied. Gill lysosomal acid phosphatase and liver microsomal glucose-6-phosphatase were significantly stimulated in both species, whereas liver acid phosphatase and intestinal alkaline phosphatase were significantly stimulated in *Heteropneustes* and significantly inhibited in *Sarotherodon*. In both species serum glucose levels were significantly increased and liver glycogen levels decreased, while muscle glycogen levels were unaffected.

Gill & Pant (1985) exposed *Barbus conchonius* to concentrations of mercuric chloride of 36, 60, or 181 µg/litre, the highest dose corresponding to the 96-h LC_{50} for the species. Acute exposure to 181 µg/litre for 24 or 48 h led to deformities in the erythrocytes: vacuolation, nuclear deterioration, microcytosis, and collapsed cytoplasmic membranes. There was also significant thrombocytosis and neutropenia. Chronic exposure to 36 or 60 µg mercury/litre led to poikilocytosis, hypochromia, fragmentation and nuclear displacement of erythrocytes, thrombocytosis, lymphocytosis, neutropenia, and mild basophilia.

When Ramalingam & Ramalingam (1982) exposed the catfish *Sarotherodon mossambicus* to a concentration of mercuric chloride of 0.09 mg mercury/litre, they found no effect on the liver or muscle total protein content over 24 h. There were, however, significant decreases after both 7 and 15 days.

Verma et al. (1984) dosed the lungfish *Notopterus notopterus* with mercuric chloride concentrations of 0.017-0.088 mg/litre for up to 60 days. Concentrations of 0.022 or more caused significant increases in serum glutamic oxaloacetic transaminase and serum glutamic pyruvic transaminase activities within 15 days. The lowest dose took at least 30 days to significantly increase the activity of the same enzymes.

O'Connor & Fromm (1975) exposed rainbow trout *Salmo gairdneri* to methylmercuric chloride, at 10 µg mercury/litre, in a flow-through system. The fish were killed and assayed at 4, 8, and 12 weeks. There was no significant difference in plasma electrolyte concentrations (Na^+, K^+, Cl^-, Mg^{2+}, and Ca^{2+}) or between the *in vitro* oxygen consumption of excised gill filaments from control and mercury-treated fish determined in 10% or 100% phosphate-buffered saline.

In studies by Sastry et al. (1982), the frehwater murrel *Channa punctatus* was exposed to mercury either directly once into the intestinal sac (0.001-10 mmol/litre) or in the water at 3 µg/litre for 15 or 30 days. A significant decrease in the rate of intestinal absorption of glucose, fructose, glycine, and trytophan occurred at the higher concentrations of 0.1, 1.0, and 10 mmol/litre. At 0.01 and 0.001 mmol/litre there was a reduction in absorption but this was not significant except at 0.01 mmol/litre in the case of tryptophan. There was a significant decrease of the absorption rate of all four nutrients in the mercury solution, but only after a 30-day exposure.

Dawson et al. (1977) exposed juvenile striped bass *Morone saxatilis* to 1.0, 5.0, or 10 µg mercuric chloride/litre for between 30 and 120

days. The fish were then allowed to recover for a further 30 days in clean running sea water. Fish exposed to the lowest dose did not differ significantly from controls with regard to respiration rate. Exposure to 5 μg/litre for 30 days significantly lowered the respiration rate but the effect had disappeared after 60-days exposure. Fish exposed to 10 μg mercury/litre showed a decreased respiratory rate after 30 days, which was reversed until a significant increase in rate was observed after 120 days of exposure. Mercury exposure did not significantly affect liver activities of aspartate aminotransferase, glucose-6-phosphatase, malic dehydrogenase, or magnesium activation of aspartate aminotransferase.

Christensen (1975) examined a range of biochemical parameters in brook trout *(Salvelinus fontinalis)* embryos and alevins exposed to methylmercuric chloride at concentrations from 0.01 to 1.03 μg mercury/litre. The fish were exposed as eggs for 16-17 days and then for a further 21 days as alevins. There was a significant decrease in glutamic oxaloacetic transaminase activity in embryos after exposure to 1.03 μg/litre, and a significant increase in its activity in alevins at 0.93 μg/litre. The alevin effect was accompanied by a significant decrease in weight. Christensen et al. (1977) exposed brook trout to methylmercuric chloride concentrations of 0.01 or 0.03 μg/litre and 2.93 μg/litre, for either 2 or 8 weeks. After 8 weeks, they found no significant effects on body weight, body length, blood plasma glucose, chloride or sodium, or plasma lactic dehydrogenase, and glutamic oxaloacetic transaminase activities. There were, however, significant increases in haemoglobin and blood plasma sodium and chloride after 2 weeks, but no effect on the other parameters measured.

Varanasi et al. (1975) noted structural alterations in the epidermal mucus of rainbow trout exposed to 1 mg of mercuric chloride/litre. Mercury accumulated in the mucus and altered the physical characteristics of the layer, which is important for locomotion and protection of the fish. Lock & Overbeeke (1981) studied the effects of methylmercuric chloride and mercuric chloride on mucus production in rainbow trout. Of three measurements made, density of mucus cells, mucus in the tissue, and release of mucus into water, only the latter was affected by mercury. The effect was less with organic than with inorganic mercury, where mucus production was increased significantly. Exposure to 10 μg inorganic mercury/litre for 4 h increased mucus production, and greater exposure concentrations and times enhanced the effect. Opercular movements increased with increased mucus production, suggesting mucus-induced hypoxia. Lock et al. (1981) attributed the osmoregulatory effect of mercury on fish as an effect on the permeability of the gill to water, rather than as an effect on active ionic transport.

Roales & Perlmutter (1977) found that methylmercury (9 μg /litre), or methylmercury and copper combined, resulted in a decrease in the immune response of blue gourami *(Trichogaster trichopterus)* to both infectious pancreatic necrosis (IPN) virus and *Proteus vulgaris*. The

two toxicants jointly produced no greater or lesser effect than when each was added alone.

6.4 Toxicity to Amphibia

Mercury has a toxicity for amphibian tadpoles similar to that for fish. There is considerable species variability in susceptibility to the metal. Sublethal effects and developmental effects have been reported. There is no information on effects on adult amphibians.

Acute toxicity of mercury to amphibian tadpoles is summarized in Table 8.

Birge et al. (1979) conducted embryo-larval bioassays on 14 species of amphibia. Exposure to inorganic mercury was maintained from fertilization to 4 days after hatching, using static renewal procedures (Table 9). Gastrophryne and five species of *Hyla* were the most sensitive, with LC_{50} values ranging from 1.3 to 2.8 μg/litre, compared to an LC_{50} value of 4.7 μg/litre for rainbow trout (the exposure period was shorter than for the trout; 6.6 to 7.4 days compared to 28 days.

Chang et al. (1974) dosed leopard frog *(Rana pipiens)* tadpoles with methylmercuric chloride, either via the water at concentrations of 0-1.0 mg mercury/litre or via injections of 0.025 mg mercury/day for 10 days. There was 100% mortality after 48 h at a water concentration of 50 μg/litre or more. At 1-10 μg/litre there was total arrest of development and differentiation after 48 h, which continued for 3 to 4 months. Mercury-injected tadpoles showed extensive deposition of blood pigment in their livers. The authors suggest that this was due to haemolysis of red blood cells caused by mercury, followed by severe peripheral oedema and haemopoietic reactions in the kidneys of the tadpoles. Dial (1976) exposed *Rana pipiens* embryos (at the cleavage, blastula, gastrula, and neural-plate stages of development) to concentrations of methylmercuric chloride of 0.5-200 μg/litre. Concentrations of 40 μg/litre or more were lethal to embryos treated at the cleavage stage. Embryos at the blastula, gastrula, and neural-plate stages were treated for 5 days at concentrations of 5-30 μg/litre. Tadpoles treated with 5 μg/litre showed only minor effects, whereas 10, 15, or 20 μg/litre caused various effects, including exogastrulae, poor tail development, and poor general development. Death rates increased with exposure time and concentration. At 30 μg/litre many defects were observed after 24 h and all tadpoles had died within 3 days.

6.5 Toxicity to Aquatic Mammals

There appears to be only a single experimental study on the effects of methylmercury on aquatic mammals. Ronald et al. (1977) fed harp seals on herring dosed with methylmercuric chloride. Two animals were used as controls, two were fed 0.25 mg/kg body weight per day and two

Table 8. Toxicity of mercuric chloride to amphibians

Organism	Life-stage	Stat/flow[a]	Temperature (°C)	Alkalinity[e]	Hardness[e]	pH	Parameter	Water concentration (µg/litre)	Reference
Frog[c]	tadpole	stat	13-16	24-40	13-80	6.2-6.7	24-h LC_{50}	762 (677-837)	Khangarot
(Rana hexadactyla)	tadpole	stat[b]	13-16	24-40	13-80	6.2-6.7	48-h LC_{50}	121 (93-151)	et al. (1985)
	tadpole	stat[b]	13-16	24-40	13-80	6.2-6.7	72-h LC_{50}	68 (57-85)	Khangarot
	tadpole	stat[b]	13-16	24-40	13-80	6.2-6.7	96-h LC_{50}	51 (33-53)	et al. (1985)
Clawed toad *(Xenopus laevis)*	3- to 4-week larva	stat	19-21				48-h LC_{50}	100	de Zwart & Slooff (1987)
Toad[d]	tadpole	stat	29-34	120-160	165-215	7.1-7.6	12-h LC_{50}	69.8	Khangarot &
(Bufo melanostictus)	tadpole	stat	29-34	120-160	165-215	7.1-7.6	24-h LC_{50}	52.8 (43.6-61.5)	Ray (1987)
	tadpole	stat	29-34	120-160	165-215	7.1-7.6	48-h LC_{50}	45.6 (40.9-56.7)	Khangarot &
	tadpole	stat	29-34	120-160	165-215	7.1-7.6	96-h LC_{50}	43.6 (36.8-58.5)	Ray (1987)

[a] stat = static conditions (water unchanged for duration of test).
[b] static conditions but test water renewed every 24 h.
[c] tadpole length 15-25 mm, weight 350-800 mg (wet weight).
[d] tadpole length 18-22 mm, weight 90-120 mg (wet weight).
[e] alkalinity & hardness expressed as mg $CaCO_3$/litre.

Table 9. Toxicity of inorganic mercury to the embryo-larval stage of amphibians

Organism	LC_{50} (μg/litre)	95% confidence limits
Narrow-mouthed toad (*Gastrophryne carolinensis*)	1.3	0.9-1.9
Southern grey tree frog (*Hyla chrysoscelis*)	2.4	1.5-3.4
Squirrel tree frog (*Hyla squirrella*)	2.4	1.5-3.8
Barking tree frog (*Hyla gratiosa*)	2.5	1.7-3.4
Grey tree frog (*Hyla versicolor*)	2.6	1.2-4.2
Spring peeper (*Hyla crucifer*)	2.8	1.9-3.9
Leopard frog (*Rana pipiens*)	7.3	4.8-10.0
Cricket frog (*Acris crepitans blanchardi*)	10.4	8.5-12.6
Red-spotted toad (*Bufo punctatus*)	36.8	18.3-51.1
Green toad (*Bufo debilis debilis*)	40.0	25.6-52.2
River frog (*Rana heckscheri*)	59.9	53.8-65.9
Fowlers toad (*Bufo fowleri*)	65.9	44.0-84.0
Pig frog (*Rana grylis*)	67.2	54.3-79.5
Marbled salamander (*Ambystoma opacum*)	107.5	72.5-153.5

Exposure was under static conditions (but water renewed every 12 h), and was initiated 30 min to 2 h after spawning and continued to 4 days post-hatching. Hatching times varied from 2.6 to 3.4 days, therefore total exposure was between 6.6 and 7.4 days. (Birge et al., 1979).

fed 25.0 mg/kg body weight per day. Various blood parameters were monitored and found to be unaffected by the lower dose. The two animals on the higher dose died after 20 and 26 days of dosing. Prior to death these animals exhibited toxic hepatitis, uremia, and renal failure.

7. TOXICITY TO TERRESTRIAL ORGANISMS

7.1 Toxicity to Terrestrial Plants

Appraisal

The main problem with studies on the effects of mercury on terrestrial plants is their relevance to the natural situation. Mercury normally binds to soil particles, which may reduce its availability to plants. In most studies, mercury has been administered as a solution in hydroponic culture. Most of the experiments have been on crop plants; wild plants might behave differently.

Oberlander & Roth (1978) measured the uptake and translocation of potassium and phosphate, into the roots and shoots of 7-day-old barley plants, from doubly labelled (^{42}K, ^{32}P) nutrient solutions containing mercuric chloride. Uptake and translocation was monitored over 5 h during exposure to mercury at 10^{-4}mol/litre. Potassium and phosphate uptake was significantly reduced to 21% and 31%, respectively, of the control level. Potassium and phosphate translocation was also significantly reduced to 6% and 8%, respectively, of the control level.

Barker (1972) exposed explants of cauliflower inflorescence stem, lettuce stem, secondary phloem of carrot root, and tubers of potato for 20 days to mercuric chloride at concentrations between 0.005 and 50 mg mercury/litre of medium. There was a significant reduction in growth (measured as mean fresh weight) after exposure to 0.5 mg/litre or more, although carrot and potato showed significant increases in growth at low levels (0.005 mg/litre) of mercury.

Mhatre & Chaphekar (1984) exposed young plants of three species (a cereal *Pennisetum typhoideum*, a forage crop *Medicago sativa*, and a vegetable *Abelmoschus esculentus)* to solutions containing mercuric chloride at 1-1000 µg mercury/litre for 24 h. They then estimated the percentages of leaf area injured and number of leaves injured. *Abelmoschus* was found to be the least sensitive of the plants, showing no damage at 10 µg/litre, whereas the other two species showed injury at this concentration. All species showed increasing percentages of leaf area injury and number of leaves injured with increasing mercury exposure. At the highest dose, 1000 µg/litre, all leaves were injured in *Pennisetum* and *Abelmoschus* and 50% of the leaves of *Medicago*.

7.2 Toxicity to Terrestrial Animals

7.2.1 Toxicity to terrestrial invertebrates

Appraisal

The experimental information available on the effects of mercury on terrestrial invertebrates is insufficient to make any proper appraisal.

Marigomez et al. (1986) fed the terrestrial slug *Arion ater* for 27 days on a diet containing mercuric chloride at 0, 10, 25, 50, 100, 300, or 1000 mg/kg. The number of slugs dying was low in all treatments (a maximum of three deaths out of 24 animals per treatment) and unrelated to the dose. The results indicated that exposure of slugs to mercury at levels likely to be found in the environment will not kill them. A significant reduction in food consumption was noted at mercury exposures > 10 mg/kg diet, the effect being dose-related. A significant dose-related reduction in growth rate also occurred. Only at the highest dose (1000 mg/kg diet) did mercury severely disrupt growth.

Abbasi & Soni (1983) kept the earthworm *Octochaetus pattoni* in cement tanks at a density of 120 animals/m^3, the average density of the species in the wild, and mixed mercuric chloride, into the soil and animal dung mixture in the tanks to dose levels of 0, 0.5, 1.0, 2.0, or 5.0 mg mercury/kg. The experiment ran for 60 days and estimates of mortality were used to give LC_{50} values. There was less than 50% mortality within 5 days. The LC_{50} was 2.39 mg/kg at 10 days and had fallen to 0.79 mg/kg over a 60-day exposure period. As the mortality of adult earthworms progressed throughout the experimental period, so the earthworms still alive reproduced more than the controls. The reason for this effect is unclear; that the animals were stressed by the metal is evidenced by the continuing deaths. Beyer et al. (1985) exposed the earthworm *Eisenia foetida* to soil containing methylmercuric chloride at 0, 1, 5, 25, or 125 mg/kg. All worms dosed at 25 or 125 mg/kg died within 12 weeks. Survival at 12 weeks was 97%, 92%, and 79%, respectively, for doses of 0, 1, and 5 mg/kg. Regeneration of amputated segments was normal after treatment with methylmercuric chloride at 1 mg/kg soil, but reduced or eliminated by 5 mg/kg.

7.2.2 Effects of mercury on birds

Appraisal

Interpretation of the results of laboratory experiments on birds should take into account that practically all studies have been carried out using gallinaceous birds, which are unrepresentative of bird species as a whole.

Birds fed inorganic mercury show a reduction in food intake and consequently in growth. Many other sublethal effects have been reported. Organomercury compounds are more toxic to birds and cause reproductive impairment.

Acute toxicity to birds is summarized in Table 10. The majority of tests have been carried out using organic mercury compounds, which are generally much more toxic than inorganic salts. The 5-day dietary toxicity of mercuric chloride was in excess of 3000 mg/kg diet for those species tested. The organic mercury fungicidal preparations were the most toxic, with 5-day dietary LC_{50}s as low as 50 mg/kg diet.

Table 10. Toxicity of mercury to birds

Species	Age	Compound[a]	Parameter[b]	Concentration (mg/kg)	Reference
Japanese quail	14 days	methyl mercuric chloride	acute LD_{50}[c]	18 (14-24)	Hill & Soares (1984)
(Coturnix coturnix japonica)	14 days	mercuric chloride	acute LD_{50}[c]	42 (33-54)	Hill & Soares (1984)
	2 months	ceresan M	acute LD_{50}[c]	668 (530-842)	Hudson et al. (1984)
	4 months	ceresan L	acute LD_{50}[c]	1498 (1190-1888)	Hudson et al. (1984)
	14 days	methyl mercuric chloride	5-day LC_{50}	47 (36-60)	Hill & Soares (1984)
	14 days	mercuric chloride	5-day LC_{50}	5086 (3743-6912)	Hill & Soares (1984)
	14 days	methoxyethylmercury chloride	5-day LC_{50}	~1750	Hill et al. (1975)
	14 days	phenyl mercuric acetate	5-day LC_{50}	614 (496-761)	Hill & Camardes (1986)
	14 days	morsodren	5-day LC_{50}	45 (40-52)	Hill & Camardese (1986)
	14 days	ceresan M	5-day LC_{50}	147 (120-180)	Hill & Camardese (1986)
Pheasant	12 months	ceresan M	acute LD_{50}[c]	360	Hudson et al. (1984)
(Phasianus colchicus)	3-4 months	ceresan L	acute LD_{50}[c]	1190	Hudson et al. (1984)
	3-4 months	phenyl mercuric acetate	acute LD_{50}[c]	169 (101-283)	Hudson et al. (1984)
	10 days	mercuric chloride	5-day LC_{50}	3790 (2768-5541)	Hill et al. (1975)
	10 days	methoxyethylmercury chloride	5-day LC_{50}	1102 (957-1263)	Hill et al. (1975)
	10 days	phenyl mercuric acetate	5-day LC_{50}	~2350	Hill et al. (1975)
	10 days	morsodren	5-day LC_{50}	64 (55-73)	Hill et al. (1975)
	10 days	ceresan M	5-day LC_{50}	146 (127-167)	Hill et al. (1975)
Mallard duck	6-8 days	ceresan M	acute LD_{50}[c]	> 2262	Hudson et al. (1984)
(Anas platyrhynchos)	3 months	ceresan M	acute LD_{50}[c]	> 2262	Hudson et al. (1984)
	3 months	ceresan L	acute LD_{50}[c]	> 2000	Hudson et al. (1984)
	3-4 months	phenyl mercuric acetate	acute LD_{50}[c]	878 (169-4558)	Hudson et al. (1984)
	10 days	mercuric chloride	5-day LC_{50}	> 5000	Hill et al. (1975)
	10 days	methoxyethylmercury chloride	5-day LC_{50}	~280	Hill et al. (1975)
	10 days	phenyl mercuric acetate	5-day LC_{50}	~1175	Hill et al. (1975)
	5 days	morsodren	5-day LC_{50}	51 (43-60)	Hill et al. (1975)
	10 days	morsodren	5-day LC_{50}	60 (47-76)	Hill et al. (1975)
	5 days	ceresan M	5-day LC_{50}	~54	Hill et al. (1975)
	10 days	ceresan M	5-day LC_{50}	~50	Hill et al. (1975)

Table 10 (Contd).

Bobwhite quail (*Colinus virginianus*)	2-3 months 14 days	ceresan L ceresan M	acute LD_{50}[c] 5-day LC_{50}	1060 (841-1330) ~70	Hudson et al. (1984) Hill et al. (1975)
Prairie chicken (*Tympanuchus cupido*)		ceresan M	acute LD_{50}[c]	360 (233-566)	Hudson et al. (1984)
Chukar partridge (*Alectoris chukar*)	4 months	ceresan M	acute LD_{50}[c]	841	Hudson et al. (1984)
Grey partridge (*Perdix perdix*)	9-20 months	ceresan M	acute LD_{50}[c]	550 (385-786)	Hudson et al. (1984)
Rock dove (*Columba livia*)		ceresan M	acute LD_{50}[c]	714 (437-1164)	Hudson et al. (1984)
Fulvous whistling duck (*Dendrocygna bicolor*)	3-6 months	ceresan L	acute LD_{50}[c]	1680	Hudson et al. (1984)

[a] morsodren = cyano methylmercury guanidine (1.51% mercury);
ceresan M = N(ethylmercury)-p-toluenesulfonalide (3.2% mercury);
ceresan L = methylmercury 2,3-di-hydroxyl propyl mercaptide + methylmercury acetate (2.25% mercury).

[b] concentrations expressed as mg/kg food, unless stated otherwise.

[c] concentrations expressed as mg compound per kg body weight in a single oral dosage (i.e., birds were fed with a dosed diet for 5 days followed by a 'clean' diet for 3 days).

7.2.2.1 Inorganic and metallic mercury

When Beliles et al. (1967) exposed male Carneaux pigeons to mercury vapour (0.1 mg/m^3) for 6 h per day over 20 weeks, no behavioural, histological, or gross signs of mercury toxicity were noted. Armstrong et al. (1963) trained pigeons to respond to coloured lights to obtain food. The birds were then exposed to mercury vapour (17 mg/m^3) for 2 h daily (5 days/week) for 30 weeks. Marked changes in behaviour were observed, as measured by a decrease in the averaged response rate. A return to normal response was found when exposure to mercury ceased.

Ridgway & Karnofsky (1952) injected chicken eggs, after 4 and 8 days of development, with mercuric chloride solutions into the yolk sac and, after 8 days of development, into the chorio-allantoic membrane, and estimated LD_{50}s. These were 0.3, at day 4, and 3.1, at day 8, expressed as molar equivalents of mercury, for the yolk sac route, and 0.21 Meq, at day 8, for the chorio-allantoic route. The result on day 4 is equivalent to a dose of 0.08 mg mercuric chloride/egg. Birge & Roberts (1976) injected chicken eggs (into the yolk sac), immediately prior to incubation, with mercuric chloride and obtained an EC_{50} for hatchability of 1.0 mg/litre yolk.

Grissom & Thaxton (1985) exposed 4-week-old male chickens to mercuric chloride (0 or 500 mg mercury/litre) in their drinking water for up to 15 days. Rates of growth, together with feed and water consumption, decreased significantly within 3 days of the beginning of mercury treatment and remained depressed throughout the study. Mortality was greater in the mercury-treated group. Red blood cell numbers, haematocrit, mean corpuscular volume, and haemoglobin level increased within 3 days of the start of treatment. Mean corpuscular haemoglobin concentration (as pg/cell) was unchanged, but mean corpuscular haemoglobin (as % of cell) decreased.

Grissom & Thaxton (1984) investigated the interaction of mercury treatment (as mercuric chloride in the drinking water) and water deprivation in chickens. Birds (3-weeks-old) were treated at a rate of 500 mg/litre water over 15 days. One group had water *ad libitum*, while a second group were given limited water by intubation. Water consumption increased as the birds grew during the experiment. Monitored water intake was 25, 55, 70, 50, and 80 ml/kg body weight at 0-3, 3-6, 6-9, 9-12, and 12-15 days into the experiment for the mercury-treated birds. Birds on water by intubation were given 20, 35, 60, 70, and 70 ml/kg water for the same periods of the experiment. Water limitation resulted in a significant inhibition of the growth rate of untreated birds within the first 3 days of the experiment and this inhibition continued throughout the experiment. Mercury did not cause a significant inhibition of growth until between 12 and 15 days after the beginning of treatment. The only significant interaction between the effects of mercury and water deprivation occurred at 15 days. Food consumption was significantly reduced in water-deprived birds. Mercury caused a significant reduction in food intake during the 9-12 and 12-15 day periods. Dehydration increased mortality of the

groups to 10% compared with 3.75% for controls on water *ad libitum*. Mercury results in birds refusing to take water or food contaminated with the metal. Therefore, the effects of mercury can be direct or indirect. Direct mercury effects appear to need more than 2 weeks of exposure to develop. Examination of the birds during a 14-day recovery period on clean water showed incomplete restoration of normal patterns of food and water consumption over this time.

Brake et al. (1977) treated juvenile chickens with mercuric chloride in the drinking water (300 mg/litre) or by injection (5 consecutive days at 3 or 12 mg/kg body weight). Growth was retarded by the chronic treatment in drinking water and by the higher of the two injection rates. Relative heart weights (the ratio of heart weight to body weight) were increased by mercury in drinking water, decreased by the higher injected dose, and unchanged by the lower injected dose. Similar results were reported for relative aorta weights. Electrocardiograms showed a consistent decrease in the amplitude of R-S and T waves, with the greatest effect in the injected birds (both doses). Histological examination of the hearts of treated birds showed myocardial histopathological changes described as a myocarditis with polymorphonuclear and lymphocytic infiltration and fatty degeneration. The authors concluded that mercury causes cardiovascular disturbance in chickens even when administered at doses which do not inhibit growth.

Hill & Shafner (1975) fed Japanese quail from hatching to one year of age on a diet containing mercuric chloride (0, 2, 4, 8, 16, or 32 mg mercury/kg). Food consumption, growth rate, weight maintenance, hatchability, and eggshell thickness were unaffected. As dietary mercuric chloride increased, so initial oviposition occurred at a younger age. The average rate of egg production was also positively related to the concentration of mercuric chloride. The rate of egg fertilization, however, was generally depressed for all mercury treatments above 4 mg/kg.

Kosba et al. (1982) dosed 8-month-old hens with mercuric chloride in drinking water at 0, 150 or 250 mg mercury/litre. Dosing at 250 mg/litre caused a slight, but insignificant, decrease in body weight and egg numbers. Birds given the maximum dose consumed less food than controls, but birds on 150 mg/litre consumed more food than controls. All treated birds laid significantly smaller eggs than controls. Fertility and hatchability were adversely affected by mercury, and chicks hatched from eggs laid by treated birds were lighter.

Hill & Soares (1984) studied the sublethal effects of feeding 9-week-old Japanese quail with mercuric chloride in the diet. They calculated EC_{50}s (a reduction to 50% of the activity of controls) for the activities of aspartate aminotransferase, alpha-hydroxybutyrate dehydrogenase, lactate dehydrogenase, and ornithine carbamoyl-transferase, in blood plasma, of 8.6, 11.2, 3.0, and 62.8 mg/kg diet, respectively.

Dieter (1974) fed male Japanese quail for 12 weeks on a diet containing mercuric chloride at concentrations of 2, 4, and 8 mg/kg. The dosed diets did not significantly effect the carcass or liver weights or the blood haematocrit, and, although there was a significant decrease in haemoglobin at the 4 mg/kg treatment, this was not reflected in the other treatment groups. The treatments had no significant effect on the activity of the plasma enzymes creatine kinase, asparate aminotransferase, or fructose-diphosphate aldolase, but cholinesterase and lactate dehydrogenase activities were altered. The maximum decrease in cholinesterase activity amounted to 25% below that in controls, and showed almost a linear relationship with the logarithm of the dose. Irrespective of the mercuric chloride dose, lactate dehydrogenase activity increased 3-fold above control values.

In studies by Scott (1977), Japanese quail were fed diets containing mercuric sulfate (0, 100, or 200 mg mercury/kg). With the highest dose, there was a significant reduction in the hatchability of fertile eggs and the strength of the eggshells. There were no significant effects on daily food intake, egg production, average egg weight, or percentage of fertile eggs.

Nicholson & Osborn (1984) found kidney lesions in juvenile starlings *(Sturnus vulgaris)* fed on a commercial diet contaminated by mercury. Analysis of the food showed mercury levels at 1.1 mg/kg. No signs of overt toxicity were seen in the birds. Damage to the kidney was mainly confined to the proximal tubules, and was similar to that found in mercury-contaminated sea birds in the field.

Bridger & Thaxton (1983) demonstrated the effects of mercuric chloride on the humoral immune response of chickens. Three treatments were employed: chronic treatment with mercuric chloride at 300 mg/litre of drinking water; acute low dose with five consecutive daily injections of 3 mg mercury/kg body weight; and acute high dose with five daily injections of 12 mg/kg. The drinking-water treatment was inhibitory to growth, while the acute treatments were not. Chronically treated birds also showed suppressed primary and secondary responses to a challenge with sheep red blood cells. Immunoglobulin M levels were reduced to a greater extent than immunoglobulin G in chronically treated birds. The primary response to *Brucellus abortus* was also suppressed in chronically treated birds, but the secondary response was enhanced, with a greater titre of circulating antibodies. Bridger & Thaxton (1982) exposed chicks to either mercuric chloride in drinking water (300 mg/litre) or five consecutive daily injections of mercuric chloride into pectoral muscle (3 or 12 mg mercury/kg body weight). The authors found that these treatments did not significantly affect cell-mediated immune responses, in contrast to the effects on humoral immune responses.

7.2.2.2 Effect of organic mercury on birds

When Birge & Roberts (1976) injected chicken eggs, immediately prior to incubation, with methylmercuric chloride, into the yolk sac the EC_{50} for hatchability was 0.1-0.5 mg/litre yolk.

Haegele et al. (1974) dosed female mallard ducks with 200 mg/kg diet of Ceresan M (3.1% ethylmercury) and measured eggshell thickness on days 76 and 85 of treatment. No significant effects were found. When mercury was added to the diet along with DDE at 40 mg/kg, mercury did not increase the effect of the organochlorine on shell thickness.

Mullins et al. (1977) dosed captive hen pheasants with phenylmercuric acetate (PMA) either in capsules (20 mg/kg body weight) or added to the diet (at the normal fungicidal treatment rate of 14.18 g/bushel of seed wheat). Birds given mercury by capsule showed significant decreases in egg hatchability, eggshell thickness, and chick weight and survival, but no effect on egg production, egg volume, fertility, or chick behaviour. The mercury-dosed diet had no effect on any of these reproductive parameters.

Hill & Soares (1984) studied the sublethal effects of feeding 9-week-old Japanese quail with methylmercuric chloride in the diet, and calculated EC_{50}s for the activity of asparate aminotransferase, alpha-hydroxybutyrate dehydrogenase, lactate dehydrogenase, and ornithine carbamoyltransferase, in blood plasma, of 4.8, 6.1, 1.2, and 3.5 mg/kg diet, respectively.

In studies by Scott (1977), Japanese quail were fed diets containing methylmercuric chloride (0, 10, or 20 mg mercury/kg). The daily food intake, egg production, average egg weight, percentage of fertile eggs, and the hatchability of fertile eggs were all significantly reduced at 10 mg/kg. There were greater effects on all these parameters with 20 mg/kg, but the difference was not significant relative to the lower dose in terms of percentage fertility or hatchability of fertile eggs. The strength of the eggshell was significantly reduced by the 10 mg/kg dose after 3 weeks of dosing. Insufficient eggs were laid by the group dosed at the higher rate to monitor this factor.

Tejning (1967) studied the effects on domestic fowl of methylmercuric-dicyandiamide (MMD)-treated grain (0-18.4 mg mercury/kg diet). Food consumption was unaffected in birds treated with 0 or 4.4 mg mercury/kg, but fell gradually over 50 days, in birds treated with 9.2 or 18.4 mg/kg. Food consumption returned to normal after about 60-65 days, but then fell below control levels again later. Egg production (eggs/hen per day) was unaffected by 4.4 mg mercury/kg or by 8.8 or 9.2 mg/kg for the first 40 days of exposure. After treatment at 17.6 or 18.4 mg/kg diet, egg production gradually fell over the period of exposure. There was no effect on body weight of any of the treated birds. Some birds on the highest doses showed ataxia with difficulty in walking. In a study comparing three treatment levels of MMD (0, 9.2, and 18.4 mg mercury/kg diet, various reproductive parameters were monitored. There was an increase, relative to controls, in the number of soft-shelled eggs of 17.1% at the highest dose and 1.4% in the 9.2 mg/kg group. Percentages of deaths of embryos in shell during the first 5 days of incubation were also increased (values were 10.5% in controls, 43.7% in the birds dosed with 9.2 mg/kg, and 62.1% in the 18.4 mg/kg group). Mortality later in the incubation period was

similar in all groups. Overall hatchability was reduced from 60% in the controls to 16% in the 9.2 mg/kg group and 10% in the 18.4 mg/kg group.

Fimreite (1970) exposed leghorn cockerels to a diet dosed with Panogen 15 (2.5% MMD) at concentrations of 6, 12, and 18 mg MMD/kg for 3 weeks, from 2 weeks of age. The total intake of mercury, based on monitoring food consumption, was calculated to be 1.7, 3.4, and 5.1 mg/chick, respectively, for the three dosing levels. All treated birds showed significant reductions in weight, but only at the highest dose was there a significant increase in deaths. Fimreite (1971) fed penned pheasant *(Phasianus colchicus)* breeder ration and treated grain containing MMD at 2.25, 4.5, or 9 mg mercury/kg, for 2, 4, or 12 weeks. There was no weight reduction amongst adults, and food consumption was only adversely affected by the highest dose. Some hens fed the highest dose showed extensive demyelination of the spinal cord. All treated birds showed reduced hatchability and egg production, with a large number of shell-less eggs. There was a significant reduction in the weight of eggs laid by mercury-treated birds. The highest dose group laid eggs of an abnormal colour.

Spann et al. (1986) fed 12-day-old bobwhite quail on diets containing methylmercuric chloride at 0, 5.4, or 20 mg/kg (equivalent to 0, 4.3, or 16 mg mercury/kg). Birds dosed at the lower rate showed low mortality, not significantly different from controls, whereas birds dosed at the higher rate showed high mortality after 6 weeks (at between 55% and 80% for three different vehicles: no solvent; corn oil; and propylene glycol). When acetone was used as carrier, deaths were significantly reduced (to about 30%), deaths in the control group being < 10%.

When Mykkanen & Ganther (1974) fed 1-day-old Japanese quail a diet containing 0-30 mg mercury/kg (as methylmercury hydroxide) for up to 32 days, no effect on erythrocyte glutathione reductase activity was found.

Fimreite & Karstad (1971) dosed chicks with MMD and then fed them to red-tailed hawks for up to 12 weeks. Mercury levels in the liver of the chicks were between 3.9 and 10 mg/kg. Three of the six birds, given chicks with mercury in the liver at 10 mg/kg and died, one bird out of six, given chicks with mercury in the liver at 7.2 mg/kg, died. All the poisoned birds showed neurological symptoms, weakness in extremities, and impaired coordination of muscular movement, and, although the hawks did not lose their appetite, they had difficulty feeding. There was no effect on food consumption, even poisoned birds maintaining appetites until in an advanced stage of poisoning. Only birds with overt signs of poisoning showed substantial body weight loss. Borg et al. (1970) fed chickens with 8 mg MMD/kg diet for 5 to 6 weeks, and muscle and liver from the contaminated chickens were fed to goshawks *(Accipiter gentilis gentilis)*. Three goshawks receiving muscle and liver averaging 13 mg mercury/kg died within 30, 38, and 47 days. One goshawk receiving muscle only (10 mg mercury/kg) died within 39 days. The major clinical symptoms, appearing after about two

weeks, were inappetance, muscular weakness, ataxia, and loss of body weight. Autopsy revealed that the dominating effect was muscular atrophy, which was presumably the main cause of weight loss. Pronounced histological changes included demyelination and nerve cell degeneration of the cerebellum and medulla oblongata and demyelination of peripheral nerves. No lesions were found in the cerebrum.

When Heinz (1974) fed mallard ducks a dry mash diet containing MMD (0.5 or 3.0 mg mercury/kg) for 21 weeks, the lower of the two dose levels had no effects on reproduction but the higher reduced egg laying and increased embryonic and duckling mortality. Eggs laid by controls tended to be heavier than eggs laid by treated birds, but there was no effect on eggshell thickness. Heinz (1976a) fed mallard for 2 consecutive years on the same doses of MMD as above. There was no significant effect on egg production or hatching success or on approach behaviour of ducklings. Ducklings from females fed 3.0 mg/kg were less likely to survive to 1 week than those from other groups. Ducklings from parents fed the highest dose were hyper-responsive in avoidance behaviour. Heinz (1976b) fed ducklings (from 9 days of age) whose parents had been fed MMD at 0.5 mg/kg diet, on the same dosed diet. Dosed second generation females laid a greater proportion of their eggs on open ground outside the nest boxes. They also produced fewer ducklings surviving to 1 week. In ducklings from second generation females, there were no significant differences in behaviour patterns such as approach response to maternal calls, avoidance response to frightening stimuli, and open-field behaviour. There was a reduction in growth of third generation ducklings. Heinz (1979) dosed three generations of mallard with 0.5 mg MMD/kg diet. As in the second generation, females laid a greater number of eggs outside the nest box. They also laid fewer eggs and produced fewer ducklings. There was some eggshell thinning in the third generation and a reduced response of ducklings to maternal calls.

Prince (1981) tested mallard ducks through four generations in an attempt to establish if resistance to the reproductive effects of methylmercury was developed. The parental generation was exposed to two doses of 8 mg methylmercuric chloride within a 2-week period. The parents were split into two groups on the basis of the survival of ducklings after exposure to mercury. Three further generations of each line were produced. The percentage survival of ducklings exposed, via the parent, to mercury tended to increase in the "resistant" strain in successive generations. This suggested an ability of the birds to adapt to mercury exposure over time.

Ganther et al. (1972) fed Japanese quail with diets containing up to 20 mg methylmercury/kg. Some groups of quail were given tuna fish as 17% of the total diet, while other groups had corn-soya instead of the tuna. Mortality in the group fed corn-soya with 20 mg methylmercury/kg diet was 61% over 6 weeks, the majority (52%) of deaths occurring between 4 and 6 weeks of dosing. The same amount of methylmercury added to the tuna diet led to only 14% mortality over the 6-week-period of dosing. The authors ascribe the protective effect of

the fish diet to the high selenium level in the tuna. Selenium, which in these diets amounted to 0.3-0.6 mg/kg, becomes toxic to birds only at dietary concentrations more than 10 times higher than this.

7.2.3 Effects of mercury on non-laboratory mammals

Appraisal

Few studies have been published on truly wild, non-laboratory mammals. Work is of most value when done not on mammal species that have been changed by generations in captivity but on those that are still found in the wild, or are genetically close to wild forms. The only work in this last category is on mink and prairie vole. The available evidence indicates that toxic effects, including reproductive changes, can be produced. Methylmercury has been found to be more toxic than inorganic mercury.

Aulerich et al. (1974) dosed the diet of mink with either 5 mg methylmercury/kg (as contained in Ceresan L, which contains 2.25% mercury) or 10 mg mercuric chloride/kg. No adverse effects attributed to these diets were observed for 3 weeks. After 25 days, the mink dosed with organic mercury showed signs of lack of coordination, loss of balance, anorexia, and loss of weight. Within 4 days, ataxia, paralysis, tremors, and, finally, death were observed. Attempts to arrest these symptoms in the least affected mink by reverting to a control diet, with either EDTA or methionine injections, had no effect and the mink still died. Mink dosed with inorganic mercury showed no clinical signs. The mercuric chloride treatment did not affect reproductive performance and no teratological effect was noted. There was a significant reduction in the weight of the kits from treated parents, at birth, but this had recovered by 4 weeks of age.

Wren et al. (1987a) fed adult mink a daily diet containing 1 mg methylmercury/kg for 3 months. Later, because of mortality, the dosed diet was administered every other day for a further 3 months. The initial, daily-dosed diet resulted in the death of 8 out of 12 females and 1 out of 4 males. There were no observed effects of the treatment on the thyroid, pituitary, or adrenal glands or on serum triiodothyronine (T3) or thyroxine (T4) levels during the experimental period. Mortality was thought to be caused by a combination of mercury poisoning and cold stress (the animals were kept outside during the winter). Under laboratory conditions, 1 mg/kg would not be considered fatal to mink (Wobeser et al., 1976). Under the same experimental conditions, Wren et al. (1987b) found that the fertility of adult male mink, percentage of females whelped, and number of kits born per female were not affected by the mercury treatment.

Hartke et al. (1976) calculated an acute LD_{50} of 10 mg/kg body weight for phenylmercuric acetate (PMA) in female prairie voles *(Microtus ochrogaster)*, after intraperitoneal injection. Female voles were also injected on days 8, 9, and 10 of gestation with

0.06-5.0 mg PMA/kg body weight. Some normal foetuses and some resorption sites (where implantation had occurred but the foetal material had been reabsorbed) were found in voles injected with 0.5 mg/kg or less on days 8 and 9 of gestation. Animals treated with > 1.0 mg/kg had no live foetuses, but all had resorption sites in the uterus. Similar results were found for voles treated on day 10. No resorption sites were found in voles treated with < 0.25 mg/kg. To study the effects of dose and the stage of gestation when dosing occurred, the authors further injected voles with 0.5 mg/kg on days 7, 11, and 12 of gestation. Normal embryos and some resorption and abortion sites were found after dosing on days 7 and 11. Dosing on day 12 of gestation produced no resorption or abortion and the numbers of live foetuses accounted for all *corpora lutea* in the ovary.

8. EFFECTS OF MERCURY IN THE FIELD

Appraisal

Fatalities and severe poisonings in birds have been reported in association with outbreaks of human poisoning. In addition, the agricultural use of organomercury fungicides has caused poisoning in birds. A statistical association has been reported between the mercury content of birds' eggs and reproductive failure. These eggs also contained organochlorine residues, but these residues did not correlate with the observed reproductive effects.

Methylmercury levels in fish in Japan have caused a major problem for human health. During these incidents, there were also reports of direct effects of mercury on wildlife in the area. Fish carrying methylmercury were found dead or showed symptoms of mercury poisoning. Fish-eating birds and scavenging birds were also killed (Harada, 1978). Birds found dead in the area showed the characteristic pathological changes in the central nervous system of Minamata disease, but no measurement of mercury content was made (Takeuchi et al., 1957).

The use of organic mercury compounds as a fungicidal seed dressing has led to deaths in the field of birds, mostly grain-eating species. Some raptors, feeding on the poisoned birds, were also casualties (Borg et al., 1969). Koeman et al. (1969) reported large numbers of birds of prey killed by indirect poisoning with organomercury fungicides in the Netherlands.

Mercury contamination has been implicated in the breeding failure of some raptor species both in Europe and North America, where residues have equalled those found to cause reproductive impairment in laboratory species. These birds also contained organochlorine insecticide residues and the separation of effects is difficult (Newton, 1979). More recent work suggests more strongly that mercury affects the breeding of birds of prey in the field. Merlins sampled in Scotland contained organochlorines along with mercury in their eggs. Statistical analysis of the data showed a clear inverse relationship between mercury content of eggs and brood size; the higher the mercury content, the less likelihood of successful breeding. Productivity fell markedly when mercury residues in eggs exceeded 3 mg/kg. Productivity, that is the number of young successfully reared, showed no statistically significant relationship with residues of other chemicals present in the eggs. Levels of mercury were highest in birds sampled in Orkney and Shetland, but the relationship between mercury residue and productivity remained when these, particularly high, residue levels were excluded from the analysis (Newton & Haas, 1988). The merlins were feeding on wading birds in estuaries and this was presumed to be the source of the mercury. A similar, but not quite significant, relationship was found in peregrin falcons breeding near the coast.

Jefferies et al. (1973) sampled small mammals from fields sown with mercury-treated grain. They express the view that residues were sufficiently high to have caused deaths in small mammals feeding on the grain. Some mammals were found dead and deemed to have been killed by mercury poisoning.

9. EVALUATION

In evaluating the environmental hazard of mercury it is necessary to extrapolate from laboratory experiments to ecosystems. This must be done with extreme caution for the following reasons.

(i) Speciation of mercury and its adsorption to environmental components such as soil, sediment, organic matter, and biota limit its availability to organisms in the environment.

(ii) Environmental variables such as temperature, pH and chemical composition of water, soil type, and geology have been shown in limited studies on a narrow range of species to affect both uptake and effect of mercury. There is insufficient information to fully assess the probable affects of, for example, tropical conditions and acid precipitation.

(iii) There are few data measuring mercury availability to organisms. Most data represent nominal or total metal concentration, rather than that component which could be taken up by organisms. True exposure is, therefore, difficult to assess.

(iv) There are limited data on the behaviour of mixtures of metals from controlled experimental work; organisms in the environment are exposed to mixtures.

(v) Experimental work seldom, if ever, is conducted on species or communities that are either representative or key components of natural communities and ecosystems. Studies do not consider all of the interactions between populations and all of the environmental factors affecting these populations.

It is probable that subtle disturbances to the community occur at much lower concentrations than those suggested in laboratory studies on acute effect, perhaps as much as one order of magnitude lower.

9.1 The Marine Environment

Marine aquatic organisms at all levels accumulate mercury into tissues. This mercury is retained for long periods if it is in an organic form. A number of factors affect the susceptibility of aquatic organisms to mercury. These include the life-cycle stage (the larval stage being particularly sensitive), the development of tolerance, water temperature, and salinity. Some incidents of severe pollution have resulted in the death of fish at that time. Few follow-up studies have been reported so that it is impossible to assess the long-term hazards. Toxic effects have been produced experimentally only at concentrations much higher than those found in the non-polluted marine environment. Furthermore, most of the studies have been on acute lethality and have used inorganic mercury compounds in the main. Birds, particularly coastal species or those eating prey that feed in

estuaries, have been affected by mercury contamination. It has adversely affected breeding and may have influenced population stability.

9.2 The Freshwater Environment

Mercury compounds are acutely toxic to freshwater microorganisms. Using photosynthesis and/or growth as parameters, the NOTEL (No-observed-toxic-effect-level) for inorganic mercury lies between 1 and 50 μg/litre, depending on the organism, density of cells in culture, and experimental conditions. Diversity of species in mixed culture may be affected by 40 μg mercuric chloride/litre. For organomercury compounds, the NOTEL is 10-100 times lower.

Aquatic plants sustain damage after exposure to inorganic mercury at concentrations of 800 to 1200 μg/litre. Organomercury produces toxic effects at concentrations 10-100 times lower.

Many aquatic invertebrates are sensitive to mercury toxicity, particularly as larvae. Organic mercury compounds are toxic at concentrations 10 to 100 times less than inorganic mercury. For the most sensitive species, *Daphnia magna*, the NOTEL for reproductive impairment is 3 μg/litre for inorganic mercury and < 0.04 μg/litre for methylmercury.

Freshwater fish show lethal responses to mercury in acute nominal concentrations from approximately 30 μg/litre. Larvae under the same static conditions are 10 times more sensitive. In flow-through tests, fish are up to 100 times more sensitive. In both static and flow-through tests, organomercury compounds are approximately 10 times more toxic than inorganic compounds. The NOTEL for the most sensitive parameters may be well below 0.01 μg/litre.

Aquatic developmental stages of amphibia show sensitivity to mercuric compounds similar to that of fish.

9.3 The Terrestrial Environment

Based on the current state of knowledge, it is not possible to determine the true exposure or concentration of mercury available to terrestrial organisms. It can, however, be stated that exposure via soil, soil water, and food is most important; exposure via open water and air is less important.

Mercury has been shown, in laboratory studies, to be toxic to terrestrial organisms over a broad range of concentrations. However, most of these studies are at high exposure levels (birds) or environmentally unrealistic exposure routes (hydroponic culture of plants).

It can be stated that acute effects would not be seen in terrestrial plants growing in natural soils, nor in terrestrial birds or mammals, other than by exposure to mercurials used as fungicidal seed-dressings. Other effects seen in birds derive from mercury in the marine environment.

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