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JOINT GROUP OF EXPERTS ON THE SCIENTIFIC ASPECTS OF MARINE POLLUTION
- GESAMP -

REPORTS AND STUDIES

No. 22

REVIEW OF POTENTIALLY HARMFUL SUBSTANCES – CADMIUM, LEAD AND TIN

WORLD HEALTH ORGANIZATION

CONTENTS

NOTES

1. GESAMP is an advisory body consisting of specialized experts nominated by the Sponsoring Agencies (IMO, FAO, UNESCO, WMO, WHO, IAEA, UN, UNEP). Its principal task is to provide scientific advice on marine pollution problems to the Sponsoring Agencies and to the Intergovernmental Oceanographic Commission (IOC).

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Definition of Marine Pollution by GESAMP

"POLLUTION MEANS THE INTRODUCTION BY MAN, DIRECTLY OR INDIRECTLY, OF SUBSTANCES OR ENERGY INTO THE MARINE ENVIRONMENT (INCLUDING ESTUARIES) RESULTING IN SUCH DELETERIOUS EFFECTS AS HARM TO LIVING RESOURCES, HAZARDS TO HUMAN HEALTH, HINDRANCE TO MARINE ACTIVITIES INCLUDING FISHING, IMPAIRMENT OF QUALITY FOR USE OF SEA WATER AND REDUCTION OF AMENITIES".

For bibliographic purposes, this document may be cited as:

GESAMP (IMO/FAO/UNESCO/WMO/WHO/IAEA/UN/UNEP) Joint Group of Experts on the Scientific Aspects of Marine Pollution. Review of Potentially Harmful Substances: Cadmium, Lead, and Tin. Reports and Studies (22).

I. INTRODUCTION

1. Background

At the Eighth Session of GESAMP (Rome, 21-27 April, 1976) a Working Group on the Review of Harmful Substances was established with the following terms of reference:

- to update the Review of Harmful Substances (GESAMP Reports and Studies No. 2, New York, 1976) with greater emphasis on the human health aspects of marine pollution;

- to continue to include consideration of the other aspects of the subject, namely: harm to living resources, reduction of amenities, and interference with other uses of the sea.

The meeting recommended that priorities should be set in order to focus initial attention upon those agents of particular significance to human health, and also that:

(a) WHO prepare a list of agents to be evaluated on the basis of actual and potential human health hazards associated with the marine environment;

(b) the proposed working group endeavour to design and use uniform data sheets; and

(c) long-term and chronic effects such as carcinogenesis and mutagenesis be adequately considered whenever possible.

The meeting also recommended that in selecting agents and in making their risk evaluation, the following factors should be considered:

(a) the total quantities discharged, fluxes and/or concentrations of harmful substances before they enter the sea. Purposes for which they were originally used and their physico-chemical characteristics including, probably, reaction paths, should also be stated;

(b) the routes by which they may enter the sea and the likelihood of such entry, taking into account both sea-based and land-based sources and atmospheric fallout. The characteristics of the sea in the area where the introduction takes place should be noted;

(c) the degree of human exposure to these agents. Evaluation should be on the basis of their distribution and the amounts present in sea water, sediments, flora, and fauna. Especially important are marine products likely to reach man. The presence of by-products and transformation products of the original substance also require consideration;

(d) the stability of the agents and their derivatives, and the possibility of their causing environmental modifications (e.g., eutrophication);

(e) their bioaccumulation, especially along critical ecological paths;

(f) their toxicity profile including, whenever possible, such factors as structural considerations, general and specific toxicity, long-term and mutagenic effects, interaction between toxic agents and between toxic agents and the environment;

(g) the distinct importance of sensitive groups of the population and of particular pathways; and

(h) an assessment of the scale of risk for the human population considered at large.

At the Tenth Session of GESAMP (Paris, 29 June - 2 July, 1978), the terms of reference of the Working Group were redefined as:

1. To prepare short and referenced reviews on selected substances which include an assessment of the following factors:

(a) the total amount of the particular substance(s) which reach(es) the marine environment (on a local, regional, and global scale) with particular attention being given to the relative importance of land-based sources;

(b) the fate (transport, distribution, and transformation) of the substance in the marine environment; and

(c) the effects of the substance on the marine environment and adjacent coastal areas, including direct and indirect effects on living resources, human health, and amenities;

2. To produce a scientific evaluation of the harmful effects of substances released into the marine environment on living resources, human health, aesthetics, and other legitimate uses of the marine environment and adjacent coastal areas.

The Eleventh Session of GESAMP (Dubrovnik, 25-29 February, 1980) decided that, in the selection of substances to be reviewed, priority should be given to:

(a) previously identified priority chemicals that show increased levels in the environment; and

(b) chemicals that are not covered by existing conventions and previous hazard evaluations.

Furthermore, it was suggested that the evaluation of effects should concentrate on those effects that are observed under field (natural) conditions.

On the basis of data profiles prepared by UNEP's International Register of Potentially Toxic Chemicals (IRPTC) and the intersessional work of the Working Group, the Twelth Session of GESAMP (Geneva, 22-29 October, 1981) decided that first priority should be given to an evaluation of cadmium, lead, and tin and, later, if time and resources permitted, arsenic and mercury should be evaluated. Subsequently, it was also recommended that, toxaphenes, phtalates, organosilicons, chlorinated and brominated aromatics, PNAHs, and nutrients (phosphorous, etc.) should be considered on the basis of the information in WHO Environmental Health Criteria documents, data profiles prepared by IRPTC, and other relevant publications.

The Thirteenth and Fourteenth Sessions of GESAMP (Geneva, 28 February - 4 March, 1983 and Vienna, 26-30 March, 1984) reviewed progress of the Working Group and endorsed publication of a document covering cadmium, lead, and tin after an edited final draft was circulated for comments and clearance to all GESAMP members.

This publication has been prepared and issued in conformity with that decision.

2. Evaluation Mechanisms

The collaboration and support of IRPTC was offered at GESAMP XII and data profiles on cadmium, lead, and tin were prepared and made available in 1982. From an examination of these, other data profiles, and available critical reviews of published data, significant papers were selected for thorough evaluation. These papers, together with recent and pertinent publications, then formed the basis of this review. It is recognized, however, that these papers provide only a partial coverage of the world literature. Information was lacking in several areas essential to an environmental hazard evaluation of these substances, and these areas were identified in the reviews.

3. Working Procedures of the Group

The method and approaches applied by the Working Group were discussed and agreed upon at a planning session in Stockholm, 24-25 September, 1982. This was attended by the chairmen of GESAMP and of the Working Group, and by international agency representatives.

For each substance, selected experts prepared draft sections of the review. The reviews for cadmium, lead, and tin were then critically examined and revised by the Working Group members (Annex I) in a meeting at WHO in Geneva on 31 January - 4 February, 1983. The Working Group was chaired consecutively by Messrs B.H. Ketchum, A. Jernelöv, and L. Friberg. The Working Group was sponsored by UNEP, FAO, and WHO. WHO acted as the lead agency. After further revision, the final draft was submitted to GESAMP for

consideration and adoption. This was followed by its formal publication and general distribution.

4. Quality of Data Base

4.1 Analytical quality control

Many studies conducted in the various countries aimed at evaluating normal and elevated levels of trace metals in different media. Unfortunately, most published reports lack quality assurance data, and valid comparisons cannot, therefore, be made. Furthermore, results from several inter-laboratory comparisons amplify the need for quality control. A review of such comparison studies, with particular emphasis on lead and cadmium in blood, has recently been published in connection with a UNEP/WHO Biological Monitoring Project on Assessment of Human Exposure to Lead and Cadmium through Biological Monitoring (Vahter, 1982). Various intercalibration exercises with those laboratories engaged in the determination of trace metals in commercially important marine organisms from the North Atlantic were also organized, since 1971, by ICES, the International Council for Exploration of the Sea (Topping, 1983). These reviews clearly show that errors may be large even in "experienced" laboratories. Since 1975, the IAEA's International Laboratory of Marine Radioactivity has also operated a large global analytical quality control programme for metals and chlorinated hydrocarbons in marine organisms and sediments. A similar programme has been run by the Intergovernmental Oceanographic Commission for Seawater Samples.

The introduction of sophisticated and increasingly sensitive analytical techniques has made it possible to measure trace substances in extremely low concentrations. Simultaneously, however, the risks of interference from competing factors has increased considerably. Although the awareness of the need for quality control has also increased during recent years, it is not possible to state, generally, that analyses carried out during the last 5-year period, for example, are always more reliable than earlier analyses.

Analytical problems may occur with any matrix to be examined. Particular problems arise, however, when analysing biological media or matrices which have very low trace metal concentrations (picograms/g). Schaule & Patterson (1980) showed that, for example, the lead concentrations in seawater samples have been overestimated by factors of up to 5000 and, that the lead concentrations reported for marine organisms are, with very few exceptions, several orders of magnitude higher than the actual concentrations present. These high concentrations are caused by the sample becoming contaminated during analysis. The importance of implementing rigid quality assurance programmes was amplified in 2 recent trace metal programmes sponsored by UNEP/WHO. One measured lead and cadmium in blood, and cadmium in kidneys (Vahter, 1982; Friberg & Vahter, 1983). The other measured trace metals in food (National Food Administration, 1982). In the first programme, it was rare that a laboratory met the criteria for data acceptance throughout the training phase, and gross errors were often recorded. The food study noted that the results of current analytical quality control analyses allowed few

conclusions to be drawn concerning the reliability of previously collected data. In addition to the various forms of analytical error, there is the possibility of contaminating biological samples, for example, by use of unsuitable sample collection vials and contaminated chemicals. Furthermore, errors due to adsorption and desorption on the walls of containers may cause inaccurate results.

The various sources of error which are possible make it necessary to exercise great caution when evaluating analytical data. In particular, it is more the exception than the rule that data on quality assurance are presented as part of published studies. Such caution has been exercised in the evaluation carried out by the Working Group, but there is still no guarantee that all the data used in this evaluation are completely valid. If rigid quality assurance criteria had been required, the analytical data available for use in the evaluation would have been extremely limited.

4.2 Ecotoxicological quality aspect

Experiments on marine organisms were carried out using many different procedures and techniques, and the usefulness of the results to the present review was critically examined. In particular, only a few experiments offered analytical confirmation of the concentrations and various forms of a substance that the subject was exposed to. There was little data on proven harmful effects resulting from chronic exposure. Therefore, extrapolations from the limited data base in order to predict whether environmental effects are likely to occur have to be treated with caution. In this respect, there is also a need for reliable analytical data on environmental concentrations for some species of the substances evaluated.

4.3 Quality of human toxicological data base

The quality and quantity of toxicological data show substantial variation from one marine pollutant to another. Ideally, the evaluation of the health hazard presented by a certain pollutant ought to be based on data which include a comprehensive dose-effect relationship. For a selected and preferably critical effect, a reliable dose-response curve should be provided. Equally important is an established correlation between the concentration of the toxic chemical (or one of its metabolites) in an index medium and the effects and responses. Some examples relevant to the substances selected by GESAMP are presented in the following paragraphs.

For one of the most widely studied metals, lead, the relationship between blood lead and the effect of lead on the synthesis of haemoglobin is well established, but the relationship between blood lead and the effect of lead at the lower end of exposure on the development of the Central Nervous System (CNS) in children is a question of controversy. The relationship between oral lead intake and blood lead concentration has not been investigated, and the lack of this information hinders the prediction of blood lead concentration from daily dietary intake and vice versa.

The corresponding correlation between blood cadmium and current exposure has been established. In a condition of changing exposure, however, blood cadmium may not correlate with the renal concentration of cadmium. However, the quality of data that predicts kidney accumulation of cadmium from dietary intake has recently become more reliable.

In many cases, the evaluation is dependent on experimental animal data, especially in experiments which aim to study those quantitative relationships which can be extrapolated to man. Unfortunately, in the cases of tin and particularly of organotins, even the animal data base is not sufficient for extrapolation, and dose-related human data are totally absent.

5. Dietary Intake Considerations

5.1 Basis for total dietary intake estimates

Accurate estimates of dietary intake of food contaminants are difficult to make for the general population because of the large variability in both environmental contaminant levels and rates of consumption. It is possible, however, to estimate intake from measurements of the concentration of contaminants in specific foods and the amounts of food consumed. Alternatively, measurements may be made with composite samples of the total diet. This approach requires fewer measurements and may reflect actual intake more closely, but it has the disadvantage of disregarding the contribution of single foods to the total dietary intake of contaminants. The calculated dietary intake may be made for representative population exposures, or it may be made with special reference to critical groups, for example, children, pregnant women, high consumers, etc. From contributions of particular food items to the total dietary intake, it is useful to note the "critical" foods to which more attention should be given in surveillance programmes.

Comparisons between calculated dietary intake and tolerable intake limits may indicate safety levels, or the incidence of risk for the exposed population. Due to the large variations in food consumption patterns among countries and to the wide variations in food contaminant concentrations, mean dietary intakes should be calculated and evaluated at the national or even local level.

Finally, the total energy value and composition of the diet should be taken into account in an evaluation of potential risks for population groups. It has been demonstrated that not only total food intake but also components such as fat, calcium, iron, and zinc can influence a subject's susceptibility to toxicity of contaminants by modifying the degree of gastrointestinal absorption.

5.2 Seafood consumption patterns

Seafoods do not represent a significant component of the diet for much of the world's population. Marine foods consumed by man include many trophic levels. Seaweeds are eaten mainly in the Far East, but also in Europe, for

example, as laverbread or agar. Phytoplankton and zooplankton are not themselves consumed, but their predators, such as oysters, mussels, clams, scallops, herring, and sardines are. However, krill is used for animal fodder. Higher trophic levels include the carnivorous gastropods, clams, cephalopods, crabs, and shrimp, and foremost, fish. The world (population 4150 million in 1977) average of fish and shellfish protein consumption is 3.8 g per person per day of a total of 68.8 g total protein per person per day (FAO, 1980a). These data can also be converted to and analysed in terms of the fresh weight of marine foods. The world's average daily consumption of 3.8 g protein corresponds to about 20 g edible fish and shellfish per day or 140 g edible tissue, which is equal to about one meal of fish and shellfish, per week. Aquatic plants and seaweeds make up only 4% of the world's harvest of marine and freshwater foods. Of the total catches of aquatic animals, 10% are caught in fresh water and the rest is marine. Seventy-seven percent of the world's landings are marine fish, 7% are molluscs, and 4% are crustaceans, with other marine animals contributing 1% or less. The northwest Pacific and northeast Atlantic are the most productive areas (FAO, 1980b).

From the Table on seafood consumption in selected countries (Table 1), it can be seen that the small populations on the islands mentioned eat relatively large amounts of marine foods. According to FAO's food balance sheets (1980a), the population (40 000 inhabitants) of the Faeroe Islands consumes, on the average, 38.6 g protein (equal to 193 g fresh weight of edible tissue) originating from marine foods. Assuming that 150 g of edible tissue constitues one meal, this corresponds to more than one meal per day of seafood. The average Japanese intake is the highest among larger populations. By comparison, the average consumption of marine foods in Australia, the USA, and the USSR is small. However, certain individuals are reported to consume much greater amounts. Fisherman at sea, especially in less affluent regions, will consume exclusively marine foods. For example, about 800 g per day were consumed on fishing boats in southern Ital7 (Bernhard & Renzoni, 1977). Canandian Indians are believed to have an intake of up to 1300 g per day during the fishing season, while 800 - 1500 g per day were eaten by fishermen and their families at Minemata (Review: Piotrowski & Inskip, 1981). Average consumption levels have been estimated on a global basis to be 23 g per day per person, with 38 g per day as the value for Europe.

Attention must be paid to situations in which seafood consumption represents a significant part of the diet, or when the concentration of a particular contaminant in consumed seafoods from heavily-polluted areas is relatively high. This may, for example, be the case with mercury in certain species of fish, or that with cadmium in oysters. It has been calculated, for example, that, while the contribution of seafoods to the total dietary intake of cadmium and lead is only 6% for the general population in Italy, the percentage can increase up to 25% for cadmium and 40% for lead in fishermen living in the Italian coastal villages where fish consumption is about 10 times that of the average quantity of seafood consumed in the rest of Italy.

Table 1. Consumption of fish and seafood (g fresh weight) (living weight) and in percent of different types of marine food[a]

	Population	Total marine protein	Seafood in fresh weight	% of different types of marine foods on protein basis		
Australia	13.8	3.4	17	68	24	
Bermuda	0.06	15.8	79	87	13	
Faeroe Islands	0.04	38.6	193	82	16	3
Iceland	0.1	19.2	96	83	13	
Japan	113.9	22.5	113	77	4	10
Maldives	0.14	37.1	186	100		
Portugal	8.8	10.2	51	87	2	3
USA	216.8	3.2	16	63	22	6
USSR	258.9	9.4	47	81		1
Vanuatu	0.1	23.2	116	99	2	
Yemen Republic	1.9	13.0	65	100		

[a] From: Bernhard & Andreae (1983).

Note: The protein values (FAO, 1980a) have been converted into fresh weight, assuming that 100 g fresh weight contain 20 g protein.

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II. CADMIUM

1. Cadmium in the Marine Environment

1.1 Reference documentation

The major reviews and reference works used were Aylett (1973) on the chemistry of cadmium, Frei & Hutzinger (1976) on analytical techniques, Webb (1979) on general chemistry and biology, WHO (1979) on environmental health criteria, Friberg et al. (1974) and Fleischer et al. (1974) on cadmium in the environment and its impact, and Simpson (1981) for a critical review of cadmium in the marine environment. On bioaccumulation, reviews by Alabaster (1978) and Coombs (1979) were consulted. The kinetics of uptake were reviewed by McLeese (1980), and George (1980) reviewed the pertinent literature on mussels.

Many other individual papers by research workers were consulted and are listed in the reference section.

1.2 General facts

Cadmium (Cd) (Greek Cadmean (earth), calamine) is in subgroup IIb, Zn, Cd, Hg of the transition series in the Periodic Table of Elements. It has atomic number 48 and atomic weight 112.40. Cadmium was first isolated and identified by F. Strohmeyer in 1817 from the zinc ore smithsonite ($ZnCO_3$). It has been released into the environment since the early days from the smelting of a variety of ores and the burning of wood and coal. Cadmium is among the rarer trace elements and is seldom found in pure minerals. It is extracted commercially from zinc ores, e.g., zincblende (ZnS), in which it occurs at 0.1 - 5.0%.

Cadmium is more mobile in non-polluted, undisturbed soils than, for example, lead. It is even more accessible and more mobile in cultivated soils under the many influences of soil chemistry (Page et al., 1981).

The predominant state of oxidation in nature is Cd^{2+}, which is a borderline Type (b) cation. In freshwater, cadmium is extensively associated with colloidal and particulate matter, and soluble speciation is confined to the free Cd^{2+} ion together with small amounts of $CdCl_2$ and $CdSO_4$. In the sea, some 66% of cadmium is present as free Cd^{2+} together with $CdCO_3$ (26%), $Cd(OH)_2$ (5%), $CdCl_2$ (1%), and $CdSO_4$ (1%) (Whitfield et al., 1981). In coastal and estuarine waters, a high proportion of cadmium is associated with particles and is present as complexes (Nriagu, 1980; MacKay, 1983).

1.3 Sources

Typical cadmium concentrations found in igneous rocks are 0.001 - 1.8 $\mu g\ g^{-1}$ (mean, 0.15 $\mu g\ g^{-1}$), in metamorphic rocks 0.04 -

1.0 μg g^{-1}, in sedimentary rocks, 0.3 - 11 μg g^{-1}, in shales, up to 90 μg g^{-1}, in marine clays, 0.4 μg g^{-1}, and in marine phosphorites, 60 - 340 μg g^{-1} (Page et al., 1981; Simpson, 1981). Agricultural soils from unpolluted areas usually contain less than 1 μg g^{-1}. These data, derived from sundry original sources, reflect the extreme values that were found and may possibly include analytical discrepancies. Areas in which enhanced levels of cadmium are found are usually linked with the occurrence of zinc-rich ore bodies, zinc smelting, and other zinc-related manufacturing processes and metal plating operations. Localized and naturally high cadmium concentrations may be found near deposits of sulfide ores such as sphalerite, phosphorite, hydrothermally-mineralized rocks, and some black shale deposits such as in the United Kingdom and California, USA.

Smaller but important sources of cadmium are a by-product of copper refining and, to a lesser extent, lead processing. Natural emissions of cadmium to the atmosphere are associated with volcanic eruptions, such as that of Mount Etna (Buat-Menard & Arnold, 1978), and forest fires and windblown dusts (Simpson, 1981; Hutton, 1982). Similarly, cadmium is released into the atmosphere by power generation facilities which use fossil fuels, and by the burning of agricultural and municipal wastes, including dried sewage sludge (Hutton, 1982).

Cadmium in water comes from contaminated agricultural soils, mining wastes, mine waters, and the industrial use of cadmium. An important source is municipal sewage effluents and sludges, including those of domestic origin.

World production of cadmium metal was about 18 900 tonnes in 1979. The main producers are the USA, the USSR, Japan, and Canada, followed by Belgium and France.

The main anthropogenic sources relate to ore mines, metallurgical industries, and to the disposal of sewage sludges. Cadmium concentrations in the fumes of copper, lead, nickel, and zinc sulfide smelters can be relatively high due to the high volatility of the metal (Fleischer et al., 1974). Other major atmospheric inputs come from the combustion of fossil fuels in industries using coke and from the incineration of domestic refuse (Fleischer et al., 1974), as shown in Table 2. These atmospheric inputs have demonstrated effects on agricultural soils and products in the surrounding areas (Peterson & Alloway, 1979). In addition, cadmium may be introduced by urban and motorway dusts, cadmium-contaminated phosphorus-containing fertilizers, and sewage sludge applications on land.

Cadmium is a scarce and fairly expensive metal of low mechanical strength. It is released slowly into the environment from widespread sources. Cadmium is mainly applied via electroplating or dipping to another metal as a thin film coating for protection against corrosion. It is also much used as a pigment in yellow or brown paints and cadmium metal is used in special alloys and solders. Seldom is it possible to recover the metal economically. Use of cadmium in alkaline Ni-Cd rechargeable batteries has potential environmental hazards in view of the amounts of nickel and cadmium

Table 2. Summary of current cadmium inputs to the environment of the European Commission Countries (tonnes year^{-1})[a]

Source	Compartment		
	Air	Land	Water
Volcanic action	20	ND	ND
Non-ferrous metal production			
zinc and cadmium	20	200	50
copper	6	15	ND
lead	7	40	20
Production of cadmium-containing materials	3	90	108
Iron and steel production	34	349	ND
Fuel combustion			
coal and lignite	8	390	ND
oil and gas	0.5	14.5	-
Waste disposal	31	1434	ND
Sewage sludge disposal	2	130	33
Phosphate fertilizers	-	346	62
Totals	132	3009	273

[a] From: Hutton (1982).

involved in discarded used batteries. It is practical and economical, however, to collect worn-out batteries where fixed units are routinely serviced, for this enables about 30% of the cadmium to be re-cycled. World usage of cadmium is given in Table 3.

There is a growing application of organocadmium compounds in the plastics industry. Alkyl-cadmiums are used as polymerizing catalysts in pvc manufacture. Cadmium laurate, stearate, palmitate, myristate, and others are used to reduce weathering effects on plastics. The rate of release of organocadmium complexes to the aquatic environment from these sources is likely to be low. However, the simpler methyl-cadmium (Me2Cd) is decomposed rapidly in air and water.

Table 3. World consumption of cadmium by main uses, 1965, 1970, and 1975[a]

Use	1965		1970		1975	
	tonnes	%	tonnes	%	tonnes	%
Batteries	669	7	842	8	1102	14
Pigments	2463	25	2733	25	1980	25
Stabilizers	905	9	2089	19	1249	16
Plating	4518	47	4068	37	2614	33
Alloys	804	8	803	7	658	8
Others	333	4	382	4	300	4
Total	9692		10 917		7903	

[a] Sources: S.A. Hiscock, Cadmium Association, 34 Berkeley Square, London.

The reasons why sewage (domestic and mixed) may contain high proportions of cadmium relative to other trace metals are not clear, nor is the reason why the cadmium content varies irregularly (Peterson & Alloway, 1979). The cadmium content of 189 samples from 150 wastewater treatment plants in the USA ranged from 3 to 3410 $\mu g\ g^{-1}$ dry weight sludge, with a strong positive correlation to the degree of industrialization in the area observed. The mean value was 16 $\mu g\ g^{-1}$ (Sommers, 1977). Förstner & van Lierde (1979) list values for Sweden, England, and Wales, and Michigan, USA in the lower part of this range, and the more recent ranges for England (Murray et al., 1980) are lower than the above average. The US Food and Drug Administration recommends, along with other restraints, an upper limit of 29 $\mu g\ g^{-1}$ for sewage sludge applied to agricultural land.

Relatively high levels of cadmium are found in dredged spoils. Data from dredging in US waters (Krenkel et al., 1976) range from 0.6 to 4.1 $\mu g\ g^{-1}$ dry weight (8 sites) with 17.6 $\mu g\ g^{-1}$ near Long Island Sound. Ranges were found in the Clyde and other Scottish estuaries for various harbour silts of 5.6 - 6.8 $\mu g\ g^{-1}$. Murray & Norton (1979) report many values in the range 0.2 - 8.2 $\mu g\ g^{-1}$. Using their data, they calculated an annual input of 17 tonnes cadmium for 1977 in the United Kingdom, on the basis that a total of 28×10^6 tonnes soil (14×10^4 tonnes dry solids) is dumped into the North Sea and Irish Sea from harbour and channel dredging in the country.

Studies on the transfer of the different forms of cadmium from freshwater to the sea are inconclusive. Certain estuaries are conservative in most trace

metals (Bewers & Yeats, 1981). The daily inputs of cadmium to inshore waters of the North Sea from the Humber estuary (Table 4) have been assessed by Murray et al. (1980). The relative proportions and magnitudes of these inputs should be of general interest to other fairly heavily urbanized and industrialized estuary areas.

Table 4. Comparison of cadmium inputs to the Humber Estuary[a]

Rivers	15 kg day^{-1}	40.4%
Sewage discharges	2 kg day^{-1}	5.4%
Industrial discharges	8 kg day^{-1}	21.5%
Sewage sludge dumping	0.3 kg day^{-1}	0.8%
Industrial waste dumping	0	0%
Dredged spoil	6.8 kg day^{-1}	18.3%
Atmospheric input	5 kg day^{-1}	13.5%
Direct coastal discharges	0.05 kg day^{-1}	0.1%
Total	37.15	100%

[a] Extract from Table 5 of Murray et al. (1980).

The estimated inputs to the environment of the European Community (Table 2) give a firm indication of emissions to the air and disposals to land but only an incomplete account of aquatic inputs. The generalized global anthropogenic input of cadmium to the ocean is calculated, on an even less firm basis, to be about 50% of the 9.25 x 10^3 tonnes $year^{-1}$ total quoted by Simpson (1981). Mohlenberg & Jensen (1980) give a detailed assessment of cadmium inputs to Danish marine areas.

1.4 Transport, transformation, and bioaccumulation

1.4.1 Transport

Cadmium enters the seas and oceans from the air mainly in particulate form and, to a lesser extent, dissolved in rain and snow. Wittmann (1979) quotes the enrichment of cadmium in atmospheric particulate matter relative to the earth's crust as 300 in north Atlantic westerly winds and 1900 in the urban air of the USA. In some remote areas, volcanic emissions may be the principal source of enrichment. The concentration of cadmium in samples of air, above the Atlantic Ocean between Iceland and the Bermudas, ranged from 0.003 to

0.62 ng m^{-3} (Duce et al., 1976). This concentration range is comparable with that of samples collected in rural areas of the USA (Fassett, 1980). Much higher levels of cadmium in air are observed in urban and industrialized areas, particularly near metal refining and processing plants. Airborne cadmium is a principal source of input to offshore and oceanic waters.

The transport of cadmium from freshwater to the sea occurs either in particulate or soluble form. The specific form depends on the state of the river, its mineralization and its sources of pollution, as well as on unidentified local factors. Quantification is difficult. The general range for cadmium content in clean rivers and lakes is about 0.1 - 1.2 µg $litre^{-1}$; in polluted industrial rivers in the United Kingdom, the USA, and in Europe, it is 1 - 36 µg $litre^{-1}$ (Coombs, 1979). Freshwaters respond to natural or anthropogenic exposure to metalliferous ores, soils, and sediments. Values of 1 - 9 µg $litre^{-1}$ were found for the Jintsu river in Japan which flows through the area where the Itai-itai disease occured; 30 µg $litre^{-1}$ was found in the drainage streams of a nearby ore mine. In the United Kingdom, 3 - 95 µg $litre^{-1}$ was found in streams in North Wales, while 5 - 20 µg $litre^{-1}$ was found in areas in Cornwall affected by ore mining. Rivers are subjected to affected by cadmium inputs from raw and treated sewage, for example, the River Rhine has a range of 1 - 10 µg $litre^{-1}$ (Coombs, 1979).

River sediments generally reflect the neighbouring soils and mineral workings. High cadmium levels are invariably accompanied by high levels of other trace metals. As a result of these inputs, there are enhanced levels of cadmium in near-shore sediments and sea waters (see below).

1.4.2 Transformation

The transformation of inorganic forms of cadmium in sea water scarcely affects its solubility. Biomethylation appears to be a possibility when considering the properties of the element, but this has not been demonstrated. Organic chelates, such as humates, are likely to liberate bound cadmium as the result of dilution and degradation in sea water. Cadmium is held in sewage sludge partly in combination with carbonates and sulfides, and partly in complex organic combinations. In the latter example, cadmium combines with the sulfur-rich fractions of organic matter of which there is great excess, for the inorganic and organic contributions are widely variable (Stover et al., 1976; Sommers et al., 1977). Since most biogenic cadmium-organic complexes, including metallothioneins, are fairly easily biodegradable (Coombs, personal communication, 1983), there is a ready release of cadmium into aerobic waters and sediments; under anaerobic conditions, insoluble cadmium carbonates and sulphides may persist in sediments.

1.4.3 Bioaccumulation

Simpson (1981) regards the processes of uptake (or absorption) by phytoplankton, followed with grazing by herbivores and the subsequent elimination of cadmium in faecal pellets, as a major contributory factor

affecting cadmium distribution in the photic zone. Such a mechanism might also be the cause of elevated concentrations (up to 60 µg g^{-1}) of cadmium in superficial sediments of the Walvis Bay, and might possibly explain the unusually high concentration, up to 600 µg g^{-1}, for Red Sea sedimentary deposits in an anaerobic environment created by a massive bloom.

Many estimates are available for bioaccumulation in marine flora and fauna (Coombs, 1979). For many species, covering most phyla, accumulation factors are of the order of thousands. For some molluscs and some arthropods, they are tens of thousands, and for certain tissues (of which few, if any, are usually eaten by man), they are hundreds of thousands.

Aquatic organisms can be exposed to cadmium in the ambient water, in sediments, and in their diet. The bioavailability of cadmium and, consequently, its accumulation in tissues depends on a number of factors. Non-complexed cadmium added to a rapidly growing algal culture has slightly less effect on the growth rate than when added at the time of inoculation of the medium (Kayser & Sperling, 1980). In experiments with the American oyster, complexed cadmium was found to be less readily accumulated (Yen-Wan Hung, 1982); the lower concentration used, however, 40 - 60 µg cadmium $litre^{-1}$, may also have contributed to this effect.

Ray et al. (1980) found that the rate of cadmium uptake by the polychaete Nereis virens, when exposed to contaminated sediments (1 - 4 mg cadmium kg^{-1}), was the same as its uptake from solutions that contained the same cadmium concentrations (30 - 100 µg cadmium $litre^{-1}$) as that present in the water overlying the sediments. Further studies by Ray et al. (1981b) showed that little accumulation from cadmium-contaminated sediments occurred in Nereis virens, Macoma balthica, and Crangon septemspinosa. Similar studies showed that N. virens, Mercenaria mercenaria, and Palaemonetes pugio did not accumulate cadmium from contaminated sediments during a 100-day exposure period (Rubinstein et al., 1983). Hardy et al. (1981) exposed excised gills of the clam Protothaca staminea to contaminated sediments and interstitial water and concluded that "low level additions of cadmium to sea water are not likely to lead to significant bioaccumulation through the gills of suspension-feeding bivalves". Therefore, cadmium bound in contaminated sediments does not appear to be bioavailable to marine organisms. However, molluscan herbivores may accumulate cadmium from littoral algae, and this may be accumulated, in turn, by carnivores feeding on these organisms (Davies, 1981; Simpson, 1981).

Algae

Phytoplankton can accumulate significant concentrations of cadmium (Kremling et al., 1978; Kayser & Sperling, 1980), although decomposing cells rapidly release cadmium into the water. It is assumed that the metal is loosely bound to the cell's surface, and this process appears to be important in the biogeochemical cycling of cadmium in the marine system. Laminaria saccharina, in common with other seaweeds, can also accumulate significant concentrations (Markham et al., 1980).

Crustaceans

Shrimps and lobsters do not appear to accumulate cadmium from ambient aqueous concentrations of less than about 2 µg litre^{-1} (data reviewed in McLeese, 1980), although White & Rainbow (1982) found some evidence of accumulation at lower concentrations in artificial seawater. The hepatopancreas is a major site of accumulation of cadmium from polluted waters containing higher concentrations (Ray et al., 1981a); studies by Davies (1981) have shown that accumulation at this site is derived from cadmium-contaminated food and not from cadmium in solution. Depuration rates in clean water range from a half-life of 11 days to no loss (data reviewed in McLeese, 1980). Von Bias (1981) found that the amphipod Corophium volutator accumulated cadmium more readily at low salinities, and that no more than 50% was lost on return to clean water.

Molluscs

Greatest attention has been given to this group of organisms, especially bivalves, because of their linear rate of cadmium uptake and their tolerance to high body burdens which makes them good sentinel species. Linear uptake rates have been recorded by von Westernhagen et al. (1978) in Mytilus edulis exposed to 5 µg cadmium litre^{-1} for 163 days; exposure of this species to 10 and 100 µg cadmium litre^{-1} for 17 days also resulted in linear uptake. Body burdens of up to 150 mg kg^{-1} did not affect respiration or growth rates (Poulson et al., 1982). Uptake rates increased with temperature in Crassostrea virginica (Zaroogian, 1980) and Saccostrea echinata (Denton & Burden-Jones, 1981). These 2 authors also recorded increased uptake at lower salinities. Uptake rates can depend on feeding intensity which may be reduced slightly at high cadmium concentrations (Ward, 1982) and are related to ventilation rates as well (Janssen & Scholz, 1979); intermittent emersion of Mytilus edulis appears to increase the rate of uptake during the immersed period as compared with those which are continuously immersed (Coleman, 1980).

Cadmium appears to be bound to the metallothionein in mollusc tissues. In the kidney tissue of Bay scallop (Argopecton irradians) exposed for 5 days to 700 µg cadmium litre^{-1}, the concretions (or granules) contained 60% of the accumulated cadmium, and only 2% was bound to low molecular weight proteins (Carmichael & Fowler, 1981). In addition, in Mytilus edulis which was exposed to 100 µg cadmium litre^{-1} for 3 months, George & Pirie (1979) found that 85% of the cadmium in membrane-limited granular structures may have been associated with metallothionein. In the same species, Marshall & Talbot (1979) found cadmium associated with sulfur and sometimes phosphorus in membrane-bound vesicles.

It is evident that cadmium accumulated from low environmental concentrations can be rapidly bound into a non-toxic complex (Carpene & George, 1981) which is retained within the body and excreted very slowly, if at all. Mowdy (1981) found that 50% of accumulated cadmium in Crassostrea virginica was lost in 60 days in clean water (the rate of loss being slower at low salinites), and George & Coombs (1977) showed that the cadmium excretion

rate in Mytilus edulis was 18 times slower than the uptake rate. Denton & Burden-Jones (1981), also, found that the cadmium half-life in Saccostrea echinata was very long. Mussels loaded with 564 mg cadmium kg^{-1} dry weight lost 47 mg kg^{-1} in a 42-day depuration period, in which time the fraction bound to metallothionein rose from 22 to 78% (Kohler & Riisgard, 1982). However, because accumulation occurs in tissues which may be sites of toxic action, harmful effects may follow when the cadmium binding sites are saturated.

Fish

Cadmium accumulates in the liver and gills of plaice (Pleuronectes platessa) but not in their muscle; liver concentrations began to increase only after 70 days exposure to 5 µg cadmium $litre^{-1}$ (von Westernhagen et al., 1978). Similar data were obtained for plaice by Pentreath (1977) and for dabs (Limanda limanda) by von Westernhagen et al. (1980). Few data are available for depuration rates of cadmium from fish. However, Noel-Lambot (1981) found that the gut of several species of fish contained "intestinal corpuscles" - a mixture of mucous cells, mucous, and granules - which have a high affinity for cadmium. These intestinal corpuscles are passed out with the faeces and may form a detoxification mechanism for fish that have ingested cadmium in sea water. Pentreath (1977) found that 4 days after feeding plaice with cadmium-loaded Nereis, about 5% of the ingested cadmium was associated with the gut wall and none detected in other internal organs. After defaecation, the half-life of the remaining cadmium was between 100 and 200 days.

Birds and mammals

Elevated concentrations of cadmium have been found in the liver and kidney of sea birds (Bull et al., 1977), seals, and porpoises (Falconer et al., 1983), but in no instances have these levels been identified as causing harmful effects. The levels are thought to result from a natural accumulation through the food chain.

1.5 Cadmium in sea water, sediments, and marine biota

1.5.1 Sea water

Many observations of the cadmium content of sea water in estuaries, inshore waters, seas with restricted circulation, shelf waters, and open oceans are detailed in the review by Simpson (1981). In general, the concentration in sea water is about 0.01 - 0.1 µg cadmium $litre^{-1}$, (Preston et al., 1972; Campbell & Loring, 1980; Magnusson & Westerlund, 1980; Simpson, 1981). Values which are about 5 - 10 times higher, 0.2 - 0.4 µg cadmium $litre^{-1}$, have been reported from certain coastal areas, such as the Oslofjorden, Norway, and Liverpool Bay, United Kingdom (Preston et al., 1972; Rojahn, 1972). The very local nature of estuarine variability was already described, and the broad pattern of declining land influences can be traced in decreasing cadmium concentrations towards the open ocean (Table 5).

Table 5. A summary of cadmium distribution in saline waters[a]

	Background	"Normal" range (dissolved, μg litre^{-1})	Suspected contamination	Top of range	Background	"Normal" range (sediment, μg g^{-1})	Suspected contamination	Top of range
Estuaries and closed bays	0.01	0.05 - 0.2	> 0.2	45.7	< 1.0	0.1 - 2	> 2.0	50 000
Bays and coastal waters	< 0.01	0.01 - 0.15	> 0.15	10.3 (100)	< 1.0	0.1 - 1.5	> 1.5	60
Open sea	< 0.01	0.01 - 0.1	> 0.1	1.6 0.65	< 1.0	0.1 - 1.0	> 1.0	(600)
Open ocean	< 0.005	0.01 - 0.1	> 0.1	(1.61)	< 1.0	0.1 - 1.0	> 1.0	0.977

[a] From: Simpson (1981).

In the more stable water regime of the open ocean a significant reduction in cadmium, parallel to the reduction in phosphate and nitrate, can be demonstrated in the upper water layer which is influenced by the photic zone and phytoplankton productivity (Simpson, 1981).

1.5.2 Sediments

The same progression is even more evident in the cadmium content of bottom sediments (Nicholson & Moore, 1981; Simpson, 1981). Extremely high values are reported for enclosed waters affected by local ore mining (US east coast, before dredging, 30 - 18 400 $\mu g\ g^{-1}$; after dredging, 40 - 5000 $\mu g\ g^{-1}$), but values in fjords, harbours, and estuaries with marked industrial and urban influence are more commonly in the range 10 - 1000 $\mu g\ g^{-1}$. In many of the cleaner inshore areas, a fairly broad range (about 0.2 - 5.0 $\mu g\ g^{-1}$) is common at each site, with upper values extending to 10 $\mu g\ g^{-1}$ where mineralization and other influences, such as oil activities, are present. Sediments in the Baltic Sea range from 0.2 - 2.2 $\mu g\ g^{-1}$, while the values for the North Sea are below 1.0 $\mu g\ g^{-1}$ for 80% of the samples (Nicholson & Moore, 1981), with indications of higher values near sites of oil-related activity.

In the open oceans, most values are below 0.5 $\mu g\ g^{-1}$; Aston et al. (1972) report a mean of 0.23 $\mu g\ g^{-1}$ for cadmium in deep-sea sediments from the North Atlantic and a higher mean of 0.65 $\mu g\ g^{-1}$ for samples taken near the mid-Atlantic Ridge. An important feature to take into account is the influence of upwelling. Diatomaceous ooze from Walvis Bay, a major upwelling area, contained 3 - 60 $\mu g\ g^{-1}$. This compared with samples farther offshore of diatomaceous ooze which showed 0.17 - 0.88 $\mu g\ g^{-1}$ and of radiolarian ooze which had 0.13 - 0.98 $\mu g\ g^{-1}$. An extensive Atlantis II survey of the deeps in the Red Sea also yielded high values: 2 - 600 $\mu g\ g^{-1}$ and 30 - 3900 $\mu g\ litre^{-1}$ in the interstitial water (Simpson, 1981).

1.5.3 Marine biota

Phytoplankton

It is particularly difficult to determine accurately the cadmium content of the phytoplankton which are the first stage in the marine food web. Likely values do not appear to exceed a few $\mu g\ g^{-1}$ on a dry weight basis (Kremling et al., 1978; Kayser & Sperling, 1980), and much of the cadmium taken up is rapidly released from decomposing cells. The attached green and red macroalgae do not accumulate above about 2.1 $\mu g\ g^{-1}$, but brown algae contain a wide range (0.2 - 26 $\mu g\ g^{-1}$), which is believed to reflect the ambient cadmium concentration.

Zooplankton

The cadmium content of protozoa, parazoa, cnidaria, euphausids, and chaetognaths is normally well below 2 $\mu g\ g^{-1}$, although a single sample of

copepods (crustacea) had a content of 9.8 µg g^{-1}. Arthropods such as the sea-skaters (Halobates sp) have shown a notably high cadmium content with values of 50 - 210 µg g^{-1} (Bull et al., 1977) and 1.7 - 120 µg g^{-1} (Schulz-Baldes & Cheng, 1980). These insects live on and feed in the upper microlayer of the seas or oceans where the atmospheric cadmium input may have a special influence.

Molluscs

Among the molluscs, the edible gastropods such as limpets (Patella sp), ormers (Haliotis sp), and whelks (Buccinum sp), which live in the intertidal and subtidal zones, can contain exceptionally high levels of cadmium, namely 0.2 - 295 µg g^{-1} for limpets and ormers, and 0.05 - 730 µg g^{-1} for whelks. The bivalves, shellfish which are commercially important, can also contain the following moderately high cadmium concentrations: mussels (Mytilus edulis) and scallops (Pecten maximus) have values of 0.04 - 140 µg g^{-1}, cockles (Cardium edule and other species), oysters, and clams show 0.3 - 170 µg g^{-1}. Other important commercial shellfish (cephalopods), cuttle-fish, squids, and octopuses can have exceptionally high liver cadmium contents, 23 - 1100 µg g^{-1}, but the decapods (shrimps, prawns, lobsters and, particularly, crabs) have a lower range, 0.5 - 3.3 µg g^{-1}. The cadmium value of the brown meat of Cancer pagurus has values up to 9.1 µg g^{-1} wet weight in certain areas (Davies, 1981). Most concentrations of cadmium in molluscs vary greatly with location. In fact, mussels are widely used in monitoring and are useful indicators of pollution. The Joint Monitoring Programme of European Countries reports general results for Mytilus edulis of less than 0.7 µg g^{-1} wet weight. Values up to 15 µg g^{-1} have, however, been reported in localized regions (Paris Commission, 1983).

Fish

Regular monitoring has ascertained that most commercial fish species contain very low concentrations of cadmium (Murray, 1979; Davies, 1981). A recent comprehensive study from the Swedish National Food Administration, accompanied by a quality assurance programme, showed mean cadmium levels ranging from 1 to 27 µg cadmium kg^{-1}. There are reports of somewhat higher levels (Coombs, 1979), but there is no information related to quality assurance. The results of the Joint Monitoring Programme of European Countries for 1978-80 has shown generally very low concentrations of cadmium in muscle of fish, independent of species. The values were mostly less than 20 µg kg^{-1} wet weight (Paris Commission, 1983).

Mammals and birds

Cadmium levels are not markedly high in the top predators such as the common porpoise (Phocoena phocoena). Mean values found are, for male liver, 0.15, for male kidney, 1.1, for female liver, 0.27, and for female kidney, 2.7 µg g^{-1} wet weight (Falconer et al., 1983). Value ranges for the grey seal (Halichoerus grypus) are, for liver, 0.07 - 8.5 µg g^{-1} wet weight

and, for kidney, 0.10 - 15 μg g^{-1} wet weight (McKie et al., 1980). Somewhat higher cadmium levels are reported for Atlantic seabirds (fulmar, Manx shearwater, puffin, Leach's petrel, storm petrel, razorbill). For example, in the liver were found 1.4 - 57 μg g^{-1} dry weight and, in the kidney, 15 - 240 μg g^{-1} dry weight were found, allowing for wet/dry weight basis (Bull et al., 1977).

2. Effects on Marine Biota

2.1 Reference documentation

Information consulted included the reviews by Alabaster (1978) and the US EPA (1980) and the tabulations of data on concentrations and effects summarized by Taylor (1981) and IRPTC (1981).

2.2 Effects on marine biota

Data given in the summaries by Taylor (1981) and IRPTC (1981) indicate that cadmium is not very toxic within short exposure periods, and the 96-h LC_{50}s for a wide range of species are usually in excess of 1 mg cadmium $litre^{-1}$. Similarly, chronic effects usually become apparent at concentrations greater than 50 μg cadmium $litre^{-1}$. It is most unlikely that these concentrations will occur in the future, even in the most polluted situations.

However, some species have been reported to be affected at cadmium concentrations less than 15 μg $litre^{-1}$ and usually after prolonged exposure under laboratory conditions. It is these data which form the basis of this critical review.

Algae

The growth rate of the dinoflagellate Prorocentrum micans was inhibited by 1.2 μg cadmium $litre^{-1}$ with resulting cell numbers in the cultures being less than one tenth of the control values; no effect was found at 0.4 μg cadmium $litre^{-1}$ (Kayser & Sperling, 1980). However, Prévot (1980) found that the growth rate of this species was only slightly affected at 5 μg cadmium $litre^{-1}$ and then only after 22 days exposure. A 50% reduction occurred at 60 μg $litre^{-1}$ with 30 days exposure. The reasons for this difference in results are not known. Concentrations greater than 10 μg cadmium $litre^{-1}$ were found to increase the vacuolation and number of lysosomes in this species (Soyer & Prévot, 1981). Li (1980) recorded a reduced growth rate of Isochrysis galbana when exposed to 1 μg cadmium $litre^{-1}$ for 10 days. Kayser (1982) found that 10 μg cadmium $litre^{-1}$ temporarily reduced the growth rate of Scrippsiella faeroense, but there was no such effect at 2.0 μg $litre^{-1}$. In contrast, Fisher & Froud (1980) found that 25 μg cadmium $litre^{-1}$ had no effect on the growth of 4 other species of diatoms, and Kremling et al. (1978) found that 1 μg cadmium $litre^{-1}$ had no effect on phytoplankton communities in a mesocosm. Other data indicate that concentrations greater than

100 µg cadmium litre^{-1} are required to produce effects on a wide range of algal species.

Coelenterates

At a salinity of 10 °/oo and at 17.5 °C, irreversible retraction of 50% of hydranths of Laomedea loveni occurred at about 3 µg cadmium litre^{-1}, whereas the same effect was produced at 15 µg litre^{-1} at 25 °/oo salinity (Theede et al., 1979). Other hydrozoa tested appear to be more resistant to cadmium.

Growth and survival of the ctenophore Pleurobrachia pileus were adversely affected by 1 µg cadmium litre^{-1} in a marine mesocosm (Kuiper, 1981).

Annelida

This group of organisms appears to be very resistant to cadmium, no effects being recorded at concentrations less than 100 µg litre^{-1} (Kuiper, 1981).

Crustacea

Embryonic development of the mud crab Eurypassopeus depressus to the megalopa stage was not affected by 10 µg cadmium litre^{-1}, but subsequent development to the crab stage was delayed and increased mortalities were noted. Phototactic swimming rates at Stages II and III were higher in larvae exposed to cadmium (Mirkes et al., 1978). Copepod populations grew in a mesocosm when exposed to 5 µg cadmium litre^{-1} due to a decrease in predation by ctenophores; but exposure to 50 µg cadmium litre^{-1} reduced their numbers (Kuiper, 1981).

The estuarine mysid (Mysidopsis bahia) appears to be very sensitive to cadmium with a 17-day LC_{50} of 11 µg litre^{-1}, and a 50% reduction in numbers of young produced per female occurred in 6.4 µg litre^{-1}, although there was no effect at 4.8 µg litre^{-1} (Nimmo et al., 1978). Further studies by Gentile et al. (1982) on this species, and also on M. bigelowi, showed that carapace malformation occurred at 10 µg cadmium litre^{-1} and that this affected brood production, but there was no such effect at 5 µg litre^{-1}.

Experiments with the lobster Homarus americanus showed that malate dehydrogenase activity intensified with exposure to 6 µg cadmium litre^{-1} for 30 days; the MDH:LDH ratio was also higher (Gould, 1980). Similar experiments showed that isolated gill respiration rates increased when lobsters were exposed to 3 µg cadmium litre^{-1} for 30 days, but no effects on osmoregulation were found (Thurberg et al., 1977). Using the brown shrimp Crangon crangon, Price & Uglow (1980) found an increase in scaphognathite rate during a 13-day exposure to 5 µg litre^{-1}. This is said to be close to the incipient lethal level, although no experimental data are given.

Molluscs

Although molluscs are resistant to cadmium toxicity, with effects on adults and larval stages being recorded at concentrations usually greater than 70 µg litre^{-1}, Zaroogian & Morrison (1981) found that 5 µg litre^{-1} slightly delayed the development of 10% of Crassostrea virginica larvae. However, after 3 weeks of exposure, the growth rates in both 5 and 15 µg cadmium litre^{-1} solutions were similar to those of the controls. Similarly, Watling (1982) found that 10 µg cadmium litre^{-1} caused a reduction in growth rate of C. margarita, which increased to above the control rate when returned to clean water. Much higher concentrations were required to produce this effect in C. cucullata and C. gigas. Stromgren (1982), too, found that 10 µg cadmium litre^{-1} reduced the growth rate of mussels (Mytilus edulis) within a 9-day exposure period; 5 µg litre^{-1} had no significant effect, and 2 µg litre^{-1} stimulated growth to 145% of the control value.

Echinoderms

This group is resistant to cadmium. No harmful effects were recorded at concentrations less than 100 µg cadmium litre^{-1}; this included genotoxicity (Pagano et al., 1982).

Tunicates

Recent experiments show that Botryllus schlosseri is very resistant to cadmium (Kayser, 1982). Sub-lethal effects occurred only after prolonged exposure to more than 5 mg cadmium litre^{-1}.

Fish

Ojaveer et al. (1980) found that eggs of Baltic herring began to hatch earlier when exposed to 5 µg cadmium litre^{-1}, and newly-hatched larvae were 10% smaller than the controls; the significance of these small differences is not clear. Plaice (Pleuronectes platessa) exposed for 280 days to 5 µg cadmium litre^{-1} grew more slowly than the controls between the 70- and 130-day exposure period; the feed ration in both groups was reduced during this period. After 130 days, the growth rate of all fish were similar (von Westernhagen et al., 1978). Hyperactivity of plaice was noted among the cadmium-exposed fish; a similar effect was noted in flounders (Platichthys flesus) that were exposed to 500 µg cadmium litre^{-1} but not those exposed to 50 µg cadmium litre^{-1} (Larsson et al., 1981). Exposure of plaice to 50 µg cadmium litre^{-1} resulted in a 90% mortality rate within 96 days and an LT_{50} of 30 days. Mortality among dabs (Limanda limanda) similarly exposed was 30% in 96 days, but no effects on growth of the survivors were noted (von Westernhagen et al., 1980). Mortality was thought to be caused by secondary infection of fin erosions. Weis & Weis (1976) noted that 10 µg cadmium litre^{-1} temporarily inhibited caudal fin regeneration in the killfish Fundulus heteroclitus.

Larsson (1975) found that the blood haematocrit and haemoglobin content of flounders was unaffected in a 4-week exposure to 5 µg cadmium litre^{-1}, but a 26% reduction from control values occurred within the next 5 weeks. On the other hand, Calabrese et al. (1975) found that 5 and 10 µg cadmium litre^{-1} had no effect on the haematological parameters of the winter flounder Pseudopleuronectes americanus within 60 days, although the oxygen consumption rate of excised gills was 15% lower than the control values. In similar experiments with this species, Gould (1977) found that production of the zinc-containing enzymes leucine aminopeptidase and carbonic anhydrase was stimulated, but the sensitivity of G6PdH to magnesium was lost; these may have been adaptive responses. Larsson et al. (1981) found a reduction in the calcium and potassium content of blood plasma in flounders exposed to 5 µg cadmium litre^{-1} for 9 weeks, but increases in phosphorus and magnesium took place only at 50 µg cadmium litre^{-1}. There was no effect on bone structure. Oxygen consumption of excised gills of striped bass (Morone saxatilis) exposed to 5 µg cadmium litre^{-1} for 30 days was 27% lower than the control values, although this effect was neither apparent at 90 days nor was it apparent in gills from fish exposed to 2.5 µg cadmium litre^{-1}. No effect on the enzymes AAT and G6PdH was noted during the exposure period, although there was a decrease in their concentration when fish exposed to 5 µg cadmium litre^{-1} were returned to clean water for 30 days (Dawson et al., 1977). It is not clear whether any of these changes can be considered as harmful.

3. Human Health Aspects

3.1 Reference documentation

The major reviews on cadmium consulted in the preparation of this section include Friberg et al. (1974), Tsuchiya (1978), Simpson (1981), Friberg et al. (in press), and the interim report on Environmental Health Criteria for Cadmium (WHO, 1979).

3.2 Toxicokinetic properties

The average absorption of cadmium from food is approximately 5% (Rahola et al., 1972), but people suffering from anaemia or those on a calcium-deficient diet may have a considerably higher rate of absorption (up to 20%) (Flanagan et al., 1978).

Cadmium is transported via the blood to other parts of the body. In blood, cadmium is mainly found in the red cells, where it is bound to a protein of low molecular weight, metallothionein. Most cadmium is stored in liver and kidneys where, after long-term low-level exposure, approximately 50% of the cadmium is to be found. About one third of the body burden is located in the kidneys, where the concentration in kidney cortex is probably about 1.25 times the average concentration in the whole kidney (Kjellström et al., 1984). Accumulation also takes place in certain other tissues, such as the muscle (Kjellström, 1979), where the biological half-time is long;

concentrations are usually low, but with large amounts of tissue, an important part of the body burden is accounted for.

The placenta serves as an effective barrier against cadmium uptake, and the newborn is practically free from cadmium. The total body burden at birth is only about 1 µg, but continuous accumulation takes place in the body up to about the age of 50. At this age, the total body burden is between 10 and 30 mg with concentrations in the kidney cortex of 15 - 50 mg cadmium kg^{-1}. In countries where exposure via food is high, such as Japan, the total body burden and concentrations in kidney cortex may be considerably higher.

The biological half-time in the kidneys is long, approximately 20 years and possibly somewhat shorter at old age, which explains the accumulation of cadmium. In blood, part of the cadmium is related to body burden and has a long biological half-time. As a rule, the major part of cadmium in blood is related to recent exposure and has a half-time of about 2 - 3 months. The concentration in blood is, therefore, a useful indicator of exposure during recent months. The concentration in blood is low, usually below 1 µg cadmium $litre^{-1}$ in non-smokers, but may reach several µg cadmium $litre^{-1}$ in smokers. If exposure remains constant, blood levels may also be used for evaluation of long-term risks (WHO, 1980).

Due to the long biological half-time of cadmium in the body, only a small part of cadmium attributable to long-term low-level exposure will be excreted. Excretion takes place via faeces and urine and comprises only 0.005 - 0.1% per day of the total body burden. Since only a minor portion of ingested cadmium is absorbed, the amount excreted via faeces can be used to estimate total daily intake. Concentrations in urine will increase with age. As for long-term low-level exposure, excretion is related to body burden and concentrations in the kidney cortex. Cadmium levels in urine are not usually good indicators of recent exposure. When concentrations in kidney cortex reach critical levels and signs of kidney dysfunction occur, the excretion of cadmium increases dramatically. Concomitantly, the cadmium concentration in the kidneys decreases. This explains why people displaying advanced signs of cadmium intoxication may have cadmium concentrations in the kidneys which are very low. At high exposure levels, such as can be seen in workers of certain industries, metabolism is different. Relatively more cadmium is accumulated in the liver, and concentrations in the blood and urine may be more difficult to interpret as indicators of exposure and body burden. For a detailed discussion of the metabolic model for cadmium, reference is made to Kjellström & Nordberg (1978) and Camner et al. (1979).

Very limited data are available on the different forms of chemical binding of cadmium to proteins or other compounds in foodstuffs and the influence this may have on the toxicokinetics of cadmium. Recent studies show cadmium is bound to different proteins in different species of oysters. In New Zealand Bluff oysters, cadmium is bound to a protein similar to metallothionein. People with extreme consumption of such oysters, leading to a daily cadmium intake of 200 - 500 µg cadmium, were found to have disproportionally low blood cadmium levels (McKenzie et al., 1982). This may indicate that the

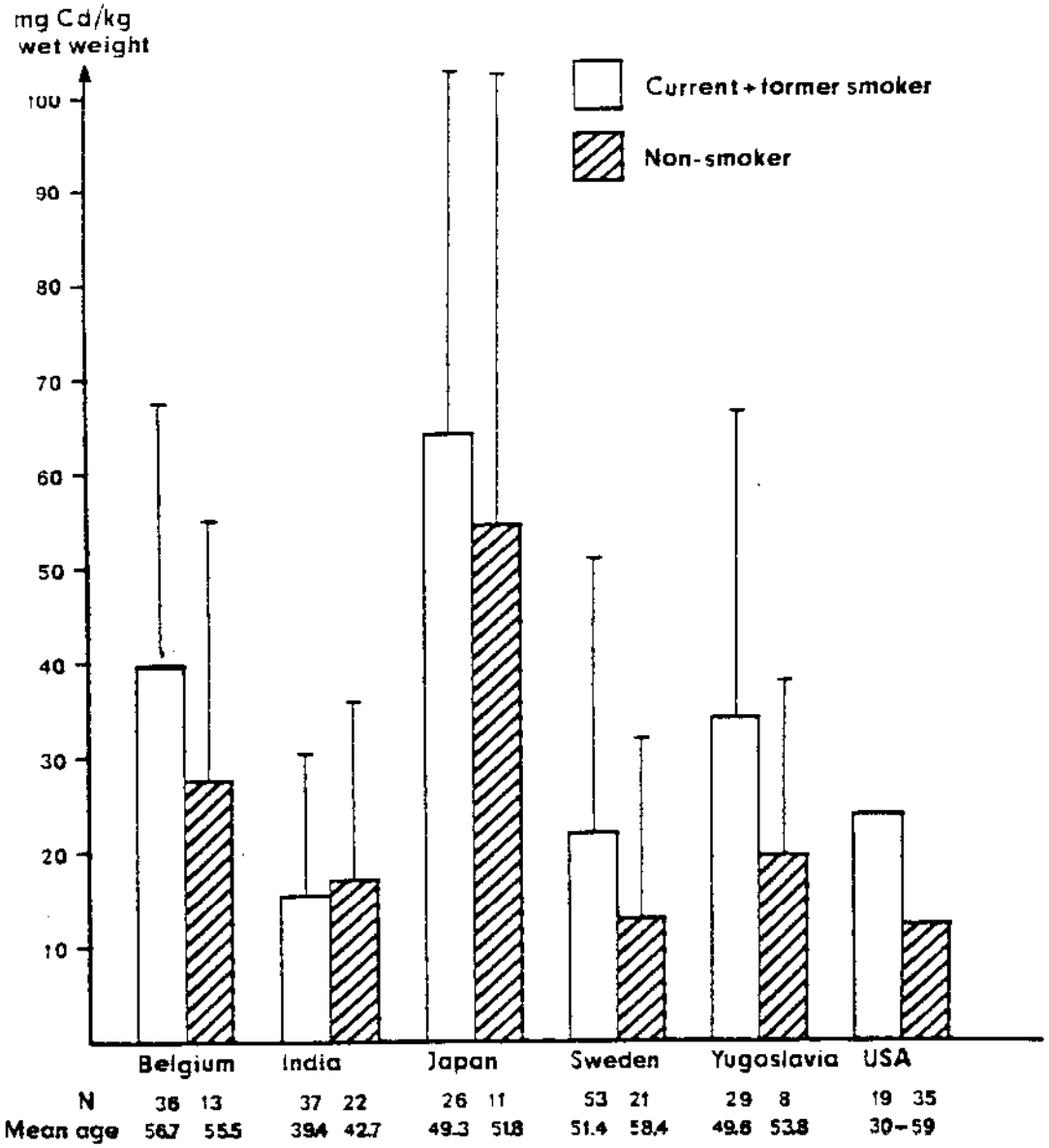

Fig. 1. Concentration of cadmium in kidney cortex (geometric mean values with 1.28 times the geometric standard deviations indicated) in relation to smoking habits among the subjects (30 - 69 years of age) studied in Belgium, India (data from Ahmedabad, Bangalore, and Calcutta pooled), Japan, Yugoslavia. Swedish data from Elinder et al. (1976). Number of smokers (including former smokers) and non-smokers as well as mean age of subjects in each subgroup are indicated under the bars.

distribution of cadmium bound to metallothionein is different from unbound cadmium. Animal data support this view. Cadmium metallothionein administered parenterally or perorally is transported directly to the kidneys (Cherian & Shaikh, 1975; Nordberg et al., 1975; Cherian et al., 1978). Thus, blood cadmium may not reflect the daily intake in the same way for all foodstuffs. Contrary to this, cadmium absorption via tobacco smoking gives rise to very high blood cadmium levels which may not be accompanied by a proportionate increase in kidney burden (Elinder et al., 1983).

3.3 Health effects

Ingestion of highly contaminated food and drink may give rise to local gastrointestinal symptoms including vomiting, diarrhoea and, in severe cases, shock. Contamination of food may arise from cadmium-containing solders in water pipes, cooling or heating devices, or from dissolution of cadmium in pottery painted with cadmium-containing pigments. Formerly, the use of cadmium-plated cooking utensils was a common source of acute cadmium intoxication. Concentrations of 15 mg cadmium $litre^{-1}$ in water may give rise to acute symptoms including vomiting. Acute manifestations of cadmium intoxication arising from marine pollution have not occurred.

Chronic cadmium intoxication may be a result of long-term exposure via inhalation of cadmium fumes or dust, or from peroral exposure to contaminated food or beverages. The critical organ, i.e., the organ in which the first signs of adverse effects may be seen, is the kidney. The critical effect is a decrease in renal tubular reabsorption of proteins. One major sign of this effect is an increased urinary excretion of low molecular weight proteins, such as $beta_2$-microglobulin and retinol-binding proteins. A continuous catabolism of the cadmium metallothionein takes place after reabsorption, and cadmium is split from the metallothionein and bound to newly formed metallothionein in the tubular cells. It is supposed (Friberg et al., 1974; Nordberg, 1978; Nomiyama & Nomiyama, 1982) that kidney damage is prevented until such a stage is reached that the kidneys no longer produce enough metallothionein, and the free cadmium ions become very toxic to enzymatic processes. In more advanced cases, more extensive kidney damage occurs. This type of cadmium intoxication has frequently been observed following inhalation of cadmium in certain industries and after ingestion of contaminated food, particularly rice where, for example, contaminated water has been used for irrigation in certain areas of Japan. In contaminated areas, concentrations in certain crustaceans may also contribute significantly to high exposure levels. Once tubular proteinuria is manifest, it persists even though exposure ceases.

Other signs of cadmium intoxication which may occur at a later stage include anaemia and liver disorders. At a very late stage, effects on the bone in the form of osteoporosis and/or osteomalacia have been observed for cases of industrial exposure and in Japan as a result of ingestion of contaminated food. In Japan, the ensuing disease has been called Itai-itai, the manifestations of which are a combination of severe renal tubular damage and osteomalacia.

Even mild trauma may give rise to multiple fractures of the skeleton. The detailed pathogenesis of Itai-itai disease is not clear. Factors other than cadmium, such as low intake of calcium, proteins, and vitamin D, have been of importance, but cadmium is a necessary factor for the development of the disease. At present, the occurrence of osteomalacia appears to be rare.

Some animal experiments indicate that hypertension can be induced by cadmium, but there are no convincing data indicating that cadmium can give rise to such symptoms in human beings.

Animal data show conclusively that injection of cadmium may cause sarcoma at the site of injection and also interstitial tumours of the testes. In a recent study, long-term inhalation of a cadmium chloride aerosol resulted in a pronounced and dose-related increase in the incidence of lung cancer (Takenaka et al., 1983). There is as yet no evidence from animal experiments which indicate that peroral exposure to cadmium increases the risk of developing cancer. Some epidemiological evidence exists, however, which suggests that cadmium may contribute to the development of cancer of the prostate as judged from studies on heavily-exposed workers (Belman & Nordberg, 1981). There are also some data suggesting a possible role of cadmium in the development of lung cancer in exposed workers. The recent animal data referred to above strengthen the suspicion that inhaled cadmium may be a human carcinogen for lung cancer. IARC (1976) concluded that occupational exposure to cadmium in some form, possibly the oxide, increases the risk of prostatic cancer and that one study also suggested an increase of respiratory cancer.

3.4 Total exposure to cadmium

Man is exposed to cadmium from the working environment, ambient air, drinking-water, tobacco, and food. For the non-occupationally exposed, food is the major source of intake. Among non-smokers, food could contribute 80 - 90% of the total intake of cadmium. In countries where intake via food is low, smoking is a major source of exposure and may contribute about half the body burden of cadmium. Normally, 0.1 - 0.2 µg cadmium is inhaled by smoking one cigarette. Concentrations of cadmium in ambient air are on an average approximately 5 ng m^{-3} and will not contribute significantly to the daily intake of cadmium.

Méranger et al. (1981) reported on cadmium concentrations in raw, treated, and distributed water from 71 municipalities in Canada. The mean cadmium concentration was $\leq$ 0.02 µg cadmium $litre^{-1}$ with a range of $\leq$ 0.02 - 0.9 µg cadmium $litre^{-1}$.

In the absence of specific sources of contamination, most foodstuffs will contain less than 0.5 mg cadmium kg^{-1} wet weight (Friberg et al., 1974; FAO/WHO, 1982; Elinder, in press). Low concentrations (1 - 50 µg kg^{-1}) are reported for meat, fish, and fruit. Somewhat higher concentrations have been reported for vegetables and cereal crops, e.g., wheat and rice, where concentrations in unpolluted areas may range from 0.01 to 0.15 mg cadmium kg^{-1}. In polluted areas, concentrations often reach 0.3 - 0.5 mg

cadmium kg^{-1}. High concentrations (0.01 - 1 mg cadmium kg^{-1}) are found in the liver and kidneys of adult animals. Adult horses may contain up to 10 mg cadmium kg^{-1} in liver and 10 - 150 mg cadmium kg^{-1} in kidney cortex.

Data on total daily intake may also be unreliable due to a lack of quality assurance. Available data indicate, however, that the daily intake of cadmium in the USA and Europe is in the order of 20 µg cadmium, although large individual variations exist. In Japan, the average cadmium intake is 40 - 50 µg cadmium day^{-1}. In contaminated areas, the daily cadmium intake may be several times greater (Elinder, in press).

3.5 Contribution of cadmium from marine food

Data on cadmium concentrations in marine food indicate the general levels of exposure that may be expected. Most fish species contain relatively low cadmium levels of less than 0.4 µg g^{-1}. Some species, however, have somewhat higher concentrations; for example, flounder can contain up to 7 µg g^{-1}. For average per capita consumption of fish, however, this is a minor source of cadmium intake.

Certain species of mussels, scallops, and oysters often have cadmium concentrations exceeding 1 mg cadmium kg^{-1} (FAO/WHO, 1982). In New Zealand oysters, cadmium concentrations ranging up to 8 mg cadmium kg^{-1} wet weight have been found (Nielsen, 1975). Brown meat of crabs may contain 1 - 30 mg cadmium kg^{-1} (UK Ministry of Agriculture, Fisheries and Food, 1973). Examples were given above in section 1.5.3 of even higher concentrations of cadmium in some molluscan species. It is important to determine the forms of cadmium in these organisms and their availability following ingestion by man. This source of cadmium could be of significance to total cadmium intake for particular regional population groups.

3.6 Evaluation of potential health effects

For most people, food is the major source of cadmium exposure. Special risk groups are smokers, who readily acquire blood cadmium levels which, on an average, are twice those in non-smokers. The average absorption of cadmium via food is probably around 5%, but with low body iron stores or calcium deficiency, absorption can increase up to 10 - 20%. There are, however, no large scale epidemiological studies to confirm the role of nutritional deficiencies. Analyses of cadmium in blood and kidney cortex in normal people show that 90 percentile values for kidney cortex levels are often about twice the median values. The difference between 90 percentile values and median values for blood levels is often greater. In some studies, the quotient is 5 or higher (Vahter, 1982; Friberg & Vahter, 1983).

Cadmium has been called the dissipated element because of its widespread occurrence in the environment and in different products. This implies the achievement of reduced cadmium exposure, measures have to be taken against a number of sources in order to minimize environmental contamination. For the average individual, uptake of cadmium in staple foods such as wheat and rice

from contaminated soil is probably of greatest importance. For people with special food habits, other foods may be of equal or even greater importance. Thus, extensive long-term consumption of liver and kidney from certain animal species may considerably increase the daily intake of cadmium. Similarly, certain marine species, such as mussels and oysters, may contain high concentrations of cadmium and there are examples where extreme consumption of such oysters has led to a daily cadmium intake of 200 - 500 µg cadmium, which is more than 10 times the average daily intake of cadmium usually found.

In cadmium intoxication, the kidney cortex is the critical organ, the first signs being tubular dysfunction. The critical concentration is established on an individual level and varies among individuals. A WHO Task Group (WHO, 1977a), engaged in the preparation of WHO Environmental Health Criteria for Cadmium, has estimated the most likely critical concentration to be 200 µg g^{-1} wet weight in kidney cortex. At that time, the variations among individuals were not taken into consideration. Friberg & Kjellström (1981) and Kjellström et al. (1984) proposed a "population critical concentration" (PCC) taking into consideration the distribution of the critical concentrations within a population. In vivo analyses of cadmium in the renal cortex of workers with and without cadmium-induced renal tubular damage have produced the following estimates of the average critical concentration: (PCC_{50}) of 332 µg cadmium g^{-1} (Roels et al., 1983) and 319 µg cadmium g^{-1} (Ellis et al., 1981). The individual variation is considerable in both studies, and Roels et al. (1938) estimated the PCC_{10} at 216 µg cadmium g^{-1} wet weight. It should be pointed out that the effects measured in these studies may not have been the earliest signs of cadmium intoxication. Furthermore, the empirical data referred to above by Ellis et al. (1981) and Roels et al. (1983) should be reduced by about 20%, since they used a factor of 1.5 in calculating concentrations in the kidney cortex from concentrations in the whole kidney. Recently, it has been observed that the factor should more likely be 1.25 (Svartengren et al., in press).

If 5% absorption of cadmium is assumed with 30% distributed to the kidneys, and a one-compartment metabolic model were used (half-time = 20 years), an intake of 200 - 400 µg cadmium day^{-1} would result in approximately 200 µg cadmium g^{-1} in the kidney cortex after approximately 50 years of exposure (Friberg et al., 1974). An absorption of 10% would correspondingly give 200 µg g^{-1} in the kidney following an intake of 100 - 200 µg cadmium d^{-1} over a 50-year period. Epidemiological data relating to exposure and effects after peroral intake are available from Japan. In the interim report by the above-mentioned WHO Task Group, it was estimated that, using the most sensitive analytical methods, a daily intake of approximately 200 µg cadmium day^{-1} had caused the occurrence of an increased excretion of low molecular weight proteins. Thus, estimates based on critical concentrations in kidneys and metabolic models, together with epidemiological data, are in fairly close agreement.

4. Conclusions on Cadmium

4.1 Potential harm to living resources

Cadmium inputs to the marine environment are derived from both natural (volcanoes, dusts, and runoff) and anthropogenic sources. A major industrial use of cadmium is in electroplating, and discharges can occur from this activity. Other point source inputs are derived from zinc ore-smelting and phosphate fertilizer manufacture. Diffuse atmospheric inputs are derived from combustion of fossil fuels and domestic wastes. Estimates of oceanic fluxes of cadmium indicate that 50% is of anthropogenic origin, and this may have increased slightly in recent years. The normal range of cadmium in offshore water is 0.01 - 0.1 µg litre^{-1}; higher levels occur near point source inputs. Cadmium may be released into the water during dredging and disposal of dredged spoil, although the extent to which this occurs is not known.

Organocadmium compounds may be discharged to the marine environment. There is evidence, however, that alkyl compounds are rapidly degraded by abiotic processes in the environment.

Data from a large number of laboratory experiments show that a wide range of marine species are not acutely affected at cadmium concentrations less than 15 µg cadmium litre^{-1}. Moreover, very few of the species tested exhibited chronic effects from long-term exposure below this concentration, although one or two species of algae, two species of coelenterate, and one crustacean were harmed by concentrations within the range of 1 - 15 µg cadmium litre^{-1}; it is likely that there are other, untested species of similar sensitivity. However, it would appear that the present level of cadmium in offshore water is below that which would cause harmful effects to marine biota in general. Elevated concentrations in the vicinity of point source discharges could, however, cause harm to local species. Since estuaries and coastal waters are frequently the recipients of point source inputs, the human consumption of fish and shellfish from these areas may be affected either directly or indirectly.

Some resistant species, such as crabs and molluscs, may accumulate high levels of cadmium in their soft tissues without apparent detriment to their well-being. This accumulation may be derived from soluble cadmium in the water, or through the food chain via cadmium-contaminated algae, and this can occur even from low concentrations, in water not affected by discharges. The element can be complexed in tissues, but the availability of such cadmium to natural predators of these organisms is not known.

4.2 Potential hazards to human health

On an average, approximately 5% of ingested cadmium is absorbed, but individuals with a low iron store or on a calcium-deficient diet may have considerably higher absorption (up to 20%). About one third of the body burden of cadmium is located in the kidneys, where the concentration in kidney cortex is about 1.25 times the average concentration in the whole kidney. The

biological half-time is long, approximately 20 years, and cadmium is, therefore, accumulated in the body. The total body burden at birth is only 1 µg, but continuous accumulation takes place in the body and, at age 50, the total body burden is between 10 and 30 mg, with concentrations in the kidney cortex of 15 - 50 µg cadmium g^{-1} wet weight.

The first sign of cadmium intoxication is a dysfunction of the kidneys in the form of a decreased renal tubular absorption of proteins. This is the critical effect of cadmium with long-term exposures. If such an effect were prevented, even more serious effects could be avoided.

A WHO Task Group engaged in the preparation of the WHO Environmental Health Criteria for Cadmium has estimated the most likely critical concentration (when a small percentage of exposed people will show effects) to be 200 µg g^{-1} wet weight in the kidney cortex. If a 5% absorption of cadmium is assumed, this concentration will be reached after an approximately 50-year exposure period to 200 - 400 µg cadmium day^{-1}. If a 10% absorption is assumed, the corresponding daily intake would be 100 - 200 µg cadmium day^{-1}.

For the general population, food is the major source of cadmium exposure. Available data indicate that, although the daily intake of cadmium in the USA and Europe is in the order of 20 µg, although large variations among individuals exist. In Japan, the average cadmium intake is 40 - 50 µg day^{-1}. A Joint FAO/WHO Expert Committee in 1972 recommended a provisional tolerable weekly intake of 400 - 500 µg of cadmium (FAO/WHO, 1972).

Generally, low concentrations (less than 0.4 mg cadmium/kg) are reported for fish meat. Certain species of molluscs, scallops, and oysters, however, may often have cadmium concentrations exceeding 1 mg cadmium kg^{-1}. In New Zealand oysters, cadmium concentrations ranging up to 8 mg cadmium kg^{-1} wet weight have been found. The brown meat of crabs may contain 1 - 30 mg cadmium kg^{-1} wet weight.

Assuming that an average fish eater consumes 40 g of fish per day (actual fish consumption values are given in Table 1), cadmium in fish will constitute only a small fraction of the total daily intake. Only under the most exceptional circumstances (very high consumption and very high cadmium levels in fish) will the cadmium intake from fish constitute an important part of the total daily intake via food. It is possible to find examples, however, where the high consumption of certain shellfish may considerably increase the total daily intake of cadmium. It has been reported recently that people consuming large numbers of certain New Zealand oysters have a weekly intake of 1400 - 3500 µg cadmium, which is considerably above the provisional tolerable weekly intake.

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III. LEAD

1. Lead in the Marine Environment

1.1 Reference documentation

The major reviews consulted in the preparation of this section were The Biogeochemistry of Lead in the Environment (Nriagu, 1978), Lead in the Marine Environment (Branica & Konrad, 1980), and Lead: Environmental Health Criteria (WHO, 1977). Information on sources of lead contamination in the terrestial environment was obtained from Friedman & Hutchinson (1981). Other papers that were used are listed in the reference section.

1.2 General facts

Lead has the chemical symbol Pb (Latin: plumbum). It is possibly the first metal discovered and worked, and it is the heaviest element in the Periodic Table Group IVb. Its atomic number is 82, and the atomic weight is 207.19 (depending on source). Lead has 2 oxidation states, Pb^{2+} and Pb^{4+}, and Pb^{2+} greatly predominates in the aquatic environment. In clean-fresh water at pH 9, $PbCO_3$ is the main inorganic species (88%); the remainder is $Pb(OH)_2$. At pH 6, $PbCO_3$ (15%), $PbSO_4$ (3%), and $PbCl_2$ (1%) are the prevailing ligands. In sea water, $PbCl_2$ (43%), $PbCO_3$ (42%), and $Pb(OH)_2$ (9%) (Whitfield et al., 1981) are found, but different authorities give varying speciation and ratios.

In aqueous solution, Pb^{2+} is a borderline Type (a) cation. Under appropriate conditions, alkyl-lead compounds can be formed in the environment (Harrison & Laxen, 1978), and this has been demonstrated in the laboratory (Wong, 1975; Chau & Wong, 1980), but these organo-metals may not be stable (Wood, 1980). Various lead sulphides are formed under anaerobic conditions in sediments.

"High lead" can occur as a result of one or more factors. These include Pb-mineralization, low pH from rock characteristics or the presence of organic acids arising from peat or tree cover, chelation, high chloride, bicarbonate, or nitrate content, intrusion of thermally active water, and/or deficiency of alkaline minerals. "Low lead" can result from Pb-deficient mineralization, a pH > 7, pressure of carbonate, agricultural application of lime, temporary uptake by profuse aquatic vegetation, and/or contact with marl, chalk, or other soils or sediments rich in alkaline minerals. Efficient removal of Pb from raw water requires extra processes carefully matched to the water characteristics, since routine purification processes are often ineffective.

1.3 Sources

Rocks containing small amounts of lead are common and widespread. Typical concentrations range from 10 to 20 $\mu g\ g^{-1}$ in many igneous and metamorphic

rocks, from 10 to 70 μg g^{-1} in carbonaceous shales, and about 100 μg g^{-1} or more in some phosphate rocks.

Lead is recovered commercially from a range of locally-occurring ores of which galena (PbS) and, to a lesser extent, cerrusite ($PbCO_3$), anglesite ($PbSO_4$), and others are important. Often, deposits are also rich in zinc and zinc-copper. These mixed ores give us significant amounts of silver, gold, bismuth, antimony, arsenic, cadmium, tin, gallium, indium, germanium, and tellurium. Sources of lead are found in many countries of the world, and some ores (especially galena) are of high purity. The most important lead mining sites (i.e., those producing more than 10^5 tonnes $year^{-1}$) are found in Australia (10% of global production), Bulgaria (3%), Canada (9.6%), China (3.8%), Mexico (4.5%), Peru (5.5%), the USA (16%), the USSR (14.5%), and Yugoslavia (3.5%). In addition, 15% of the mining production is distributed on a minor scale among about 50 other countries. Substantial amounts of lead are recycled (estimated to be about 33% for some countries) and reclaimed from waste recovery processes. Global lead production in 1975 was 3.6 x 10^6 tonnes (all these data from WHO, 1977b and Koeppe, 1981), and this production, together with recycled and recovered sources, meets a world usage of 4.1 x 10^6 tonnes which remains constant. The mining, smelting, and refining processes can give rise to water and air pollution, and further pollution is derived from the manufacture, use, and discarding of goods, alloys, and organo-lead compounds. Lead is released into the environment by the combustion of coal, wood, and other organic matter including city garbage (DOE, 1974).

Synthetic alkyl-lead fuel additives are produced on a large but slowly declining scale, (1973: 378 000 tonnes; 1974: 357 000 tonnes; 1975: 301 000 tonnes). Those additives are widely regarded as a serious source of atmospheric lead pollution. Efforts are being made to progressively reduce and, when possible, eliminate world-wide use of lead as a motor fuel additive. In 1969, the proportion of alkyl-lead to total lead consumed was 20% in the USA, 11% in the United Kingdom, 11% in Italy, and 5% in France, other countries remaining within this range of consumption.

Of much current concern is the use of lead in drinking-water distribution and domestic plumbing. This lead usage contributes to water pollution in rivers, estuaries, and the sea. Lead is a common contaminant of sewage wastes. As far as is known, lead, a potentially toxic and highly available element, is generally not essential to man or living organisms.

Nriagu (1978) estimates that the global atmospheric lead emissions from man-made sources were 438 x 10^3 tonnes $year^{-1}$ for the years 1974-75, in contrast to only 18.55 x 10^3 tonnes $year^{-1}$ from natural mobilization and inputs. Atmospheric discharges from the combustion of lead in petrol (61%), from steel and base metal production (23%), and from the mining and smelting of lead (8%) are the major contributors, with coal combustion and numerous minor activities comprising the remainder.

The main industrial uses of lead are summarized in Table 6. Many of these promote widespread dispersal of lead as do, for example, the emissions from leaded petrol and coal combustion; to this must be added other uses such as lead solders in canning. The recycling of metallic lead from domestic and industrial uses, such as in lead-acid batteries, removes a significant environmental threat.

Table 6. Lead consumption by use in Europe, the USA, and Japan (x 1000 metric tonnes)[a]

Use	1964	1969	1974	1980 (estimated)
Batteries	772	992	1390	1700
Pigments, chemicals	248	289	360	500
Tetraethylleads	254	319	317	200
Alloys	303	307	288	300
Cable sheathing	427	352	322	200
Pipe and sheet	298	266	173	200
Miscellaneous	206	192	258	200
Total	2508	2717	3108	3300

[a] Sources: International Lead-Zinc Study Group, World Bureau of Metal Statistics; Lead Industries Association; US Bureau of Mines; Noranda Sales Corporation. From: Robinson (1978).

In addition to the many uses of lead metal and alloys, there are inumerable applications of inorganic, often highly toxic compounds, from lead acetate to zirconate (Lutz et al., 1970), of which some are biocides. In some countries, due to the health hazard involved, lead pigments are no longer used in interior paints, but remain a component of some exterior paints and protective coatings for metals. Alkyl-lead antiknock derivatives are produced on a massive scale and are a cause of environmental concern; other organoleads used in biocides, detonators, plastics, and catalysts are produced on a minor scale and, biocides excepted, have less access to the environment.

There is a wide range of lead concentrations in the soil (2 - 200 $\mu g\ g^{-1}$) with considerable areal heterogeneity. Some geologically-unusual soils from diverse countries contain up to 30000 $\mu g\ g^{-1}$ (Nriagu,

1978). Usually, lead in the soil is virtually immobile and barely soluble, and lead in drainage waters is readily adsorbed by hydrous metal oxides, clay minerals, and organic-suspended particles.

Lead in rivers is found in the form of poorly soluble species together with many complexes such as organic acids, amino acids (often complexed with cysteine, which is exceptionally stable), and colloidal ones combined with peptides, proteins, and other natural macromolecules. Treated or untreated sewage residues and sewage sludge all possess enhanced levels of diverse organic lead compounds. Soluble lead concentrations vary widely, but typical ranges for the world's major rivers are 1 - 10 µg litre^{-1}, 1 - 55 µg litre^{-1} for upland streams, 0.5 - 180 µg litre^{-1} for rivers and streams in populated areas, with mineralized acid streams having upwards of 1000 µg litre^{-1}, and for run-off water from city centres 100 - 12 000 µg litre^{-1}, of which about 10% may be alkyl leads (Chow, 1978).

The lead concentration in river sediments is < 10 µg g^{-1} for remote Arctic regions, an average of 23 µg g^{-1} for those of the world's major rivers and a range of 50 - 500 µg g^{-1} for large rivers traversing populous areas which rises to 3800 µg g^{-1} for heavily-mineralized streams (Nriagu, 1978). Brackish, estuarine, and inshore waters show less variability in the lead content of sediments; typically, < 10 - 50 µg g^{-1} and up to 850 µg g^{-1} are found in the estuaries of polluted rivers such as the Rhine, or in those with limited water exchange or exposed to the effects of ore mining.

The dumping of sewage sludge may create local hot spots; extreme ranges of 85 - 10 000 µg g^{-1} dry weight have been quoted (Johnson et al., 1974; Nelmes et al., 1974), and more recently 50 - 3000 µg g^{-1} (Förstner & van Lierde, 1979).

The lead content of dredged spoil from harbours, channels, and estuaries has a reported range of 10 - 1450 µg g^{-1}, and other town and industrial wastes dumped at sea may also contribute to the lead content (Stanford et al., 1981).

There is much dispute about the fate of lead in estuaries, about how much lead enters the littoral and pelagic biomasses and how much of it is locked away in sediments; this is important because, as a source of lead, anthropogenic input can be several hundred times greater than natural silt input. An example of lead inputs to an estuary in an urbanized and industrialized area in the United Kingdom is given in Table 7 (Murray et al., 1980).

1.4 Transport, transformation, and bioaccumulation

1.4.1 Transport

Atmospheric transport is a major consideration in lead cycling. Various estimates suggest background levels in air, at points remote from man's

Table 7. Comparison of lead inputs to the Humber Estuary[a]

Rivers	44 kg day^{-1}	7.3%
Sewage discharges	23 kg day^{-1}	3.8%
Industrial discharges	9 kg day^{-1}	1.5%
Sewage sludge dumping	14 kg day^{-1}	2.3%
Industrial waste dumping	0.3 kg day^{-1}	0.05%
Dredged spoil	460 kg day^{-1}	76.3%
Atmospheric input	52 kg day^{-1}	8.6%
Direct coastal discharges	0.7 kg day^{-1}	0.15%
Total	603	100%

[a] From: Murray et al. (1980).

activities, to be of the order of 0.1 - 1.0 ng m^{-3} (Chow et al., 1969; Murozumi et al., 1969; Egorov et al., 1970; Jernigan et al., 1971), 0.6 ng m^{-3} (Patterson, 1965), with 8 ng m^{-3} (Chow et al., 1972) over uninhabited mountain areas of southern California. Duce et al. (1974) report a gradient over the western North Atlantic ocean of 2 ng m^{-3} offshore increasing to 50 ng m^{-3} towards the eastern shore of the USA. Much higher atmospheric concentrations generally affect coastal seas and waters; for example, Cambray et al. (1975) estimate the total deposition of lead in the North Sea to be 8.1 x 10^3tonnes $year^{-1}$. High inputs are also suggested for Mediterranean coastal bays (Fukai, 1980).

On the basis of measurements of stable and radioactive ^{210}Pb isotopes, Schaule & Patterson (1980) estimate that about one-half of the flux of airborne lead to the sea is derived from alkyl-lead antiknock compounds. According to the authors, the natural influx of lead in rivers is much less significant, considereing the size of the north Pacific Ocean, than the natural atmospheric input (Fig. 2).

Estimates of global fluxes into the oceans of the northern hemisphere are continually being revised. Chow (1978), using data from Chow & Patterson (1962), suggests a pre-industrial background input of 1.1 x 10^9 tonnes $year^{-1}$, supplemented in modern times by a 21 x 10^9 tonne $year^{-1}$ input through rain and a 17 x 10^9 tonne $year^{-1}$ river input. It is possible that only 1% of riverborne lead is carried beyond the continent shelf. Settlement of lead associated with biological debris (4 x 10^{10} tonnes $year^{-1}$ with a

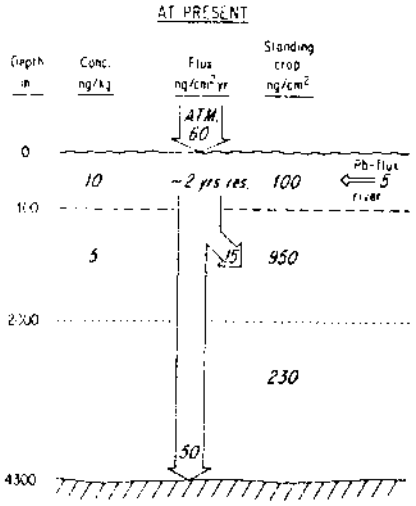

Fig. 2. Oceanic outputs, river and atmospheric inputs, and reservoirs of soluble lead at present times in the open North Pacific (Schaule & Patterson, 1980).

lead content of 1 - 10 μg g^{-1}) is between 4 and 40 x 10^4 g $year^{-1}$; much of this is within shelf areas.

1.4.2 Transformation

Atmospheric alkyl-lead contributions fall from high values near petrol handling sites to 1 - 15% of the total, usually in busy cities. It is generally agreed that alkyl leads are degraded to inorganic forms with "a relatively short period" (Nriagu, 1978) in the atmosphere.

There are indications that the survival time of alkyl-lead compounds in surface waters is only a matter of days or weeks and, therefore, values found in sea water or marine organisms ought to be very low. Published high values are suspect (Bernhard, 1980). Although tetra-alkyl leads are the most toxic of the alkyl lead compounds, they are rapidly converted in water to the less toxic tri-alkyl compounds. Biomethylation of lead has been demonstrated in vitro in marine sediments.

1.4.3 Bioaccumulation

A review of several aspects of lead chemobiokinetics has been given by Branica & Konrad (1980) but, in general, little attention has been paid to lead in comparison to other heavy metals. Lead can be taken up, either in the inorganic form or as organolead compounds, from ambient water, sediments, or through diet. The following paragraphs indicate the paucity of information available on these pathways.

Bioavailability of lead discharged in estuaries is discussed by Rickard & Nriagu (1978). Most lead-containing estuarine particulates liberate a major proportion of lead on treatment with weak organic acid. Such indications may be misleading regarding the availability of lead in soluble form, since the normal range of sea water pH would be about 7.6 - 8.2, but this might have some relevance with regard to lead uptake through the gut of animals.

Few experiments have been carried out on the toxicity of complexed lead to marine organisms, probably because the concentrations of lead required to produce harmful effects are so high that only a small proportion of lead in natural sea water could be in a complexed form. Canterford & Canterford (1980) estimated that the amount of "free" lead required to reduce by 50% the growth of the algae Ditylum brightwellii, in a synthetic sea water medium containing EDTA, was 1.0 - 1.3 µg litre^{-1}; comparison with other algae indicates that either this species is abnormally sensitive or that the remaining EDTA-complexed lead was toxic and, thus, by implication, capable of bioaccumulation.

1.4.3.1 Inorganic lead

Bryan (1976) found that Nereis diversicolor from sediments containing 8 g lead/kg dry weight contained 1 g kg^{-1} lead dry weight in their tissues. Ray et al. (1981) also found that N. virens accumulated lead from sediments containing 243 mg lead kg^{-1} dry weight but not from sediments containing 96 mg kg^{-1}. However, Macoma balthica and Crangon septemspinosa both accumulated lead from the lesser contaminated sediment. It is not clear whether the lead was absorbed from interstitial water or from sediment particles, although it is most probably the latter, since most of the lead could be extracted by EDTA. Luoma & Bryan (1978) found that the uptake of lead from sediments by Scrobicularia was inversely related to the concentration of iron present.

Crustacea

Little is known of the ability of this group of animals to accumulate lead. Weis & Weis (1979) found that the fiddler crab (Uca pugilator) exposed to 100 µg lead litre^{-1} in sea water accumulated 2 mg kg^{-1} in 14 days.

Molluscs

Most attention has been given to this group, especially bivalves, and uptake appears to be linear in all species tested. Mussels (Mytilus edulis) showed a linear uptake during a 40-day exposure, even at the highest concentration tested, i.e., 5 mg lead litre^{-1}. They accumulated 3.7 g kg^{-1} wet weight in 39 days (Schulz-Baldes, 1974). Similarly, Philipps (1976) exposed mussels to 10 and 20 µg lead litre^{-1} for 35 days and found accumulations of 10.7 and 13.7 mg lead kg^{-1} wet weight, respectively. In these experiments, lead uptake was not affected by zinc, copper, or cadmium in the water. Using a very high ambient concentration of 10 mg lead litre^{-1},

Marshall & Talbot (1979) showed that uptake by the gills of mussels plateaued after 50 days, at which time the gills contained 1 mg lead kg^{-1}.

Linear uptake was also demonstrated in the American oyster (Crassostrea virginica) when it was exposed to concentrations between 25 and 200 mg lead $litre^{-1}$ over a period of 10 weeks (Shuster & Pringle, 1969). Accumulation factors ranged from 1160 to 1400, which was similar to that for mussels.

Young abalone (Haliotis sp.) were fed for 6 weeks on brown algae (Egregia laevigata) which had been exposed to 1 mg lead $litre^{-1}$ and which had accumulated up to 21 mg lead kg^{-1} wet weight in their tissues (Stewart & Schulz-Baldes 1976). This was not found to have any effect on their growth or activity. Denton & Burden-Jones (1981) found that the Black-lip oyster (Saccostrea echinata) accumulated more lead at a salinity of 20 °/oo than at one of 36 °/oo, whereas Philipps (1976) found that the lower salinity reduced uptake by the mussel.

In mussels exposed to high ambient concentrations, lead has been found as crystalline extracellular deposits in the capillary walls of the gill (Marshall & Talbot, 1979). These deposits are possibly a mixed or complexed carbonate with calcium. George (1980) reports the presence of lead in membrane-limited vesicles in the gill, and indicates that such granules are excreted by the kidney into the urine. Where half-lives have been measured, these appear to be relatively short. Denton & Burden-Jones (1981) found for Black-lip oysters a half-life of 26 - 34 days which was unaffected by temperature or salinity. Schulz-Baldes (1974) found that mussels lost 33% of accumulated lead with a 40-day exposure to clean water. It is possible that the rate of loss of lead depends, in part, on initial exposure concentrations and on specific detoxification mechanisms.

Echinoderms

Uptake of lead by Lytechinus pictus embryos was linear in solutions containing 100 - 1000 µg lead $litre^{-1}$ (Nash et al., 1981).

Fish

There are few data on the uptake of lead by fish. Somero et al. (1977) exposed an estuarine fish, Gillichthys mirabilis, to lead acetate concentrations of up to 2.6 mg lead $litre^{-1}$. After 100 days, the highest accumulation factors were found in the spleen, gill, and fin tissue, but the factor for muscle tissue was close to unity. Reduction in salinity to 25% sea water doubled the muscle bioaccumulations.

1.4.3.2 Organic lead compounds

These have been little studied in marine organisms. The most comprehensive data have been provided by Maddock & Taylor (1980). The

following accumulation factors were found for 3 species exposed to their 96-h LC_{50} for 4 days (Table 8).

Table 8. Accumulation factors for 3 species exposed to their 96-h LC_{50} for 4 days

	Shrimp (Crangon crangon)	Mussel (Mytilus edulis)	Plaice (Pleuronectes platessa)
Tetramethyl lead	20	170	60
Tetraethyl lead	650	120	130
Trimethyl lead	1	24	1
Triethyl lead	2	10	2

Further experiments with trimethyl lead showed that a plateau concentration occurred in mussels within about 10 days, and depuration was rapid with a half-life of about 3 days. In contrast, depuration of trimethyl and triethyl lead from dabs (Limanda limanda) was very slow, with half-lives in excess of 41 days; with this species, accumulation factors were about 2 and 12 after 41 days' exposure to trimethyl and triethyl lead, respectively.

Food chains

The transfer factor (concentration in consumer/concentration in prey) for trophic chains in the River Loire (France) was found to be < 1 (Amiard et al., 1980; Amiard-Triquet et al., 1980). So lead does not appear to be accumulated in greater concentrations by top aquatic predators.

However, recent concern has been expressed about 2 potential sources of lead poisoning in birds. There is some evidence that mute swans (Cygnus olor) ingest lead shot or anglers' discarded lead weights which is retained and slowly ground in the gizzard (Birkhead, 1982). Dead swans with lead in their gizzards contained median concentrations of 908 mg lead kg^{-1} dry weight in the kidney compared with 8 mg kg^{-1} in dead swans without lead. However, experiments with White Chinese Geese (Johnson & Damron, 1982) fed with lead shot failed to show a lethal effect, and it was proposed that diet may be an important contributing factor.

In the autumn of 1979, over 2000 birds, mainly waders and gulls, were found dead or ill in the Mersey Estuary (United Kingdom); affected birds contained elevated alkyl lead levels; concentration of alkyl lead in the liver of dead dunlin (Calidris alpina) averaged 11 mg kg^{-1} wet weight. Industrial discharges to the estuary include trialkyl lead, and the mollusc Macoma bathica was found to contain about 1 mg lead kg^{-1}, mostly in the form of alkyl lead. It is thought that the contaminated invertebrate food was the cause of the bird's mortality (Bull et al., 1983).

1.5 Lead in sea water, sediments, and marine biota

1.5.1 Sea water

Since it is clearly important to understand how atmospheric inputs of lead (and other trace metals) interact with the surface microlayer of the sea, attention must be drawn to the recent work of Hunter (1980) on samples collected inshore and in the North Sea. With few exceptions, lead was enriched in all microlayer samples compared to sub-surface concentrations, probably as a result of flotation of particles attached to rising bubbles and perhaps assisted by direct influx of atmospheric particles.

According to Burnett & Patterson (1980), the shallow waters of the open (Pacific) ocean contain only 10 ng lead kg^{-1} and deep ocean waters, 1 or 2 ng lead kg^{-1}. The range of values for the North Atlantic Ocean and around the British Isles is large and, 50 - 1200 ng $litre^{-1}$ (Topping et al., 1980).

The generalized depth profile for lead in the northeast Pacific Ocean (Fig. 3(a)) closely resembles those for ^{210}Pb and tritium (Fig. 3(b)). These figures are interpreted by Schaule & Patterson (1980) as demonstrating the contemporary incorporation of airborne lead mainly from lead in petrol simultaneously with ^{210}Pb from continental ^{222}Rn emanations and tritium from nuclear bomb test debris.

Earlier estimates of the residence time for lead in the ocean were of the order of 10^3 years (Chow, 1978). Recently, Schaule & Patterson (1980) have lowered the estimate to only 2 years in the surface layer, and about 200 - 400 years in deep water.

1.5.2 Sediments

From high values in certain estuaries, the lead content of open sea sediments, 8.4 - 60 µg g^{-1} (Nriagu, 1978), falls off away from the coast but retains considerable spatial heterogeneity. Deep-sea sediments have ranges of 13 - 17 µg g^{-1} for oozes, 47 - 61 µg g^{-1} for clays, and 740 - 1250 µg g^{-1} for manganese nodules. Local enhancement is reported for areas influenced by hot brine deposits and volcanic activity.

Several reports confirm the recent enhanced deposition of lead in sediment cores from ponds, lakes, and the marine environment from many areas. One example (Fig. 4) is quoted from Bertine (1980) which considers a variety of depositional areas.

1.5.3 Marine biota

Data on the lead content of marine biota were compiled by Eisler (1983), but as already mentioned in the introduction, most of the data should be considered as too high due to sample contamination. Similar reserves have to be made for the extensive surveys of trace metal concentrations in commercial

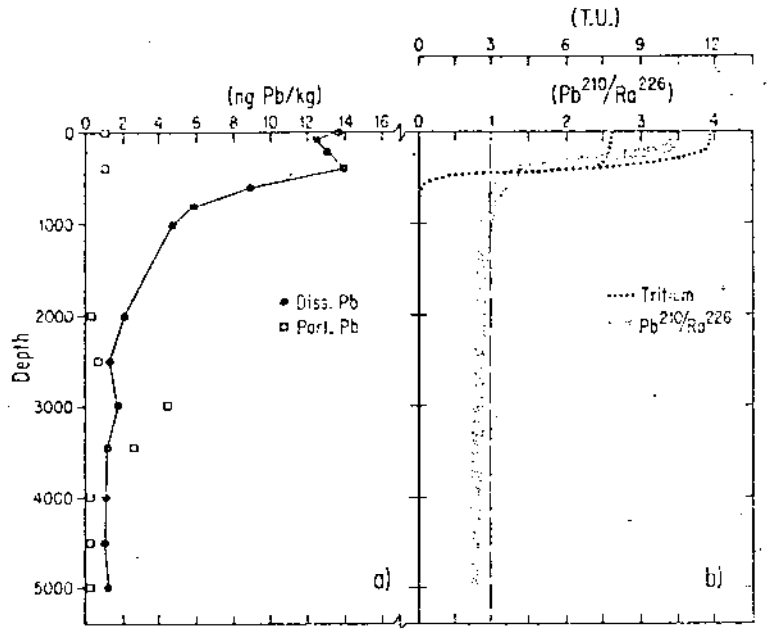

Fig. 3. (a) Generalized depth profile of common lead concentration in the central northeast Pacific Ocean.
(b) General structure of tritium and ^{210}Pb distribution in the central northeast Pacific Ocean.
From: Schaule & Patterson (1980).

fish and shellfish species coordinated by ICES in the northern Atlantic, although the participating laboratories intercalibrated (ICES, 1974, 1977a,b,c, 1980). Most of these concentrations reported are below the detectable limit of 0.01 µg g^{-1} fresh weight, and probably the only conclusion that may be drawn from these data is that the lead levels cannot be higher. The very low values of Patterson & collaborators, which may have been accepted generally as true levels, because they were carried out under ultra-clean conditions (Patterson & Seattle, 1976; Burnett & Patterson, 1980) may serve as a guideline for the concentrations to be expected:

Species	µg/g fresh weight
Valonia, alga	0.2
Abalone, muscle	0.004
Mytillus, muscle	0.025
Spiny lobster, muscle	0.005
Tuna, muscle	0.0003

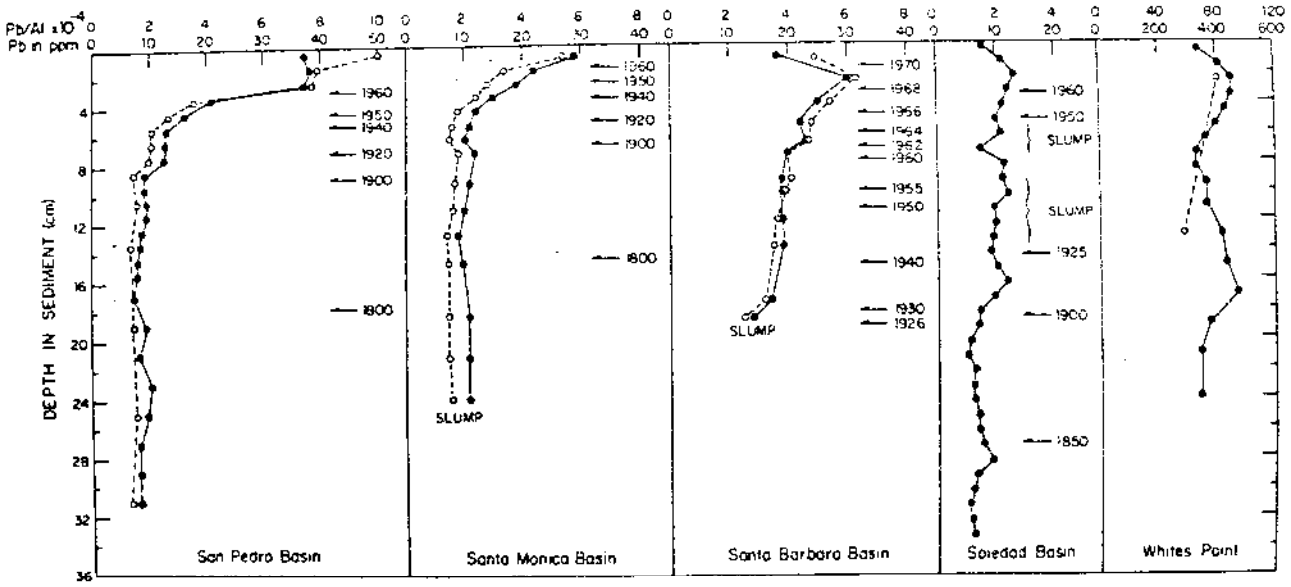

Fig. 4. Lead content (•) and lead/aluminium ratios (o) in Southern California Basin sediments, and Whites Point sewer outfall sediments; note change of scale, Whites Point. Vertical dating of sediments is indicated. From: Bertine (1980).

Davies (1981) and Murray (1979) provide in-depth data from routine surveys of northern and southern North Sea commercial fish and shellfish. The coverage is unequal with regard to the components of the food web for representative sea areas and, for the most part, it is inadequate when defining the contributions made by species, age, sex, feeding habits, maturation, exposure to natural or anthropogenic lead levels, and so on. In particular, the data on lead in phytoplankton and its members, which is critical in defining the role of primary production on the fate of lead in the sea, is almost totally lacking because of uncertainties in sampling and contamination.

Phytoplankton

Using Eisler (1983) data, except where otherwise indicated, the only guideline value for lead in phytoplankton is 40 µg g^{-1} dry weight.

Macrophytes

A selection of macrophytes, including edible seaweeds, exhibit accumulation factors from 1200 to 82 000. Red, green, and brown seaweeds commonly have lead contents of 3.0 - 20 µg g^{-1} dry weight. Lead contents can reach a maximum of 300 µg g^{-1} dry weight depending on species and ambient lead concentrations under either natural or anthropogenic influences. Sea grasses are important food for certain wild fowl, and their typical lead

concentrations (μg g^{-1}) are 5.0 for Spartina, 1.6 - 8.4 for Zostera, and up to 1800 (all dry weight) for Zostera (entire).

Zooplankton

Values given for pelagic zooplankton are: anomalocera 3.5 - 6.0, copepods 3.3, meganyctiphanes 2.2, and euphausids 2.1 μg g^{-1} dry weight. All suggest moderate lead accumulation. Pelagic carnivores such as Aurelia sp. (0.8 μg g^{-1} dry weight), Beröe sp. (6.0 μg g^{-1} ash weight), and Cyanea sp. (6.0 μg g^{-1} ash weight) have low lead contents.

Crustacea

Larger filter feeders such as shrimp (0.2 - 1.2 μg g^{-1} wet weight) and rock shrimp (1.6 μg g^{-1} dry weight) also have low lead contents in their soft parts, with somewhat higher levels in the exoskeleton. The important benthic crustacean, Nephrops norvegica (< 0.1 - 0.5 μg g^{-1} wet weight), Cancer pagurus (< 0.1 - 0.3 μg g^{-1} wet weight), and Homarus vulgaris (0.4 μg g^{-1} wet weight) (Davies, 1981) sampled from commercial fishing areas all have very low lead contents.

Molluscs

Common benthic animals, such as corals with 2 - 42 μg g^{-1} dry weight show response to depth; Alcyonium (24 μg g^{-1} weight), abalone, cardium, chlamys, oysters, littorina, modiolus, and Mya sp. are all well below 5 μg g^{-1} wet weight. Several species, at least, show high values in specific organs, e.g., Chlamys kidney, 830 μg g^{-1} dry weight; Pecten kidney, 137 000 μg g^{-1} dry weight. For Mytilus sp., used in the Mussel Watch Programme (Goldberg et al., 1978), very many lead values are given. There is a reasonably consistent relationship between lead in Mytilus and lead measured or anticipated in ambient water and sediment. Most values, including the main harvested areas, have lead contents not exceeding 1 μg g^{-1} wet weight. In the Eisler (1983) compilation, values up to 450 μg g^{-1} dry weight are reported. Accumulation factors for soft parts range from thousands to tens of thousands. Accumulation is variable in the shell but, in gills and visceral tissues, it is usually considerable. Some squids and octopuses which are also commercially fished have values of < 0.4 μg g^{-1} wet weight.

Fish

The lead contents of all common commercial fish species sampled in the United Kingdom are consistently low and typically lower than the detection limit of approximately 0.1 μg g^{-1} wet weight (Murray, 1979; Davies, 1981). Diverse observations on elasmobranchs and many teleost species give most values at < 0.5 μg g^{-1} wet weight, and extreme values which seldom exceed 1.0 μg g^{-1} wet weight. Exceptions are Ciliata sp. at 8.1 - 25 μg g^{-1} dry weight, Chinocottus sp. at 0.6 - 4.9 μg g^{-1} wet weight, "flounder meal" with total lead at 5.3 μg g^{-1} and tetraalkyl lead at 4.8 μg g^{-1} wet weight, and Platichthys sp. at 14 - 28 μg g^{-1} dry

weight. The lead content of flesh (muscle) is usually lower than that of skin and internal organs, but the disproportion is minor, unlike in molluscs, and the response to environmental lead is small for most edible species (perhaps with the exception of flounder).

Birds

In the kidney and liver of sea birds, lead contents are modest, namely <2.1 and < 5.3 μg g^{-1} wet weight, but information is meagre.

Mammals

Fish-eating mammals (4 spp) show very low values except for the harbour seal (Phoca). It has a lead accumulation in the kidney ranging from 0.08 - 0.60 and in the liver from 0.09 - 5.3 μg g^{-1} wet weight. Higher levels are common in the hard tissues.

2. Effects on Marine Biota

2.1 Reference documentation

A summary of the information on acute and chronic effects of lead on marine organisms has been published by the US EPA (1980), and tabular summaries listing aqueous concentrations and their effects on organisms have been prepared by Taylor (1981) and IRPTC (1981). Individual research papers are listed in the reference section, but the following critical review is based mainly on those studies in which low concentrations have been shown to have an effect.

2.2 Effects on marine biota

2.2.1 Inorganic lead

Algae

Although most of the published data indicate that concentrations greater than 100 μg lead $litre^{-1}$ are required to produce acute or chronic effects on algae, there are a number of papers which report effects at lower levels.

Using a natural population of mixed algal species in enriched sea water, Hollibaugh et al. (1980) found that Chaeterceros sp. were affected by 60 μg lead $litre^{-1}$. Other algae increased in numbers, and a slight growth reduction of Thalassisira aestevalis occurred at 100 μg $litre^{-1}$. Similar results were obtained for Phaeodactylum tricornutum (Woolery & Lewin, 1976) in that 100 μg lead $litre^{-1}$ (as $PbCl_2$) reduced photosynthesis (but not respiration) to 70 - 80% of control values. Using synthetic sea water, Rivkin (1979) found that 4.4 - 7.8 μg lead $litre^{-1}$ caused a 50% reduction in chlorophyll and cell numbers (compared with controls) of Skeletonema costatum. However, minimum growth inhibition was observed for this species in enriched natural sea water at 1000 μg lead $litre^{-1}$ (Berland et al.,

1976). Using EDTA-complexed lead in synthethic sea water, Canterford & Canterford (1980) estimated that only 1.0 - 1.3 µg "free" lead $litre^{-1}$ was required to reduce growth of Ditylum brightwellii to 50% of control values. The significance of these low effect levels in synthetic sea waters is unclear.

Protozoa

The growth rate of Cristigera was slightly reduced by 300 µg lead $litre^{-1}$ (Gray & Ventilla, 1973).

Annelida

This group of organisms appears to be resistant to lead toxicity. Juvenile polychaetes (Neanthes arenacoedentata) exposed to dilutions of lead citrate in sea water had a 28-day LC_{50} of 2.5 mg lead $litre^{-1}$, which compared with 3.2 mg $litre^{-1}$ for adults of the same species (Reish et al., 1976). These authors also found that the 96-h LC_{50} for trochophore larvae of Capitella capitata was 1.2 mg lead $litre^{-1}$, which compared with a 28-day LC_{50} of 1.0 mg $litre^{-1}$ for the adult. Similar experiments by Reish & Carr (1978) on Ctenodrilus serratus showed that the 96-h LC_{50} was > 20 mg lead $litre^{-1}$, and that concentrations > 1.0 mg $litre^{-1}$ reduced population sizes over a 21-day exposure period. The population size of Ophryotrocha diadema (96-h LC_{50} of 11 mg lead $litre^{-1}$) was also reduced by lead concentrations in the range 1 - 5 mg $litre^{-1}$. Brown & Ahsanullah (1971) found that 1.0 mg lead $litre^{-1}$ killed less than 80% of a batch of Ophryotrocha labronica in 25 days, and 10 mg $litre^{-1}$ did not suppress growth within an 8-day exposure period. Therefore, this group of organisms appears to be resistant to lead.

Crustacea

These organisms, too, appear to be resistant to lead toxicity. Embryonic development of the mud crab Rhithropanopeus harrisii to the megalopa stage was delayed from 14.3 days to 15.4 days by 50 µg lead $litre^{-1}$ (Benijts-Claus & Benijts, 1975); the biological significance of such a short delay is unclear. Martin et al. (1981) found that the 48-h LC_{50} for Cancer magister zoea was 575 µg lead $litre^{-1}$. Chaisemartin et al. (1978) found increased production of aspartic amino-transferase in the hepatopancreas of the crab Macropodia rostrata exposed to 102 µg lead $litre^{-1}$. However, Zencirci (1980) found that 100 µg lead $litre^{-1}$ produced 100% mortality of Gammarus locusta in 18 days. The 24-day LC_{50} for Artemia salina was found to be 1.0 mg lead $litre^{-1}$, but this concentration did not reduce growth rates within a 10-day exposure period (Brown & Ahsanullah, 1971).

Mollusca

Juvenile stages of molluscs are usually more sensitive to toxic substances than are adults. However, Calabrese et al. (1973) found that the no-observed-effect level for oyster (Crassostrea virginica) embryos was 500 µg $litre^{-1}$ for a 48-h exposure period. Similar tests with C. gigas and Mytilus edulis

embryos gave 48-h EC_{50}s of 758 and 476 µg lead $litre^{-1}$, respectively (Martin et al., 1981), 760 µg lead $litre^{-1}$ for C. virginica, (unpublished MAFF data), and 780 µg lead $litre^{-1}$ with a no-effect level of 400 µg $litre^{-1}$ for Mercenaria mercenaria (Calabrese & Nelson, 1974). Using Mytilus galloprovincialis, Hrs-Brenko et al. (1977) found that embryo development in lead solutions was affected by an increase in temperature and a reduction in salinity. With a salinity of < 32.5 °/oo, abnormalities increased at > 17.5 °C and 100 µg lead $litre^{-1}$ and at 15 °C with 250 µg lead $litre^{-1}$.

Lethal concentrations reported for juvenile and adult molluscs are in excess of 1 mg lead $litre^{-1}$. Exposure of Macoma balthica to 500 µg lead $litre^{-1}$ for 24 h at a salinity of 6 °/oo, and 6 °C slightly reduced burrowing activity when they were returned to clean water; a concentration of > 5 mg $litre^{-1}$ was required to damage the siphons (Eldon et al., 1980). Data on the long-term accumulation of lead by molluscs, reviewed in a previous section, provides additional evidence of resistance of these species with prolonged exposure.

Echinoderms

Tests with developing sea urchin embryos over a 12-h exposure period indicated lead concentrations of between 1.1 and 2.2 mg lead $litre^{-1}$ (added as lead acetate) at a maximum no-observed-adverse effect on eggs of Anthocidaris crassispina to reach the gastrula stage (Kobayashi, 1971). Retardation of plutei development of Arbacia punctulata was observed at 10 mg lead $litre^{-1}$ (or $PbCl_2$) over a 13.5-h exposure period (Waterman, 1937), and the true exposure concentration was probably lower.

Fish

Few experiments have been carried out with marine fish. In Fundulus heteroclitus, exposure to 1 mg lead $litre^{-1}$ did not retard fin regeneration within a 14-day exposure period (Weis & Weis, 1979). When mullet Mugil auratus were exposed to about 470 µg lead $litre^{-1}$, the blood lead levels rose linearly for 28 days, whereas a decrease in erythrocyte ALA-D and haemoglobin content plateaued within this period (Krajnovic-Ozretic & Ozretic, 1980). After 80 days exposure, blood haemoglobin fell from 8.8 to 5.5 g 100 ml^{-1}, and ALA-D activity decreased by about 50%.

2.2.2 Organic lead

The toxicity of the various alkyl lead compounds are summarized in Table 9.

Although tetra-alkyl leads are the most toxic form of lead, they are rapidly converted to the less toxic tri-alkyl compounds in water. There appears to be agreement between the 2 sets of data. Although the studies on tetra-alkyl lead by Maddock & Taylor (1980) were under continuous flow conditions of test (the remaining ones being static), those of Marchetti (1978) were static, but in closed vessels. Although the toxicity of the

Table. 9. The toxicity of various alkyl-lead compounds

	Maddock and Taylor (1980)				Marchetti (1978)		
	96-h LC_{50} (mg $litre^{-1}$)			6-h EC_{50} (mg $litre^{-1}$)	48-h LC_{50} (mg $litre^{-1}$)		48-h EC_{50} (mg $litre^{-1}$)
	Shrimp	Mussel	Plaice	Alga[a]	Artemia	Morone labrax	Alga[b]
Tetra-methyl lead	0.11	0.27	0.05	1.3	0.25	0.10	1.65
Tetra-ethyl lead	0.02	0.10	0.23	0.1	0.085	0.065	0.15
Tri-methyl PbCl	8.8	0.5	24.6	0.8			
Tri-ethyl PbCl	5.8	1.1	1.7	0.1			
Dimethyl $PbCl_2$			300				
Diethyl $PbCl_2$			75				

[a] Phaeodactylum tricornutum; 50% reduction in photosynthetic activity.
[b] Dunaliella tertiolect; 50% reduction in photosynthetic activity.

tetra-alkyl leads was greater than that of the inorganic forms, the bioconcentration factors were much lower. Tests with oyster larvae (unpublished MAFF data) showed that the 48-h EC_{50} for tri-methyl lead was 0.1 mg lead $litre^{-1}$.

3. Human Health Aspects

3.1 Reference documentation

The following section reviews the human health aspects of lead and evaluates the potential risk of health effects by contribution of lead from the marine environment. Several reviews on health effects of lead have been published (US EPA, 1977; WHO, 1977; Jaworski, 1979; Bornschein et al., 1980; DHSS, 1980; Needleman, 1980; Rutter, 1980; WHO, 1980; Ratcliffe, 1981; Chisolm & O'Hara, 1982; Oskarsson & Camner, 1983; Rutter & Russel-Jones, 1983). Due to the great number of recent scientific reports not included in the reviews, the following section is primarily based on original publications which are listed in the reference section.

3.2 Toxicokinetic properties

The absorption of lead from the gastrointestinal tract varies considerably. Balance studies in human beings have shown that about 10% of the lead in food is absorbed through the gastrointestinal tract (WHO, 1977b). In addition to individual variation, the absorption is highly dependent on the presence of food in the gastrointestinal tract. Flanagan et al. (1982) studied the retention of radioactive lead in 85 fasting subjects and found that approximately 60% of an oral dose of 4 - 400 µg of lead was retained. The effect of minerals on uptake of lead in the gastrointestinal tract was studied by Heard & Chamberlain (1982), who found that addition of calcium and phosphate in doses equivalent to that which a normal meal contains reduced the uptake of lead from 60 to 10%. The chemical species of lead have some influence on the absorption, and water soluble lead compounds are more easily absorbed than those which are less soluble (Chamberlain et al., 1978).

The effect of age on gastrointestinal absorption of lead has been studied in experimental animals. Kostial et al. (1971) demonstrated that 5 - 7 day-old rats absorb about 55% of a single oral dose of radioactive lead. There are very few data on the gastrointestinal absorption of lead in children. Ziegler et al. (1978) performed 89 balance studies with 12 healthy infants ranging in age from 14 days to 2 years. With lead intakes above 5 µg kg^{-1} day^{-1}, they reported a mean absorption of 41.5% with large individual variations. Eleven lead balance studies were performed on 8 healthy children ranging in age from 3 months to 8.5 years (Alexander et al., 1973, 1974). An absorption of 53% was reported, and the mean retention, calculated as intake minus total excretion, was 18% with a range of -4% to 37%. The small number of measurements and the large range of values do not allow any conclusions to be drawn about the relation between gastrointestinal absorption of lead and age in children.

The body burden of lead is divided into 2 fractions: one firmly bound to bone and another loosely bound to blood and soft tissues. The bone fraction constitutes about 90% of the total body burden of lead. The mean retention of lead in blood and soft tissues is about 3 weeks to 1 month and, in bone, about 5 years (Chamberlain et al., 1978; Schütz et al., 1981).

Lead is excreted via urine and faeces. The faecal excretion is mainly unabsorbed lead and could be used as an indicator of the oral intake of lead.

Lead in blood (PbB) is a good indicator of current lead exposure. PbB can be related to the intake of lead, the concentrations in air, diet, or water, and to different health effects due to lead exposure.

PbB levels vary in different parts of the world. A study was performed on PbB levels in the populations of big cities in member countries of the European Community (EEC) (Berlin, 1982). UNEP/WHO initiated a global study in which the PbB levels in teachers, living in big cities in different parts of the world, were measured (Vahter, 1982). The analyses were made with emphasis on quality control in order to obtain accurate and intercomparable results.

Figs. 5 and 6 summarize the results from the 2 studies. The lowest PbB levels were found in Tokyo, Peking, Stockholm, and Baltimore, where the median levels were below 10 µg/100 ml. The US national estimates of PbB levels from 1976 to 1980 have been reported recently (Mahaffey et al., 1982). Blood samples were analysed from a total of 27 800 persons, representing age groups from 6 months to 74 years of age. The results show that 22% of the whole population had PbB levels under 10 µg/100 ml and 1.9% had levels above 30 µg/100 ml.

Among children in Sweden, Finland, and Denmark, the PbB levels are about the same as or a little lower than in adults. The mean PbB levels in children living in urban or rural areas of Finland were 6 - 7 µg/100 ml (Taskinen et al., 1981).

The lowest reported PbB levels are from a population living in a remote mountain area in Nepal (Piomelli et al., 1980). The concentration of lead in air was below the detection limit of 0.004 µg m^{-3}. The mean value of PbB was 3.4 µg/100 ml. A study on PbB levels in children living in rural areas of Papua New Guinea reported a mean value of 5.2 µg/100 ml (Poole et al., 1980).

A study in Sweden showed no significant difference in PbB levels in persons living in city areas with high traffic density compared to persons from low traffic areas (Elinder et al., 1983). Tobacco smoking and alcohol consumption have been demonstrated to increase the PbB levels (Olsen et al., 1981; Shaper et al., 1982; Elinder et al., 1983).

It is of great importance to determine the relationship between dietary lead intake and PbB. Chamberlain et al. (1978), using the same model that had been used for inhalation of lead, predicted an increase in PbB of 3.6 µg/100 ml per 100 µg day^{-1} of lead in the diet. The calculations are based on a 15% gastrointestinal absorption, a 50% distribution into a blood volume of 5.4 litre, and an 18-day half-life of lead in blood. The results from 4 human experimental studies cited by Chamberlain showed a range of 1.4 - 4.3 µg/100 ml per 100 µg day^{-1} of lead in the diet. However, this calculation does not seem to be generally valid for estimating the resulting PbB level from a certain intake of lead. A low daily intake of lead of, for example, 30 µg day^{-1}, as in Sweden, will give too low a PbB value of about 1 µg/100 ml compared to the measured value of approximately 8 µg/100 ml. If it can be assumed that the intake rate has been correctly determined, the discrepancy can be explained by a non-linear relationship between ingested lead and PbB. The value predicted by Chamberlain et al. (1978) is valid for the determination of the increase of PbB after addition of lead to the diet in the high exposure range. Curvilinearity means that, as the dose increases, the rise in PbB becomes progressively smaller. Such a relationship has been demonstrated by Moore et al. (1977) between lead in drinking-water and PbB, and by Hammond et al. (1981) for the relationship between lead in air and PbB. According to the DHSS (1980), using the equation from Moore et al. (1977), a small increase of lead by ingestion increases PbB by about 7 µg/100 ml per 100 µg day^{-1} of ingested lead when starting from a baseline PbB of 18 µg/100 ml. This becomes 2 µg/100 ml per

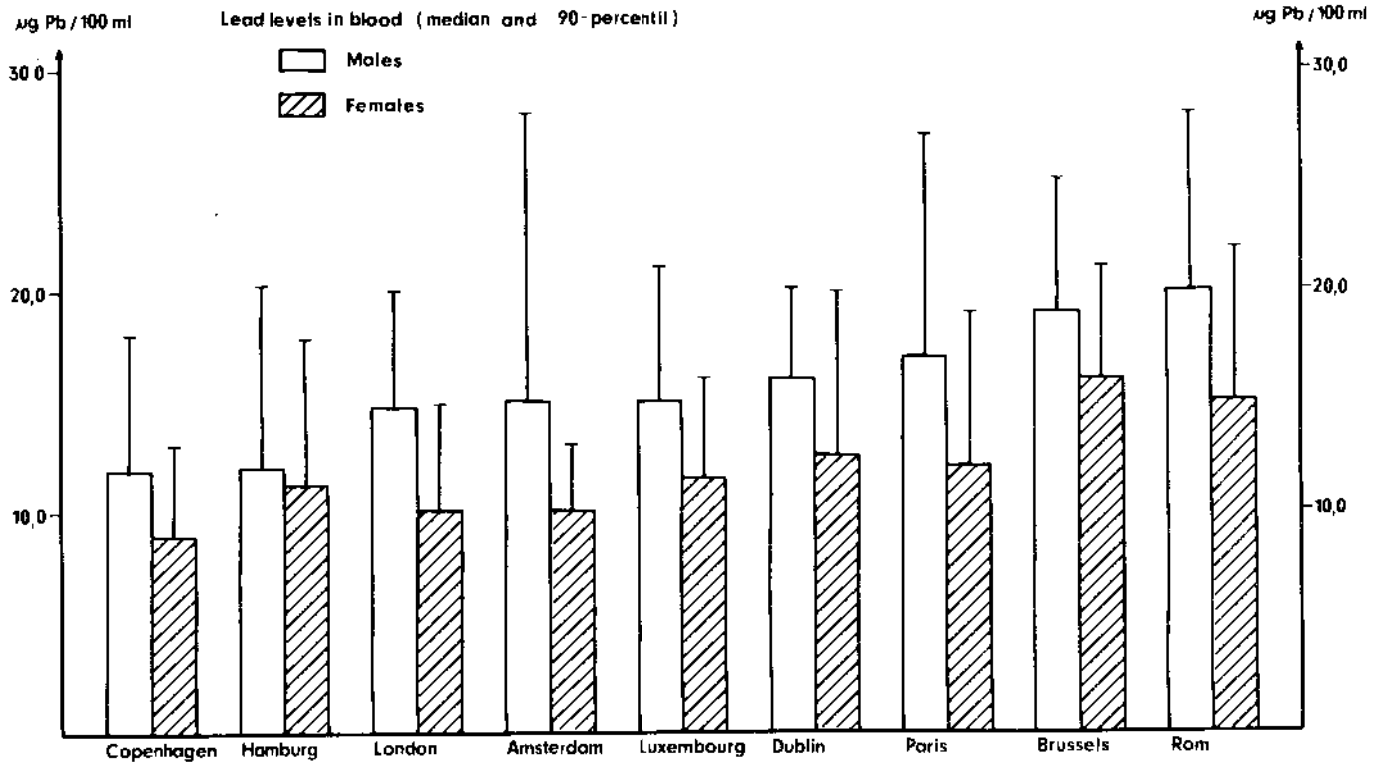

Fig. 5. Lead levels in blood (median and 90-percentile in the population of European cities. From: Berlin (1982).

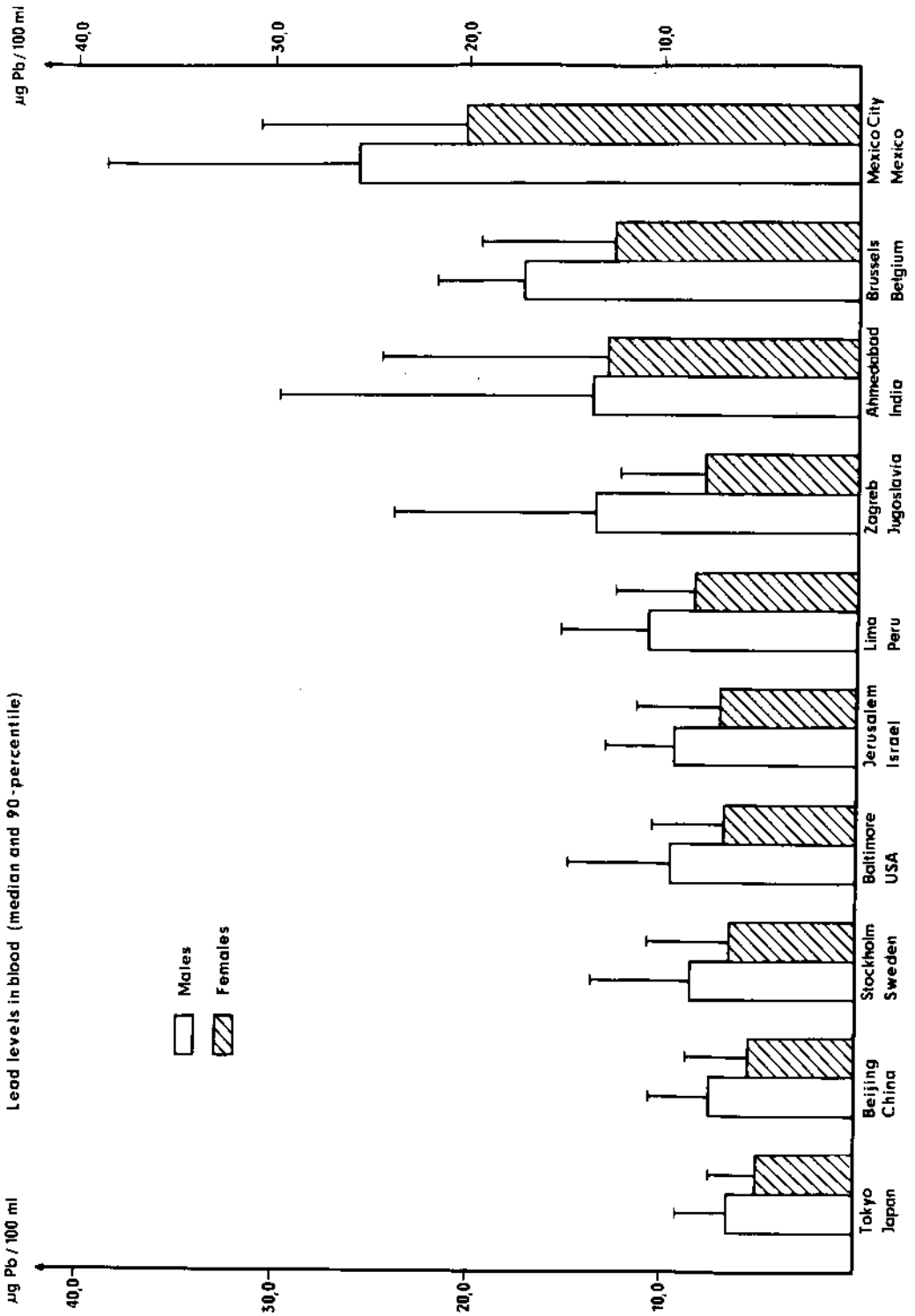

Fig. 6. Lead levels in blood in teachers (Stockholm, a randomly-selected population) (median and 90-percentile). From: Vahter (1982).

100 µg day litre^{-1} when starting from a baseline of 24 µg/100 ml, and it is about 1 µg/100 ml per 100 µg day^{-1} when starting from a baseline of 30 µg/100 ml. The relationship between first draw water which is in the equation and the ingestion rate of lead was, however, not specified.

3.3 Health effects

Early biochemical changes due to elevated lead exposure occur in the haematopoietic tissues where the biosynthesis of haem is disturbed. The haem biosynthesis pathway and the effect of lead in different steps are summarized in Fig. 7. The most sensitive effect is the inhibition of ALA-D, which has been observed at PbB levels above 10 µg/100 ml (Nordman, 1975). The decreased activity of ALA-D results in increased urinary excretion of ALA, and this has been reported at 40 µg/100 ml blood (Zielhuis, 1975). Increased levels of free protoporphyrins in the erythrocytes (FEP) have been associated with PbB levels of about 20 - 30 µg/100 ml in adult females, about 25 - 35 µg/100 ml in adult males, and about 15 µg/100 ml in children (Roels et al., 1975; Zielhuis, 1975; Piomelli et al., 1982). These changes in haem biosynthesis are usually not considered to be adverse health effects as such, but they can be taken as indicators of biological response to elevated lead absorption (US EPA, 1977). At higher PbB levels, there is an effect on the overall haemoglobin synthesis and anaemia will result. Lead-exposed workers developed anaemia at PbB levels of 60 - 80 µg/100 ml (Baker et al., 1979). Children are more sensitive and anaemia has been reported at PbB levels above 40 µg/100 ml (Betts et al., 1973).

Lead exposure may have serious effects on the central and peripheral nervous systems. CNS effects are most frequent in children, and PNS effects occur after long-term exposure in adults. PNS defects range from paresis to slight functional impairment. The major neurophysiological disturbances consist of slowing of the motor conduction velocity (especially of the slower fibres), slowing of the sensory conduction velocity, and electromyographic disturbances (WHO, 1980). Reduced conduction velocities begin to occur in the PbB range 40 - 50 µg/100 ml and become more prominent in the range 50 - 70 µg/100 ml (WHO, 1980).

Lead in high doses causes encephalopathy. The symptoms are ataxia, coma, and convulsions. In children, lead encephalopathy has occurred at PbB levels above 60 µg/100 ml and, in adults, above 80 µg/100 ml (WHO, 1977b). Neurological sequelae can follow in severe or repeated episodes of lead encephalopathy. The sequelae are commonly of a subtle nature involving impaired learning ability, motor incoordination, disturbed sensory perception, and inability to concentrate (WHO, 1977b). Such disturbances are suspected to follow lower-dose lead exposure than those which cause encephalopathy. The major concern today is the sub-clinical effects of lead on the developing CNS. Effects of lead exposure on intelligence and behaviour has been the subject of many epidemiological studies.

Reports in the older literature suggested that occupationally lead-exposed women had a higher frequency of stillbirths and miscarriages than normal (Rom,

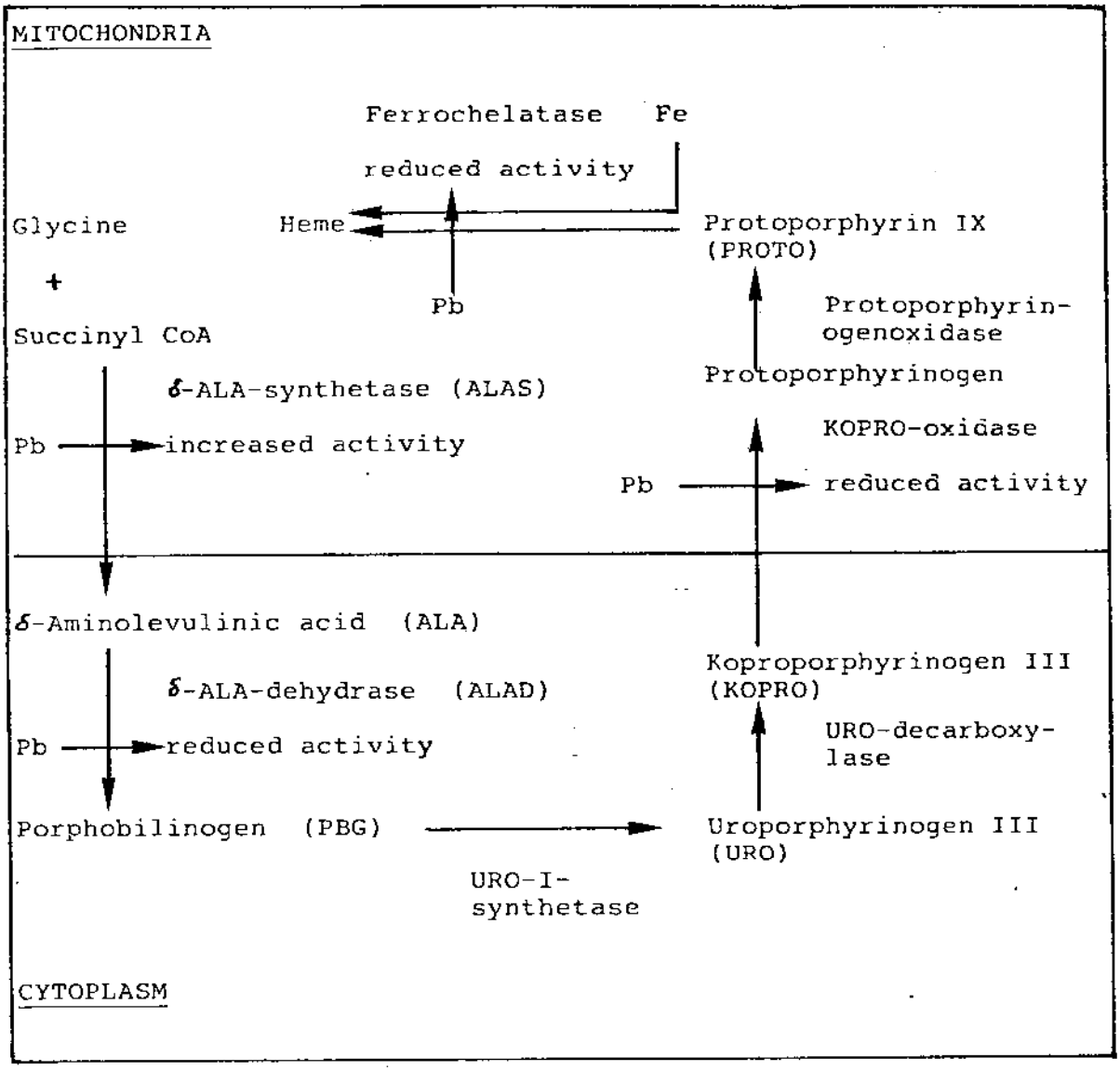

Fig. 7. Haem biosynthesis and the effect of lead.

1976). PbB levels were not reported, but these women were probably exposed to very high levels of lead. From animal experiments, it is known that lead passes the placental barrier and can cause CNS damage to the exposed fetus. What effects lower levels of lead exposure have on reproduction in human beings are not known, but it is suspected that lead exerts toxic effects on the developing CNS of the fetus.

The literature concerning the chromosomal abnormalities in persons exposed to lead is controversial. Most studies have not been able to exclude the possibilities of concurrent exposure to other metals. Beckman et al. (1982) did not find the frequency of chromosomal aberrations in lead workers to be related to the extent of lead exposure. Animal studies have shown that some lead compounds induce renal tumours after peroral administration. In epidemiological studies, it has not been confirmed that lead is also a human carcinogen. Due to the lack of adequate human data, IARC (1980) recommended that, based on experimental animal data, lead acetate, lead subacetate, and lead phosphate be considered as carcinogens.

The major concern in studying health hazards resulting from low-level lead exposure is the possibility that subtle effects on the developing nervous system may result from absorption of lead in smaller quantities than those known to give symptoms. There have been a number of studies conducted on children exposed to lead where intelligence and behaviour have been measured. There are methodological problems with these studies due to imprecise measurements of the intensity and duration of the lead exposure, varying definitions of the nature of the insult, and several confounding variables which are hard to control. Several critical reviews of the literature on this subject have been published lately (Bornschein et al., 1980; DHSS, 1980; Needleman, 1980; Rutter, 1980; Ratcliffe, 1981; Rutter & Russel-Jones, 1983).

The most common indicator of lead exposure is PbB. A single blood sample gives an indication of the present lead exposure but does not show the exposure in earlier childhood. A normal PbB level does not rule out previous chronic lead exposure, and an elevated PbB value may reflect an occasional high exposure. Repeated measures of PbB levels over a long time-span would be preferable. Determination of lead levels in dentine could be a useful measure of past exposure. There is a moderate but not high correlation between dentine and blood lead levels.

A battery of tests for measures of intelligence, general cognitive development and behaviour, as well as some for neurologic and psychomotor functions, have been used. Diverse tests have been used in different studies, and the results are hard to compare.

In all probability, there are disparities between test and control groups other than PbB levels which may affect the performance of the neurological and behavioural experiments. Confounding variables such as age, sex, socio-economic status, and parental IQ should be controlled. There are other variables more difficult to measure which also affect development and behaviour, such as the quality of the care-giving environment (Milar et al., 1980).

Taking all evidence into consideration from recent animal as well as human studies (US EPA, 1977; WHO, 1977; Needleman et al., 1979; Ernhart et al., 1981; Otto et al., 1981; Yule et al., 1981; Rice, 1982; Silbergeld, 1982; Lansdown et al., 1983; Winneke et al., 1983), it appears that severe encephalopathy has occurred at around 60 µg/100 ml in blood. Minor

neuropsychological effects may occur at considerably lower blood lead levels (around 35 µg/100 ml), and there are some indications that effects may occur in sensitive groups already at levels of 15 - 30 µg/100 ml. Scientific consensus on effects at these low levels has, however, not yet been reached.

3.4 Total exposure to lead

Of the 3 routes of exposure, food, water and air, food is considered the major contributor to the total lead exposure (NF, 1982). Lead in food can be derived naturally or as a result of man's activities from direct (e.g., dustfall on crops) or indirect contamination (e.g., dustfall on soil, contaminated water for irrigation, or sewage sludge used as fertiliser). Furthermore, lead can get into food through food processing. Lead-soldered cans for food packaging have been shown to increase the lead content in the food to a great extent (DHSS, 1980; NF, 1982).

The DHSS (1980) estimated the contribution of lead from different sources to the body burden. Based on the mean blood lead and the contribution from air as described in section 3.3.4.2, the percentage of blood lead from food varied between 44% and 91%, the lower percentage calculated from an air lead level of 1 µg m^{-3} and a water lead level of 50 µg/litre (DHSS, 1980). These calculations are, of course, approximate. Schaffner (1981), citing Mahaffey (1981), estimated that 55 - 85% of a person's daily exposure originates from food. Tsuchiya (1979) gave an estimation of 80 - 85%.

3.4.1 Dietary lead intake

Daily dietary intakes of lead in different countries and age groups are shown in Table 10. There are 2 ways of estimating the lead intake from food (DHSS, 1980). One way is to calculate the lead content in foods made up to represent an average national diet (total diet studies), the other is to analyse duplicate portions of food actually consumed (individual diet studies). The mean daily dietary intake of lead calculated from total diets was 113 µg in Great Britain compared to 75 µg when calculated from individual diets (Table 10). There are many factors to consider when estimating the dietary intake of lead. For example, differences in the composition of diet in various parts of the world could account for differing intakes of lead. In some cases, the variations among countries might partly be explained by methodological differences. Very seldom has the analytical quality been controlled and, therefore, reliable comparisons between different studies can hardly be made.

A provisional, tolerable weekly intake of 3.0 mg of lead for adults was recommended by the Joint FAO/WHO Expert Committee on Food Additives (JECFA) in 1972 (FAO/WHO, 1972). This is equivalent to about 430 µg day^{-1}. The recommendation does not apply to children and any increase in the amount of lead derived from drinking-water or inhaled from the atmosphere will reduce the amount tolerated through food intake. What PbB level such an intake corresponds to is unknown and, therefore, it is not possible to relate this intake level to the health effects of lead.

Table 10. Estimated average daily intakes of lead (μg person^{-1} day^{-1})

Country	Age, sex	Lead intake (μg/day)	Reference
Canada	0 - 5 months	33	NF (1982)
Canada	40 - 64 years; male	113	NF (1982)
Canada	40 - 64 years; female	89	NF (1982)
Finland	adults	66	Varo & Koivistoinen (1980)
Italy	adults	400	Tomassi, National Institute of Nutrition, Italy (personal communication)
Sweden	adult; male	27	Slorach et al. (1982)
Sweden	50 - 60 years; male	33	Schütz (1979)
United Kingdom	0 - 4 months	17	DHSS (1980)
United Kingdom	adults[a]	113	DHSS (1980)
United Kingdom	adults[b]	75	DHSS (1980)
USA	0 - 5 months	20 - 46	NF (1982)
USA	2 - 6 years	60 - 70	NF (1982)
USA	teenage; male	79 - 95	Jelinek (1982)

[a] Calculated from total diets.
[b] Calculated from individual diets.

Total dietary lead intake in children is lower than in adults. Lead intakes in μg day^{-1} increase with age, but on body weight basis (μg kg^{-1} day^{-1}) decreases with age. Thus, for one-year-old children, the mean lead intake was 61 μg day^{-1} or 5.4 μg kg^{-1} day^{-1}, while for adults 40 - 64 years old, the mean lead intake was 113 μg day^{-1} (males) or 1.6 μg kg^{-1} day^{-1} (NF, 1982). A factor of great importance in the comparison of dietary lead intake is the proportion of canned foods included in the diet.

Lead-soldered tin cans constitute a major source of lead in foods. Acidic foods, particularly, can dissolve lead from the lead solder in the side seam of the can. The inclusion of canned foods in the studied diets will considerably increase the dietary intake. Schütz (1979), in examining dietary intake of lead in Sweden, found 2 diets containing canned fruits that contributed 157 and 167 μg day^{-1} compared to the average 33 μg day^{-1}. Slorach et al. (1982) estimated dietary intake of lead in Sweden to be 27 μg day^{-1}. Very few foods from lead-soldered cans were included in their study. Jorhem & Slorach (1979) compared lead levels in canned and fresh

fruits and vegetables and found a 6- to 28-fold higher level in the canned products. For children 0 - 6 years old, lead from canned foods contributed 17 - 28% of the mean total dietary lead intake (NF, 1982). In the United Kingdom, lead-soldered cans are estimated to contribute approximately 15% of the total dietary intake of lead (DHSS, 1980).

Table 11 summarizes some of the data on lead levels in foods and compares lead levels in fresh and canned food (Jelinek, 1982; NF, 1982). Canned foods contain markedly higher lead levels than fresh foods. This is most evident in fruits.

Because of the toxicity of lead to the developing nervous system, there has been great concern over the need to reduce the levels of lead in foods for infants. In the USA, lead levels in such foods have been reduced 5 - 10 times since the early 1970s, mostly due to the introduction of steel cans or glass containers instead of welded cans (Jelinek, 1982).

Table 11. Lead content in foods ($\mu g\ g^{-1}$ fresh weight)[a]

Food	Uncanned	Canned
Dairy products and eggs		
Milk	0.02	0.10 - 0.13
Butter	0.07	
Ice cream	0.01	
Cheese	0.05	
Eggs	0.17	
Meat and poultry		
Beef, pork, lamb, veal	0.06	0.24
Poultry	0.12	0.24
Hamburger	0.25	
Beef liver	0.09	
Cereal, nut and sugar products		
Flour, white	0.05	
Bread, white	0.08	
Cereals, breakfast	0.11	
Peanut butter	0.06	
Sugar, refined	0.03	

Table 11 (contd).

Vegetables		
Potatoes	0.05	0.12
Cabbage	0.01 - 0.04	0.08
Lettuce	0.12 - 0.15	0.39
Beans	0.01 - 0.04	0.16 - 0.32
Peas	0.03	0.27
Carrots	0.14	0.13
Onions	0.18	0.32
Tomatoes	0.05 - 0.08	0.30 - 0.37
Cucumbers	0.02	
Fruits		
Citrus (oranges, lemons)	0.01	0.39
Apples	0.02	0.22
Cherries	0.02	0.39
Pears	0.02	0.18 - 0.19
Fish and shellfish		
Salmon	0.39	0.72
Mackerel	0.40	0.99
Tuna		0.45
Cod	0.06	
Flounder	0.10	
Oysters	0.17	
Clams	0.21	

[a] From: NFPA/CMI Report for the US FDA (1980) cited in NF (1982); Jelinek (1982).

Lead levels in Swedish human breast milk have been analysed in a study initiated by WHO (Larsson et al., 1981). The median lead content in 41 samples was 0.002 μg g^{-1} with a range of 0.0005 - 0.009 μg g^{-1}. The calculated daily intake for a 3-month-old infant who consumed only breast milk was 0.27 μg kg^{-1} body-weight.

Lead intake from wine can be an important contribution to the daily intake of heavy wine consumers. Noirfalise & Collinge (1982) analysed the lead content in samples of French and Italian wines and reported concentrations in the range 18 μg^{-1} to 262 μg $litre^{-1}$. The average concentration was 65 μg $litre^{-1}$.

3.4.2 Exposure from water and air

Lead from tap water not only contaminates food during cooking but also contributes to lead intake through drinking. Normally, tap water contains less than 10 μg $litre^{-1}$, but markedly higher concentrations are found where lead piping carries soft water. In Great Britain, 10% of the households have tap water with lead concentrations of more than 50 μg $litre^{-1}$ (DHSS, 1980). Based on an average daily intake of 1.25 litre water day^{-1}, the daily intake of lead from water containing 50 μg $litre^{-1}$ is 62 μg.

Most of the lead in urban air originates from the combustion of lead-containing petrol. Organic lead in the form of tetraethyl- or tetramethyl lead is added to petrol in order to increase the octane number. The added amount of lead is regulated and, in most European countries, the maximum permitted concentration is 0.15 - 0.40 g $litre^{-1}$. Lead from car exhausts can be directly inhaled or deposited, or indirectly ingested by children through intake of soil or dust or through contaminated foods. Chamberlain et al. (1978) have studied the increase in blood lead level in relation to the environmental concentration of lead in air. They concluded that a concentration of lead in air of 1 μg m^{-3} will, through direct inhalation, increase the level of lead in blood by 2 μg/100 ml.

3.5 Contribution of lead from marine food

Relatively little data is available on lead concentrations in marine food. Table 11 gives a few examples of concentrations in fish and shellfish from the USA. Cod and flounder had levels of lead comparable to meat and poultry (about 0.1 μg g^{-1}). Fresh salmon and mackerel had a higher content of lead. From the FAO/WHO Collaborating Centres for Food Contamination Monitoring (FAO/WHO, 1982), levels in fresh fish in the range of 0.04 - 0.3 μg g^{-1} were reported. Fresh crab and lobster from different waters in the United Kingdom and the USA contained median levels ranging from 0.1 to 1.2 μg g^{-1}. The lead levels were slightly higher in brown crab meat than in white. Shrimp was reported to have levels from 0.2 to 0.7 μg g^{-1} (FAO/WHO, 1982).

As for other kinds of food, concentrations in canned marine foods are much higher than in the corresponding fresh foods. Table 11 shows a more than 2-fold increase in lead concentrations in canned as opposed to fresh products.

3.6 Evaluation of potential health effects

The main source of lead intake for most individuals is through diet. A provisional tolerable weekly intake (PTWI) of 3.0 mg of lead in diet for adults was suggested by the Joint FAO/WHO Expert Committee on Food Additives (JECFA) (FAO/WHO, 1972). This intake level is not applicable to children, and any increase in the amount of lead derived from drinking-water or inhaled from the atmosphere will reduce the amount tolerated through food intake. As PbB is the best indicator of total lead exposure and can be related to the health effects of lead, it would be of great importance to be able to relate lead

intake to PbB levels. However, there are no conclusive data for such a relationship. Estimates of dietary lead absorption and retention can be applied; however, there are several modifying and interfering conditions. It is also likely that the relationship between ingested lead and PbB is not linear. The lack of data points to a great need for further studies in order to obtain information on the relationship between ingested lead and PbB.

PbB levels vary in different parts of the world, and mean levels are usually in the range of 10 - 20 µg/100 ml. Ninety percentile values above 25 µg/100 ml have been reported in many countries.

The main target tissues for damage due to lead exposure are the haematopoietic and nervous systems. The major concern about low-level lead exposure is the risk of fetuses and children developing central nervous system dysfunction. These effects include those on behaviour, learning ability, intelligence, and fine motor co-ordination. Several steps in the haem biosynthetic pathway are affected by lead. Reduced activity of ALA-D, which has been observed at PbB levels above 10 µg/100 ml, is considered to be an indicator of elevated exposure rather than an adverse health effect. Increased levels of FEP have been connected with PbB levels of about 20 - 30 µg/100 ml in adult females, about 25 - 35 µg/100 ml in adult males, and about 15 µg/100 ml in children. The dose levels which produce anaemia are not clearly demonstrated. In children, lead encephalopathy has occurred at PbB levels above 60 µg/100 ml. There are considerable uncertainties concerning the neuropsychological effects of low-level lead exposure in children due to methodological difficulties and because so many factors other than lead exposure can affect the intellectual development of children. The available data indicate that neuropsychological effects occur at PbB levels of more than 35 µg/100 ml, but more recent studies suggest harmful effects can occur at lower lead levels.

4. Conclusions on Lead

4.1 Potential harm to living resources

Lead inputs to the marine environment are derived from anthropogenic sources, including mining and the combustion of coal, wood, and other organic matter. The widespread use of alkyl-lead compounds in gasoline contributes to a diffuse atmospheric source which, following deposition or runoff, contributes in addition to the contamination of freshwater discharged to estuaries. It has been estimated that as much as 90% of the atmospheric input to the sea may be of anthropogenic origin. There is evidence from analyses of inshore sediments that lead inputs have increased in recent years. However, levels of lead in surface offshore seawater is low, and recent analyses give concentrations of 10 ng litre^{-1}. Much of the lead input to the inshore waters appears to be in sediments. This lead, to an unknown extent, is likely to be mobilized during dredging and dredged spoil dumping.

Alkyl-lead compounds are readily degraded in sea water and are not likely to be persistent. Although there is some indication that alkyl lead compounds can be formed in the environment by microbial action, this has not been shown to be a very significant factor in lead recycling.

Most of the acute toxicity experiments with marine organisms and inorganic lead indicate that effects are seen only after exposure to high concentrations, although gammarid crustacea were affected at concentrations of 100 µg/litre. Reports on the higher sensitivity of 2 algal species need to be confirmed. Even so, available data do not show that the present levels of lead in the sea constitute a hazard to marine biota. Although there are no reported observations of effects of lead in marine mammals, it cannot be excluded that these may show biochemical effects from lead corresponding to those observed in terrestrial mammals.

Marine organisms, especially molluscs, can accumulate lead from contaminated environments, but the relative importance of water, sediments, and food as pathways for lead contamination is poorly understood. There is some evidence that birds may accumulate organic lead from contaminated molluscs, but the availability of such accumulated lead to other predators is unknown.

4.2 Potential hazards to human health

Dietary intake of lead, through food and drink, is the major contributor to the total body burden of lead in the general population. Other sources of exposure include water and air, and there is a possibility of exposure through lead in paints, dust, soil, and glazed domestic vessels. About 10% of the ingested lead in food is absorbed in the gastrointestinal tract of adults. Higher absorption, about 60%, has been shown under fasting conditions. In infants (up to 2 years of age), an absorption of about 40% has been reported. It is noted, however, that there is not only wide individual variability but several other factors which affect absorption.

Body burden of lead is divided into 2 fractions, the one firmly bound to bone and the other loosely bound to blood and soft tissues. The bone fraction, which constitutes about 90% of the total body burden of lead, has a biological half-time of approximately 5 years. The mean retention time of lead in blood and soft tissues is within the range of 3 weeks to 1 month.

The adult dietary intake of lead varies in different countries from about 30 µg day^{-1} in Sweden to 400 µg day^{-1} in Italy. There is a scarcity of data on the concentration of lead in fish. Average concentrations in the range of 0.1 - 0.4 mg kg^{-1} fresh weight have been reported. In addition, some marine foods are canned for distribution, in which case lead from the cans may increase lead intake approximately 2-fold. It would normally be expected that fish does not greatly contribute to the daily intake of lead. The present PTWI dates as far back as 1972 and was based on a number of uncertain assumptions. The value is therefore considered to require re-evaluation, particularly as concerns pregnant women.

All contributions to environmental lead exposure are, thus, of concern. The neurotoxic effects of lead in human beings and, particularly, the developmental impairments in fetuses and children suggest that control of lead discharges to the environment, together with minimizing human exposure, are required.

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IV. TIN

1. Tin in the Marine Environment

1.1 Reference documentation

Apart from the individual papers referred to in the text and listed at the end, the major reference sources employed for this review were Environmental Health Criteria No. 15, Tin and Organotin Compounds (WHO, 1980), Handbook of Geochemistry (Wedepohl, 1969), and Trace Metal Concentrations in Marine Algae (Eisler, 1983).

1.2 General facts

Tin, with chemical symbol Sn (Latin Stannum), was discovered over 4000 years ago. The element belongs to Group IVb of the Periodic Table along with carbon, silium, germanium, and lead. Its atomic number is 50, and its atomic weight is 118.70. Oxidation states in the environment are Sn^{2+} and Sn^{4+}. Recent studies (Pettine et al., 1981) show the predominant forms of Sn^{2+} in aerobic sea water at pH 8.1 to be $Sn(OH)_2$, 93.8%, $Sn(OH)_3$, 2.4%, and $MeSn(OH)_3$, 3.8%. The final stable form in the sea may be as SnO_2 in bottom deposits. Sn^{2+} ionic forms must first be changed to hydrated oxides and then to Sn^{4+} hydrated oxides with a variable ionic configuration which is carried to the bottom. The oxidation-reduction potential of -0.13 V places redox reactions well within the physiological range. Tin can also be biomethylated in the environment, enhancing its toxicity and retention in biota (Hodge et al., 1979). Although ranking 21st in abundance among the 30 most familiar trace metals, tin is the eighth among trace metals in the human body. Schwartz (1974) has suggested that tin is an essential element for animals (rats), and there is evidence that it may be essential to man (Hamilton, 1979).

From considerations of the amount of input to the sea and its abundance in sea water and marine sediments, the nominal residence times quoted are 10^4 - 10^5 years (Förstner & van Lierde, 1979). More recently, Li et al. (1980) assessed the half-life of tin in the upper 350 m as only 3.5 years.

1.3 Sources

The main sources of tin are placer deposits in or derived from rivers, estuaries, and immediate offshore waters. The global distribution of commercially-useful deposits is shown by centres of production. World production in 1971 was distributed among Malaysia (32.3%), the USSR (12.1%), Bolivia (12.0%), Thailand (9.3%), China (8.7%), Indonesia (8.5%), Australia (4.1%), and others (13.0%) (which include Nigeria, Zaire, Brazil, and numerous minor contributors). Cassiterite (SnO_2) deposits, containing roughly 240 g tin m^{-3}, yield some 80% of the total production of 245 000 tonnes (1979) (Bauer, 1980); the remainder comes from various sulfide ores, some of which contain other metals as well as tin.

The mining, dredging, and beneficiation of tin are large scale operations requiring water, which inevitably causes the dispersal of tin-rich particulate matter in river and estuarine systems and, via them, to the sea. The cassiterite grains are 2.5 times more dense than sand and are separated by washing and sieving. Tin is recovered from ores by smelting techniques in which the tin is vaporized and condensed. As tin is very valuable, thorough recovery and reworking of tin residues and smelter fume is practiced.

In its metallic form, tin has many uses which promote virtually worldwide distribution; only the more massive and convenient products are retained for recycling. The main uses are as tinplate (much of which is unsuitable for re-use), solders and other alloys (bronze, babbit-metal, pewter, type metal, dentists' amalgam, special air frame alloys), for strengthening glass, as a colour base, in catalysts, as a stabilizer for perfumes, and for sundry medical and dental applications (WHO, 1980; Peterson & Girling, 1981).

In recent years, synthetic tin compounds have increased to rank fourth among organometals, and production of organotins is now, perhaps, in the region of 50 000 tonnes $year^{-1}$ (WHO, 1980 predicted value). Organotins are used as non-systemic pesticides, as catalysts, as antioxidants, in antifouling paints, and also to stabilize plastics and synthetic rubbers. Methyltins are considered biodegradable, yet complex synthetic organotins (e.g., triorganotin biocides) are resistant to environmental bacteria. Chemical attack requires either strongly acidic (pH < 1) or alkaline (pH > 13) conditions.

The passage of water through domestic plumbing and waste disposal systems greatly increases the level of tin in sewage. Trade waters from canning, dyeprinting, and laundries contain additional traces of tin.

The levels of tin (possibly including tin compounds) are relatively high in primary treated sewage, e.g., 4 - 6 mg $litre^{-1}$ in the United Kingdom and 110 - 170 µg g^{-1} in dry weight sewage solids (Sterritt & Lester, 1980); 100 - 500 µg g^{-1} in dry weight sewage solids is reported by Furr et al. (1976) for a series of USA samples. These values compare with only 3.8 µg g^{-1} in cow manure.

Assuming a quantity of 150 µg g^{-1} tin in dry weight sewage sludge (Förstner & von Lierde, 1979), the input of tin into the North Sea can be estimated at 1000 tonnes $year^{-1}$. A comparable amount could be assumed for the New York Bight (GESAMP, 1982). Clearly, even these approximate calculations demonstrate levels of local input that are important, although there is still no information about the contribution made by organotins. In the same way, large inputs of tin probably occur in channel and harbour dredgings which possess a high level of organic contamination.

1.4 Transport, transformation, and bioaccumulation

1.4.1 Transport

The rate of mobilization of tin by man (240 x 10^9 g $year^{-1}$) exceeds the rate of mobilization by natural forces 10-fold.

There is evidence that most of the transport of tin from the continents to the oceans is via the atmosphere. In the vicinity of tin smelters and recovery plants, airborne tin occurs at locally high concentrations (e.g., 3.8 - 4.4 µg m^{-3} at 700 m from a Japanese smelter) (WHO, 1980) and some tens of ng m^{-3}, in polluted environments generally. The concentration in the marine air of the Northern Hemisphere is about 1 ng m^{-3}, but it is much less in the Southern Hemisphere (Byrd & Andreae, 1982).

Recent preliminary estimates of fluxes from the main atmospheric sources are given in Table 12 (Byrd & Andreae, 1982). Rates of natural and anthropogenic inputs into the global atmosphere appear to be substantial (12 x 10^9 to 70 x 10^9 g $year^{-1}$), but estimates are speculative due to inadequate data. According to the authors, the high value attributed to waste incineration may be overestimated by up to a factor of 10.

Fluxes to the aquatic environment are just as uncertain; some examples are 0.22 x 10^9 g $year^{-1}$ soluble tin in rivers and 83 x 10*9 g yr*-1 tin attached to riverborne particles. This particulate tin is deposited almost totally in estuaries and inshore waters and contributes little to ocean input.

Freshwater transport of tin from land to sea originates from the weathering of low-level minerals, but the flux is reduced to a very low rate due to the characteristic, strong binding of tin and organotins to soil, sediment, and particulate matter. The average occurrence of tin in typical rocks is 0.5 µg g^{-1} in sandstones, 2 µg g^{-1} in igneous rocks, 3 - 4 µg g^{-1} (range = 0.9 - 51 µg g^{-1}) in coals, and 6 µg g^{-1} in shales. The concentration of tin in normal soils is usually between 1 and 10 µg g^{-1} (Peterson & Girling, 1981).

1.4.2 Transformation

In recent years, many more analyses of soluble tin and organotins in freshwater have been reported. In most of the analyses of filtered waters reported by Byrd & Andreae (1982), mostly for southeastern USA rivers, total organotins average about 5% of total tin. An exception was the relatively high levels of organotin found in the German Rhine river. Braman & Tompkins (1979), however, report about 54% organotin in some rivers and lakes in Florida, USA. The average total tin concentrations from these studies were 6 ng $litre^{-1}$ and 12 ng $litre^{-1}$, but the ranges were wide, 0.02 - 120 ng $litre^{-1}$ (Byrd & Andreae, 1982) and 1.3 - 37 ng $litre^{-1}$ with a single high value of 730 ng $litre^{-1}$ not included in the average (Braman & Tompkins). The single analysis of the polluted river Rhine (tin, 20 ng $litre^{-1}$; organotins, 300 ng $litre^{-1}$) shows a very high contribution from dimethyltin. Similarly, Hodge et al. (1979) (Table 13) report very high levels of tin in Lake Michigan with values of inorganic tin at 490 ng $litre^{-1}$ and 84 ng $litre^{-1}$, and butyltins at 2820 ng $litre^{-1}$ and 120 ng $litre^{-1}$ at 10 and 62 m depths, respectively.

In preliminary studies of the behaviour of soluble tin forms with respect to salinity in the estuary of the Ochlockonee River in Florida, Byrd & Andreae

Table 12. Estimated fluxes of tin into the atmosphere from natural and anthropogenic sources[a]

Source	Global production or consumption (10^{12} g/year)	Emission factor (g/g source)	Tin flux (10^9 g/year)
Anthropogenic input			
Coal burning	3245	0.20×10^{-6}	0.65
Coal burning	3245	0.17×10^{-6}	0.55
Oil burning	1600	0.01×10^{-6}	0.002
Wood and agricultural burning	1320	0.75×10^{-6} [b]	0.17
Wast incineration	540	8.60×10^{-5}	47
Iron and steel production	1220	0.10×10^{-6} [c]	0.12
Non-ferrous metal production	18	3.50×10^{-4} [c]	6.3
Tin production	0.24	0.005[d] (22)	1.2
Organotins	0.008	0.05	0.4
Estimated range			10 - 60
Natural input			
Sea spray	1000	2.80×10^{-10}	0.0003
Soil dust	800	0.15×10^{-5}	1.2
Volcanoes	25	0.96×10^{-5}	0.24
Forest fires	320	0.75×10^{-6}	0.24
Biomethylation			6 (?)
Estimated range			2 - 10
Total flux			12 - 70

[a] From: Byrd & Andreae (1982).
[b] Volatilization efficiency assumed to be 10%.
[c] Emission efficiency assumed to be 10%.
[d] Volatilization efficiency assumed to be 50%.

Table 13. Tin IV and organotin compounds in some environmental waters[a]

Location	Collection date	Sn(IV)	$MeSnCl_3$ - (ng/litre)[b] -	Me_2SnCl_2
San Diego Bay, California (surface water)				
32°41'45"N 117°13'52"W	10/5/78	38 ± 2	8 ± 1	45 ± 2
32°43'05"N 117°13'32"W		6 ± 1	5 ± 1	38 ± 2
32°40'42"N 117°07'26"W[c]		9 ± 1	2 ± 1	15 ± 1
32°42'07"N 117°13'42"W		14 ± 1	2 ± 1	35 ± 1
32°40'57"N 117°13'42"W		13 ± 1	4 ± 1	20 ± 1
San Francisco Bay, California				
36°48'00"N 122°28'30"W (8 M)	10/14/78	2.1 ± 0.3	0	0
36°50'41"N 122°25'00"W (12 M)		3.2 ± 0.3	0	0
California coast, off San Francisco				
37°40'N 122°32'W (12 M)	10/11-12/78	0.8 ± 0.3	0	0
37°40'N 122°34'W (17 M)		0.5 ± 0.3	0	0
37°43'N 122°37'W (12 M)		0.3 ± 0.3	0	0

[a] From: Hodge et al. (1979).
[b] Organotin compounds are assumed to be chlorides for the calculation. 0 = not detected.
[c] 10 ng/litre of a compound with the same retention time as the hydride of Et_2SnCl_2 found in the precipitate of this sample.

(1982) found some scavenging of inorganic tin and some indication that seawards, there were enhanced concentrations of methyltin compounds, especially dimethyltin. These data provide some field evidence for the formation of methyltins in estuaries. They highlight the marked local influence on tin and organotin levels of polluted freshwaters.

One of the earliest reports of biomethylation of tin comes from Huey et al. (1974), who observed the methylation of tin and of monomethyl tin by a Pseudomonas species. Subsequently, mono-, di-, and trimethyl tin compounds have been detected in freshwater, estuarine, and inshore waters (Braman & Tompkins, 1979 (Table 14); Hodge et al. 1979; Byrd & Andreae, 1982; Jackson et al., 1982), and biomethylation by certain bacteria in marine sediments has been repeatedly confirmed. Braman & Tompkins (1979) propose soil as a likely source of methyltins in freshwater.

The formation of inert volatile tetramethyltin from trimethyltin hydroxide is slow and not extensive (Guard et al., 1981), and it proceeds by biotic and abiotic action in marine sediments.

Bacterial demethylation has been demonstrated, and Jackson et al. (1982) suggest that the balance among methyltins in the environment represents a combination of methylation and demethylation activities. Exactly how aerobic and anaerobic conditions modify the formation of methyltins in sediments has not been fully explored.

1.4.3 Bioaccumulation

Very little information is available on the uptake of inorganic tin by marine organisms. Zencirci (1980) exposed the crustacean Gammarus locusta to 0.1 mg litre^{-1} as stannic chloride and found that accumulation occurred on the cuticle and in the gut. However, as the solubility of tin in sea water is about 35 µg litre^{-1}, it is possible that this accumulation could have consisted of precipitated tin. Some information can be derived from data published by Smith & Burton (1972) who found that the southern United Kingdom coastal waters contained 0.02 - 0.04 µg litre^{-1} of total tin. Algae (Phaeophyceae), gastropod, and lamellibranch molluscs from the same area contained 0.10 - 0.65, 0.33 - 0.71, and 0.23 - 0.67 µg tin g^{-1} dry weight, respectively, indicating bioaccumulation factors within the range 2500 - 30 000 on a dry weight basis. Phytoplankton from Southampton Water contained 3.5 µg g^{-1} dry weight, as compared to a concentration in the water of 0.04 µg litre^{-1}. In both cases, the contribution made by organotin compounds was unknown.

Dogan & Haerdi (1980) reported the concentrations of total tin in some plants, plankton, fishes, and sediments from one of the more contaminated Swiss freshwater lakes. By adopting a value of 500 ng litre^{-1} for contaminated lake water (Hodge et al., 1979), the indicated biological accumulation factors for total tin would be in the range 6000 - 60 000 on a dry weight tissue basis. Representative values of the biological accumulation factors for methyltins alone are not yet available; however, the data of Dogan

Table 14. Analysis of saline and estuarine water samples[a,b]

Sample	Tin (IV) (ng litre^{-1})	%	Methyl tin (ng litre^{-1})	%	Dimethyl tin (ng litre^{-1})	%	Trimethyl tin (ng litre^{-1})	%	Total tin (ng litre^{-1})
Saline waters									
Gulf of Mexico, Sarasota	62.0	73	15	18	7.0	8.3	0.98	1.2	85
Gulf of Mexico, Fort Desoto	2.2	60	ND[c]		0.74	20	0.71	20	3.6
Gulf of Mexico, St Petersburg	4.5	54	0.62	7.4	3.2	39	ND	ND	8.3
Old Tampa Bay, Oldsmar	0.3	9.7	0.86	33	0.88	34	0.61	24	2.6
Old Tampa Bay, Safety Harbor	1.4	29	0.86	17	2.0	40	0.65	13	5.0
Old Tampa Bay, Philipee Park	0.8	32	1.1	44	0.60	24	ND	ND	2.5
Old Tampa Bay, Davis Municipal	ND	ND	0.98	35	0.91	32	0.95	34	2.8
Old Tampa Bay, Courtney Campbell	2.7	54	ND	ND	1.7	34	0.61	12	5.0
Average	1.7	40	0.63	15	1.4	33	0.50	12	4.2
Estuarine surface waters									
Sarasota Bay	5.7	47	3.3	27	2.0	16	1.1	9.1	12
Tampa Bay	3.3	27	8.0	66	0.79	6.5	ND	ND	12
McKay Bay	20	88	ND	ND	2.2	9.6	0.45	2.0	23
Hillsborough Bay	ND	ND	ND	ND	1.8	71	0.71	29	2.5
Hillsborough Bay, Seddon Channel; north	12	86	0.74	5.3	0.91	6.6	0.35	2.5	14
Hillsborough Bay, Seddon Channel; south	13	83	ND	ND	2.4	15	0.31	1.9	16
Manatee River	4.8	61	1.4	17	1.1	14	0.65	8.2	7.9
Alafia River	3.4	73	ND	ND	0.75	16	0.55	12	4.7
Palm River[d]	567	98	ND	ND	4.6	0.80	4.0	0.69	576
Bowes' Creek	8.6	42	8.5	42	3.3	16	ND	ND	20
Average	7.9	63	2.4	19	1.7	14	0.46	3.7	12

[a] From: Braman & Tompkins (1979).
[b] Data are averages of duplicates.
[c] ND: less than 0.01 ng litre^{-1} for methyl tin compounds and 0.3 ng litre^{-1} for inorganic tin.
[d] This set of values was not used in computing the average.

& Haerdi (1980) suggest a multiplier of less than 10 between plankton and fish tissue concentrations.

Few data are available for the bioaccumulation of organotin. Sheepshead minnows (Cyprinodon variegatus) exposed to 1.6 µg ^{14}C-labelled tributyl tin oxide litre^{-1} for 58 days did not reach an equilibrium within that period and, at the end, the bioaccumulation factor on a wet weight basis was 2600 for the whole fish (Ward et al., 1981). This was based on ^{14}C measurements, and analysis showed that some breakdown to di-butyl and mono-butyl tin had occurred. There was a 78% loss of organotin after transfer to clean water for 28 days.

Alzieu et al. (1982) also found that organotin compounds were readily taken up and lost by oysters held in tanks containing surfaces painted with anti-fouling compounds. When tissue levels reached 110 µg tin g^{-1} dry weight, a sequence of active gel secretion on the interior surfaces of the shell followed by calcium deposition occurred to give a chambered effect. Environmental evidence for this effect by organotins used in anti-fouling paints is given by Alzieu et al. (1980). Laboratory studies showed that bioaccumulation factors for Crassostrea gigas and Ostrea edulis exposed to 0.15 µg tributyl tin oxide litre^{-1} were 6000 and 1500, respectively, reaching a plateau after 10 days exposure. Only 50% of the accumulated organotin was depurated, within 10 days, in clean water (Waldock et al., 1983).

1.5 Tin and organotin concentrations in seawater, sediments, and marine biota

1.5.1 Sea water

An average concentration of tin in surface waters of the eastern Atlantic Ocean of 10 ng litre^{-1} has been reported by Smith & Burton (1972). More recently, Hodge et al. (1979) have shown a pronounced gradient of inorganic tin from 38 ng litre^{-1} for inshore California water to less than 1 ng litre^{-1} for offshore water.

In the Ochlockonee River estuary, inorganic tin changed from about 1 to 3 ng litre^{-1} at the head of the estuary to about 1 - 4.5 ng litre^{-1} at the mouth with a hint of an overall increase in dimethyltin (Byrd & Andreae, 1982). The authors found near-surface values of inorganic tin between 1 and 3 ng litre^{-1} for 4 stations in the Gulf Stream with a pronounced decrease over the upper few hundred meters. At a depth of about 3000 m, concentrations were negligible but increased to about 0.6 ng litre^{-1} below 4500 m. The likely source of the high near-surface values is atmospheric input; various interpretations of the slightly enhanced values in deep water might include benthic input or the presence of a separate deep water mass. Mono-, di-, and trimethyl tin distributions in the Ochlockonee estuary show consistent increasing concentrations seawards with dimethyltin as the dominant form (Byrd & Andreae, 1982). Values at the seaward limit were 0.3 - 1.2 ng litre^{-1} Me_2Sn^{2+}, 0.4 ng litre^{-1} Me_3Sn^{2+}, and 0.15 ng litre^{-1} $MeSn^{2+}$.

Much higher values were found for San Diego Bay: 6 - 38 ng litre^{-1} inorganic tin, 2 - 8 ng litre^{-1} $MeSnCl_3$ and 15 - 45 ng litre^{-1} Me_2SnCl_2 (Hodge et al., 1979); concentrations in samples farther offshore were below the limit of detection (MeSn, 0.5 ng litre^{-1}; Me_2Sn, 0.5 ng litre^{-1}). Examples of reported concentrations of tin in coastal and estuarine waters of the USA are given in Tables 13 and 14. Concentrations of about 0.15 µg tributyltin litre^{-1} were found in English east coast estuaries utilized by pleasure crafts, with higher concentrations occurring in marinas (Waldock & Thain, 1983).

1.5.2 Sediments

Even after excluding coastal sites of known tin occurrence (prospected or operational), values of total tin in inshore sediments are generally much higher than in deep-sea sediments. Hodge et al. (1979) claim to have demonstrated a well-marked and statistically-significant increased tin depositional rate over the past 50 years in the upper part of sediment cores from Narragansett Bay by a dating technique using ^{210}Pb. Tin concentrations in the upper 0 - 10 cm lie in the range 20 - 14 µg g^{-1} (dry weight). These concentrations are much lower than those found by Dogan & Haerdi (1980) for Lake Leman (55 µg g^{-1} in sandy samples and 90 µg g^{-1} in sediments rich in humus).

Chester (1965) reported the following concentrations of tin in marine sediments: igneous rocks, 2 µg g^{-1}; near-shore sediments, 21 µg g^{-1}; manganese nodules, 300 µg g^{-1}; and deep-sea argillaceous clays (Pacific Ocean), 20 µg g^{-1}.

Smith & Burton (1972) found mean values for tin in ultramafic rocks to be 0.8 µg g^{-1}; in basalts, 1.7 µg g^{-1}; in silicic rocks, 2.5 µg g^{-1}; in red clays, 3.4 µg g^{-1}; in amphibolites, 1.2 µg g^{-1}; and in ferromanganese deposits, 0.2 - 5.8 µg g^{-1}. All of these values were found in estuarine, shelf, and Atlantic sediments.

Krauskopf (1956) suggested the possibility of co-precipitation of tin forms with $CaCO_3$ in deep water accompanied by bio-magnification in calcareous skeletons of unicellular organisms. Turekian (1965) has made a study of the distribution of tin concentration relative to the particle size of $CaCO_3$ ooze from the deep Pacific Ocean. Particles greater than about 50 µm had, on average, less than 10 µg g^{-1} tin. The tin-rich fractions (44 - 67 µg g^{-1}) tend to fall between 0.7 and 10 µm. Braman & Tompkins (1979) report average forms of tin in a collection of sea shells: total Sn, 0.88 ng g^{-1}, MeSn, 0.24 ng g^{-1}, Me_2Sn, 0.051 ng g^{-1}, Me_3Sn, <0.01 ng g^{-1} dry weight. The values for a white coral were: total Sn, 1.00 ng g^{-1}; MeSn, 0.20 ng g^{-1}, Me_2Sn, 0.21 ng g^{-1}; and Me_3Sn, < 0.01 ng g^{-1} dry weight.

1.5.3 Marine biota

Values for tin in the marine biota given in the review by Eisler (1983) are listed in Table 15. Apart from the values quoted for smoked-canned oysters, all the concentrations of total tin in edible material are moderate. The concentrations of tin and organotins in the marine food web have not been studied in detail. Samples from the Caribbean region, including fish, showed up to 8.8 ng g^{-1} organotin, but most of the samples contained only 0.25 - 1.0 ng g^{-1} total tin (Sherman & Carlson, 1980). Hodge et al. (1979) found the average total tin for 3 macroalgae (Pelagophycus, Macrocystis, Eisenia) to be 0.87 µg g^{-1} dry weight. Many more observations are needed to provide a useful framework for an assessment of the roles of tin and organotins in the marine environment.

2. Effects on Marine Biota

2.1 Reference documentation

This section is based on information given in original publications as listed in the reference section. No general reviews were found in the literature.

2.2 Effect on marine biota

2.2.1 Inorganic tin

There are very few data on the toxicity of inorganic tin to marine organisms.

Algae

In a study of microorganisms in Chesapeake Bay, Hallas & Cooney (1981) found that the microbial flora were resistant to tin, but the exposure concentration used, 75 mg tin $litre^{-1}$, was well in excess of the reported solubility of about 35 µg tin $litre^{-1}$, so that the true exposure concentration is in doubt. Saboski (1977) found that exposure of the diatom Nitzschia liebethrutti to 178 µg tin $litre^{-1}$ for 14 days caused frustular abnormalities; the significance of this response is uncertain.

Crustacea

Exposure of Gammarus locusta to 0.1 mg tin $litre^{-1}$ resulted in a 34% mortality in 16 days and 100% in 24 days (Zencirci, 1980).

Fish

No mortalities were observed among dab (Limanda limanda) exposed to a nominal 1 mg tin $litre^{-1}$ for 4 days (G. Mance, personal communication), but precipitation and settlement of tin was observed with 500 µg tin $litre^{-1}$ remaining in suspension and 35 µg $litre^{-1}$ in solution.

Table 15. Reported values for total tin in the marine biota and food preparations (ng g^{-1})[a]

Algae and macrophytes	0.5 - 101.0 dry weight	mainly 1971
Coelenterata		
Beröe	7.0 ash weight	1959
Cyanea	4.0 ash weight	1959
Pleurobrachia	20.0 ash weight	1962
Corals (34 spp)	< 5.0 dry weight	1971
Mollusca		
Clione	20.0 ash weight	1959
Mixed edible tissues	0.3 - 2.0 wet weight	1978
Mytilus	1.3 - 7.1 dry weight	1977
Omnastrephes	3.0 ash weight	1959
Smoked-canned oysters	25 - 30 wet weight	1950
Crustacea		
Calanus	< 1.0 ash weight	1959
Centropages	50 ash weight	1959
Copepods	70 ash weight	1962
Mixed edible tissues	0.6 - 2.0 wet weight	1978
Chaetognatha		
Sagitta	20 ash weight	1959
Sagitta	400 ash weight	1962
Tunicata		
Salpa	8 ash weight	1959
Teleosta		
Fish livers (26 spp)	0.2 - 0.4 wet weight	1978
Fish muscle (35 spp)	0.4 - 0.5 wet weight	1978
Fish muscle (75 spp)	0.5 - 0.6 wet weight	1978
Morone, liver	0.33 wet weight	1979
Morone, muscle	0.30 wet weight	1979

[a] Source: Eisler (1983), which gives original references. The dates are given as a guide to the likely status of methodological development.

These data are of very limited value in assessing the potential effects of elevated tin concentrations on marine organisms. However, precipitated tin in suspension does not appear to be highly toxic, and saturated solutions of tin in sea water may be toxic to crustacea only after prolonged exposure.

2.2.2 Organic tin

A number of organic tin compounds have been used in anti-fouling paint formulations, and these leach directly into sea water. Since the purpose of these paints is to kill sessile organisms which settle on the surface, the release of such substances from areas of high boat density, such as harbours and marinas, is a potential hazard to organisms in the surrounding area. The main compounds used are the trialkyl and triphenyl tins, and these form the basis of the available marine toxicity data.

(a) Alkyl tins

Bacteria

Hallas & Cooney (1981) report that the microbial flora of Chesapeake contained organisms were fairly resistant to dimethyl tin and very resistant to inorganic tin. Tests were made with solutions containing 15 mg tin $litre^{-1}$.

Crustacea

Wright & Roosenberg (1982) exposed Stage 1 zoeal larvae of the crab Uca pugilator to trimethyl tin over a range of salinities and temperatures. Significantly greater mortalities occurred in test solutions containing 20 µg $litre^{-1}$ trimethyl tin as compared to mortality in the control. Using larval stage of the shore crab (Hemigrapsus nudus) and lobster (Homarus americanus), Laughlin & French (1980) obtained the following data:

		Exposure concentration (µg $litre^{-1}$)	LT_{50} (days)
Shore crab	trimethyl tin oxide	50	6.6
	triethyl tin oxide	50	4.8
	tripropyl tin oxide	25	5.1
	tributyl tin oxide	25	6.2
Lobster	tributyl tin oxide	5	about 3
	tributyl tin oxide	1	no significant mortality

Mortalities increased at the time of shedding of the outer (or cuticular) layer. This was after 7 - 10 days of exposure for shore crab and after 3 - 5 days of exposure for lobster, which may explain the apparently greater

sensitivity of the latter. Similar results were obtained with larval Carcinus maenas when it was exposed to tributyl tin oxide, for which the 96-h LC_{50} was 10 µg litre^{-1}. Larval brown shrimp (Crangon crangon) were more sensitive with a 96-h LC_{50} of 1.5 µg litre^{-1}, although adults were 10 times more resistant (Thain, 1983).

Tests on the harpacticoid Nitocra spinipes in brackish waters (salinity 7 °/oo) gave 96-h LC_{50}s of 2 µg litre^{-1} for tributyl tin as both the fluoride and the oxide (Linden et al., 1979).

Molluscs

The correlation between enhanced environmental concentrations of organic tin compounds and shell thickening and chambering in molluscs has been mentioned in section 4.1.4.3. Excessive shell thickening and reduction in growth occurred in juvenile Crassostrea gigas exposed to 0.15 µg tributyl tin oxide litre^{-1} for 8 weeks. This effect was increased when additional suspended matter was present in the water (Waldock & Thain, 1983). Similar environmental concentrations in the vicinity of high pleasure-craft density produced the same effect on relaid oysters (Waldock & Thain, personal communication). His & Robert (1980) found that the eggs of the oyster C. gigas exposed to tributyl tin acetate did not develop at 5 µg litre^{-1}; "D" larvae suffered a 30% mortality, and the survivors showed no growth at this concentration. Thain (1983) found that the 48-h LC_{50} for developing embryos of C. gigas and Ostrea edulis was 1.6 and 2.3 µg tributyl tin oxide litre^{-1}, respectively.

Juvenile clams (Rangia cuneata) exposed to tributyl tin acetate at 20 µg litre^{-1} showed 100% mortality in 11 days, whereas adult clams were not killed by 90 µg litre^{-1} in 17 days (Good et al., 1980).

Fish

Bleak (Alburnus alburnus), when exposed to tributyl tin fluoride in brackish water (salinity 7 °/oo) had a 96-h LC_{50} of 6 - 8 µg litre^{-1}. Its 96-h LC_{50} was 15 µg litre^{-1} when it was exposed to tributyl tin oxide (Linden et al., 1979). Using a mixture of fish species (Gambusia affinis, Peocilia latipinna, and Fundulus grandis) in estuarine water, Good et al. (1980) found that 100% mortality occurred within 11 days of exposure to 20 µg tributyl tin acetate litre^{-1}. Life cycle studies with the sheepshead minnow, Cyprinodon variegatus, showed that 1 µg tributyl tin oxide litre^{-1} had no effects on growth (Ward et al., 1981). However, Thain (1983) found that larval sole (Solea solea), when exposed to this compound, had a 96-h LC_{50} of 2.1 µg litre^{-1}, whereas the corresponding concentrations for adult sole and the armed bullhead (Agonus cataphractus) were 36 and 16 µg litre^{-1}, respectively.

(b) Aryl tins

Algae

Callow et al. (1979) found that the photosynthetic activity of zoospores of Enteromorpha intestinalis was reduced by 50% when it was exposed to triphenyl tin chloride (as tin) at 0.6 µg litre^{-1}, whereas zoospores of Ulothrix flacca were similarly affected by 600 µg litre^{-1}. Subsequently, Callow & Evans (1981) showed that the chlorophyll content of the diatom Achanthes subsessilis decreased during a 5-day exposure to concentrations of triphenyl tin acetate greater than 6 µg litre^{-1} (as tin). Mortalities occurred at 120 µg litre^{-1}. There was some evidence of acclimatization to this organic tin during the exposure period.

Crustacea

Linden et al. (1979) found that the 96-h LC_{50} for the harpacticoid Nitocra spinipes when it was exposed to triphenyl tin fluoride in a brackish water (7 °/oo) was 8 µg litre^{-1}.

Fish

Further experiments by Linden et al. showed that the 96-h LC_{50} for bleak (Alburnus alburnus) when it was exposed to triphenyl tin fluoride was 40 µg litre^{-1}.

3. Human Health Aspects

3.1 Reference documentation

This section is based primarily on original publications listed in the reference section. However, publications reviewing the health effects of inorganic tin and organotin compounds, such as the WHO (1980) Environmental Health Criteria document on the subject, and reviews by Barnes & Stoner (1959), Piscator (1979), and Bennett (1981) have also been used.

3.2 Toxicokinetic properties

The kinetics of tin, including gastrointestinal absorption, depend on its chemical form.

3.2.1 Inorganic tin

Inorganic tin is poorly absorbed from the gastrointestinal tract. Hiles (1974) found that less than 1% of the environmentally-dominant Sn (IV) form was absorbed in the rat after oral administration. In man, no differences can usually be found between oral intake and faecal excretion of tin (Schroeder et al., 1964; Calloway & McMullen, 1966). According to Johnson & Greger (1982), however, the daily supplementation of diet with 50 mg tin increased urinary tin excretion from 29 µg day^{-1} to 122 µg day^{-1}.

All human tissues, with the exception of those of newborn babies, contain appreciable amounts of tin, and the highest concentration is found in the lung (Schroeder et al., 1964; Anspaugh et al., 1971). Hamilton et al. (1972, 1973) found 0.8 mg kg^{-1} tin in rib bones and lungs, 0.4 in liver, 0.2 in kidneys, and 0.005 mg $litre^{-1}$ in blood, mainly in red blood cells. The presence of tin in airborne particles, and a longer-term exposure explains the age-dependent increase in the lung concentration of tin (Schroeder et al., 1964). Bone is the major accumulation site for absorbed tin. Clearance from soft tissues is fast, but from bone it is slow (Furchner & Drake, 1976). After a fast elimination phase, approximately half of the absorbed dose is cleared both in man and experimental animals with a half-time of 400 days (Bennett, 1981).

Absorbed tin is mainly eliminated in urine. Perry & Perry (1959) and Meltzer et al. (1962) found mean values of 16.6 and 18.0 µg $litre^{-1}$ total tin in the urine of people without any occupational exposure to tin.

3.2.2 Organotin compounds

The kinetics of organotin compounds have not been studied in man and only to very limited extent in animals. Some quantitative data on gastrointestinal absorption (Bridges et al., 1967; Piscator, 1979), but mainly the differences between oral and intraperitoneal LD_{50} values (Stoner et al., 1955; Kimbrough, 1976; WHO, 1980), indicate that absorption decreases with the number of carbon atoms in the organic radical. In the case of lower alkyltin compounds, gastrointestinal absorption also decreases with the number of valencies occupied by inorganic anions.

Many of the alkylated tin compounds are dealkylated in vivo (Cremer, 1957; Bridges et al., 1967) and, at least in the case of tributyltin, inorganic tin is the final product (Iwai et al., 1981). As in the case of absorption, excretion, too, depends on the degree of alkylation; an increase in the number of alkyl groups tends to shift excretion from urine to bile (Bridges et al., 1967).

The clearance half-time of trimethyltin is approximately 14 - 20 days in the rat (Brown et al., 1979), but as trimethyltin and triethyltin have exceptionally high affinities for rat haemoglobin (Rose & Aldridge, 1968), this clearance half-time cannot be extrapolated to other species, including man.

The urinary excretion of tin was increased above 100 µg $litre^{-1}$ by toxic occupational exposure to triphenyltin (Manzo et al., 1981). Braman & Tompkins (1979) found that 18% of the tin in urine was in methylated forms, but the mean total urinary tin concentration in the 11 samples was only 1 µg $litre^{-1}$, which is very much lower than the concentration found by other researchers.

3.3 Health effects

There are qualitative and quantitative differences in the toxicities of different tin compounds.

3.3.1 Inorganic tin

The oral toxicity of inorganic tin is low due to low absorption and rapid excretion. Outbreaks of nausea, vomiting, and diarrhoea have allegedly been caused by the consumption of orange-based drinks containing 0.43 g $litre^{-1}$ tin, although in studies with human volunteers, even 0.73 g $litre^{-1}$ tin caused no ill effects (Benoy et al., 1971). It has been suggested that the gastrointestinal effect of tin correlates with the concentration of tin in the drink and not with the total amount of tin ingested (Benoy et al., 1971).

Inorganic tin can interfere with the gastrointestinal absorption of essential elements, including calcium and zinc. This effect was apparent in human volunteers given 50 mg tin per day in their diet (Johnson et al., 1982). Very much higher doses inhibited bone calcification in experimental animals (Yamaguchi et al., 1981) and decreased the compressive strength of the femoral bone (Ogoshi et al., 1981).

3.3.2 Organotin compounds

The intrinsic toxicities of several lower members of the trialkyltin series and triphenyltin are of the same order (Stoner, 1966), but there are great variations between the different compounds in the manifestation of systemic toxicity. Their common localized effect is irritation. Lyle (1958) observed eye and skin irritation and sometimes skin burns in workers handling tributyltin, dibutyltin, and triphenyltin. Similar irritative effects, including nasal irritation, were reported by others (WHO, 1980).

The use of triphenyltin acetate as a fungicide spray resulted in headache, nausea, and vomiting in 2 sprayers (Manzo et al., 1981). One of them had 48 µg $litre^{-1}$ tin in blood and more than 100 µg $litre^{-1}$ tin in urine. Exposure to a mixture of triphenyltin acetate and manganese dithiocarbamate produced similar symptoms in 6 other cases. Three of them also developed liver damage (WHO, 1980). However, as no liver damage was seen in guinea-pigs treated with highly toxic doses of triphenyltin acetate (Stoner, 1966), the hepatotoxicity of this organotin seems questionable.

The main target of trimethyltin and triethyltin is the central nervous system, but within the central nervous system their effects are different. There are 3 reported cases of trimethyltin intoxication. One chemist exposed to trimethyltin developed hyperactivity, insomnia, and absent-mindedness; his recovery was complete (Brown et al., 1979). Two other chemists, who worked in a pilot plant and were exposed to dimethyltin and trimethyltin, suffered from memory defect, loss of vigilance, insomnia, anorexia, and disorientation. These symptoms were ignored until the persons developed mental confusion with general epileptic seizures. Both patients recovered completely after removal

from exposure (Fortemps et al., 1978). In experimental animals, trimethyltin produces symmetrical neuronal damage in selected brain areas, mainly in the hippocampus but also in the neurocortex. Tremors, prostration, hyperactivity, aggression, and convulsions are the main toxic manifestations. In surviving animals, recovery can be complete (Brown et al., 1979). The marmoset, gerbil, and hamster are more sensitive than the rat because their haemoglobin does not bind trimethyltin. Based on experiments on the marmoset, Aldridge et al. (1981) predicted that the toxic dose of trimethyltin to man is 3.0 mg kg^{-1} or less.

The primary effect of human triethyltin intoxication is cerebral oedema (Barnes & Stoner, 1959) and the main clinical manifestations are headache, vomiting, disordered equilibrium, and coma (Alajouanine et al., 1958). Diethyltin capsules contaminated with triethyltin and used against boils and carbuncles caused 102 lethal and 108 non-lethal intoxications (Alajouanine et al., 1958). As the triethyltin content of capsules were not constant, the exact dose could not be calculated (Barnes & Stoner, 1959). Children seemed to be more sensitive than adults (WHO, 1980).

In experimental animals, the main pathological lesion is also cerebral oedema (Magee et al., 1957). The experiments of Stoner et al. (1955) indicate that, in the rabbit, the toxic oral dose is approximately 3.0 µg kg^{-1} triethyltin chloride. For the rat, the minimal toxic triethyltin concentration in drinking-water is 5.0 mg $litre^{-1}$. This concentration produced brain oedema after 2 months exposure (Dieckman & Butler, 1971). Because of its conversion to triethyltin (Cremer, 1958), tetraethyltin produces the same effects in experimental animals as triethyltin (Barnes & Stoner, 1959).

There are no reported cases of systemic intoxication caused by the higher alkyl analogues. On a molar basis, they are less toxic than the lower alkyl homologues and they do not produce hippocampal damage or brain oedema (Mushak et al., 1982). The same is true for dibutyltin. In experimental animals, dibutyltin salts are able to damage the biliary tract, liver, and pancreas (Barnes & Magee, 1958) and to decrease thymus weight. This latter effect is independent of the length of the alkyl radical (Henninghausen et al., 1980).

3.4 Total exposure to tin

The daily intake of tin by man ranges from 200 to 17 000 mg per head. As the contributions of water (30 µg day^{-1}) and atmospheric pollution (less than 1 µg day^{-1}) are small, the major source of tin is food. Fresh food can provide 4.0 mg day^{-1} but, when food is stored in cans or PVC containers, significantly more tin can be ingested (WHO, 1980). Organotin pesticide residues in fresh foods have only a negligible effect on exposure (WHO, 1980).

3.5 Contribution of tin from marine food

There are few data on tin concentrations in fresh marine food, and these are not representative. The flesh of fish or molluscs caught in polluted bays

and coastal areas may contain 1 mg kg^{-1} tin, and even higher concentrations can be present in fish from harbour areas (UNEP, 1981). Since per capita average daily fish consumption in Europe is 38 g day^{-1} (Hamilton and Minski, 1972/73), it seems reasonable to use 40 g day^{-1} fish consumption as a starting point for calculation. Fish consumption from products containing 1 mg kg^{-1} inorganic tin adds 40 µg to the daily tin intake from fish. This contribution is small so that a 10-fold or even 100-fold increase in fish consumption could not result in toxic inorganic tin exposure. The increased consumption of marine food products will decrease the consumption of other types of foods and, consequently, tin intake from other sources. However, tin in fish and in other marine food products can be organic, both from anthropogenic sources and synthesized in marine biota.

In harbours, bays, and off-shore areas, anthropogenic organotin biocides or their metabolites (e.g., monobutyltin from tri- and dibutylin) can be present in appreciable concentration and can enter the bioaccumulation process (Hodge et al., 1979; Seidel et al., 1980; Jackson et al., 1982). A more ubiquitous source of organotin is the gradual methylation of inorganic tin to mono-, di-, tri-, and tetramethyltin (Braman & Tompkins, 1979; Byrd & Andreae, 1982; Jackson et al., 1982). The presence of methylated tin compounds in seashells (Braman & Tompkins, 1979), macro algae, and invertebrates (Seidel et al., 1980) has been demonstrated, and this makes their presence in marine food products probable. At present, lack of data prevents the assessment of any hazard to human health.

When marine food products are used for feeding livestock, the restricted absorption of tin from the gastrointestinal tract limits, as far as inorganic tin is concerned, the transfer of marine tin to meat products.

3.6 Evaluation of potential health effects

It seems very unlikely that inorganic tin in marine food products, directly or indirectly, presents any hazard to human health. However, neither exposures nor health hazard presented by the possible accumulation of organotin compounds, especially trimethyltin and tetramethyltin, can be assessed.

4. Conclusions on Tin

4.1 Potential harm to living resources

World production of tin is about 0.25 million tonnes per annum, and the substance is used widely both as metal and as organic tin compounds. Evidence from tin concentrations in estuarine sediments indicate that the discharge of tin to the marine environment has increased steadily during the past 100 years. Much of the input is derived from diffuse sources, such as domestic and industrial wastes (including those from tin-mining), which may be discharged via rivers or directly, including by the dumping of sewage sludge.

Specific point sources include offshore tin mining of those sediments in which primary tin deposits are present in a potentially soluble form (e.g., stannite and varlamoffite) as occurs in Malaysia.

The use of organic tin compounds in anti-fouling paints for ships may give rise to elevated concentrations in harbours and estuaries where shipping is concentrated.

Most of the data for tin concentrations in seawater refer to total tin and reported concentrations range from 1 to 5 ng litre^{-1} in offshore waters with up to 40 ng litre^{-1} in inshore waters. More recently, some analyses have been made of organotin concentrations, which are important because of the high toxicity of some of the different tin species and compounds.

Soluble inorganic tin entering estuaries may be adsorbed or co-precipitated on particles, and there is some evidence that such tin can become methylated by bacterial action. The extent to which this occurs is not known. Similarly, organotin compounds can be slowly degraded by abiotic and biotic processes but, again, there is little quantitative information on degradation rates.

The data available for this review indicates that inorganic tin and a range of organotin compounds have widely different toxic properties. Few experiments have been carried out with inorganic tin and these have been at concentrations above the solubility limit of 35 µg litre^{-1}. Neither precipitated tin nor saturated solutions have been shown to be acutely toxic to a limited range of marine organisms, although effects on crustaceans have been recorded with chronic exposure. Since this solubility limit is a 1000 times greater than recorded total tin concentrations inshore, it is unlikely that existing levels of contamination are approaching harmful concentrations.

Organotins used in anti-fouling paints are much more toxic than inorganic tin. Although the data on alkyl and aryl tins are not extensive, they are reasonably consistent and show that concentrations greater than 1 µg litre^{-1} are harmful to a range of species. Early life stages of organisms, particularly molluscs and moulting crustacea, may be harmed at lower concentrations.

The use of organotins in antifouling paints is a direct source of hazard to marine organisms in the vicinity of harbours, marinas, and shipping lanes. At present, only commercially-exploited shellfish are known to have been harmed, but it is likely that other species are also at risk. On the evidence available, it is not possible to derive a concentration below which the biota would not be harmed.

Depending on the extent to which methylation of inorganic tin occurs, areas containing tin-contaminated sediments, derived from domestic and industrial inputs, may form a potential source of toxic organotin compounds which could also occur in offshore mining areas.

4.2 Potential hazards to human health

Tin is acquired by many individuals through ingestion of food without occupational exposure. Tin in the food can be in inorganic or organic forms. The gastrointestinal absorption of inorganic tin is low, probably below 5%, and animal experiments indicate that the gastrointestinal absorption of the organotins with longer alkylchains is similarly low. There is only indirect evidence to support the suggestion that gastrointestinal absorption of the lower homologues is higher and increases with increasing alkylation.

In man, the highest concentrations of tin are found in bones and in lungs (0.8 $\mu g\ g^{-1}$), while concentrations in blood are less than one hundredth of this (5 $ng\ ml^{-1}$). Increases in oral intake of inorganic tin is not usually, but may be, associated with corresponding increases in faecal tin excretion and with some increases in urinary excretion.

At present, there is no established relationship between exposure to tin and the blood concentration or excretion rates. Depending on its chemical form, tin exerts different toxic effects. Acute inorganic tin intoxication after oral ingestion is manifested by nausea, vomiting, and diarrhoea caused by the local effect of tin on the gastrointestinal mucosa. A diet supplemented with 50 mg tin per day for 20 days had no adverse effect but did cause an increase in faecal zinc excretion.

The toxicity of organotin compounds shows wide variation. Cases of intoxication have been reported only for triethyltin, trimethyltin, and triphenyltin. The most toxic, trimethyltin and triethyltin, damaged the central nervous system, while triphenyltin caused only general symptoms such as headache, nausea, and vomiting. Children seem to be more sensitive to triethyltin than adults, but in none of the cases of organotin poisonings was dose defined or biological parameters monitored.

The dietary intake of tin shows wide variation depending on the regional distribution of tin in soil, the use of tin in industry, and its use in agriculture. The greatest increase in dietary tin, however, is caused by the release of tin from cans or plastic containers. The dietary intake of tin is increased by the predominant use of tinned food from less than 1 $mg\ day^{-1}$ to well over 100 $mg\ day^{-1}$, but without any noticeable adverse effect.

The contribution of fresh fish or other seafood to the daily intake of tin is low. If one assumes 40 $g\ day^{-1}$ fish consumption from products containing 1 $mg\ kg^{-1}$ inorganic tin, the consumption of tin is only 40 $\mu g\ day^{-1}$, a small proportion of the normal daily tin intake. However, tin in fish and in other marine food products can be organic, both from anthropogenic sources and synthesized by marine biota. The decomposition of anthropogenic organotin compounds in the marine environment, the synthesis of trimethyltin in the marine biota, and the irreversibility of the damage inflicted by doses as low as 3.0 $mg\ kg^{-1}$ trimethyltin on the CNS in experimental animals, make trimethyltin the most important organotin compound in the marine environment in terms of potential toxicity. However, at present it is difficult to

predict whether or not trimethyltin in marine food alone, or in conjunction with other tin compounds, exhibits any hazard to human health. The reason for this is 2-fold: (1) the lack of concentrated data on marine food products; and (2) the lack of quantitative human toxicological data both on toxic doses and clearance half-time.

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ANNEX I: FIRST SESSION OF THE GESAMP WORKING GROUP ON REVIEW OF POTENTIALLY HARMFUL SUBSTANCES: CADMIUM, LEAD, AND TIN (Geneva, 31 January - 4 February, 1983)

Participants

Dr B.G. Bennett, Monitoring and Assessment Research Centre, The Octagon Building, London, United Kingdom

Prof L. Friberg, National Institute of Environmental Medicine, Department of Environment Hygiene of the Karolinska Institute, Stockholm, Sweden (Chairman)

Prof A. Furtado Rahde, Brazilian Drug and Poison Information System, Fundaçnao Oswaldo Cruz, Ministry of Health, Porto Alegre, Brazil

Prof A. Jernelöv, Swedish Environmental Research Institute, Stockholm, Sweden

Mr R. Lloyd, Ministry of Agriculture, Fisheries, and Food, Fisheries Laboratory, Burnham-on-Crouch, Essex, United Kingdom

Dr L. Magos, Medical Research Council, Toxicology Unit, Carshalton, Surrey, United Kingdom

Prof. S.P. Meyers, Department of Food Science, Louisiana State University, Baton Rouge, Louisiana, USA

Dr A. Oskarsson, National Institute of Environmental Medicine, Department of Environmental Hygiene of the Karolinska Institute, Stockholm, Sweden

Dr P.M. Sivalingam, School of Biological Sciences, University of Sciences Malaysia, Minden, Penang, Malaysia

Prof. G. Tomassi, National Institute of Nutrition, Rome, Italy

WHO Secretariat

Dr H. Galal-Gorchev, Environmental Hazards and Food Protection, World Health Organization, Geneva, Switzerland

Dr M. Gilbert, International Register of Potentially Toxic Chemicals, United Nations Environment Programme, Geneva, Switzerland

Dr R. Helmer, WHO Technical Secretary of GESAMP, World Health Organization, Geneva, Switzerland

Dr J. Parizek, International Programme on Chemical Safety, World Health Organization, Geneva, Switzerland

Dr G. Vettorazzi, International Programme on Chemical Safety, World Health Organization, Geneva, Switzerland