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

REPORTS AND STUDIES

No. 28

REVIEW OF POTENTIALLY HARMFUL SUBSTANCES - ARSENIC, MERCURY AND SELENIUM

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WORLD HEALTH ORGANIZATION
Geneva, 1986

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 Oceanograhic 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 REDUCTON OF AMENITIES."

* * *

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The development of this review document was carried out by the GESAMP Working Group on the Review of Potentially Harmful Substances which was organized by WHO in collaboration with FAO and UNEP. Under the chairmanship of Professor L. Friberg draft evaluations were prepared as follows:

Arsenic the marine aspects of arsenic were covered by Dr J.S. Edmonds whereas Dr M. Vahter dealt with the human health aspects

Mercury the first review of mercury was prepared by the Monitoring and Assessment Research Centre of Chelsea College, London. The marine part of mercury was subsequently reviewed by Dr M. Bernhard, and the human health aspects by Dr T. Clarkson

Selenium The evaluation of selenium was based on drafts prepared by Dr A.V. Holden for marine aspects and by Dr O.A. Levander as concerns human health.

Editorial review of the entire document was undertaken by Dr A.V. Holden and Dr L. Magos.

The efforts of all those mentioned above, as well as of those scientists who assisted in reviewing and commenting on the various drafts were most appreciated. GESAMP gratefully acknowledges their participation and dedication so necessary for the review of the three substances covered by this report.

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I. INTRODUCTION

1. BACKGROUND INFORMATION

1.1 Scope and purpose

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

- to up-date 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, icluding 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 Twelfth 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. A draft evaluation document had been prepared on mercury and arsenic for GESAMP XIV and the addition of selenium approved. Following review by GESAMP XV, New York, 25-29 March 1985 the Working Group met again in November 1985 and finalized the combined draft document on arsenic, mercury and selenium. Final approval was subsequently obtained from GESAMP XVI, London, 17-21 March 1986.

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

1.2 Evaluation mechanisms

The collaboration and support of IRPTC was offered at GESAMP XII and data profiles were prepared and made available. 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.

1.3 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 arsenic, mercury and selenium were then critically examined and revised by the Working Group members (Annex I) in a meeting at WHO in Geneva on 4 - 8 November 1985. The Working Group was chaired from 1976 to 1986 consecutively by Messrs B.H. Ketchum, A. Jernelov, 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.

1.4 Quality of data base

1.4.1 Analytical quality control

Many studies have been conducted in 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 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. For mercury in fish the Saskatchewan Department of Fisheries, Winnipeg, has been providing inter-laboratory quality control samples for many years.

Intercalibration has two important aspects: participation increases confidence in the analytical data published and it also improves the analytical techniques used, since very often errors in the analytical procedures can only be detected through participation in intercalibration. Topping (1983) describes the experiences gained during several intercalibration exercises in which the distribution of an acidified metal solution revealed that the analysts used wrong working standards. Adjusting for these differences in standards reduced the range of the mean values submitted. Comparison of the ranges of means submitted by laboratories which had participated in all of the first three exercises showed a decrease of the interlaboratory co-efficient of variation from 35 to 5 %. However, lower levels of mercury in the two samples of the fourth intercalibration resulted in increases in the CV to 33 and 50 %. The International Laboratory of Marine Radioactivity (Monaco) distributed four biological samples in the frame of the MED POL programme. The CV in the different matrices ranged from 1 to 25 % (Fukai et al., 1978; IAEA, 1978; IAEA, 1980).

A collaborative study of methylmercury by eight laboratories using electron-capture gas-liquid chromatography on blind duplicates of oyster, shrimp, tuna and swordfish samples yielded concentrations ranging from 0.15 to 2.48 mgHg/kg, with a precision of 3 to 13 %. The accuracy, by comparison with the reference values (from the Associate Referee Laboratory) ranged from 99 to 120 % (Hight & Caspar, 1983).

Much greater difficulties were encountered when distributing sea water samples for mercury for analysis. Spiked samples showed good comparability withe reference to the standard solutions used but data from the low level unspiked samples (CV about 100 %) indicated systematic errors (Olafson, 1982). Similar experiences were reported from Japan. Intercomparison between 17 Japanese laboratories for the analysis of mercury and cadmium showed a wide scatter of data, with coefficients of variation for some samples as high as 83 % (Sugawara, 1978). The scatter of data was wider in natural water samples than in synthetic solutions indicating difficulties with the natural matrix.

The results from these intercomparisons show that the data from different analysts are not easily comparable and only large differences in the mercury concentrations reported may be significant. The uncertainty increases with decreasing mercury concentration.

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 (nanograms/g). The importance of implementing rigid quality assurance programmes was amplified in two 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.

1.4.2 Ecotoxicological quality aspect

Most toxicity testing with aquatic organisms is undertaken primarily for the purpose of comparing the relative toxicity of chemicals, as required by regulatory authorities before approval in agricultural or health protection practice, or discharge to the environment. For such purposes simple screening tests are usually considered adequate, at least for initial comparison purposes.

The experimental study of the effects of toxic substances on aquatic organisms differs fundamentally from that on terrestrial species in that, in most instances, the environment is dosed rather than the test organism itself. Most routine testing is for short periods of 24, 48 or 96 hours to determine the aqueous concentration lethal to 50% of the individuals (LC_{50}). In such tests assimilation of the toxicant by the fish is via gill membranes, and, for certain substances, the body surface. In only a small proportion of the studies are the fish dissected or blood etc. extracted for autopsies or analysis to allow an assessment to be made of the tissue burden acquired at the time of death or the end of exposure. Consequently the results of most aquatic toxicity tests are not measurements of lethal doses (cf animal studies).

Tests which actually involve feeding of the test organism with food contaminated by known concentrations of the test substance are unusual. However, there is an increasing tendency towards testing in model ecosystems over long

periods (months to years). Tests over extended exposure periods are also regularly conducted on substances which have high toxicity and/or are known to occur at low concentrations in sea water. It should also be noted that a number of tests have been developed which have non-lethal end points, for example changes in enzyme functions, alteration of fish behaviour or responses to stimuli, for example swimming behaviour or gill ventilation rates (such tests usually involve individual rather than group exposure and are more complex to conduct).

The fish used for toxicity testing are usually small (e.g. 5-20 g), ten or twenty to each aquarium or test chamber, and may not be of commercial importance. Small fish are not used solely as a matter of practical convenience, often a deliberate choice is made of small young fish in order to test more positive stages in the life cycle of fish. Larval stages may also be used for the same reason. In addition to fish, some molluscs and crustacea have been used for toxicity testing.

Until recently most fish or invertebrate species used in testing have been of freshwater origin, but in the past 10-15 years estuarine or marine species of small size, or juveniles of large fish species (eg plaice), have been used to establish data on toxicity to marine species. The scale of testing in aquaria has ruled against using large fish (say 500 g), although studies on individuals of such a size (eg rainbow trout) are sometimes made. Marine species of commercial interest such as herring, cod or tuna cannot generally be held in aquaria for periods suitable for toxicity testing, on present knowledge. The individual fish are usually selected for size, sometimes implying a common age, but not for sex (usually impossible to detect in small fish). They are frequently obtained from the natural environment, and with the exception of such species as trout from fish farms, are not specially bred.

The fish or other test species are not fed during short-term testing, although they are usually held in aquaria for an acclimation period of several days before exposure to the toxicant, during which they may be fed and sorted for suitability for use. In long-term testing, when feeding is essential, unless a flow-through system is used, the introduction of food pellets into aquaria raises problems of contamination of the water and alternative pathways for the toxicant.

Especially during long-term tests protection against disease may be necessary, involving prophylactic measures, but otherwise fish are assumed to be in good health. The length/weight ratio of a sample may be the only criteria of fitness. A major problem, especially with tests conducted under static conditions or only infrequent changes of the test solutions, is that the toxicant may be lost to the walls of the test chamber, to the sediment or suspended matter, or in some cases to the atmosphere, resulting in true exposure concentrations well below the nominal concentration and consequent underestimation of toxicity. It is therefore desirable that the aqueous concentration of the toxicant is monitored, to establish the actual concentration to which the test organisms are exposed, although few studies actually include this. Failure to determine the actual concentrations to which fish are exposed is responsible for much incorrect data on toxicity. Dosing aquaria with concentrations greatly in excess of the actual solubility of the toxicant in water can grossly underestimate the toxicity of the substance, much of which may be floating on the surface, on the bottom or adhering to the walls. (It may also be ingested in particulate form). Many, if not all, of these problems can be overcome by using continuous flow exposure systems.

As mentioned earlier, experimental models, using simplified eco-systems, have been devised to study the pathways by which a chemical may pass through or be retained by the environment. Such systems may involve, for example, phytoplankton and a species of zooplankton, a small plankton-eating fish and a carnivorous fish, with perhaps a sand substrate, but the distribution of the chemical between these compartments is likely to differ from the natural environment. In particular, the relationship of surface to volume, determining the proportion of water to surface-living bacteria and algae, and the ratio of sediment surface to water column, may have a considerable influence on the relative rates of uptake or transfer of a chemical between the components of the system.

For certain purposes it may be necessary to establish whether a toxicant has an effect through long-term low-level exposure and to this end effects on growth and reproduction have been measured. For obvious reasons, such tests are usually conducted with species which breed at short intervals, the brine shrimp Artemia salina is one such species.

1.4.3 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 substance 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 concentrations has not been investigated, and the lack of this information hinders the prediction of blood lead concentration from daily dietary intake and vice versa.

2. DIETARY INTAKE CONSIDERATIONS

2.1 Basis for total dietary intake estimates

Accurate estimates of dietary intake of chemical 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 "high-risk" groups, for example, children, pregnant women, high consumers (above a certain percentile). etc. In radi-

ation protection the concept of "critical group" is often used (ICRP, 1977). Such a group should be "representative of those individuals in the population expected to receive the highest dose equivalent" and it should be "small enough to be relatively homogenous with respect to age, diet and those aspects of behaviour which affect the dose received". 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.

An alternative approach for estimating dietary intakes of chemical food contaminants is to use biological monitoring. This is possible when a good indicator material is available, e.g. mercury in hair is a good indicator of daily intake of methylmercury via fish in people with no other major exposure to mercury compounds. Biological monitoring has the advantage of integrating the total exposure from all food sources. Exposures from other sources (water, air, workplace) are also integrated and need to be considered if biological monitoring is aimed specifically at food contaminants. In addition, biological monitoring can give more accurate individual intake estimates, which makes it possible to estimate the intake distribution in the population and the intakes of defined extreme groups according to percentiles.

Comparisons between calculated dietary intake and tolerable intake limits may indicate a risk of health effects for the exposed population. 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.

2.2 Seafood consumption patterns

2.2.1 Overall intake and individual variability of consumption

The importance of seafood as a source of toxic element in the human diet is dependent on a number of factors. These include the quantity of seafood consumed, whether for short or long periods, the particular species involved, the concentration of the element in the organs or tissues consumed, the form or forms in which the element exists in the tissue, the presence of possible protective substances, or of substances which might enhance the toxicity, and the intake of the element in question, or of other substances influencing toxicity, derived from other sources (whether food or the environment). A diet restricted to one type of seafood may present a particular risk if the concentration of the toxic element is high.

The level of consumption of seafood varies widely both between and within countries. Island nations or coastal communities may be particularly dependent on fish, shellfish or marine mammalian tissue (from seals or whales). Estimates of the daily or weekly average intake of fish have been made in a number of countries, but by various methods, from specific diet studies involving cooked meals to the averaging of the national sales of fish among the total population. Some data include both fish consumers and non-consumers (the latter possibly representing a large proportion of the population), other data are confined to fish consumers only. In some studies the form of seafood is defined (fish, shellfish, crustacea, seal meat) and in a very few instances the species are identified.

The average consumption data rarely indicate the highest consumption rates, but two studies have estimated the likely intake of the small section of a population with the highest consumption rate. In the United States, a study of almost 38,000 individuals showed that an extreme consumption of a particular food (the 95th percentile of consumption) was roughly three times the mean consumption. This applied for a variety of foods, the value for fish and shellfish being 2.7 and for canned tuna 2.3. A more limited study in the United Kingdom, involving only consumers of particular food, indicated that the 90th percentile of the number of consumers ate approximately 1.75 times as much fish and 2.33 times as much shellfish as the average. (The 95th percentiles averaged 2.17 and 2.70, and the 99th percentiles 3.60 and 4.55 respectively) (WHO, 1985).

In an Australian survey of nearly 20,000 fish consumers (Australian Working Group, 1980), the 90th percentile represented those consumers eating at least twice the average. 0.9 % of the population sample ate more than 1,000 g/week (140 g/day). (Only 4 of 284 extreme consumers ate more than 2,000 g/week).

The estimated daily average consumption rates for seafood (including both finfish and shellfish) from a number of countries are given in Table 1. They are derived from FAO statistics of fish (FAO, 1984) based on the assumption that 50 % of the per capita supply to national populations is consumed by the average citizen (fish consumer and non-fish consumer). Some published data from special consumer studies tend to give higher values, but in most instances the FAO data are reasonably representative averages. Many of the values (largely from Western countries) are of the order of 20 g/day (or about one seafood meal per week), but for several countries 60 g/day is more representative. In addition, there are a few references in the literature of unusually high consumption levels by particular individuals or groups. Bernhard & Andreae (1984) refer to Italian fishermen at sea consuming about 800 g/day, Canadian Indians reaching 1,300 g/day during the fishing season, and 800-1,500 g/day by Japanese fishermen and their families. Doi & Ui (1975) reported one Japanese fisherman eating 1,200 g/day. These high rates can be represented by an average intake of 1,000 g/day.

Any risks which may be incurred from the ingestion of a particular substance by consumers of seafood are likely to be confined to the upper percentile of the population which has a consumption rate several times the average. Such a fraction is likely to be represented by a relatively large number of people. For example, in the United Kingdom only about one third of the population are regular consumers of fish products. If the total quantity of fish consumed is assumed to be eaten solely by this group, the average daily intake (calculated to be 20 g/day for the entire population) will be 60 g/day. The 97.5th percentile of these consumers eat at least three times the average, or 180 g/day. In the United Kingdom this group represents about 450,000 people. It is not possible to extrapolate accurately beyond the 99th percentile (with four times the average intake) but it is not inconceivable that a small fraction of the 99th percentile may eat up to ten times the average, perhaps as weight watchers, or devotees of fish.

The tables given in the reviews of the elements arsenics, mercury and selenium (chapters II to IV) indicating daily intake at various levels of seafood consumption show such intakes for 20 g seafood per day (about one meal per week); 60 g/day (three meals per week), 150 g/day (one meal per day) and for the extreme consumer 1,000 g/day.

Table 1. Fish consumption in various countries

Estimated Mean Value (g fish weight per capita per day) *)

COUNTRY	MEAN DAILY INTAKE	COUNTRY	MEAN DAILY INTAKE
Algeria	3	Lebanon	8
Argentina	8	Liberia	22
Australia	19	Libya	11
Bahamas	17	Madagascar	7
Bahrain	28	Malaysia	61
Belgium	26	Maldives	93
Bulgaria	7	Martinique	64
Burma	19	Mexico	14
Canada	29	Morocco	8
Chile	38	Mozambique	4
China	6	Netherlands	13
Cuba	23	New Zealand	14
Cyprus	12	Nigeria	22
Czechoslovakia	11	Norway	67
Denmark	62	Panama	10
Egypt	7	Papua New Guinea	12
Faeroes	136	Peru	40
Fiji	58	Philippines	42
Finland	40	Poland	23
France	33	Portugal	38
Gambia	32	Roumania	8
German DR	18	Senegal	34
Germany FR	13	Seychelles	97
Ghana	26	Singapore	43
Greece	22	Solomon Islands	67
Hong Kong	66	Spain	43
Hungary	5	Sri Lanka	19
Iceland	122	St. Lucia	41
India	4	Sweden	42
Indonesia	16	Thailand	26
Ireland	21	Tonga	48
Italy	17	UA Emirates	34
Jamaica	24	United Kingdom	23
Japan	114	USA	22
Korea (DPR)	53	USSR	35
Korea (Republic of)	57	Vanuatu	46
Kuwait	13	Venezuela	16

*) based on the assumption that consumption equals 50 % of supply (FAO, 1984a)

2.2.2 Review of selected data on high-consumption populations

The seafood consumption rates in selected countries show that small island populations can consume relatively large amounts of seafoods (Table 1). According to FAO food balance sheets (FAO, 1984b) Iceland's 231,000 inhabitants consume 1,022 g (fresh weight) edible seafood per week or 6.8 meals of 150 g each i.e. about one meal of seafood per day. 93 percent were marine fish and the rest marine crustaceans. Also, the people (41,000 inhabitants) of the Faeroe Islands consume quite large amounts: on average 950 g FW edible seafood per week. This corresponds to 6.3 meals per week or about one meal per day of seafood. The Japanese average is the highest among larger populations with 850 g/week (fresh weight) (5.6 meals per week). By comparison, the average consumption of marine foods in the USSR and the USA is small. The different food habits are also illustrated in the relative consumption rates of fish versus crustaceans and molluscs.

However, the average consumption rates even in populations with high average consumption rates are usually much lower than the consumptions of certain "critical groups". Fishermen at sea, especially in less affluent regions, will satisfy their protein requirement exclusively with seafoods. Estimates range from 800 g/day on fishing vessels in southern Italy (Bernhard & Renzoni 1977), to 800 to 1,500 g/day for Canadian Indians, and fishermen and their families at Minamata (review: Piotrowski & Inskip, 1981). Unfortunately, few data are based on surveys actually carried out.

Turner et al. (1980) studied two fishing villages and one inland village in northern Peru. The average consumption rate of this critical population was 1,600 g seafood per week, while the residents of the inland village consumed on the average only 310 g seafood per week. Since these data are reported per family (average size 6.2 persons) most likely containing several children of different ages the food intake by adults must be greater and that of the children smaller than the average. Turner et al. (1974) surveyed 88 Korean fishermen based at American Samoa and 45 Samoan factory workers. The fishermen were aboard 47 weeks per year and lived mainly on fish and rice. The fishermen consumed on the average 294 g fish per day. The male factory workers consumed 200 g and the female 105 g of fish per day. The fish consumed included tuna and swordfish. Suzuki et al. (1976) estimated from the available catch (i.e. the supply) that populations living on small Japanese islands consumed on average between 104 g to 395 g per season daily or between 2 and 11 meals per week.

Paccagnella et al. (1973) studied the population of Carloforte (Sardinia) because its average consumption of seafoods was 3.5 times (300 g/week) the national Italian average and because during the summer months fresh tuna meat was available from the 'tonnare' situated there. From the 6,200 residents 195 were chosen at random who agreed to give information about their food habits, take a medical examination and allow blood and hair analyses. About 65 % of these reported that they ate seafood more than 3 times a week. 11.7% consumed 7 and more seafood meals per week and 1.5 % as many as 13 to 14 meals per week, equal to about 2,100 g edible seafood.

The 1973-74 Seafood Consumption Study (National Purchase Diary Panel, 1975) surveyed 26,848 individuals in the USA. They found that 93 % of those interviewed consumed seafoods. Tuna was the most common, eaten by 61.5 %. The average consumption of seafood was 17.6 g/day with a maxiumum of 7,000 g/month (230 g/day). The maximum tuna consumption was 5,730 g tuna per month (190 g/day).

Preston et al. (1974) and Haxton et al. (1979) approached the problem of critical groups more systematically. Arguing that due to physiological differences betweeen individuals, the person consuming the highest amounts of seafood may not be the most exposed to contaminants, and that an average consumption rate of a critical group may be more representative, the authors try to separate such critical population groups from the general public. Surveying 500 individuals from North-East Irish communities with special emphasis on fishing communities, they found that the seafood consumption pattern for most of the population fitted a log-normal distribution. At the low and the high end there was some deviation from this distribution. Four persons consumed on average 410 to 510 g of seafoods per day and one individual on average 835 g/week. The authors concluded that due to the restricted number of observations available they were not able to select objectively a critical population group and suggested using the exceptional individual in a representative sample of the exposed population for an estimation of highest possible exposure. Haxton et al. (1979) repeated the survey on two high exposure groups in the North-East Irish Sea and the Southern English Channel comparing results obtained by interview with the duplicate diet method (a duplicate of the amounts eaten is preserved for analysis). They observed that interviewing resulted in a higher consumption rate than actually determining the amounts of seafoods eaten. In the interview, some consumers reported that they had eaten up to about 800 g (fresh weight)/day but in the duplicate diet study on the same subjects the maximum amount consumed was only 225 g/day. The authors concluded that the discrepancy between the consumption data actually found and that stated during the initial interview reflects inherent inaccuracies in arriving at true consumption rates simply by interview, rather than the failure to supply an exact duplicate of a meal.

In Australia, the Working Group on Mercury in Fish conducted two surveys on heavy fish consumers: people buying and selling fish at wholesale markets, commercial and leisure fishermen, teenagers, fishshop customers, people who were dieting, or eating fish with a high mercury concentration etc. (Australian Working Group, 1980). 13 % of 259 interviewees consumed more than 1,000 g of seafood per week and 0.3 % between 3,000 and 4,000 g/week. People of Mediterranean background consumed higher amounts than those of British background. Those with the highest consumption rate did not necessarily ingest the largest amounts of pollutants because different seafoods had different mercury concentrations. The following shows how the mercury concentration in the seafood influences the intakes.

Fish consumption g/week	weight of consumer	calculated mercury intake ug/kg body weight/week
3,580	73.1	3.3
2,840	74.7	8.5
2,000	95.4	5.0
1,440	54.5	10.4

As can be seen, the person eating only 1,440 g of fish had about 3 times the mercury intake of the person eating about three times that amount of fish. The Australian survey identified the highest consumers. They were in order of consumption: a person on a medical diet, a Greek fish shop proprietor, a fish wholesaler, a Laotian restaurant employee, a Greek "fish and chip" shop owner, a Chinese cook, a Greek salesman, an Australian housewife, a

German female clerk, a fish salesman, a Greek unemployed person and a German lady on a slimming diet. However, this survey also showed that interviewing people on their food habits did not produce very reliable estimates: those interviewed about their frequency before the survey started overestimated the number of meals they had eaten regularly.

Probably the highest seafood consumption rates were observed in Japanese tuna fishermen and fish retailers. In their review Doi & Ui (1975) give details on 13 tuna fishermen, who worked up to 2 years mainly in the Indian Ocean, and 34 retailers. The majority of fishermen are reported to have eaten about 300 g of tuna daily with a range from 50 to 400 g tuna per day. 21 out of 34 retailers consumed 100 to 200 g fresh tuna (range of all 34 retailers 50 to 300 g/day) 7 times a week in addition to a daily diet of 100 to 200 g of other seafoods (range 70 to 1,000 g/day). One man is reported to have eaten daily 200 g fresh tuna plus 1,000 g of other seafoods.

In conclusion it may be said that a true consumption rate can probably be estimated only through actual measured seafood intakes, but that there may exist extreme consumers of seafood (fish and shellfish) who reach a daily intake of 1 kg (7 kg/week) and perhaps even more.

In view of the extremely high MeHg concentrations in the muscle, and the high inorganic mercury level in the liver of marine mammals, special attention has to be given to critical groups (e.g. Inughuits) eating muscle, heart, liver, blubber etc. of these marine mammals. In his review of the changes in Inughuit utilization of Arctic wildlife, Riewe (1981) points out that their consumption pattern is rapidly changing. Before the introduction of the snow-mobile and other technology, and before they began to gather in permanent settlements during the winter, the Inughuits typically joined together in communal camps on the flat sea ice to hunt ringed seals (Phoca hispida) at seal breathing holes. In May, the in large winter camp split into smaller family units which consumed seals taken as they basked on the ice surface. During the first two weeks in July, when the arctic char (Salvelinus alpinus) began to migrate downstream, some families fished at stone weirs. During August and September the people turned further inland and hunted barren-ground caribou (Rangifer tarandus) from kayaks at water crossings. During October and November the Inughuits camped along rivers and fished through the thin ice. By December they moved back into the winter sealing camp. In more recent times i.e. 1960 at Point Hope the harvest of wildlife consisted of 56 % of ringed and bearded seals, 22 % bowhead whale and white whales, 19 % caribou and 3 % fish. Adult Point Hope Inughuits are estimated to consume 2,260 g wild meat per day and children consumed about 1,500 g/day. Approximately 50 % of the caloric intake (2,300 to 4,500 kcal: mean 2300) were derived from fat and 30 to 35 % from protein. Carbohydrates accounted for only 15-20 % of their calories, largely in the form of glycogen from the meat they consume (Ho et al. 1972). If one considers that whale meat contains 1,250kcal/kg (fresh weight) (5,225 kJ) (Documents Geigy, 1969) then 2260 g fresh meat from marine mammals should correspond to about 2,800 kcal (11,700 kJ) for a man and 1,875 kcal (7,840 kJ) for a child. This seems not to be excessive considering that this is the only energy source in a rigorous climate. Ho et al. (1972) found that during 1960 only one-fifth of the food consumed was imported. Southampton Island Inughuits consumed an average of 374 g/day consisting of 48 % of ringed seal, 16 % bearded seal, 8 % of polar bear and 7 % of white whale. In addition, they consumed arctic char, wild-fowl, arctic hare, eggs and caribou as well as imported foods. Also, Inughuits from southern Ellesmere Island in 1970 relied on ringed seal (47 %),

bearded seal (13 %), polar bear 12 %, walrus 9 %, harp seal 8 % and narwal (5 %). Men consumed on average about 2,700 g/day. Near Sachs harbour, on the other hand, caribou was the main food item (73 %), followed by fish (7 %) and snow goose (7 %) and only a small part was from ringed seal, ptarmigan (northern goose), polar bear, arctic hare, eider ducks and snowy owls. Poorer families who cannot afford to buy imported food ate higher proportions of wild meat than more affluent families. These data indicate that Inughuits who are more integrated in the "Western society" and dispose of higher monetary income will consume lesser amounts of marine mammals than those still living in remote locations with little cash income.

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II. ARSENIC

1. ARSENIC IN THE MARINE ENVIRONMENT

1.1 Reference Documentation

The major reviews and reference works used in the preparation of this section were the IRPTC Data Profile on arsenic compounds (UNEP, 1983), the WHO Environmental Health Criteria on Arsenic (WHO, 1981), the National Academy of Sciences review of arsenic (NAS, 1977), Penrose (1974), Ferguson & Gavis (1972) and the Handbook of Chemistry and Physics (Weast, 1976). Many other individual papers by research workers were consulted and are listed in the reference section.

1.2 General facts

Arsenic (As): atomic number 33; atomic weight 74.9216; valency 3, 0, +3, +5. Arsenic, in group Va of the periodic table, is classified as a metalloid and exhibits both metallic and non-metallic properties. The element occurs in two solid forms: grey (metallic) and yellow, with specific gravities of 5.73 and 1.97 respectively. With an average concentration in the earth's crust of 1.5 to 2 mg/kg, arsenic ranks 20th in abundance in relation to other elements (NAS, 1977). It is present in rocks and ores and is found native in the sulphides realgar (As_4S_4) and orpiment (As_2S_3), as arsenides and sulpharsenides of heavy metals, as the oxide, and as arsenates. Mispickel or arsenopyrites (FeSAs) is the most common mineral.

Oxide forms of arsenic are usually found in sedimentary deposits. The elemental oxidation state, though stable in reducing environments, is rarely found. Soils generally contain 4 mg/kg of arsenic with an average background level of about 7 mg/kg.

The arsenic concentration of surface freshwaters in unpolluted areas varies, but typical values seem to be a few micrograms per litre or less. Quentin & Winkler (1974) found an average value of 3 µg/litre in river water and 4 µg/litre in lake water in the Federal Republic of Germany. Mean arsenic concentrations of 0.02 to 1.05 µg As/litre were reported for some Norwegian rivers (Lenvik et al., 1978). The relative importance of the particulate-bound, complexed and dissolved components in river-borne arsenic is variable (Ferguson & Gavis, 1972; de Groot, 1973; Crecelius et al., 1975; Waslenchuk & Windom, 1978; Langston, 1980; Lemmo et al., 1983).

Arsenic is present in sea water predominantly as arsenate and at the pH and Eh of normal sea water will be partially protonated i.e. it will exist as $HAsO_4^{2-}$ (Ferguson & Gavis, 1972). Other forms of arsenic are present in sea water as a result of biological mediation (see below). Arsenic has a rich and complex organic chemistry and this is reflected to some extent in its environmental behaviour.

The formulae of various arsenic compounds referred to in this report are given in Table 1.

Table 1. Formulae of some arsenic compounds referred to in the text.

Arsenous oxides	As_2O_3 (or As_4O_6)
Arsenite	AsO_3^{3-}
Arsenate	AsO_4^{3-}
Methylarsonic acid (MMA)	$CH_3AsO(OH)_2$
Dimethylarsinic acid (DMA)	$(CH_3)_2AsO(OH)$
Arsenobetaine	$(CH_3)_3As^+CH_2COO^-$
Arsenocholine	$(CH_3)_3As^+CH_2CH_2OH\ X^-$
Dimethylarsinoylethanol	$O=As(CH_3)_2CH_2CH_2OH$
Trimethylarsine	$(CH_3)_3As$
Trimethylarsine oxide	$(CH_3)_3As=O$

Arsenic has long been known as a poison and a therapeutic agent. Arsenous oxide (As_4O_6) is the most important compound in terms of industrial production and is the starting point for the manufacture of other arsenicals used as pesticides, herbicides, cotton desiccants and wood preservatives; in the manufacture of glass, ceramics, semiconductors and dyestuffs; and as additives in alloys to increase hardness and heat resistance.

1.3 Sources

Total world production of arsenic in 1975 was around 60,000 tonnes/year (Nelson, 1977; WHO, 1981). The main producers are China, France, Federal Republic of Germany, Mexico, Namibia, Peru, Sweden, U.S.A and U.S.S.R. Much of this arsenic is intentionally released into the environment (as pesticides for example). Arsenic is also released unintentionally as a result of smelting or roasting sulphide minerals, the combustion of fossil fuels, leaching of exposed wastes from mining activity, and accelerated erosion of land. Volcanoes, the burning of vegetation and continental weathering are major natural contributors of arsenic to the marine environment. It has been considered, though, that releases resulting from the activities of man may exceed those due to natural processes (Ferguson & Gavis, 1972; Mackenzie et al., 1979). There is still uncertainaty about the relative importance of the various sources of arsenic (Walsh et al., 1979; Chilvers & Peterson, 1984). Ferguson & Gavis (1972) considered that the influence of the increase in arsenic concentration on a well-mixed ocean would be negligible for many thousands of years. However, Mackenzie et al, (1979) calculated a net gain by oceanic waters of 5.66×10^4 tonnes/year. A similar result has been obtained by Chilvers & Peterson (1984). They calculated a net gain in the arsenic contents of the oceans of 13.7×10^4 tonnes/year, which represents a concentration increase of 0.1% per year. Aspects of the global flux of arsenic have also been considered by Walsh et al. (1979).

The most important local inputs of arsenic into the marine environment result from the smelting of non-ferrous ores and from the river drainage of areas with substantial arseniferous ore deposits. Various local sources of arsenic contamination to the marine environment have received attention in the literature. The largest of these is a copper smelter which discharges liquid waste and crystalline slag particles into the Puget Sound, north-western Washington State, U.S.A. (Crecelius et al., 1975). All U.S.A. commercial production of arsenic is from this smelter and it amounts to about one quarter of all world production (Nelson, 1977). Contamination of the Gulf of Bothnia by a large copper smelter at Skellefteo, Sweden, has also been reported (Lindau, 1977; Hallberg, 1979). Klumpp and Peterson (1979) and Penrose et al. (1975) have measured the distribution of elevated levels of arsenic in water, sediments and organisms affected by drainage from areas of mining and land containing arseniferous ores in south-west England and Newfoundland, Canada, respectively. Rivers draining large industrial areas are also likely to carry increased arsenic burdens from a variety of industrial and domestic sources. For example, the Rhine contains arsenic at a concentration five times as high as in oceanic water (Weichart, 1973). Other inputs of arsenic into the marine environment, localized, but nevertheless possibly severe in their effects, include accidental emissions of the type documented by Ambrosi & Amicarelli (1982) when several tonnes of arsenic were accidentally released in the region of Manfredonia, Italy and, in the past, dumping (Bryan, 1976), although this is no longer thought to be a significant source.

1.4 Transport, transformation and bioaccumulation

1.4.1 Transport

Arsenic enters the oceans from the atmosphere, and is in freshwater associated with particulates and in the dissolved phase (de Groot, 1973; Crecelius et al., 1975; Waslenchuk & Windom, 1978). Langston (1980) reported that arsenic entered estuaries in particulate form, from weathering of ores such as arsenopyrites, and as arsenate and arsenite which may be scavenged by hydrated iron oxides in estuarine sediments. However, arsenic borne by rivers of south-eastern U.S.A. was complexed to low molecular weight dissolved organic material and passed through the estuaries without being precipitated (Waslenchuk & Windom, 1978). The precipitation of arsenic entering natural waters by hydrated ferric compounds has also been considered by Ferguson & Gavis (1972) and Lemmo et al. (1983).

Arsenic in oceanic waters is predominantly in dissolved form (Johnson & Pilson, 1972; Gohda, 1975), although it may be removed from solution by adsorption or co-precipitation with iron and aluminium compounds (Crecelius et al, 1975; Andreae, 1979). As the distribution of total arsenic levels in the oceans shows little variation (see Section 1.5.1.), movement of arsenic with bodies of water will have little or no effect on overall total arsenic distribution. However, the biologically facilitated transport of the various arsenic species in sea water may have some effect on overall arsenic distribution within a certain locality (Johnson & Pilson, 1972; Andreae, 1978, 1979; Peterson & Carpenter, 1983).

1.4.2 Transformation

Transformations of arsenic in the marine environment are biochemically mediated, with the exception of arsenite-arsenate conversions facilitated by the oxic-anoxic status of the water. Four species of arsenic (arsenite, arsenate, monomethylarsonate and dimethylarsinate) have been detected in sea water (Braman & Foreback, 1973; Andreae, 1978, 1979) and although in oxic waters arsenate predominates, arsenite is always present at greater than thermodynamic equilibrium concentrations (Johnson & Pilson, 1972; Andreae, 1978, 1979) (see Section 1.4.3). Johnson (1972) and Johnson & Burke (1978) have demonstrated the ability of bacteria to reduce arsenate in sea water to arsenite. Evidence that marine waters of the photic zone contain arsenite, monomethylarsonate and dimethylarsinate indicates that algae play a major role in the production of reduced and methylated forms of arsenic in sea water (Andreae, 1978, 1979; Andreae & Klumpp, 1979; Sanders & Windom, 1980). Other organisms may also transform arsenate to arsenite and/or simple methylated forms (Pilson, 1974; Vidal & Vidal, 1980). Sanders (1979a) and Scudlark and Johnson (1982) have also shown a microbial role in the demethylation and oxidation of methylated arsenicals in sea water. Thus all stages in the sequence arsenate $\Longrightarrow$ arsenite $\Longrightarrow$ $CH_3AsO(OH)_2$ $\Longrightarrow$ $(CH_3)_2AsO_2H$ have been shown to involve possible biological mediation.

Much work has been undertaken in an effort to identify arsenic compounds in marine organisms as well as to understand the mechanisms for their formation (e.g. Lunde, 1975, 1977; Penrose et al., 1977; Wrench et al., 1979; Cannon et al., 1981; Edmonds et al., 1982a; Edmonds & Francesconi, 1981a, 1983). Laboratory experiments carried out on marine animals have shown that food is more important as a source of arsenic than sea water (section 1.4.3). Therefore the precursors of arsenobetaine, the major form of arsenic in marine animals at the top of the food chain, is most likely formed along the food chain. The analysis of biota associated with Sargassum weed indicated that substantial amounts of arsenic were present in forms other than the inorganic or methylarsenic compounds (Johnson & Braman, 1975). Only small amounts of simple methylated arsenicals were present. Water-soluble and lipid-soluble forms of organic arsenic have both been detected in algae, their relative proportions varying in different forms of algae (Irgolic et al., 1977; Cooney et al., 1978; Klumpp & Peterson, 1981). Identification of organic arsenic compounds other than the methylated arsenic acids in algae is to date confined to arsenic-containing derivatives of ribofuranoside (ACR) isolated from the brown kelp Ecklonia radiata (Edmonds & Francesconi, 1981b; 1983). Acylation by long chain fatty acids of the relevant free hydroxyl groups in the appropriate ACR would produce a phospholipid analogous to lecithin (Edmonds & Francesconi, 1983). Thus lipid-soluble as well as water-soluble ACR derivatives may exist.

The conversion of ACR derivatives, which are possibly common to all marine algae (Edmonds et al., 1982a; Knowles & Benson, 1983), to arsenobetaine in organisms along the food web has been studied by Edmonds et al. (1982b). Anaerobic decomposition of Ecklonia quantitatively converted ACRs into 2-dimethylarsinoylethanol and it has been proposed that this compound has a key position in the biosynthesis of arsenobetaine (Edmonds et al., 1982b). Thus a microbially mediated stage in anaerobic sediments is apparently necessary for the production of arsenobetaine by this scheme, with arsenobetaine or its precursors becoming available to the food chain via detritivores. Animals based on food chains lacking such a microbially mediated stage are likely to

contain either algal derived arsenic compounds or as yet unidentified compounds (Klumpp & Peterson, 1981; Shiomi et al., 1983b) formed by their degradation or conversion. Thus, filter feeding organisms deriving nutrition from planktonic organisms (particularly phytoplankton) and other animals based on food chains lacking a detrital stage cannot accumulate arsenobetaine by the sequence outlined above. Indeed, it has been shown (Edmonds et al., 1982a) that the edible adductor muscle of the giant clam Tridacna maxima contains its arsenic as ACRs and arsenobetaine is absent. In addition, Shiomi et al. (1983b) have demonstrated that the arsenic in a tunicate (Halocynthia roretzi) was present as one acidic and two basic compounds, of which none was arsenobetaine. The studies of Klumpp & Peterson (1981), on a short macroalgae-based food chain lacking a detrital stage, also demonstrated an absence of arsenobetaine in snails feeding on Fucus spiralis. It has been shown, furthermore, (Cooney & Benson, 1980) that organo-arsenicals biosynthesised by, and contained in, the unicellular alga Dunaliella tertiolecta were not metabolised to arsenobetaine by the American lobster Homarus americanus although it was demonstrated (Edmonds & Francesconi, 1981b) that the native arsenic in H. americanus is present as arsenobetaine.

Arsenobetaine has been found to be the major form of arsenic in marine animals contributing to the human diet, e.g. western rock lobster, dusky shark (Cannon et al., 1981), American lobster (Edmonds & Francesconi, 1981a), several teleost fishes, crabs and shrimps (Luten et al., 1982; Norin & Christakopoulos, 1982; Luten & Riekwel-Booy, 1983; Shiomi et al., 1984). Arsenobetaine is the arsenic analogue of betaine, a common osmolyte in marine organisms (Yancey et al., 1982).

Evidence that extracts of the shrimp Pandalus borealis contain small amounts of arsenocholine in addition to the arsenobetaine has been presented (Norin & Christakopoulos, 1982; Norin et al., 1983). Other unidentified methylated arsenic compounds, have been shown to be present in scallops (Pecten alba) and prawns (Penaeus latisulcatus) (Maher, 1985). In addition, Whitfield et al. (1983) have reported low levels (0.002-0.98 µg/kg) of trimethylarsine in a range of crustaceans with the higher levels in the red prawn Aristeomorpha foliacea, and some species of fish taken from marine, brackish and freshwaters have been found to contain a small percentage (in general less than 10%) of their total arsenic burden as trimethylarsine oxide (Norin et al., 1985a). Penrose (1975) with trout and Klumpp & Peterson (1981) with snails have demonstrated the conversion of administered radio-labelled arsenate into organic, cationic, arsenic compounds. The relationships of the above compounds to the metabolism of arsenobetaine remains to be demonstrated.

The oil-rich tissues from some marine animals (e.g. fish liver) contain lipid-soluble arsenic in addition to any arsenobetaine that might be present in muscle tissue (Lunde, 1968; Kurosawa et al., 1980) and it is likely that some oil-rich fish such as herring (Clupea harengus) contain lipid-soluble arsenic in their muscle tissue (Lunde, 1968). Further study is required to identify these compounds. Lipid-soluble conjugates of arsenobetaine or phospholipids of the type discussed by Irgolic et al. (1977) are possible candidates.

1.4.3 Bioaccumulation

Marine algae contain arsenic at concentrations 2,000 to 5,000 times greater than those in sea water (see sections 1.5.1 an 1.5.3).

Uptake of ^{74}As by the brown macroalgae Fucus spiralis and Ascophyllum nodosum was found to increase in direct proportion to increasing temperature and was greater in the presence of photosynthetic inhibitors or in the dark than under illuminated conditions (Klumpp, 1980a). Fucus spiralis accumulated four times more arsenate than arsenite. Uptake will therefore be affected by the prevailing physical and chemical conditions of the sea water, notably its oxic-anoxic status (see Section 1.4.2.). However, Klumpp's (1980a) studies showed that variations of pH or salinity did not affect arsenic incorporation. The accumulation of ^{74}As reached a steady state in 1 to 8 days, depending on the species and external arsenic concentration. At steady state the accumulated arsenic was proportional to the external arsenic concentration. Sanders & Windom (1980) also found that arsenate uptake depended on the concentration present in the media, both in terms of the uptake rate and the total arsenic absorbed, for the phytoplankton Skeletonema costatum. In addition, uptake and total arsenic absorbed were affected by the phosphate concentration of the media in S. costatum which suggested to Sanders & Windom (1980) a common mechanism of phosphate and arsenate uptake. Klumpp (1980a) found no evidence for this in his studies with macroalgae, although high concentrations of phosphate (40 to 400 µM) initially inhibited arsenate uptake.

Experimental studies on marine animals have indicated the food chain (i.e. dietary intake) to be of far greater importance than ambient water as a source of arsenic. It is therefore likely that environmental factors affecting arsenic absorption by algae will be critical in determining arsenic concentrations at all levels of the food chain.

Klumpp (1980b), for example, reported that when marine snails Littorina littorea and Nucella lapillus were held in sea water containing 3 µg/litre radio-labelled arsenate (approximately the normal ocean water concentration), they achieved steady-state tissue ^{74}As concentrations two orders of magnitude below the actual arsenic levels in nature. Fowler & Unlu (1978) concluded, as a result of experiments with radio-labelled arsenate, that levels of arsenic in the shrimp Lysmata seticaudata would be difficult to achieve by the direct absorption of arsenic from water and concluded that food is the means by which shrimp accumulate arsenic in the natural environment. They supported this hypothesis with data showing a high degree of assimilation and long retention of arsenic ingested by shrimp from food. Pentreath (1977) studied the accumulation of ^{74}As-labelled sodium arsenate from sea water by plaice (Pleuronectes platessa) and thornback ray (Raja clarata) and made comparisons with accumulation of arsenic from labelled food. He found that accumulation direct from sea water was slow. In contrast, retention of ^{74}As from labelled food was high for the ray (85%) but low (10%) for the plaice. Both species contained the largest fraction of the ^{74}As body burden in muscle. However, it is most unlikely (see Section 1.4.2.) that fish would receive food containing arsenic as arsenate in natural circumstances.

It is possible, in the light of general conclusions about the importance of food versus water to arsenic accumulation, that detailed kinetic studies of absorption of arsenic from water by marine animals will have little relevance to natural situations. Nevertheless, Klumpp (1980b) showed that a number of environmental factors influenced the uptake of arsenic by marine snails. The uptake of arsenic by Littorina littorea was reduced at low salinity and by phosphate levels between 9 and 17 µM, and increased by higher temperatures and arsenate concentrations. However, the uptake of arsenic from water by Lysmata sticaudata (Fowler & Unlu, 1978) was greater at low

salinities. Large individual variations (mostly caused by moulting) masked any effect that temperature might have had on accumulation. Unlu & Fowler (1979) showed that the absorption of arsenate by the mussel Mytilus galloprovincialis increased with increasing concentration of arsenic in the water, increased with higher temperatures, and was three times as high at a salinity of 19% than at 38%.

Arsenic is not biomagnified in marine foodchains (Ferguson & Gavis, 1972; Kennedy, 1976; Klumpp & Peterson, 1979; Sanders, 1980; Klumpp, 1980b). Never- theless, a number of studies have demonstrated positive relationships between size of marine animals and their arsenic concentrations, indicating that several species are able to bioaccumulate arsenic. Bohn (1975) reported sig- nificant relationships between fish size and arsenic level for wolffish (Anarhichas minor), halibut (Reinhardtius hippoglossoides) and shorthorn sculpin (Myoxocephalus scorpius) from West Greenland. Kennedy (1976) demonstrated significant positive correlations between animal size and arsenic concentrations for two species of pandalid shrimps (Pandalus borealis and Pandalus montagui) from several localities in Newfoundland and Labrador (Canada). Bohn & McElroy (1976) and Bohn & Fallis (1978) reported similar relationships for arctic cod (Boreogadus saida) and shorthorn sculpin (Myoxocephalus scorpius), respectively, from northern Canadian waters. A teleost fish (Sillago maculata) from Cockburn Sound, Western Australia was shown to display a positive relationship between size and arsenic level (Edmonds & Francesconi, 1981c). Bohn (1975) and Klumpp & Peterson (1979) have shown elevated levels of arsenic in biota from areas of abnormally high environmental levels. Langston (1980) showed that concentrations of arsenic in estuarine organisms correlated more significantly with the As/Fe ratio in sediments than with arsenic levels alone.

1.5 Arsenic in sea water, sediments and marine biota

1.5.1 Sea water

Early analyses of British coastal and English Channel waters determined a mean total arsenic concentration of 2.6 µg/litre (Armstrong & Harvey, 1950; Smales & Pate, 1952; Portmann & Riley, 1964). More recent studies have reported rather lower values for Pacific and Atlantic ocean waters. Some studies have also provided details of the contributions of the four arsenic species (arsenate, arsenite, monomethylarsonate and dimethylarsinate) in sea water to the total arsenic concentration. Burton et al. (1980) reported 1.3 to 1.7 (mean 1.49) µg/litre for Atlantic waters (Cape Basin) above 110 m, while deeper waters contained 1.3 to 2.1 (mean 1.58) µg/litre. Waslenchuk (1978) stated that arsenite contributed about 20% of total arsenic (mean 1.1 µg/litre) in continental shelf waters of the southeastern U.S.A. Gohda (1975) obtained values of 1.4 to 2.4 µg/litre for 12 samples of Japanese coastal waters in which arsenate predominated (80 to 97%) with arsenite forming the balance (apart from a trace of arsenic in particulate material). The first report of the presence of monomethylarsonate and dimethylarsinate in sea water was that of Braman & Foreback (1973). They determined the levels of arsenate, arsenite, monomethylarsonate and dimethylarsinate in three samples of saline water from the Florida coast. Total arsenic ranged from 1.48 to 2.28 µg/litre with arsenate contributing 24 to 82% and dimethylarsinic acid 11 to 68%. Andreae (1978) showed Pacific ocean waters off southern California to contain 1.4 to 1.8 µg/litre total arsenic. Waters

below the photic zone (with a few exceptions) contained virtually all arsenic as arsenate. Waters of the photic zone contained dimethylarsinate (0.1 to 0.3 µg/litre), arsenite (up to c. 0.9 µg/litre) and monomethylarsonate (up to c. 0.1 µg/litre) as well as arsenate (see Section 1.5.2.).

Elevated levels of arsenic (as measured against the consistent background of oceanic waters given above) have been reported for certain estuarine or coastal locations where a specific source was implicated. For example, Klumpp & Peterson (1979) reported up to 42 µg/litre in water of the Carnon River draining an area that supported non-ferrous metal mining in the southwest of England. Penrose et al. (1975) gave 5.3 µg/litre as the highest value in sea water associated with a mining drainage site. Zingde et al. (1976) recorded arsenic concentrations of up to 66 µg/litre in water from estuarine and coastal sites in Goa, but apart from noting the presence of a manganese ore body did not suggest reasons for such high readings. Brügmann (1981) reported 0.5 to 7.5 µg/litre for waters of the Baltic Sea and suggested that arsenic could represent a pollution problem in parts of the Gulf of Bothnia.

1.5.2 Sediments

Arsenic concentrations in sediments are given on a dry weight basis. Onishi & Sandell (1955) discovered arsenic concentrations between 0.58 and 20.0 mg/kg for 13 deep sea sediments from the West Pacific Ocean and the Sea of Japan, the highest values being found in Pacific samples. Boström & Valdes (1969) found the following values for the arsenic content of deep ocean pelagic sediments (on a carbonate free basis): North Atlantic Ocean, 18 mg/kg; Indian Ocean, 16 mg/kg; equatorial Pacific, 121 mg/kg; south Pacific Ocean, 40 mg/kg; with average for world oceans of about 40 mg/kg. They noted that their average value differed from that of Turekian & Wedepohl (1961) who recorded an average of 13 mg/kg for pelagic sediments. Boström & Valdes (1969) also noted that if pelagic deposits in adjacent seas were also included, their average value of c. 40 mg/kg might well have dropped to 20 to 30 mg/kg. They considered that the highest arsenic values in sediments were found on or close to active oceanic ridges and noted some evidence that arsenic levels seemed to co-vary with iron or manganese.

Uncontaminated sediments from coastal areas and estuaries appear to contain rather lower concentrations of arsenic than deep sea sediments. Leatherland & Burton (1974) reported an arsenic concentration of 14 mg/kg in bottom muds from Southampton Water, U.K., and noted that this value was very close to the average concentration in shales and unconsolidated muds estimated by Onishi (1969). Crecelius et al. (1975) reported that concentrations of arsenic in non-contaminated surface sediments from Puget Sound, Washington State, U.S.A., ranged from 3 to 15 mg/kg. Kennedy (1976) recorded arsenic levels of 3.2 to 5.0 mg/kg for 6 samples of muds taken from two locations in northern Newfoundland and southern Labrador, Canada.

However, as has been described elsewhere in this report, discharges of arsenic-enriched effluents of largely anthropogenic origin can cause local (Penrose et al., 1975) or more widespread (Langston, 1980; Crecelius et al., 1975) elevations of arsenic concentrations in sediments. Sediment arsenic concentrations in the range 50 to 300 mg/kg were recorded for virtually the entire Gulf of Bothnia, in the Baltic Sea, not just in the immediate vicinity of discharge outfall from a smelter at Skelleftea, Sweden (Hallberg, 1979).

1.5.3 Marine biota

There is now a considerable body of data on arsenic concentrations in marine organisms, particularly on components of the human diet. In addition to reports on total concentrations of arsenic in marine organisms, there are also several papers that deal with the chemical form of the arsenic present. These may be classified into two groups: first, those that measure the relative proportions of inorganic and organically bound arsenic present; and second, those that identify the chemical form of the organic arsenic.

Algae

In general, brown algae contain considerable amounts of arsenic, typically in the range 10 to 100 mg/kg dry weight (Dhandhukia & Seshadri, 1969; Lunde, 1970; Tagawa & Kojima, 1976). These values are somewhat higher than those detected in red or green (1 to 20 mg/kg dry weight) algae (Dhandhukia & Seshadri, 1969; Tagawa & Kojima, 1976; Sanders, 1979b).

Inorganic arsenic may form a substantial proportion of the arsenic content of some marine algae. Of particular interest is the brown alga Hizikia fusiforme, an important foodstuff (Hijiki) in Japan, which has been reported to contain 60 to 80% of its arsenic load as inorganic forms, mainly arsenate (Tagawa, 1980; Fukui et al., 1982; Shinagawa et al., 1983). However, the latter two groups consider the possibility that part of the arsenic was originally present as organic compounds which were hydrolysed to inorganic forms by the acidic conditions employed during analysis. Sanders (1979b) found considerable differences in arsenic form in a study which examined 16 species of green and 15 species of red algae. The percentage of total arsenic present in inorganic form differed markedly between 11 to 96% in the green algae and 19 to 83% in the red. Other studies have reported the majority of arsenic in algae (Laminaria hyperborea, L. japonicus, Undaria pinnatifida) to be organic (Lunde, 1973; Shinagawa et al., 1983).

The only organic arsenic compounds (other than the simple methylated arsenic acids) that have been identified in algae are the arsenic-containing ribofuranoside derivatives isolated from the brown kelp Ecklonia radiata (Edmonds & Francesconi, 1981b, 1983, see Section 1.4.2.). These authors report the lability of such compounds at extremes of pH, with release of dimethylarsinic acid. Thus reports (e.g. Tagawa, 1980; Maher, 1981) of dimethylarsinic acid in algae extracts where acidic conditions may have been involved in processing, may be indicative of the presence of arsenic originally bound in larger, comparatively unstable compounds.

Crustacea

High arsenic concentrations have been reported for crustaceans, with 1 to 50 mg/kg wet weight being representative of the range of normal values. Data on total arsenic content are available for many species, including shrimps (Kennedy, 1976; Munro, 1976), krill (Stoeppler & Brandt, 1979), prawns (Bohn, 1975; Egaas & Braekkan, 1977) and lobsters (Munro, 1976; Egaas & Braekkan, 1977). In addition, the relative proportions of organic and inorganic arsenic have been determined in commercially important species of East Australian crustacea, such as prawns, crabs, crayfish (Flanjak, 1982; Table 2). The ratio of inorganic:organic arsenic was less than 0.05 in 97% of the samples (overall mean 0.02). Numerous species from several other

Table 2. Organic and inorganic arsenic in some commercial East Australian crustacea (Flanjak, 1982)

		Arsenic (mg/kg, fresh weight basis)			
		Inorganic		Organic	
Sample and locality	Number	Range	Mean	Range	Mean[a]
School prawns					
Brooklyn	3	ND[c]	ND	3.5-5.1	4.2
Palm Beach	1		0.13		2.0
Clarence River	1		0.07		ND
Sydney Harbour	1		0.15		3.3
King prawns					
Sydney Harbour	3	ND-0.3	0.02	12.1-14.6	13.1
Jarvis Bay	6	0.05-0.10	0.08	2.8-4.0	3.5
Ballina	6	ND-0.05	0.03	3.5-7.0	5.1
Sydney fish markets[b]	3	0.03-0.07	0.05	2.8-5.2	4.3
Royal Red prawns					
Wollongong	3	0.03-0.05	0.04	3.5-5.1	4.2
Blue Swimmer crab					
Botany Bay	6	ND-0.18	0.07	1.2-5.9	3.1
Eastern Common crayfish					
Wallis Lake	29	0.19-0.41	0.30	19.0-54.1	35.5
Coffs Harbour	10	0.12-0.41	0.26	11.9-31.3	18.2
Sydney fish markets[b]	6	0.13-0.24	0.18	18.3-24.1	20.0
Mud crab					
Coffs Harbour	8	ND-0.13	0.05	ND-4.0[d]	1.9

a = In calculating mean 'non detected' (ND) was taken as half the detection limit
b = Exact origin of samples purchased is not known
c = ND 0.03 mg/kg for inorganic arsenic
d = ND 0.8 mg/kg for organic arsenic

localities are also represented by these figures (Lunde, 1973; Reinke et al., 1975; Maher, 1983; Shinagawa et al., 1983). Arsenobetaine has been reported as the sole or major arsenical in crustacea (Cannon et al., 1981; Edmonds & Francesconi, 1981c; Norin & Christakopoulos, 1982; Luten & Riekwel-Booy, 1983). It has also been reported in conjugated form in dried shrimp (Fukui et al., 1982).

Molluscs

Gastropod and bivalve molluscs have arsenic concentrations of 1 to 25 mg-/kg wet weight although the higher values may reflect coastal water pollution (Karbe et al., 1977). Cephalopod molluscs have occasionally been reported to contain higher concentrations (Leatherland & Burton, 1974). Data have been published from a number of coastal locations in Europe (Peden et al., 1973; Leatherland & Burton, 1974; Karbe et al., 1977; Kosta et al., 1978), North America (LeBlanc & Jackson, 1973; Munro, 1976; Hall et al., 1978) and elsewhere (Bohn, 1975; Zingde et al., 1976).

A limited number of studies have indicated the prevalence of organic arsenicals (90%) in bivalve and gastropod (Brooke & Evans, 1981; Shinagawa et al., 1983) and cephalopod molluscs (Shiomi et al., 1983a, Shinagawa et al., 1983). The presence of arsenic-containing ribofuranoside derivatives in the adductor muscle of the giant clam Tridacna maxima is thought to be due to the presence of unicellular green algae in the clam tissues (Edmonds et al., 1982a). It remains to be seen in what form bivalve molluscs and other filter-feeding organisms, deriving nutrition largely from phytoplankton, contain their arsenic burden.

Fish

Many references provide information on the arsenic content of marine fish. Data have been reported for teleosts in the Pacific: 0.3 to 11.5 mg/kg wet weight (LeBlanc & Jackson, 1973; Zingde et al., 1976; Kobayashi et al., 1979) with means of 0.6 in black marlin (Mackay et al., 1975) and 1.0 in several species (Bebbington et al., 1977; Powell et al., 1981). Values in the Atlantic ranged from 1 to 9 mg/kg dry weight (Leatherland et al., 1973; Windom et al., 1973) and 1 to 7 mg/kg wet weight (Hall et al., 1978). Mean values in the North Sea for several species of teleost fish were 5 mg/kg dry weight (Leatherland & Burton, 1974) and 23 mg/kg wet weight for plaice (Luten et al., 1982). In the Antarctic, Stoeppler & Brandt (1979) found concentrations of 0.3 to 1.6 mg/kg, mean 0.8 mg/kg wet weight, in a few species of teleost fish.

In general, teleost fish contain 1 to 10 mg/kg wet weight of arsenic with some species, usually of demersal habit, producing individuals of very much higher concentration. Luten et al. (1982) have reported a sample of plaice (Pleuronectes platessa) which contained 160 mg/kg wet weight, while Westöö & Rydälv (1972) recorded a range of 0.78 to 25 mg/kg (mean 5.9) for 78 samples of the same species.

Rather less information exists on the arsenic content of elasmobranchs, although in general they seem to contain higher levels than teleosts. Concentrations of 5 to 30 mg/kg wet weight appear normal (Hall et al., 1978; Glover, 1979; Cannon et al., 1981; Kurosawa et al., 1980) although the data of Le Blanc & Jackson (1973), Windom et al. (1973) and Powell et al. (1981) are at the lower end of this range.

Table 3. Arsenic concentrations in some fish species from fresh water and seawater in Sweden (Westöö and Rydälv, 1972).

Species	Number of samples	Arsenic level, mg/kg fish flesh	
		Range	Average
Pike, fresh water	92	0.004-0.64	0.08
Pike, Baltic	20	0.07-2.8	1.1
Baltic herring	38	0.15-1.4	0.59
Herring	19	0.38-2.9	1.5
Flounder	24	0.28-3.2	1.0
Plaice	78	0.78-25	5.9
Cod	10	0.82-2.4	1.5
Baltic cod	24	0.32-1.1	0.65

More than 80% of the total arsenic present in many commercially important fish species (cod, haddock, herring, mackerel, perch, pike, plaice) is reported to be in organic form (Lunde, 1973; Brooke & Evans, 1981; Norin et al., 1985b). Arsenobetaine is the major or sole arsenical in both teleost (Edmonds & Francesconi, 1981c; Luten et al., 1982; Luten & Riekwel-Booy, 1983; Shiomi et al., 1983b) and elasmobranch fishes (Kurosawa et al., 1980; Cannon et al., 1981). Norin et al. (1985b) reported that fish from brackish water polluted with arsenic contained slightly less (4-10%) of their arsenic burden as inorganic arsenic than did fish from unpolluted water (6-13%).

Concentrations of arsenic in fish commonly used as human food are given in Tables 3 to 5.

Marine birds and mammals

Data for marine birds and mammals are few, but indicate that levels of arsenic in tissues are generally low. Several species of shore birds (gulls, sandpipers, yellowlegs, oyster catchers, etc) have been reported to contain 0.01 to 1.50 mg As/kg wet weight in their livers and 1 mg/kg in their feathers (Turner et al., 1978; White et al., 1980). Ospreys (Pandion haliaetus) from estuaries in the eastern U.S.A. had 0.03 to 16.7 (mean 2.4) mg As/kg wet weight in their livers and 0.05 to 2.0 (mean 0.64) mg As/kg in their kidneys (Wiemeyer et al., 1980). The narwhal (Monodon monoceros), a marine mammal, contained similar levels in liver (1.27±0.70 mg/kg dry weight), kidney (1.61±0.79 mg/kg dry weight) and muscle (0.58±0.41 mg/kg dry weight). Higher concentrations were detected in blubber (2.93±12.1 mg/kg wet weight) (Wagemann et al., 1983).

Table 4. Concentrations of arsenic in some Norwegian fish products (Egaas and Braekkan, 1977).

Fish product	Arsenic Wet weight, mg/kg
Fresh/frozen	
Cod	0.2-3.41
Coalfish	0.56-2.22
Halibut	1.19
Plaice	1.38
Whale meat	0.15
Smoked	
Cod	5.95
Greenland halibut	7.84
Mackerel	3.36
Salmon	2.59
Salt-cured	
Herring	3.18
Crustacea	
Deep sea prawn meat	18.7
Lobster meat	4.40
roe	3.68
Canned/preserved fish products	
Cod	0.37-6.22
Coalfish	0.19-0.54
Haddock	0.91
Mackerel	1.16-2.91
Herring	0.87-1.34
Brisling	2.15-4.25
Crab	10.41-11.8

Table 5. Typical mean arsenic levels in Canadian fish and fishery products (Munro, 1976)

Fish/fish product	Arsenic, mg/kg
Freshwater	
Pickerel	0.4
Pike	0.3
Smelt	0.5
Whitefish	0.2
Marine	
Groundfish	
Cod	2.7
Halibut	4.9
Flounder (nonmineral area)	2.5
(mineral area)	18.3
Grey sole	56.4
Pelagic	
Herring	0.8
Salmon (Atlantic)	0.3
Tuna (bluefin)	0.6
Crustaceans and Molluscs	
Clams	1.2
Lobster	5.9
Scallops	1.2
Shrimp	10.9

2. EFFECTS ON MARINE BIOTA

2.1 Reference documentation

The major reviews and reference works used in the preparation of this section were the IRPTC Data Profile on arsenic compounds (UNEP, 1983) and the National Academy of Sciences review of arsenic (NAS, 1977).

2.2 Effects on marine biota

Arsenic contamination of the marine environment may lead to deleterious effects mainly from localized inputs (of industrial origin) of arsenous oxide/arsenite which may be acutely or chronically toxic to marine life within affected areas.

Reports of the effects of arsenic on marine organisms are surprisingly few and are summarized in Table 6. Most work on aquatic biota has concentrated on freshwater organisms. Not all classes of marine organisms have been subjected to study and the majority of work has been concentrated on algae, particularly phytoplankton. No studies have been carried out on the long-term effects of elevated arsenic levels on marine ecosystem. In addition, many studies have used arsenic concentrations unlikely to be encountered in marine systems, even under conditions of gross contamination. It must be stressed, therefore, that data on which to base a hazard evaluation for arsenic in the marine environment are less than adequate.

This chapter is limited to measurable, deleterious effects on marine organisms. However, algae (and micro-organisms) do respond to the ambient arsenic to which they are subjected by absorption and/or transformation of arsenic species (see Section 1.4.2.).

Algae and micro-organisms

It has been demonstrated (Sanders, 1979b; Sanders & Vermersch, 1982) that the growth of some marine phytoplankton (there is considerable interspecific variation (Sanders & Vermersch, 1982)) is inhibited by concentrations of arsenate at, or a little above, ambient levels when phosphate concentrations are low. Other studies (Bottino et al., 1978; Hollibaugh et al., 1980) with nutrient enriched sea water have shown As(III) and As(V) to reduce and delay growth at higher concentrations. Low levels of arsenate affect the species composition and succession of natural assemblages of phytoplankton (Hollibaugh et al., 1980; Sanders & Vermersch, 1982) and it is not possible to predict the response of communities by studies with single algal cultures (Sanders and Vermersh, 1982). It is apparent that As(V) is as toxic as As(III) to phytoplankton (possibly more toxic) (Bottino et al., 1978; Hollibaugh et al., 1980; Sanders, 1979b; Sanders & Vermersch, 1982). It is likely that arsenite is toxic because of its reactivity with sulfhydryl functions whereas arsenate exerts a more immediate effect by competitively replacing the chemically similar phosphate in mechanisms for phosphate absorption and transport (Sanders, 1979b).

Table 6. Effects of arsenic on marine biota

Location	Type of organism	Species	Type of study and arsenic compounds involved	Brief summary of results	Reference
Canada	Phytoplankton	Mixed natural populations including *Skeletonema costatum*, *Thalassiosira spp.*, *Chaetoceros spp.*, etc. Also *Thalassiosira aestevalis* alone	Growth inhibition by As(III) and As(V) of phytoplankton in nutrient-enriched seawater	Mixed natural population: As(V) at a concentration of 75 µg/litre resulted in decreased growth yields, and was slightly inhibiting at 22.5 µg/litre. *T. aestavalis*: growth was repressed when As(III) was present at 22.5 µg/litre and when As(V) was present at 22.5 µg/litre in low phosphate (0.5 µg As/litre medium). As(V) not toxic at 75 µg/litre in high phosphate medium	Hollibaugh et al. 1980
USA	Phytoplankton	*Tetraselmis chui*, *Hymenomonas carterae*	Effects of arsenate and arsenite on growth and morphology	Arsenate delayed and diminished growth of *T. chui* but tended to increase growth of *H. carterae* at concentrations of 10 mg/litre. Arsenite did not substantially affect growth patterns but caused morphological changes. Only arsenate affected both growth and morphology	Bottino et al. 1978
USA	Phytoplankton	*Skeletonema costatum*	Examine effects of arsenate, arsenite, dimethylarsinic acid on algal productivity	Arsenate inhibited primary productivity at concentrations as low as 9.3 µg/litre when phosphate levels were low. A phosphate enrichment of 28.5 µg/litre alleviated this inhibition. Arsenite was toxic to *Skeletonema* at similar concentration. Dimethylarsinic acid did not affect cell productivity at the levels examined	Sanders 1979c

Table 6. continued

Location	Type of organism	Species	Type of study and arsenic compounds involved	Brief summary of results	Reference
USA	Phytoplankton	Amthidinium carterae, Chaetoceros pseudocrinitum, Cylindrotheca closterium, Skeletonema costatum, Thalassiosira pseudonana, Isochrysis galbana, Tetraselmis contracta Natural assemblages dominated by microflagellates (37%) Chaetoceros spp., Chroomonas amphioxeia (11%) and Skeletonema costatum (8%)	To determine the degree of growth inhibition by arsenate of unialgal cultures. To determine whether low levels of arsenate can cause shifts in dominant species or in species succession in multi-algal cultures	Single species cultures: growth of Isochrysis galbana, Amphidinium carterae and Chaetoceros pseudocrinitum terminated by 3-15 pg/cell over background levels (0.2 pg/cell). Skeletonema costatum was greatly affected, Thalassiosira pseudonana and Tetraselmis contracta slightly affected and Cylindrotheca closterium most resistant to arsenate additions of up to 20 pg/cell. (Phosphate - not added - present at 47.5 µg/litre initial cell densities $5x10^3$ cells/ml). Natural phyto-plankton assemblages: specific growth-rate depressed, species composition affected by arsenate addition. S. costatum was greatly affected (abundance 33% of cellnumbers in flasks with highest arsenate - 25 µg/litre). Chaetoceros spp. dominatedthe assemblages. Microflagellates largely unaffected by addition of arsenate. (Phosphate - not added - present at 52 µg/litre initial cell densities $1.8x10^3$ cells/ml.	Sanders & Vennersch 1982
USA	Marine yeast	Similar to Rhodotorula rubra	Inhibition of phosphate uptake and transport by arsenate	Reduced phosphate uptake at 95 µg/litre arsenate and high death rates in cultures with normal growth rates were induced by 0.95 to 9.5 µg/litre arsenate in phosphate deficient systems	Button & Dunker 1971 Button et al. 1973

Table 6. continued

Location	Type of organism	Species	Type of study and arsenic compounds involved	Brief summary of results	Reference
USA/ Atlantic	Brown macro-algal community	*Sargassum* sp. community including hydroids and shrimps (especially *Leander tenuicornis*)	To measure oxygen production: consumption in a pelagic *Sargassum* community, with arsenic additions	Arsenic addition had no measurable effect upon the metabolism of the community but when the "steady state" distribution of arsenic species was altered, there was a rapid response to re-establish the ambient As(III)/As(V) ratio	Blake & Johnson 1976
USA	Shrimp	*Penaeus setiferus*	Acute toxicity trials with arsenic trisulfide	96 hour LC_{50} 40.6 mg/litre (i.e. 24.7 mg As/litre)	Curtis et al. 1979
USA	Bivalve mollusc embryos	American Oyster - *Crassostrea virginica*	Acute toxicity trials with sodium arsenite	48 hour LC_{50} 7.50 mg/litre	Calabrese et at. 1973
USA	Bivalve mollusc juveniles	Bay scallops - *Argopecten irradians*	Acute toxicity trials with sodium arsenite Also to determine uptake	96 hour LC_{50} 3.49 mg/litre	Nelson et al. 1976
USA	Bivalve molluscs Crustacean	*Crassostrea gigas* *Mytilus edulis* *Cancer magister*	Determination of EC_{50}s of arsenite for mollusc larvae: LC_{50} of arsenite for crab zoeae	EC_{50}s (abnormal development) *C. gigas* 326 µg/litre *M. edulis* <3000 µg/litre. LC_{50} for *C. magister* zoeae 232 µg/litre	Martin et al. 1981
USA	Gastropod mollusc	Mud snail - *Nassarius obsoletus*	Measurement of behaviour and oxygen consumption of mud snails exposed to sodium arsenite	Oxygen consumption depressed after exposure to arsenic at concentrations <2.0 mg/litre. Animals distressed at exposure to <3.0 mg/litre arsenic	MacInnes & Thurberg 1973

Table 6. continued

Location	Type of organism	Species	Type of study and arsenic compounds involved	Brief summary of results	Reference
Canada	Teleost fish fry	Oncorhynchus keta (chum salmon)	Acute toxicity trials with sodium arsenite	48 hour TLm 11 mg/litre As_8O_3	Alderdice & Brett 1957
USA/ Florida	Teleost fish	Red Drum - Sciaenops ocellata	Field observations and analyses of tissues from fish apparently killed by metal poisoning	Arsenic levels in some tissues higher than in controls but other metals possibly more involved in deaths of fish	Cardeilhac et al. 1981
USA	Teleost fish	Pink salmon Oncorhynchus gorbuscha	Acute toxicity of As(III)	Total kill of fish in 7 days with concentration of 9.5 mg/litre 2.6-5.3 mg/litre tolerated (10 days plus observation)	Holland et al. 1960
Italy (Mediterranean)	Echinoderms	Sea urchins Paracentrotus lividus & Sphaerechinus granularis	Action of As(III) & As(V) on gametes and embryos	Exposure of embryos and pretreatment of sperms and eggs with As(III) & As(V) at concentrations of 0.37 to 3.75 mg/litre resulted in abnormalities. Data might suggest an as yet overlooked hazard of As(V)	Pagano et al. 1982

Macroalgae appear to be more resistant to the effects of arsenate. Klumpp (1980a) considered that if phosphate and arsenate share a common uptake system in the macroalgae Fucus spiralis and Ascophyllum nodosum then the process is unsaturated at levels of phosphate and arsenate likely to be encountered environmentally and phosphate levels will have little effect on arsenate uptake (and, presumably, the converse). Unnaturally high (1.6 mg/litre) phosphate was necessary to inhibit arsenate uptake. Blake & Johnson (1976) have reported that the overall metabolism of a pelagic Sargassum community was unaffected by arsenic additions.

Dimethylarsinic acid was non-toxic to phytoplankton up to a level of 46.9 µg/litre (Sanders, 1979c). Higher concentrations were not studied. Thus the synthesis and release of dimethylarsinic acid (presumably via arsenite (Antonia et al., 1979) and possibly via arsenic-containing ribofuranosides (Edmonds & Francesconi, 1981b) may represent a detoxification process.

The single example of a marine yeast examined (Button & Dunker, 1971; Button et al., 1973) to date behaved in a way similar to unicellular algae in its response to low levels of arsenate.

Molluscs

Studies on the acute effects of sodium arsenite on American oyster (Crassostrea virginica) larvae (Calabrese et al., 1973) and juvenile bay scallops (Argopecten irradians) (Nelson et al., 1976) showed 48 hour LC_{50}s of 7.50 and 4.40 mg/litre respectively. Martin et al. (1981) reported the concentrations of arsenite that caused abnormal development in 50% of individuals for two species of bivalve mollusc larvae to be 326 µg/litre (Crassostrea gigas) and 3000 µg/litre (Mytilus edulis). Sublethal studies (MacInnes & Thurberg, 1973) on the mud snail Nassarius obsoletus showed that arsenic (effects at 2 to 3 mg/litre) required a higher concentration than copper, silver or cadmium to cause depressed oxygen consumption or retraction, but lower concentrations than zinc.

Crustacea

A study (Curtis et al., 1979) on the shrimp Penaeus setiferus demonstrated a 96 hour LC_{50} of 40.6 mg/litre for arsenic trisulphide. As the solubility of As_2S_3 is only 0.5 mg/litre at 18°C, most of the compound would have been in suspension. Martin et al. (1981) reported that the LC_{50} of arsenite to zoeae of the crab (Cancer magister) was 232 µg/litre.

Echinoderms

Pagano et al. (1982) reported that As(III) and As(V) produce abnormalities in sea-urchin gametes and embryos at levels of 0.37 to 3.75/litre. Competitive inhibition by phosphate and arsenate has been observed (Chambers & Whiteley, 1966) for the eggs of the sea-urchin Strongylocentrotus purpuratus.

Fish

Acute toxicity effects of As(III) on chum salmon (Oncorhynchus keta) fry (Alderdice & Brett, 1957), and pink salmon (O. gorbuscha) (Holland et al., 1960) show a 48 hour TLm of 11 mg/litre and the LC_{100} in 7 days was

9.5 mg/litre respectively. On this limited basis As(III) must be considered to be as acutely toxic to fish as mercury, cadmium, chromium, zinc or copper (Portmann & Wilson, 1971; Negilski, 1976).

Arsenic has occasionally been implicated in fish kills (Zingde et al., 1979; Cardeilhac et al., 1981; Kilikidis et al., 1981) and arsenic levels have been reported as higher in dead fish than in controls. However, in each case, other factors were evident that were considered more likely to have caused death. Thus, Zingde et al. (1979) reported a general decline in water quality in the coastal waters of Goa relevant to their study, and Cardeilhac et al. (1981) and Kilikidis et al. (1981) considered other substances, notably copper, to have contributed to the fish deaths in their areas of interest (Florida and Crete, respectively).

3. HUMAN HEALTH ASPECTS

3.1 Introduction and reference documentation

When evaluating the health consequences of exposure to arsenic it is important to take into account the pronounced differences in metabolism and toxicity between different compounds of arsenic. Human exposure may be to inorganic compounds with arsenic in trivalent or pentavalent oxidation states or to many different organic arsenic compounds.

Reference documentation used in preparing this review include NAS (1977), Pershagen & Vahter (1979), IARC (1980), Fowler et al. (1979), WHO (1981), Fowler (1983), and Vahter (1983).

3.2 Toxicokinetic properties

3.2.1 Absorption

The absorption of inorganic arsenic compounds from the gastrointestinal tract depends to a great extent on solubility and on whether the arsenic compound is ingested in dissolved or undissolved form. When ingested in dissolved form, inorganic arsenic compounds are readily absorbed. Following oral exposure to arsenite or arsenate, less than 10% of the dose is recovered in the faeces both of animals and humans (Bettley & O'Shea, 1975; Charbonneau et al., 1978; Vahter & Norin, 1980; Pomroy et al., 1980). In contrast, gastrointestinal absorption of less than 30% has been reported following administration of a suspension of arsenic trioxide in gum solution to rats and rabbits (Ariyoshi & Ikeda, 1974) and no increase in urinary arsenic was found in a man who had ingested powdered arsenic selenide, which has a very low solubility in diluted hydrochloric acid (Mappes, 1977).

Following inhalation of soluble arsenic compounds, like arsenic trioxide, most of the deposited arsenic will rapidly be absorbed from either the respiratory or the gastrointestinal tract (Inamasu et al., 1982; Pershagen et al., 1982). Absorption probably also takes place through the skin (WHO, 1981).

The most common arsenic compound in the marine environment to which man is exposed is arsenobetaine, present in various seafoods (section 1.5.3). In 15 human subjects, each ingesting about 10 mg of arsenic with witch flounder (Glyptocephalus cynoglossus), less than 1% was recovered in the faeces within

8 days, indicating a high absorption (Tam et al., 1982). Similarly, a high degree of absorption has been observed in mice and rats following oral administration of ^{73}As-arsenobetaine or ^{73}As-arsenocholine (Vahter et al., 1983; Marafante et al., 1984). Studies on mice and rats have indicated that about 70 to 80% of dimethylarsinic acid is absorbed in the gastrointestinal tract (Stevens et al., 1977; Siewicki & Sydlowski, 1981; Vahter et al., 1984). In contrast, arsinilic acid and related arsenic compounds, used as feed additives for poultry and swine, seem to be considerably less available following ingestion, as based on reported faecal elimination of about 70% of an oral dose (Calesnick et al., 1966; Calvert, 1975).

3.2.2 Biotransformation

The tissue distribution, retention and toxicity of the various arsenic compounds are greatly influenced by their in vivo biotransformation. Like bacteria and moulds, mammals are able to methylate inorganic arsenic. In man, when not excessively exposed to inorganic arsenic, urinary excretion consists of 10 to 20% inorganic arsenic, 10 to 15% MMA and 60 to 80% DMA (Crecelius, 1977b; Smith et al., 1977; Buchet et al., 1980a, b; Yamauchi & Yamamura, 1979; Tam et al., 1979). Following exposure to relatively low doses of arsenite or arsenate, 35 to 40% of the dose is methylated and excreted in the urine as MMA and DMA within 4 to 5 days (Tam et al., 1979; Buchet et al., 1981a). In mice, rats, hamsters, rabbits and dogs exposed to arsenite or arsenate, DMA is the main metabolite in the urine and at the most a few per cent of the urinary arsenic is in the form of MMA (Charbonneau et al., 1979; 1980; Vahter, 1981; Marafante et al., 1982). There are also some indications that a further methylation to trimethylarsine oxide occurs in rodents, although in very small amounts (Odanaka et al., 1983; Yamauchi & Yamamura, 1985). The methylation efficiency varies considerably between the species and seems to be the highest in mice and dogs, in which about 80% of the absorbed inorganic arsenic is methylated and excreted in the form of DMA within 48 hours (Charbonneau et al., 1979; Vahter, 1981). The marmoset monkey is the only species which so far has been found unable to methylate arsenic (Vahter et al., 1982; Vahter & Marafante, 1985). In both man and rodents, the methylation efficiency decreases with increasing dose levels (Vahter, 1981; Mahieu et al., 1981) and studies on rat liver in vitro have shown that the formation of DMA, but not that of MMA, is inhibited by trivalent arsenic (Buchet & Lauwerys, 1985). Arsenite is methylated to a higher degree than arsenate (Vahter, 1981; Vahter & Marafante, 1983), which partly is due to the fact that arsenate is reduced to arsenite before being methylated (Wood et al., 1978; Vahter & Marafante, 1985). The site and mechanism for the methylation in mammals is not yet fully elucidated but it seems likely that the liver is one major site of methylation (Lerman & Clarkson, (1983; Lerman et al., 1983; Marafante & Vahter, 1984; Buchet & Lauwerys, 1985). This would explain the findings that the methylation is higher following oral than parenteral administration (Vahter, 1981; Buchet et al., 1982). MMA and DMA have a lower affinity for tissue constituents than inorganic arsenic, especially the trivalent form, resulting in an increase of the urinary excretion (Buchet et al., 1981a, Vahter & Marafante, 1983). Transformation of DMA to cacodyl (dimethylarsine), cacodyl oxide and inorganic arsenic in man treated with drugs containing cacodylate has been suggested (Goodman and Gilman, 1955; Wade & Reynolds, 1977), but in experimental studies where DMA has been administered in low doses to man (Buchet et al., 1981a), mice or rabbits (Vahter & Marafante, 1983; Vahter et al., 1984) DMA has been found to be excreted essentially unchanged in the urine. MMA is partially methylated to DMA in vivo (Odanaka et al., 1978; Buchet et al., 1981a).

Another important biotransformation reaction involving inorganic arsenic is the reduction of arsenate to arsenite, which has been demonstrated in mice, rabbits and marmoset monkeys (Vahter & Envall, 1983; Vahter & Marafante, 1985). It can be estimated that more than 50%, maybe as much as 80%, of the administered arsenate is initially reduced to arsenite (Vahter, 1983; Vahter & Marafante, 1985). As in the case of exposure to arsenite, the biosynthesized arsenite is partly methylated. Following administration of arsenite, some oxidation to arsenate occurs in addition to the methylation.

The changes of valence state of inorganic arsenic is important from the toxicological point of view since arsenite has a higher toxicity than arsenate (section 3.3.1.). The methylation can be considered as a detoxification of inorganic arsenic, since MMA and DMA also have a lower acute toxicity than inorganic arsenic (Fairchild et al., 1977; NAS, 1977).

Arsenobetaine is excreted in the urine of mammals without biotransformation (Vahter et al., 1983). Arsenocholine is to a great extent oxidized to arsenobetaine in mammals, probably by a mechanism similar to that of choline (Mann et al., 1938; Marafante et al., 1984). In mice, rats and rabbits, 55 to 70% of the administered arsenocholine was oxidized and excreted in the urine as arsenobetaine within 3 days (Marafante et al., 1984). Unmetabolized arsenocholine was found in the urine on the first day only (about 10% of the dose). The mammalian metabolism of various arsenosugars, the main arsenic compounds in certain brown kelp, is not known.

3.2.3 Tissue distribution

The concentration of arsenic in tissues (autopsy samples) of human subjects exposed to normal environmental levels are shown in Table 7 (Liebscher & Smith, 1968). Hair and nails show the highest arsenic concentrations, while fairly high concentrations are found in skin and lungs. Since the data in the table represent total arsenic concentrations, the accumulation form of arsenic in the various tissues is not known. However, it seems likely that the high levels in hair, nails and skin are due to the exposure to inorganic arsenic. Experimental studies on mammals exposed to inorganic arsenic show that the tissues with the longest retention of arsenic are skin, hair, squamous epithelium of the upper gastrointestinal tract (oral cavity, tongue and oesophagus), stomach wall, intestinal walls, epidermis, thyroid, skeleton and lens (Du Pont et al., 1941; Deak et al., 1976; Lindgren et al., 1982). The half-time in skin seems to be more than 1 month (DuPont et al., 1941). All the tissues mentioned, except the skeleton, have higher concentrations following administration of arsenite than that of arsenate (Lindgren et al., 1982). For the skin, hair, upper gastrointestinal tract and probably also the intestinal walls, this can be explained by the binding of arsenite, possibly after first being reduced to arsonous acid (Knowles, 1982), to SH-groups of keratin (Atalla et al., 1965; Webb, 1966) which is present in high amounts in these tissues (Sun et al., 1979). The relatively high concentrations in these tissues after exposure to arsenate are probably a result of the reduction of arsenate to arsenite *in vivo*. The accumulation in the thyroid and lens may be due to retention of the DMA-metabolite (Vahter et al., 1984). High arsenic levels in the human lungs may be caused by long-term retention of inhaled inorganic arsenic compounds (Brune et al., 1980). It may be that the uptake of arsenate in the skeleton occurs by the same mechanism as for phosphate, due to the chemical similarities between the two compounds. Animal experiments have shown that 2,3-dimercapto-1-propanol (BAL), which traditionally has been

Table 7. Concentration of arsenic in human subjects (healthy adults who died as a result of violence), mg As/kg tissue, dry weight
From Liebscher & Smith (1968)

Tissue	No. of samples	Maximum	Minimum	Median	Geometric Mean
Adrenal	22	0.293	0.002	0.029	0.029
Aorta	29	0.570	0.003	0.031	0.035
Blood (whole)	12	0.920	0.001	0.038	0.036
Bone	20	0.240	0.010	0.057	0.053
Brain	19	0.036	0.001	0.013	0.012
Breast	3	0.221	0.030	-	-
Hair	1250	8.17	0.020	0.460	0.460
Heart	23	0.078	0.002	0.024	0.021
Kidney	25	0.363	0.002	0.033	0.026
Liver	27	0.246	0.005	0.028	0.034
Lung	56	0.514	0.006	0.082	0.078
Muscle (pectoral)	24	0.431	0.012	0.063	0.062
Nail	124	2.90	0.020	0.300	0.283
Ovary	13	0.260	0.013	0.037	0.048
Pancreas	30	0.410	0.005	0.045	0.047
Prostate	10	0.090	0.010	0.046	0.039
Skin	76	0.590	0.009	0.090	0.080
Spleen	23	0.132	0.001	0.020	0.017
Stomach	21	0.104	0.003	0.037	0.022
Teeth	75	0.635	0.003	0.050	0.049
Thymus	11	0.332	0.003	0.015	0.019
Thyroid	22	0.314	0.001	0.042	0.042
Uterus	23	0.188	0.010	0.031	0.037

Administration of ^{73}As-arsenocholine to mice, rats and rabbits caused higher tissue concentrations and longer retention of ^{73}As than did administration of ^{73}As-arsenobetaine (Marafante et al., 1984), probably due to the incorporation of arsenocholine in phospholipids in the same way as the endogenous choline (Mann et al., 1938). Accumulation of ^{73}As was found in muscles and several parenchymatous and endocrine organs, i.e. epididymis, semen ducts, testes, prostate, parathyroid, pancreas, adrenal cortex, liver, lungs, salivary glands and thymus. Rabbits had on average five times higher and rats three times higher tissue concentrations than mice.

3.2.4 Excretion and biological half-time

The major route of excretion of most arsenic compounds is via the kidneys. Significant biliary excretion has been reported only for rats exposed to arsenite (Klaassen, 1974). The faecal excretion of arsenic after parenteral exposure to inorganic arsenic, arsenobetaine or arsenocholine is at the most about 5% of the dose. Loss of arsenic through desquamation of skin, hair growth and sweating are excretory routes of minor importance (WHO, 1981).

used in the treatment of arsenic poisoning, increases the arsenic content in the brain following exposure to arsenite (Aposhian et al, 1984). Other metal complexing agents, like dimercaptosuccinic acid (DMSA) and 2,3-dimercapto-1-propanesulfonic acid (DMPS), which both showed a higher protective effect against arsenic toxicity than BAL, did not give rise to increased concentrations in the brain.

There are major species differences in the metabolism of arsenic compounds (see e.g. Vahter, 1983). Specific binding of arsenic to rat haemoglobin, for example, results in accumulation in the blood of a great part of an administered dose of inorganic arsenic (Lanz et al, 1950; Rowland & Davies, 1982) or DMA (Stevens et al., 1977; Siewicki, 1981). Similarly, specific binding of inorganic arsenic in the liver occurs in the marmoset monkey (Vahter et al., 1982).

There are only a few reports available on the accumulation of arsenic in tissues after prolonged exposure. In experimental animals continuously exposed to arsenic via drinking-water or inhalation, the tissue levels increased for about 2 weeks, whereafter they decreased in spite of the ongoing exposure (Katsura, 1958; Bencko et al., 1968; Bencko & Symon, 1969, 1970). Furthermore, mice given arsenite via drinking-water for 4 days had significantly higher ^{74}As-levels in liver and kidney after a single parenteral administration of ^{74}As-arsenate than control mice or mice given the arsenite-rich water for 64 days (Bencko et al., 1973). The mechanism for these observations is not known but may represent a facet of the adaptation or tolerance which has been reported for arsenic in relation to medication (Goodman & Gilman, 1955) or among the so-called "arsenic eaters" in Styria (Steiremark), Austria (Lewin, 1929).

Data on the tissue retention of organic arsenic compounds ingested with seafoods are limited. In mice and rabbits the tissues with longest retention of ^{73}As-arsenobetaine were cartilage (especially in mice), epididymis, testes, semen ducts, thymus and muscles (rabbits) (Vahter et al., 1983). Siewicki (1981) administered a flounder diet containing 29 mg As/kg to rats for 42 days. Arsenic levels in the liver and spleen of treated rats were sevenfold and two-fold higher, respectively, than in control rats.

The rate of excretion of arsenic in the urine varies with the form of arsenic absorbed and the animal species exposed. Arsenate is rapidly cleared from blood and most tissues and the urinary excretion is somewhat faster after exposure to arsenate than to arsenite, in spite of the more efficient methylation of arsenite than of arsenate (Vahter & Marafante, 1983). In certain species there are specific sites of binding of arsenic, e.g. the red blood cells in the rat and the liver of the marmoset monkey, which give rise to long biological half-times. In other animal species the variation in the rate of excretion is greatly influenced by variations in methylation efficiency. Thus in mice, which have a very efficient methylation of inorganic arsenic, more than 95% of the dose of arsenite or arsenate is eliminated with half-time of about 4 hours. Almost 2% is excreted with a half-time of 2 days and less than 1% with a half-time of 15 days (Vahter, 1983). A similar rate of clearance of inorganic arsenic has been reported for dogs (Charbonneau et al., 1979). The excretion of arsenic in man is slower than in most animal species. In six human subjects who ingested ^{74}As-arsenate (0.01 µg As/man), 38% of the dose was excreted in the urine within 48 hours

and 58% within 5 days (30% DMA, 12% MMA and 16% inorganic arsenic) (Tam et al., 1979). Pomroy et al. (1980) found that the whole-body clearance of arsenic followed a three component exponential function and 66% was eliminated with a half-time of 2.1 days, 30% with a half-time of 9.5 days and 4% with a half-time of 38.4 days. In three subjects, each of whom ingested 500 µg arsenic in the form of arsenite via drinking-water, about 33% of the dose was excreted in the urine within 48 hours (Buchet et al., 1981a) and 45% within 4 days (24% DMA, 10% MMA and 11% inorganic arsenic). Following ingestion of the same amounts of arsenic in the form of MMA or DMA, 75 and 78% of the dose, respectively, were excreted in the urine within 4 days.

In human subjects ingesting fish with high arsenic content, about 70% of the ingested arsenic is excreted in the urine within 3 days; total range 52 to 85%, n = 24 (Westöö & Rydälv, 1972; Freeman et al., 1979; Tam et al., 1982). About 76% (range 61 to 86%) of the arsenic ingested with witch flounder (10 mg As) was excreted in the urine within 8 days (Tam et al., 1982). Less than 1% was recovered in the feces. A similar rate of excretion of arsenic in the urine has been reported for cynomologus monkeys given a single test meal of arsenic-containing fish (63% of the ingested arsenic was excreted within 3 days and 67% within 14 days; Charbonneau et al., 1978), as well as for rabbits given a single oral dose of ^{73}As-labelled arsenobetaine (75% of the dose was excreted in the urine within 3 days; Vahter et al., 1983). The urinary excretion of ^{73}As-arsenobetaine in mice and rats was found to be essentially complete within 3 days (Vahter et al., 1983). Following administration of ^{73}As-arsenocholine, almost 80% of the dose was excreted in the urine in mice and rats and 66% in rabbits within 3 days (Marafante et al., 1984).

3.2.5 Indicators of exposure

The major part of both inorganic and organic arsenic in blood is cleared fairly rapidly in man, which means that arsenic in blood will reflect exposure for only a short period following absorption. Only if exposure is continuous and steady, as is sometimes the case with exposure through drinking-water, will arsenic reach a steady-state in the blood and, thus, make it possible to arrive at a relationship between blood arsenic levels and exposure to inorganic arsenic (Cebrian et al., 1985). However, the possibility of interferences from "seafood arsenic" has to be considered.

Arsenic is normally found in higher concentrations in human hair and nails than in other parts of the body. This has been explained by the high content in these tissues of keratin, the SH-groups of which may bind trivalent inorganic arsenic (Shapiro, 1967; Hopps, 1977). The methylated metabolites of inorganic arsenic and the seafood arsenic are not accumulated in hair (Vahter et al., 1983, 1984). Normally the concentrations of arsenic in hair and nails are less than 1 mg As/kg (Smith, 1964; Liebscher & Smith, 1968; Hopps, 1977). In subjects exposed to arsenic occupationally or via arsenic-containing medicine or drinking-water hair levels may be as high as 50 mg As/kg (for review see WHO, 1981). However, arsenic in hair and nails may have other sources than the blood. The most important is probably external contamination via air, water, soaps, shampoos etc., There is at present no method available to remove arsenic from exogenous sources or to separate exogenous arsenic from endogenous arsenic. Thus, arsenic in hair may be used as an indicator of exposure to inorganic arsenic through ingestion if external contamination can be controlled for. For example, a good correlation between hair arsenic contents

and the dose of inorganic arsenic ingested has been reported by Pearson & Pounds (1971) and Curry & Pounds (1977). They also reported that arsenic concentrations along the length of the hair can serve to indicate the time of intake.

Since arsenic is excreted mainly via the kidneys, the concentration of arsenic in urine may be a suitable indicator of exposure. An estimate of the exposure to inorganic arsenic may be obtained from the urinary concentration of inorganic arsenic and its metabolites MMA and DMA. Exposure to MMA and/or DMA will influence the estimate, but seems to be very low in most countries. Exposure to "seafood arsenic" may be estimated from the total urinary concentration of arsenic minus the concentration of the metabolites of inorganic arsenic.

The concentration of inorganic arsenic and its metabolites MMA and DMA in urine may be determined by generation of the corresponding arsines through addition of sodium borohydride directly to the urine followed by detection of the arsenic using atomic absorption or emission spectrometry. This analytical procedure is not influenced by the presence of organic arsenic compounds, e.g. arsenobetaine, originating from the intake of seafood (Buchet et al., 1980a; Norin & Vahter, 1981). However, the possible degradation product trimethylarsine oxide is reduced to trimethylarsine by sodium borohydride and will thus be included in the measurements (Braman, 1983). The reported low concentrations of this arsenic compound in various seafood (see section 1.4.2) indicate that it will not influence the evaluation of exposure to inorganic arsenic to any significant extent. Separation of the different arsines produced by gas chromatography (Talmi & Bostick, 1975) or selective volatilization from a cold trap (Braman & Foreback, 1973) may reveal the presence of trimethylarsine oxide in the urine. Inorganic arsenic, MMA and DMA in urine may also be separated by ion exchange chromatography (Tam et al., 1978). Determination of arsenic in the different fractions by arsine generation/atomic absorption or emission spectrometry will prevent interferences from arsenobetaine, which if present, will be eluted together with the DMA (Norin et al., 1983). The total concentration of arsenic in the urine may be determined by e.g. atomic absorption after degradation of the organic arsenic to inorganic by dry ashing or strong wet digestion procedures (Lauwerys et al., 1979).

Reported normal concentrations of metabolites of inorganic arsenic and total arsenic in the urine are given in Table 8. The data indicate that the concentrations of metabolites of inorganic arsenic normally are less than 10 µg As/litre urine in the European countries, somewhat higher in USA and the highest in Japan. The concentration of organic arsenic compounds other than the metabolites of inorganic arsenic seems to be the highest in the Nordic countries. The concentration of these compounds will of course be highly dependent on the intake of arsenic-containing seafood and on the time of urine sampling after intake, since the clearance of arsenobetaine is very fast. Intake of a seafood meal may give rise to arsenic concentrations of more than 1000 µg As/litre within 24 hours. After 23 days, the concentrations have decreased to almost normal levels (Westoo & Rydalv, 1972; Freeman et al., 1979; Buchet et al., 1980a; Norin & Vahter, 1981; Tam et al., 1982). The average concentration of total arsenic among arsenic-exposed workers living near the Mediterranean Sea with high consumption of seafoods was found to be 286 µg As/litre, while that among Belgian workers, with relatively low intake of seafood but higher exposure to inorganic arsenic, was 81 µg As/litre (Buchet et al., 1980a).

3.3 Health effects

3.3.1 Inorganic arsenic compounds

The major symptoms following acute intoxication from inorganic arsenic are severe gastrointestinal irritation resulting in vomiting and diarrhoea, muscular cramps, facial oedema and cardiac abnormalities (WHO, 1981). Respiratory system disorders, peripheral nervous disturbances, primarily of a sensory type, and effects on the haematopoietic system such as anaemia and leukopenia, especially granulocytopenia, have also been observed following short-term exposure to inorganic arsenic (WHO, 1981). An ingested dose of 70 to 180 mg of arsenic(III)oxide has been reported to be fatal in man (Vallee et al., 1960). Animal data indicate that trivalent arsenic is more toxic than pentavalent arsenic (Byron et al., 1967; WHO, 1981).

Long-term exposure to inorganic arsenic through inhalation or ingestion may give rise to adverse health effects in a number of organs. Effects on the respiratory tract, including perforation of the nasal septum, laryngitis, pharyngitis, bronchitis and lung cancer have been encountered following occupational exposure to arsenic (IARC, 1980; WHO, 1981; Pershagen, 1982). Lung cancer has been considered the critical effect in the case of inhalation of arsenic (WHO, 1981). Effects on the peripheral nervous and vascular systems, as well as an increased mortality from cardiovascular diseases have been reported following occupational exposure to inorganic arsenic and following ingestion of drinking-water containing arsenic (Heymann et al., 1956; Hara et al., 1968; Lee & Fraumeni, 1969; Grobe, 1976; Hindmarsh et al., 1977; Axelson et al., 1978; Feldman et al., 1979; Lagerkvist et al., 1983) and effects on the haematopoietic system have been observed following long-term exposure via drinking-water, medication or occupation (WHO, 1981). Liver cirrhosis and a particular syndrome of portal hypertension without cirrhosis have also been associated with chronic arsenic exposure (Morris et al., 1974; Chainuvati & Viranuratti, 1979; Datta et al., 1979). Arsenic might also induce liver cancer (angiosarcoma) and leukemia (WHO, 1981).

A high prevalence of severe vascular disturbances, often leading to gangrene ("blackfoot disease"), has been observed in an area in Taiwan with high concentration of arsenic in the drinking-water taken from artesian wells (total range 0.01 to 1.8 mg/litre, 75% of the wells 0.05-0.75 mg/litre, valence form not known) (Tseng, 1977). The prevalence rate among subjects who had ingested about 20 g of arsenic over many years was 3%(WHO, 1981). It has been suggested that other factors, e.g. fluorescent compounds in the well water, may have been involved in the cause of "blackfoot disease" (Lu et al., 1975). However, WHO (1981) concluded that the data presented did not provide firm support for that hypothesis. In certain parts of Chile, where people have been exposed to drinking-water with about 0.6 mg As/litre for 10 to 15 years, Raynaud's syndrome (white fingers) and acrocyanosis have been common findings (30% and 22% incidence, respectively) (Borgono et al., 1977).

Hearing loss, brain wave and electroencephalographic abnormalities have been observed among Japanese youths several years after they, as infants, had been given arsenic-contaminated milk powder, corresponding to an ingestion of about 3.5 mg arsenic per day for one month (Yamashita et al., 1972; Ohira & Aoyama, 1972).

Table 8. Concentrations of metabolites of inorganic arsenic (iAs-met) and total arsenic in different countries

Country/ area	No.	iAs-met µg As/litre	Tot-As µg As/litre	Reference
Belgium	5	13±1.9[a] (-)*	15±2.1[a] (+)	Buchet et al., 1980a
Finland/ Helsinki	205	9.0±9.8[b] (+)	34±41[b] (+)	Valkonen et al., 1983
FRG/unspec.	37	5.8 (1.1-22.7)[c] (-)	17 (1.3-90)[c] (-)	Apel & Stoeppler, 1983
FRG/Juelich	19	9.5 (2.8-24)[c] (-)	17 (6.7-46)[c] (-)	-"-
Italy/Milan	160	5.9±2.9[b] (+)	17±11[b] (+)	Foa et al., 1984
Japan/unspec	20	57±21[b,d] (+?)		Yamauchi & Yamamura,(1979)
Sweden/ Stockholm	49	7.9 (2.3-53)[e] (+)	25 (6.6-363)[e] (+)	Vahter & Lind, 1986
Sweden/ Västeros	50	7.4 (1.7-40)[e] (+)	20 (4.4-210)[e] (+)	-"-
USA/unspec.	41	18±1.6[a,f] (-)	21±2.0[a,f] (-)	Smith et al., 1977
USA/unspec.	4	21±1.6[a] (-)		Braman & Foreback, 1973

a) geometric mean ± G.S.D
b) arithmetic mean ± S.D.
c) arithmetic mean (range)
d) adjusted to S.G. 1.024
e) median value (range) adjusted to S.G. 1.016
f) adjusted to S.G. 1.018. *) (-) no adequate quality control, (+) adequate quality control.

Cutaneous manifestations in the form of hyperpigmentation, often with paler spots (depigmentation), hyperkeratosis, especially of the palms and soles, and skin cancer have been associated with exposure to arsenic via drugs (trivalent arsenic) or drinking-water (valence form often unknown) (for review see WHO, 1981). Fierz (1965) reported that the prevalence of hyperkeratosis was more than 50% among patients who had received about 3 g of arsenic in medicine during 6 to 26 years of treatment. Based on the data reported by Tseng (1977) the WHO Task Group on Environmental Health Criteria for arsenic concluded that the lifetime risk for skin cancer due to arsenic in drinking-water is about 5% for a total dose of 10 g in an assumed life span of 70 years, which would correspond to a concentration in the drinking-water of 0.2 mg As/litre (WHO, 1981). The estimation was based on a 2-litre per day

intake of drinking-water, which seems low. The daily intake of arsenic needed to produce this skin cancer risk was probably higher than the assumed 400 µg. Skin lesions due to exposure to arsenic via the drinking-water (about 0.4 mg As/litre, range 0.16 to 0.59 mg As/litre, about 70% in pentavalent form) have recently been reported also from an area in Mexico (Cebrian et al., 1983). The estimated minimum dose at which the lesions were detected was 2 g for hypopigmentation, 3 g for hyperpigmentation and palmoplantar keratosis, 8 g for papular keratosis and 12 g for ulcerative lesions. The frequency of hyperpigmentation and keratosis increased with about 10% per 3 g of arsenic ingested.

Increased frequencies of chromosomal aberrations have been observed in lymphocytes of human subjects exposed to inorganic arsenic, e.g. patients on arsenic-containing drugs (Petres et al., 1977; Nordenson et al., 1979) and copper smelter workers (Nordenson et al., 1978; Nordenson & Beckman, 1982). Studies on experimental animals have shown that high doses (more than 3 mg As/kg body weight) of trivalent and pentavalent inorganic arsenic induce teratogenic effects (Léonard & Lauwerys, 1980; WHO, 1981).

3.3.2 Organic arsenic compounds

Although the toxicity of organic arsenic compounds in marine organisms has been very little studied it seems clear that they are considerably less toxic than inorganic arsenic. In an acute toxicity study on mice no deaths occurred at oral arsenobetaine doses of 10 g/kg body weight (highest dose used) (Kaise et al., 1985). Chronic effects, however, cannot be ruled out.

Coulson et al. (1935) fed rats a diet containing dried shrimps for one year. This diet with a concentration of 14 mg As/kg resulted in the total ingestion of 60 mg As. This exposure caused no difference in food intake or growth compared to control rats. The concentration of arsenic in the liver at the end of the feeding period was about 7 times higher than that in the controls, while rats fed the same amount of arsenic in the form of arsenic trioxide had 50 to 60 times higher tissue concentrations than the controls. No gross histological changes were seen in the spleens, livers or kidneys in any of the arsenic treated rats. In rats fed diets containing witch flounder (Glyptocephalus cynoglossus), giving rise to 8 to 30 mg As (probably mainly in form of arsenobetaine)/kg diet, for 42 days, the weight gain, feed intake and results of several biochemical tests were similar to those of control rats fed a diet containing winter flounder (Pseudopleuronectes americanus) with less than 5 mg As/kg (Siewicki, 1981; Siewicki & Sydlowski, 1981). Sheep fed lake weed corresponding to a daily dose of 1.4 mg As/kg body weight for 3 weeks showed no sign of intoxication and no gross morphological changes in the tissues at slaughter (Lancaster et al., 1971). The chemical form of the arsenic was not studied, but reported high arsenic concentrations in the wool indicate that at least part of it was in inorganic form.

Arsenobetaine and arsenocholine, the arsenic analogues of betaine and choline, were used during the late 1930s and early 1940s in research on the biological role of choline. Arsenocholine was found to have marked lipotropic and antihaemorrhagic effects, at least half that of normal choline (Welch, 1936; Welch & Welch, 1938; Welch, 1950). The arsenocholine was incorporated into phospholipids by the same mechanism as choline (Mann et al., 1938; Welch & Landau, 1942). Arsenobetaine, in contrast to betaine, showed no lipotropic or antihaemorrhagic effect. Rats fed a diet containing about 1% arsenocholine chloride for a period of one week showed no signs of toxic reactions (Welch & Landau, 1942).

Recently the potential of initiating and promoting effects of arsenobetaine have been studied in vitro (Jongen et al, 1985). No mutagenicity was observed in the Salmonella typhimurium assay (5 mg/plate), the sister chromatid exchange assay using V79 Chinese hamster cells (10 mg arsenobetaine/ml) or in the forward mutation assay on the HGPRT locus (10 mg arsenobetaine/ml). Furthermore, no inhibition of metabolic co-operation between V79 Chinese hamster cells was observed.

3.4 Total exposure to arsenic

3.4.1 Air

The airborne concentration of arsenic in unpolluted areas is generally less than a few nanograms per m^3, while as much as 1 µg m^{-3}, or more, has been recorded near point emissions (WHO, 1981). An air sampling network in urban areas of the U.S.A. reported arsenic concentrations of 10 to 750 ng m^{-3} for 133 stations during 1964 (Sullivan, 1969). 80% of the reported yearly average concentrations were 10 ng m^{-3} or less. Lower values (0.3 to 6 ng m-3) are reported for rural areas of the U.S.A., England and Canada (NAS, 1977; NRCC, 1978). The inhaled amount of arsenic is thus normally less than 0.1 µg As per day in urban areas but may reach as much as 20 µg As near point emissions.

3.4.2 Drinking water and beverages

The concentration of arsenic in drinking-water depends largely on the type of water, the arsenic content of the ground near the water source and the treatment of the water before use. In an extensive US survey, less than 1% out of 18,000 water supplies had arsenic concentrations exceeding 0.01 g/litre (McCabe et al, 1970), while in a gold mining area in Nova Scotia, Canada, 13% out of 800 wells had water containing 0.05 mg/litre or more (Grantham & Jones, 1977). Arsenic levels in the range 0.14 mg/litre due to arsenic-containing ground have also been reported from Taiwan (Tseng, 1977), Argentina (Arguello et al, 1938) and the north of Mexico (Cebrian et al., 1983). Excluding areas such as these, where the daily intake of arsenic through drinking-water may reach several hundred µg As, drinking-water normally contributes less than 10 µg As/day.

Elevated arsenic levels, 0.02 to 0.11 mg As/litre, in wine have been reported from U.S.A. (Noble et al., 1976). In another study of 19 different wines, the range of As(III) was 0.001 to 0.42 mg/litre and that of As(V) 0.001 to 0.11 mg/litre (Crecelius, 1977a).

3.4.3 Food

Arsenic levels in human food products, with the exception of seafood, are generally low. Elevated arsenic levels may be caused by industrial pollution, use of arsenic-containing pesticides or use of feed additives such as arsanilic acid and related compounds (substituted phenylarsonic acids) for poultry and swine. Tolerance levels for residues of such feed additives have been established in the U.S.A.: 0.5 mg As/kg for eggs, meat of chickens, turkeys and swine and 2 mg As/kg for edible by-products of chickens and turkeys and the livers and kidneys of swine (Jelinek & Corneliussen, 1977). Jelinek & Corneliussen (1977) reported a decrease in arsenic concentrations in most food products in the U.S.A. between 1967 and 1974, probably due to the decreased

use of arsenic-containing pesticides. Various foods within the categories meat, egg, milk, fruit, vegetable, cereal and sugar products, analysed by the U.S. Food and Drug Administration 1974/75, had average levels of less than 0.1 mg As/kg, with the exception of rice, which had slightly more. Analysis (neutron activation) of foods in the U.K. found average arsenic concentrations in cereals, fats, fruits, preserves, root vegetables, milk, meat and fish ranging from 0.05 to 2 mg As/kg (Hamilton & Minski, 1973). In an extensive survey of arsenic concentrations (neutron activation analysis) in food products in Sweden, Westöö & Rydälv (1972) reported levels of 0.05 mg As/kg in eggs, 0.05-0.25 mg As/kg in fresh fruit (including imports), 0.05 to 5 mg As/kg in dried fruit, and 0.05 to 1 mg As/kg in meat products (including poultry and liver and kidney), except chicken meat, all samples of which contained 0.265 mg As/kg. With the exception of chicken meat, most of the samples contained less than 0.25 mg As/kg.

As can be seen in Table 3 marine fish from the North Sea near the Swedish west coast have higher levels than fish from the brackish water of the Baltic, e.g. 1.5 mg/kg and 0.65 mg/kg for cod from the North Sea and the Baltic, respectively, and that pike from the Baltic (mean 1.1 mg/kg) have higher arsenic concentrations than pike from freshwater lakes (mean 0.08 mg/kg) (Westöö & Rydälv, 1972). From Tables 2, 4 and 5 giving examples of arsenic levels in seafoods from different countries, it is evident that there is a great variation between species and locations. In general, the highest concentrations are found in bottom-feeding fish and crustacea. In Table 2 (Flanjak, 1982), the arsenic concentrations are divided into inorganic and organic arsenic. The ratio of inorganic:organic arsenic was less than 0.05 in 97% of the samples (overall mean 0.02). A similar content of inorganic arsenic, about 2% of the total arsenic (range 0.1 to 5.3), has been reported for fish and shellfish in the U.K. (Brooke & Evans, 1981), while Lunde (1977) reported that about 10% of the total arsenic in Norwegian fish and crustacea was in the form of inorganic arsenic. For further details concerning total concentrations and chemical forms of arsenic in marine organisms, see Section 1.

There are very few data on the chemical form of arsenic in food products other than fish and crustacea. In some areas like eastern Asia it is common to eat certain types of seaweed, e.g. Hizikia fusiforme, which has been shown to contain high concentrations of arsenate and arsenite (see section 1.5.3). Pyles & Woolson (1982) reported on the presence of different forms of organic arsenic, methanol extractable as well as chloroform extractable compounds, in vegetables grown in arsenate-treated soil. Thus it cannot be assumed that the arsenic in terrestrial food products is in the form of inorganic arsenic.

3.4.4 Total daily intake

To enable an evaluation of possible health effects due to exposure to arsenic, the chemical forms of arsenic have to be considered. So far, data on the daily intake have been reported for total arsenic only. The average total daily intake of arsenic in Canada was reported to be 0.025 to 0.035 mg As in 1970-73 (Smith et al., 1972, 1973, 1975). The daily intake of arsenic in Belgium has been reported to be 0.012 mg As per day (Buchet et al., 1983), while that in the UK was about 0.1 mg As per day, most of which came from fish (Hamilton & Minsky, 1973; MAFF, 1982). The US Food and Drug Administration has calculated that the average daily total intake of arsenic has decreased from about 0.04 mg As in the late 1960s to less than 0.01 mg As in 1972-74 (Jelinek & Corneliussen, 1977). The daily intake of arsenic in Japan is re-

portedly 0.07 to 0.17 mg (Nakao, 1960), which probably is due to the higher average consumption of seafood in Japan than in most other countries, and the consumption of seaweed.

The WHO Task Group on Environmental Health Criteria for arsenic (WHO 1981) estimated that the daily intake of inorganic arsenic normally is less than 50 µg. Assuming that 60-70% of the daily ingested inorganic arsenic is excreted in the urine (Mappes, 1977; Buchet et al., 1981b), the data on the urinary excretion of the metabolites of inorganic arsenic given in Table 8 indicate that the average daily intake of inorganic arsenic in the general population is 0.01 to 0.05 mg As in Europe and USA and more than 0.10 mg As in Japan. Normally, the contribution from drinking-water and air is less than 0.01 mg As per day.

3.5 Contribution of arsenic from marine food

The contribution of inorganic arsenic from seafood to the total daily intake may occasionally be significant. With the assumption that 5-10% of the total arsenic in seafood is in the form of inorganic arsenic (Section 1.5.3), the world average daily intake of seafood (see Introduction, section 2.2 on seafood consumption patterns) would correspond to ingestion of 2-10 µg As inorganic arsenic. Table 9 shows the calculated daily intake of inorganic arsenic at various degree of seafood consumption and concentrations of arsenic. It can be seen that daily consumption of seafood (150 g per day) would give rise to an intake of about 15-75 µg inorganic arsenic per day, the higher figure relating to a daily intake of flatfish, crustacea and molluscs. Extremely high consumption of seafood (1 kg per day) would give rise to an intake of 100-500 µg inorganic arsenic per day. The daily intake of seaweed (Hizikia and others) in Japan is about 5 g (Watanabe et al, 1979). With an assumed average arsenic concentration in Hizikia of about 100 mg As/kg dry weight, or about 20 mg As/kg wet weight, this means that the daily intake of arsenic from seaweed might exceed 100 µg, out of which 60-80% probably is in form of inorganic arsenic (section 1.5.3).

Based on reported concentrations of total arsenic in various types of seafood (section 3.4.3) and the seafood consumption data (see Introduction, section 2.2 on seafood consumption patterns) it can be estimated that the world average daily intake of organic arsenic from seafood (most of which is arsenobetaine) is 15-20 µg As. There are great variations between countries, but in most it is less than 50 µg As. As can be seen from Table 9, daily consumption of seafood would give rise to an intake of about 135-1,500 µg organic arsenic, while extremely high consumption of flatfish or crustacea may result in the ingestion of 10,000 µg As per day. In a study performed in Sweden on the urinary concentrations of arsenic it was observed that subjects who ate flatfish or crustacea more than once a week had on average about 3 times as high concentrations of organic arsenic compounds (other than metabolites of inorganic arsenic) in the urine than subjects who seldom ate such food (Vahter & Lind, 1986).

Table 9. Total daily intake of inorganic and organic arsenic by consumers of various amounts and types of seafood (µg As/day)

Mean concentration in seafood (µg As/g)	Daily seafood consumption (g)							
	20		60		150		1000	
	Inorg.	Org.	Inorg.	Org.	Inorg.	Org.	Inorg.	Org.
1.0*	2	18	6	54	15	135	100	900
10**	10	190	30	570	75	1425	500	9,500

*) Normal concentration in most commercially important fish species. 10% is assumed to be inorganic arsenic.

**) Concentration likely to be found in bottom feeding fish (e.g. flounder, sole) and in various crustacea, 5% is assumed to be inorganic arsenic.

3.6 Evaluation of potential health effects

From a toxicological point of view there are basically two forms of arsenic in marine organisms to be considered; arsenobetaine, which is the dominant form in most seafood, and inorganic arsenic, constituting 2-10% of the total arsenic content. Inorganic arsenic is by far the most toxic form and has in several studies been shown to cause skin lesions, such as hyperkeratosis, hyperpigmentation and skin cancer, peripheral vascular disturbances, effects on the nervous system, chromosome damage, interference with immune defence and lung cancer (after inhalation of inorganic arsenic compounds). The organic arsenic compounds present in seafood have so far not been shown to cause adverse health effects in man.

Evaluation of human health effects due to consumption of inorganic arsenic has been presented by WHO (1981). It was concluded that intakes of 3 mg As/day over a few weeks may cause severe poisoning in infants and toxic symptoms in adults, and that 1 mg As/day over several years may result in skin effects in the form of hyperkeratosis, hyperpigmentaton and hypopigmentation. An estimated total intake of 10 g arsenic over a lifetime from drinking-water was related to a 5% prevalence of skin cancer (WHO, 1981). This would correspond to a daily intake of 0.4 mg As over a lifetime (70 years), or a daily intake of 1 mg As for about 25 years. The range between intakes causing subacute effects and those causing chronic effects after longterm exposure is apparently very narrow. The WHO guideline for arsenic in drinking-water has been set to 0.05 mg As/litre (WHO, 1984), which may correspond to a daily intake of 0.1-0.2 mg As.

As has been discussed in section 2.2 of the Introduction (seafood consumption patterns), the world average intake of seafood is about 20 g, while the 97.5th percentile consume at least three times the average or about 60 g per day. The world average intake of inorganic arsenic from seafood would be in the range 2-10 µg As, depending on the type of seafood consumed. It can be seen from Table 9 that a daily consumption of about 60 g seafood would correspond to a daily intake of 6-30 µg inorganic arsenic, which is less than a tenth of the amount which would give rise to a 5% increased risk for skin cancer after a lifetime. It is also less than the daily intake from drinking-water containing inorganic arsenic corresponding to the WHO guideline. However, it may contribute significantly to the total daily intake of inorganic arsenic. In case of extreme consumption of seafood, e.g. 1 kg flatfish, crustacea or molluscs per day over a lifetime, the intake of inorganic arsenic would reach the levels when a 5% increased risk for skin cancer may occur. In these calculation it has been assumed that the inorganic arsenic in seafood has the same bioavailability and toxicity as that in drinking-water.

There are no data concerning the toxicity of the organic arsenic compounds present in seafoods for man. However, it seems obvious that the toxicity, at least of the major compound, arsenobetaine, is much lower than that for inorganic arsenic. Otherwise acute effects ought to have been observed in people with high seafood consumption. The few studies performed on experimental animals show that arsenic of marine origin gives rise to much lower tissue levels than does inorganic arsenic, and has a lower acute toxicity than inorganic arsenic. Considering the high concentrations in food products eaten over a lifetime by a large number of people and the accumulation in certain tissues, e.g. in epididymis, testes and thymus, the accumulation and possible adverse effects in man following long-term exposure need to be investigated. There is also a need for data on the placental transfer. It is also important to identify the other organic compounds, water soluble as well as fat soluble, which, although present at low concentrations in the seafood, may be of greater toxicological significance than the arsenobetaine.

4. CONCLUSIONS ON ARSENIC

4.1 Potential harm to living resources

Arsenic enters the marine environment from both natural and anthropogenic sources. It has been calculated that anthropogenic contributions of arsenic to the environment exceed those resulting from natural processes. Difficulties in determining the natural contribution make this comparison uncertain.

There is a primary cycle of arsenic in marine waters involving the largely biologically mediated reduction, methylation, demethylation and oxidation of simple arsenic species, with arsenate always the dominant form in toxic waters. The biological processes are carried out by micro-organisms and by algae. Some of the arsenate absorbed by algae is converted into organic arsenic compounds, such as arsenic-containing ribofuranosides, directed into foodwebs and gives rise mainly to arsenobetaine.

Arsenic is present in oceanic waters at concentrations of 1 to 3 µg/litre and the accumulation of arsenate from seawater by algae represents a magnification in concentration of 2,000 to 5,000 times. Arsenic is

not further significantly biomagnified in the food chain. Arsenobetaine wet weight concentrations of 20 mg As/kg in crustaceans, 2 mg As/kg in bony fish and 15 mg As/kg in sharks and rays are normal in organisms from unpolluted waters.

There are few data on the effects of arsenic on marine organisms but these suggest that marine animals, even during larval stages, are not acutely affected by arsenic concentrations below 200 µg/litre. However, it has been reported that the growth of some marine phytoplankton may be inhibited at arsenate concentrations only 3 to 4 times the normally found total arsenic concentration in sea water. Long-term sublethal effects of elevated arsenic levels on marine animals have not been studied.

4.2 Potential hazards to human health

People are exposed to arsenic via air, drinking-water, beverages and food. Exposure occurs to different inorganic and organic arsenic compounds, but data on the average daily intake of arsenic has so far been reported for total arsenic only. Intakes of 0.01 to 0.1 mg As/day have been reported for Canada, a few countries in Europe and the USA, and 0.07 to 0.17 mg for Japan. Arsenic intake by air is mainly in inorganic forms and less than 0.1 µg As/day in urban areas, but can occasionally, near point emissions, reach 20 µg As/day. Arsenic in drinking-water is in inorganic forms and normally contributes less than 10 µg As/day. However, in areas with arsenic-containing groundwater the contribution can reach several mg As/day. Most food products contain less than 0.25 mg As/kg, while seafood commonly contains arsenic in the order of milligrams per kg. Seafood is the dominating source of human arsenic intake. However, most of it (90%) is in organic form, mainly arsenobetaine.

It can be calculated that the average daily intake of organic arsenic from seafood is 20-200 µg As with one meal per week, but may reach 10,000 µg As in case of extreme seafood consumption, such as 1 kg of flatfish, crustacea or molluscs per day. The daily intake of inorganic arsenic from seafood may reach from less than 10 µg As to more than 500 µg As in case of very extreme seafood consumption. In some areas of the World, notably eastern Asia, the consumption of seaweed (e.g. Hizikia fusiforme) may be a source of dietary intake of inorganic arsenic and organic compounds of unknown toxicity (e.g. arsenic-containing ribofuranosides).

Inorganic arsenic compounds may be readily absorbed in the gastrointestinal tract; absorption is dependent on water solubility. Absorbed arsenic is retained in epithelial tissue like skin, hair, mucus membranes of the gastrointestinal tract, epididymis, lens and thyroid and in the skeleton, with the highest concentrations seen in hair, nails, skin and skeleton. The distribution of arsenic after exposure to arsenate differs very little from that after exposure to arsenite. In the body, arsenate is to a great extent converted to arsenite, which is partly methylated to methylarsonic acid and dimethylarsinic acid. Methylarsonic acid and dimethylarsinic acid are excreted more rapidly than unmethylated salts and are therefore considered to be detoxification products. Following exposure to arsenite a small fraction is oxidized to arsenite in the body. After ingestion of arsenate about 40% is excreted in two days; after ingestion of arsenite about 33% is excreted within the same time.

Ingested arsenobetaine is absorbed to more than 90% and excreted in the urine in unchanged form. Retention of arsenobetaine in several organs, including male reproductive organs, has been demonstrated in animal experiments. Around 70% of ingested dose is excreted within three days by man, while 20% is still retained after 8 days.

Inorganic arsenic compounds are toxic to man. Trivalent arsenic compounds are more toxic than pentavalent compounds (lowest reported lethal dose 70 to 180 mg arsenic(III)oxide ingested). Exposure to inorganic arsenic compounds by ingestion may cause skin lesions such as palmar and plantar hyperkeratosis, hyperpigmentation and skin cancer, as well as peripheral vascular disturbances. Other effects which may occur are disturbances of the nervous system, chromosome damage, interference with immune defence and bone marrow functions and lung cancer (after inhalation of inorganic arsenic compounds). The WHO Task Group on Environmental Health Criteria for Arsenic (WHO, 1981) concluded that intakes of 3 mg As/day over a few weeks may cause severe poisoning in infants and toxic symptoms in adults, and that 1 mg As/day over several years may result in skin effects. An estimated total intake of 10 g inorganic arsenic over a lifetime, assumed to correspond to a daily intake of 0.4 mg As for a lifetime or 1 mg As for 25 years, was related to a 5% prevalence of skin cancer. The range between intakes causing subacute effects and those causing chronic effects after longterm exposure is apparently very narrow.

No toxic or adverse effects have been seen in experimental animals given seafood diets with moderate levels of arsenic (14 mg As/kg diet for one year or 30 mg As/kg for 42 days). The consequences of life-long high intake of arsenobetaine and other organic arsenicals occurring in marine organisms are unknown.

It can be concluded that seafood dominates as the source of human arsenic intake but inadequate data on the toxicology of arsenobetaine, the major arsenic compound in seafood, and the other organic arsenicals occurring in low concentrations, do not allow a reasonably accurate assessment of the possible health risks to man due to long-term intake of large amounts of seafood with high arsenic content. Considering the high concentrations in food products eaten over a lifetime by a large number of people and the accumulation of arsenobetaine in certain tissues, e.g. the male reproductive organs, possible adverse effects in humans following long-term exposure need to be investigated.

To make possible a meaningful evaluation of the arsenic exposure from various types of seafood, both the inorganic and the organic arsenic components have to be analyzed. Present analytical data indicate that a few per cent of arsenic in fish may be in inorganic form. This means that seafood consumption occasionally can contribute considerably to the daily intake of inorganic arsenic. Extreme seafood consumption, e.g. 1 kg of flatfish, crustacea or molluscs per day, may give rise to ingestion of as much as 500 µg inorganic arsenic per day. This exposure level over a life-time has been related to a 5% prevalence of skin cancer.

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III. MERCURY

1. MERCURY IN THE MARINE ENVIRONMENT

1.1 Reference documentation

A large number of studies on the distribution and transformation of Hg in the environment have been published. Some of the available reports are referred to below. This review is selective and presents detailed original data only to exemplify the issue being discussed. More comprehensive reviews of the environmental behaviour of Hg include those by Friberg & Vostal (1972), National Academy of Sciences (1978), National Research Council Canada (1979). Nriagu (1979), and Lindqvist et al. (1984).

1.2 General facts

Mercury (Hg), atomic weight 200.61, belongs to the group IIB of the periodic system together with zinc and cadmium. Air in equilibrium with metallic Hg contains 5.5 mg Hg/m^3 at 10 °C and 13.2 mg Hg/m^3 at 20 °C. Such high levels are never found in the ambient atmosphere (Matheson, 1979). Under ion equilibrium conditions the air over inorganic Hg salts can reach considerable concentrations. At equilibrium HgS reaches 100 ng Hg/m^3 in dry air and 5,000 ng Hg/m^3 in water-saturated air. Over HgO dry air contains 2,000 ng Hg/m^3, and over diluted MeHgCl solutions (0.04 to 0.08 %) the air concentrations range from 140 to 900 µg Hg/m^3 (Matheson, 1979).

Knowledge of the chemical forms of inorganic Hg in natural waters is largely based on thermodynamic calculations which predict that in practical terms Hg(I) does not exist. Hg (II) sulphide, cinnabar, has a very low solubility (solubility product: 10^{-53} M). Hg (II) forms covalent bonds and is strongly coordinated with -SH ligands of biological molecules, specially proteins.

The emphasis on methylmercury (MeHg) in the biogeochemical cycle has most probably distracted attention from the fact that dissolved MeHg is not the dominant form in natural waters. CH_3Hg^+ occurs in aqueous solutions as an aquo complex CH_3HgOH^{2+}. The cation behaves as a weak acid and has a strong preference for the addition of only one ligand. CH_3Hg^+ undergoes rapid coordination reactions with S, P, O, N, halogens, and C.

The CH_3Hg^+ unit itself is kinetically remarkably inert toward decomposition. Therefore, MeHg compounds once formed are not readily demethylated. The neutral species formed with CH_3Hg^+ are hydrophilic, and lipophilic; thus they can readily pass through boundaries.

Reactions between Hg and selenium are of great importance for the toxicity of Hg compounds (see chapter on selenium).

1.3 Sources

The major natural sources of atmospheric Hg are land and ocean degassing. Although a precise quantification is difficult, the following global values have been suggested by Matheson (1979): land degassing 17,800 t/year, open ocean degassing 7,600 t/year, coastal water degassing 1,400 t/year and volcanic activity 20 t/year. This estimate of emissions totals 26,820 t/year,

which is higher than the 18,500 t/year quoted by Miller & Buchanan (1979). There is obviously considerable uncertainty attached to these estimates, as they do not account completely for recycling and they are extrapolations from local measurements to the global totals. Last but not least there are great analytical uncertainties.

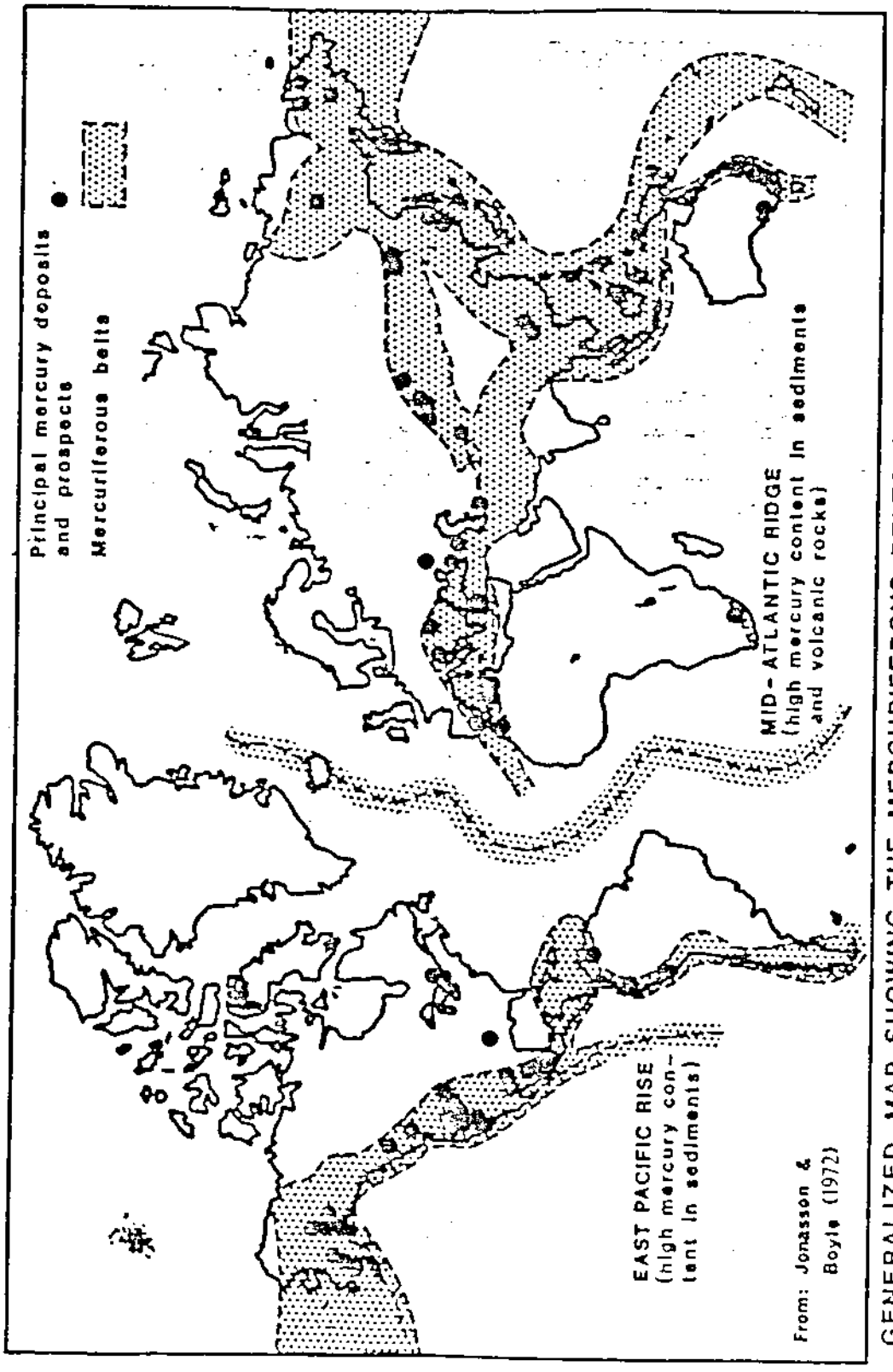
Mercury occurs naturally in the environment and is concentrated in geographical belts. Significant Hg deposits belong to one of the two Tertiary or Quarternary orogenic and volcanic belts: the Circumpacific and the Mediterranean-Himalayan belt (Fig. 1). Although many Hg minerals exist, commercial production is almost entirely from cinnabar (HgS). World production reached a peak of 10,000 t in 1971 but has since declined, totalling only 7,100 t in 1981 (Metallgesellschaft AG, 1978; U.S. Bureau of Mines, 1983). Current major producers include the USSR, Spain, the USA and Algeria. However, besides being sites of Hg mines these mercuriferous areas are also areas of higher than average natural Hg levels in the environment and, by weathering, contribute a considerable Hg input into the environment. As a consequence marine coastal areas can be strongly influenced by the runoff from these Hg anomalies resulting in elevated Hg levels in sediments and biota. The influence of the Mount Amiata and the Idrija Hg anomalies situated in the Mediterranean have been well illustrated (reviews: Bernhard & Renzoni 1977; Bernhard & Buffoni, 1982). Similar high Hg levels in the marine (and terrestrial) environment are expected in other areas under influences of the Circumpacific and the Mediterranean-Himalayan belts.

Mercury metal and Hg compounds are widely used in a variety of industrial and agricultural applications. A summary of the relative importance of different types of Hg consumption in industrialized countries (Korringa & Hagel, 1974) shows: chloralkali industry 25 %, electrical equipment manufacture 20 %, paint manufacture 15 %, industrial and scientific equipment manufacture 10 %, agricultural uses 5 %, dental uses 3 % and laboratory uses 2 %. Miscellaneous uses account for a further 20 % and these include use as catalysts, as fungicides during pulp and paper manufacture, in pharmaceutical and cosmetic preparations and in amalgamation processes (Korringa & Hagel, 1974).

Since 1974 industrialized nations have considerably reduced the Hg discharges from such industries as chlor-alkali plants, but continue emitting Hg when burning fossil fuels or sewage sludge and municipal wastes. The mean Hg concentrations in coal range from 0.01 to 3.3 mg/kg. Airey (1982) estimated that 140 to 2,700 t Hg/year is released globally from the burning of coal. Cement manufacture is estimated to emit an additional 250 t Hg/year. Fertiliser manufacture may also be an important source. The Hg emissions from each industrial sector are difficult to quantify due to changes in the production and consumption of Hg compounds, and the increasing sophistication of pollution control devices used by the respective industries.

Atmospheric emissions from anthropogenic sources are less than those from natural sources; reported ratios vary between 1/4 and 1/30 (Miller & Buchanan, 1979). However, at a local level anthropogenic emissions can be of considerably greater significance than natural emissions. Observations made near chlor-alkali plants and other sources have shown that within 5 to 10 km from the sources depositions rates can be 10 to 100 times higher than background levels (review: Lindqvist et al., 1984). However, the amounts deposited locally represent only 10 to 20 % of the total emission. The remaining 80 to 90 % of emissions contributes to regional and global deposition.

Figure 1. Generalized map showing the mercuriferous belts of the earth (WGMF, 1980)



The release of Hg compounds from industrial liquid wastes has been shown to greatly influence the immediately adjacent environment of the source. In the Minamata disease incident organic and inorganic Hg was released in wastes into Minamata Bay (Takizawa, 1979). The detection of MeHg crystals in the acetic acid equipment of the factory and the geographical distribution of the victims of Minamata disease left no doubt as to the cause of the disease. The poisoning was caused by the ingestion of fish and shellfish containing MeHgCl which had slowly accumulated in the fish and shellfish from the wastes released into the Minamata Bay from the chemical plant (Irukayama, 1967).

One explanation of the source of the MeHg in the seafood was that it was produced as a by-product during the manufacture of acetic acid from acetylene in the presence of inorganic Hg as a catalyst. This was reported by Kitamura et al. (1967) who constructed a model plant to simulate the production of acetic acid in the Minamata factory and detected an organomercurial compound. Takizawa et al. (1972) experimenting with acetaldehyde reaction fluid and waste materials from other acetaldehyde factories detected CH_3HgCl in gas-chromatograms. The bulk of the Hg in the waste water from the factory was inorganic. Another source of the high MeHg content in seafood is the methylation of Hg by algae in the water or bacteria in the bottom sediments (Section 1.4.2). The two explanations for the spread of MeHg may of course both be true, but different reports have argued that one or the other is wrong (Irukayama, 1967; Swedish Expert Group, 1971; Takizawa, 1979).

Mercury analyses of the sediments near the outlet of the Minamata plant attained a maximum of about 2,000 mg total Hg/kg DW (dry weight) and at the seaward side of Minamata Bay concentrations of about 12 mg total Hg/kg DW were detected. Similar observations have been made near chlor-alkali plants. Several authors (Renzoni et al., 1973; Cottiglia 1984; Hornung 1985; Bacci et al., 1985) investigated the influence of the release of large amounts of inorganic Hg from chlor-alkali plants. All authors found elevated Hg levels in sediments and marine organisms in the immediate vicinity of the outfall but at 10 to 20 km from the source the levels were low. Near the chemical plant discharging into the St. Gilla lagoon (Cagliari, Sardinia) sediment concentrations of about 200 mg total Hg/kg DW were observed. The biota in the lagoon had, however, only slightly elevated Hg levels. In contrast to the acetaldehyde plants chlor-alkali plants discharge only inorganic Hg. Inoko & Matsuno (1984) have carried out model experiments on the possibility of the formation and decomposition of CH_3HgCl in each process occurring in a Hg-type Cl_2-caustic soda electrolysis plant. They could not observe any formation of MeHg and observed in addition that 0.001 mg/kg of CH_3HgCl was decomposed when either chlorine evolved at the anode or when 10 to 100 mg/kg of HClO was present in the circulating brine. Consequently the probability that MeHg is formed in a chlor-alkali plant using Hg electrodes is low.

1.4 Transport, transformation and bioaccumulation

1.4.1 Transport

Mercury is transported through the environment by a variety of processes which are influenced by the form in which the Hg occurs. For example, Hg vapour is transported over considerably greater distances than Hg bound to particulate matter (Wallin, 1976). As Hg moves over continental land masses, it may be deposited and revolatilized many times. The residence time between deposition events is only a few days, yet during this time the Hg may be

transported over several hundreds of kilometres (Miller & Buchanan, 1979). Revolatilization is of more significance over land than oceans. As an air mass containing Hg from a continent moves over the ocean, evidence suggests that most of the Hg is lost to the ocean within a few hundred kilometres (Windom et al., 1975).

Nriagu (1979) reviewed the various global cycles proposed. Although the models differ in details the fluxes between the land surface and the atmosphere are an order of magnitude greater than the transport from continents to oceans. The transport between land surface and atmosphere involves mainly elemental Hg vapour, while the Hg transported from the land into oceans includes mainly Hg associated with suspended particles. Estimates of the residence time vary between 5 and 90 days and the proportion of anthropogenic Hg between 10 to 80 %.

1.4.2 Transformation

The different Hg species have different pathways and routes in the environment. Many of these routes can be taken both by biologically mediated processes and by abiological processes. Many pathways have been studied in experimental set-ups, but the ecological and environmental significance of each single route of the biogeochemical cycle of Hg is still very uncertain. In the past much less emphasis has been placed on abiological processes than on biologically mediated ones and among the biological ones the microbiological have obtained the greatest attention.

Natural foci of Hg dissemination are usually considered to be ore deposits (HgS) and non-mercury ore deposits such as Pb, As, Sn, which contain traces of Hg. Natural weathering and human exploitation of these deposits and use of Hg in chloride, caustic soda, and paper production, as well as Hg containing fertilizer, coal and municipal wastes, have introduced and continue to introduce many different forms of Hg. The most important from a health point of view are mono-methylmercury (MeHg) and di-methylmercury (Me_2Hg) because they are toxic to living organisms with a well-developed nervous system (Section 3). Inorganic Hg may be more toxic to other biota, which may or may not methylate Hg. According to general opinion the major pathways of the Hg cycle are mediated by microrganisms. However, a closer examination of the experimental set-ups used to study the transformation of Hg species, and the interpretation of field observations, may show that other biological and abiotic pathways could also be important. The most important difficulties in extrapolating the results obtained in the transformation experiments to environmental conditions are the extremely high inorganic Hg concentrations used. For example additions of 5 to 100 mg of inorganic Hg salts per kilogram of sediment are usual in these investigations. For comparison background concentrations of Hg in sediments range from 0.02 to 0.01 mg total Hg/kg DW of sediment. The very high Hg concentrations used in the experiments are selective for Hg resistant bacteria, which possibly are not the organisms which carry out Hg methylation under environmental conditions. The methylation experiments were also conducted on pure bacteria strains, which again are not typical for natural populations. With these limitations in mind our present knowledge can be summarized as follows:

Aerobes can solubilise Hg^{2+} from highly insoluble HgS by oxidizing sulphide to sulphate. It has been suggested that this is the rate-limiting step in organo-mercurial transformations (Fagerström & Jernelöv, 1972).

Not much is known about the biochemistry of the oxidation of elemental Hg to Hg^{2+} and the following methylation of MeHg and Me_2Hg. A wide range of bacteria can oxidize elemental Hg to Hg^{2+}. However, the enzyme(s) responsible for this oxidation has not yet been identified but is likely to include catalase (present in bacteria and animal tissues) (Silver 1984).

Several authors showed that microorganisms are necessary for methylation of Hg unless other methyl-metal compounds (e.g. tetramethyl lead or methyltin species) are added (Jernelöv & Martin 1975). Bacteria isolated from mucous material on the surface of fish and bacteria isolated from soil (Pseudomonas spp.) were able to methylate Hg under aerobic conditions. Methylation has been observed in fresh water and marine sediments, but even after several months only a few percent of the Hg added was methylated.

Not all bacteria can convert Hg^{2+} to MeHg and the volatile Me_2Hg. A methylating agent is methylcobalamine, methyl-vitamin B-12. All aerobic microorganisms which utilize cobalamine-dependent methionin-synthetase are capable of synthesizing MeHg. Some bacteria, such as E. coli, found in the intestinal flora, which require ß-12 for growth, can accumulate B-12 from other organisms, methylate it to methylcobalamine and subsequently carry out Hg methylation. Some anaerobic bacteria which possess methansynthetase are also capable of the methylation of Hg (reviews: Wood & Wang, 1983; Silver, 1984). Methylation of Hg has been interpreted as a detoxication mechanism, as bacteria do not have a nervous system sensitive to MeHg. It may be difficult to conceive that bacteria would produce a Hg compound more toxic to themselves, when they can convert inorganic Hg into the much less toxic elemental Hg.

At present it is believed that once MeHg is released from the microbial system into the surrounding water, it is accumulated by aquatic organisms directly from the water. Another, most probably more important, route consists in its accumulation through the food chain.

It has been shown that Hg^{2+} can also be methylated abiotically and extracellularly by methylcobalamine. The non-enzymatic methylation of Hg by cell-free extract of a methanogenic bacterium has shown to be possible with methylcobalamine as a donor for methyl groups in the presence of ATP and a mild reducing agent. Several other abiotic methylation mechanisms have been reported. DeSimone (1972) observed that water-soluble methylsilicone compounds can react with Hg^{2+} to yield MeHg. Photomethylation using methanol, ethanol, acetic and propionic acid produced MeHg from mercuric chloride (Akagi et al., 1977). Also Hayashi et al. (1979) observed photomethylation of inorganic Hg when aliphatic amino acids were irradiated with UV light for 4 hours. Both humic and fulvic acids have been shown to possess the ability to methylate inorganic Hg, albeit under conditions different from those found in the natural environment (Nagase et al., 1982) and very small amounts of MeHg were produced under these conditions.

The great attention placed on the transformation of Hg species by bacteria has diverted interests from other microorganisms. Ben-Bassat & Mayer (1975) and Betz (1977) have shown that unicellular algae can volatilise Hg, but it was not reported which Hg species were involved. Since many pelagic fish species live at a great distance from the sea bottom and still contain high proportions of Hg as MeHg, probably pelagic bacteria or phytoplankton may produce the MeHg found in these fishes. Topping & Davies (1981)

found MeHg associated with particulate matter sedimented to the bottom of large enclosed pelagic microcosms.

The data on methylation in higher organisms is still conflicting, probably because methylation is carried out in the intestine and depends on the intestinal flora. In shellfish from Minamata Bay CH_3HgSCH_3 instead of MeHg has been found (Kitamura 1968, Lofroth, 1969). This substance has not yet been conclusively identified in shellfish from other areas. A possible mechanism of its synthesis from methane-thiol (produced during decomposition of shellfish) has been described by Wood et al. (1975).

The transformations of MeHg to Hg^{2+} and then to elemental Hg are both catalysed by enzymes coded in the DNA of the bacterial plastides and transposons, and not coded in normal bacterial chromosomes of Hg resistant strains of microorganisms isolated from soil, fresh water and marine environments (reviews: Silver, 1984; Wood & Wang, 1983). Several authors have studied individual bacterial strains observing that most can convert Hg^{2+} into elemental Hg (e.g. Blair et al., 1974; Colwell et al., 1976; Olson et al., 1979; Barkey et al., 1979).

It has been reported that the marine phytoflagellate, Dunaliella, which produces H_2S, can take up Hg^{2+} and detoxify it by formation of HgS (Davies, 1976). Many invertebrate and fish detoxify Hg^{2+} by synthesis of the metal-binding protein, metallothionein (MT) (Kaegi & Nordberg 1979). Hg^{2+} will displace other metals from MT, thus in organisms where MT has also been induced by other metals, Hg^{2+} tends to accumulate as HgMT. Although Olson et al., (1978) reported the presence of MeHg-thionein in rainbow trout after Hg exposure, further investigation and evaluation of the quantitative significance is required since studies with rats have indicated that MeHg induced MT synthesis but did not bind to it (Chen et al., 1973).

In many large predatory fish, there is a direct correlation in certain organs (liver, kidney, etc.) between Hg and Se concentrations, indicating accumulation of Hg selenide. Experimental studies have so far produced equivocal results on this relationship (e.g. Ringdahl & Julshman, 1985). However, this may be due to factors other than selenide uptake being the rate limiting step in the transmethylation reaction (see chapter on selenium). The MeHg proportions are low in liver and kidney of fish and marine mammals, because these are the tissues where inorganic Hg accumulates.

In conclusion, despite the availability of a considerable amount of information on transformation mechanisms of different Hg species, their relative ecological significance can not yet be ascertained for certain, because the experimental conditions were different from those encountered in the normal environment. In particular, the origin of MeHg in marine biota living far away from the sea floor is still uncertain.

1.4.3 Bioaccumulation

Available evidence indicates that inorganic Hg uptake by phytoplankton is by passive diffusion since the uptake is directly proportional to the external Hg concentration. The internal concentration does not appear to be regulated. (Davies, 1976; Fisher et al., 1984). Results with plankton indicate a very rapid elimination of MeHg, with a biological half time (Tb) of about 3 days (Huckabee et al., 1979).

Many zooplankton are crustaceans and, therefore, it is most convenient to compare the data with higher, largely carnivorous crustaceans such as crabs, shrimps and lobsters. Studies with the shrimp (Lysmata seticaudata) have shown that MeHg is absorbed much more efficiently (10 times) than the inorganic Hg (Fowler et al., 1978). The inorganic Hg is eliminated with a (Tb) of about 110 days, but MeHg is eliminated with a very long Tb probably between 400 and 500 days. For the loss of inorganic Hg taken up Luoma (1977 a,b) estimated a loss Tb of about 16 days for Palaemon debilis and Sloan (1974) found 20 to 25 days for Cancer magister. The studies of Luoma (1977 a,b) indicated that Hg levels in the deposit feeding shrimp, Palasaman debilis, were related to availability from the water rather than from the sediment under field conditions.

Uptake of Hg by molluscs has been extensively studied (Miettenen et al., 1970; Unlu et al., 1970; Cunningham & Tripp, 1975; Fowler et al. 1978; Wrench, 1978). In the study of Fowler et al., (1978) the uptake of MeHg and inorganic Hg from water and food were compared. In both cases MeHg uptake was faster. In experiments studying the release of radioactive Hg from molluscs results have been variable (the biological half-times were for inorganic Hg from 20 to 40 days and for MeHg from 150 to 1,000 days), but in general mussels in the natural environment lose Hg faster than in the laboratory (Jaervenpaeae et al., 1975; Fowler et al., 1978; Seymour & Nelson 1971). Without data on the growth rate of the molluscs studied, loss rates are difficult to compare since the additional new tissue containing lower Hg levels will obviously decrease the Hg concentration in the organisms studied faster than when an organism loses Hg only from the same tissue into which the Hg has been accumulated (growth dilution). However, a large and significant difference between the elimination rates after accumulation of inorganic Hg and organic Hg demon- strated that no transformation between the two forms occurs in the organisms. Studies with mussels (Roesijadi, 1982), and oysters (Wrench 1978) indicate that uptake of inorganic Hg could be by pinocytosis of mucus-bound metal, this would then accumulate in lysosomes.

Transplanting mussels from sites with low to sites with high Hg levels have given similar results. For example, Majori et al. (1967) observed that, after transplanting mussels to a higher Hg site (near the mouth of the Isonzo river, which received mining wastes from the Idrija mercury mines), within one month the mussels had reached the same Hg concentrations as local mussels. On the other hand they required 6 months to lose the Hg accumulated when transplanted from a high level site to a low level one. More recently Riisgard et al. (1985) transplanted Mytilus edulis and Macoma near a chemical plant (producing Hg-containing fungicides) and near a dumping site of chemical wastes from the same chemical plant (phenyl-mercury-acetate and methyl-mercury-chloride). The chemical analysis showed that the mussels contaminated near the chemical plant contained 25 % of organic Hg and the mussels near the dumping site only 4 %. In fact, after transferring the mussels into clean sea water, the mussels from the dumping site eliminated the Hg at a markedly faster rate than the mussels from the chemical plants. Apparently even small differences in the relative amount of different Hg species will markedly influence their elimination rates.

Experimental studies of uptake, accumulation and loss of MeHg and inorganic Hg in two species of flatfish, (plaice and thornback ray) from both water and food were carried out by Pentreath (1976 a,b,c,d). Inorganic Hg uptake from water by plaice was directly proportional to the water concen-

tration up to 3 µg Hg/litre, with a Tb of 190 days. A similar Tb was observed in the ray. However, after exposure to MeHg there was no measurable loss in the ray. When Hg was fed in the form of radioactively labelled worms (Nereis) there were very low uptake efficiencies for inorganic Hg (3-14 %) and Tb's of 30 and 62 days in the two species, whereas when 80-100 % of the MeHg was taken up long Tb's of 275 and 323 days were observed. The tissue distributions of the two Hg forms were also different. When the fish were exposed to MeHg it partitioned strongly into the muscle, as observed in fish sampled from the field, whereas when exposed to inorganic Hg, this was largely found in other organs. The results are consistent with the diet being the major source of MeHg, almost complete uptake, and little or no demethylation (and subsequent elimination) in the organism. For inorganic Hg, there is poor uptake efficiency, which may be due to low absorption and a fairly rapid excretion, probably via the bile as a Hg cysteine or glutathione complex as described in mammals (Backström, 1969).

The uptake of HgS from sediments by fish has been studied (Gillespie & Scott, 1971). Although uptake from control sediments (0.024 mg Hg/kg DW) was considerable, freshwater fish exposed to sediment containing 50 mg Hg/kg as HgS accumulated still higher amounts of Hg. Whether this was direct uptake of cinnabar or MeHg formed from cinnabar, in the aquarium sediment or by gut flora, is not known.

In addition to the experimental foodchains discussed above another approach has been adopted by Buffoni et al. (1982) and Bernhard (1986). These authors have modelled the observed Hg concentrations in the foodchain of tuna. A relatively simple model of a pelagic food chain (sea water, plankton, sardine, tuna), based on general data available on Hg metabolism and specific Hg concentration in pelagic marine organisms from different sites in the Mediterranean, can show how total Hg can increase with size of the organisms, with a higher position in the food chain, and how at the same time the percentage of MeHg can increase. On the basis of their modelling simulations the authors could explain the difference in Hg concentrations observed in two bluefin populations, a low-Hg Atlantic population and a high-Hg Mediterranean one. The best fit to the Hg concentrations observed in sardines and tuna was obtained by assuming that in the Mediterranean the concentration of Hg in sea water is five times higher than the 1 ng Hg/litre generally accepted for open ocean water, even though such a difference could not be discerned from literature due to the wide spread of the data (see section 3.2). A similar difference in Hg body levels would result if the MeHg assumed in the model to be 2 % of the total Hg were five times higher i.e. 10 % in the Mediterranean. Also a combination of higher total Hg and an increased percentage of MeHg in the sea water would give the same increase in Hg body levels. The model also shows that it is not necessary to assume that higher organisms can methylate Hg. The differences in the uptake and loss kinetics between MeHg, total Hg and inorganic Hg are sufficient to explain the high MeHg enrichments observed.

Little data is available on the uptake of Hg by marine birds and mammals. Accumulation is almost entirely via the food and so the uptake is highly dependent upon the feeding behaviour. For example, the grey seal, (Halichoerus grypus), which has a diet of large fish and cephalopods, contains higher Hg concentrations than the harp seal, (Pagophilus groenlandicus), which feeds on small fish and crustaceans (Sergeant & Armstrong, 1973).

1.5 Mercury in atmosphere, seawater, sediments and marine biota

Many studies of Hg concentrations in different components of the marine environment have been carried out. For example, Eisler (1981) has collected available data on trace elements in marine organisms. Unfortunately, most of the data are not accompanied by quality control studies.

1.5.1 Mercury concentrations in the atmosphere

Atmospheric Hg occurs in particulate and gaseous phases. Although soluble and particulate Hg usually account for less than 1 % of the total Hg (Fitzgerald et al., 1983) these two forms of Hg are mainly responsible for the transport of Hg from the atmosphere to the earth's surface. Particles are easily washed out by rain or - to a lesser extent - scavenged by dry deposition. Estimates of gaseous Hg concentrations in air range from 7 ng/m^3 in urban areas (range 0.5 to 50), 4 ng/m^3 in remote, rural, continental areas (range 0.1 to 10), 1.5 ng/m^3 over continental shelf areas and 0.7 ng/m^3 over oceanic and polar regions (National Academy of Sciences, 1978). Due to the uneven distribution of land and oceans in the two hemispheres the mean level of gaseous total Hg should be about 1.5 ng total Hg/m^3 over open ocean sites in the northern hemisphere and 1 ng total Hg/m^3 over similar sites in the southern hemisphere. Over land the levels are definitely higher in urban areas and much higher in Hg anomalies. The little data (23 samples) so far collected indicates that open ocean atmospheric particulate Hg varies little in comparison with inshore variation observed over the Long Island Sound (Fitzgerald et al., 1983). These inshore variations reflect the dependence of particles on local sources and their short residence time in the atmosphere. Over the open ocean all Hg is "gaseous Hg" while over land and near anthropogenic sources, "organo-Hg species" can also occur.

Rainwater concentrations of Hg range from 1 to 85 ng/litre (Lindqvist et al., 1984). The highest concentrations of Hg are found near anthropogenic sources.

1.5.2 Mercury concentrations in sea water

The reported total Hg concentrations in sea water have been continuously lowered in recent years, mainly because of more attention to quality control and sample contamination. Like other trace metals, under natural conditions, only a very small part of Hg in fresh estuarine and sea water is likely to be present in dissolved form. Most inorganic and organic Hg appear to be associated with particles, colloids and high-molecular organic matter where it is probably co-ordinated with sulphur ligands (Mantoura, 1981; Wallace et al. 1982).

Due to the high chloride concentration in sea water, thermodynamic models of the occurrence of soluble inorganic Hg predict that, because of covalent bond formation and complex formation with so many ligands, $(HgCl_4)^{2-}$ is the dominant complex followed by $HgCl_2$, the mixed complex HgOHCl and $Hg(OH)_2$. The $Hg(II)^{++}$ cation can exist only at very low concentrations (more than 14 orders of magnitude less than $(CHgCl_4)^{2-}$ at pH 8 in sea water). Therefore, Hg^{++} cannot be considered a typical component of sea water.

It is now believed that Hg concentrations in open sea water will range from fractions of ng/litre to a few ng/litre (e.g. Bruland, 1983) and all open-ocean values above 10 to 20 ng/litre should be viewed with suspicion. However, one should not be inclined to accept the lowest data as the more accurate. Mercury adsorbs very easily on to surfaces, and although adsorption by sampling and storage bottles has not yet been investigated at nanogram levels, it may occur. In addition, many Hg species are highly volatile, so involuntary losses during sampling, storage and analysis are just as likely to happen as additions caused by sample, filtration or reagent contaminations. Unfortunately, because no sea water standard for Hg is available at the very low environmental concentrations, it is not possible to assess the accuracy of even the more recent data.

Bruland's (1983) review of metals in the oceans recognized that the distribution of Hg was not fully understood but that the recent data suggested that the Hg concentration in the open ocean was in the range 0.5 to 2 ng/litre. He noted that improvements in procedures for shipboard handling of samples and analytical techniques have largely eliminated contamination artifacts. Several reports of the levels of Hg in the North Atlantic have appeared in the literature but only since the early 1980s have the reported values started to approach the 2 ng/litre level for total Hg. Slemr et al. (1981) reported total Hg to be 1.9 to 8 ng/litre with the volatile fraction in the range 0.1 to 0.6 ng/litre in the surface waters on a transect from Hamburg to Buenos Aires. The following year Bruegmann et al. (1982) reported reactive Hg at 0.5 ng/litre and total Hg at 3 ng/litre in the waters of the North Sea, the Iceland-Faroe Ridge and the eastern North Atlantic. Similar levels of reactive and total Hg were found in the surface waters of the North Sea by Freimann et al. (1983) during the evaluation of a clean sampling device. Olafsson (1983) sampled the waters south of Iceland and found reactive Hg at 1.7±0.7 ng/litre and total Hg at 2.2±1.0 ng/litre. He also found the Hg distribution to be unlike that of the nutrient-like metals (Zn, Cd and Ni) or like aluminum, which is controlled by inorganic processes. Similar distributions were observed by Dalziel and Yeats (1985) in the waters of the Mid-Atlantic Ridge, south of the Azores, and in the Sargasso Sea, north of Bermuda. They found the level of reactive Hg to be fairly uniform with depth at 0.5±0.1 ng/litre.

A recent review by Aston and Fowler (1985) states that the open-ocean waters of the Mediterranean Sea contain 0.5-2.5 ng/litre total Hg and are not enriched relative to the open North Atlantic and other open-ocean areas. Similarly low levels of Hg have been reported for the Pacific. Fitzgerald et al. (1983) reported 0.5 ng/litre reactive Hg in surface waters from a transect from Hawaii to Tahiti. Two years later Fitzgerald and Gill (1985) studied reactive Hg in the Atlantic and Pacific Oceans and found no distinctive features in the distribution other than a higher value in the Northwest Atlantic (ca. 1.0 ng/litre) compared with the Pacific (0.35 ng/litre). Bloom and Crecelius (1983) studied the coastal waters of Puget Sound, Washington, and observed reactive Hg in the range 0.1 - 0.5 ng/litre and total Hg between 0.2 and 1.0 ng/litre.

There are recent reports on total Hg in the western Pacific which provide evidence that concentrations are significantly higher than concentrations in the eastern Pacific. Nishimura et al., (1983) reported total Hg in the range 3 to 6 ng/litre from extensive sampling in the Bering, Japan and China Seas, the North and South Pacific and Indian Oceans. Even higher levels of total Hg

were reported by Miyake and Suzuki (1983) in the western North Pacific. These authors found that surface waters contained 14 ng/litre with the concentrations decreasing to 6 ng/litre with depth. Slightly lower values were found in the subtropical region of the western North Pacific. These higher values compared with other reported for the same and comparable areas may be due to contamination in sampling and analysis.

In conclusion, the data indicate that reactive Hg in the ocean is about 0.5 ng/litre and total Hg in the range 2-3 ng/litre. It is important to note that the most recent data deal mainly with reactive Hg and more information on the concentrations of total Hg, and its partitioning, would be valuable to improve our understanding of the marine geochemistry of this element.

Suzuki & Sugimura (1985) reported that the total Hg in the surface layer of the Western North Atlantic was about 14 ng/litre and decreased rapidly with depth. Below 700 m depth the total Hg was almost uniform at about 5 ng/litre. The "organic" Hg was also higher in the surface (6 to 8 ng/litre) layer than below 700 m depth. The Hg was associated with an organic molecule (molecular size: about 9,000). Such Hg-compounds are not always defined as "organic". WHO (1976a) only included compounds in which Hg was bound to carbon with a covalent bond.

Mercury concentrations in coastal waters tend to be higher. In areas not directly affected by anthropogenic activities levels are usually less than 20 ng/litre (Fitzgerald, 1979). A recent summary of total Hg levels in coastal waters suggest the following concentrations: Denmark, 4 to 32 ng/litre; Federal Republic of Germany, 2 to 45 ng/litre; France, 10 to 390 ng/litre; Netherlands, 10 to 150 ng/litre; Norway, 20 to 50 ng/litre and Sweden, 20 to 100 ng/litre (Oslo Commission, 1984). These data may represent polluted sites and not coastal waters in general.

Concentrations in estuarine waters are more variable because of a direct relationship to an increase in the sources of contamination. Total Hg concentrations in several large estuaries draining into the North Sea and north-east Atlantic are believed to be as follows: Scheldt, 19 to 280 ng/litre; Ems, 50 to 330 ng/litre; Forth, 20 to 340 ng/litre; Seine, 10 to 140 ng/litre; Loire, 60 to 1100 ng/litre; Gironde, 20 to 400 ng/litre; Thames, 30 to 530 ng/litre and Aveiro, 20 to 1700 ng/litre (Oslo Commission, 1984).

Since MeHg predominates in marine organisms, it is the most important Hg compound from the biological and health protection point of view. Unfortunately very little MeHg data for sea water exist (Fujita & Iwashima, 1981; Egawa et al., 1982; Yamamoto et al., 1983). The values range from 0.03 % to 6 % of the total Hg present.

In the highly polluted Minamata Bay seawater concentrations of particulate and total Hg decreased with distance from the pollution source. In the inner bay total Hg reached 200 ng/litre; most of the Hg was particulate. At about 4 to 5 m distance from the source the particulate Hg approached ng levels while the total Hg still remained at 70 ng Hg/litre. Since sediment and suspended matter Hg concentrations decrease in a similar pattern the results suggest that the origin of the Hg in the suspended matter is the contaminated sediment (Kumagai & Nishimura, 1978).

1.5.3 Mercury concentrations in sediments

Sediments are the principal site of Hg accumulation in the marine environment. Background concentrations of up to 0.08 µg/g DW have been reported for a range of sediments including those from the Australian coast (0.004 to 0.005 µg/g DW), the Israeli coast (0.02 µg/g DW) various Arctic coastal regions (0.02 to 0.08 µg/g DW) and Baffin Bay near Greenland (0.07 µg/g DW) (Knauer, 1976; Campbell & Loring, 1980; Hornung et al., 1981).

Elevated concentrations are associated with sources of anthropogenic inputs. Between 0.1 and 5.4 µg/g DW occurred near the Los Angeles county sewage outfall (Hershelman et al., 1981) High concentrations also occur in other estuarine sediments, for example, 1.2 to 11.3 µg/g DW in the Mersey and 0.4 to 4.4 µg/g DW in the Clyde (Bartlett et al., 1978). In locations affected by chlor-alkali plants levels rose over relatively short distances (10-20 km) from background up to 1.4 µg/g DW on the Egyptian coast (El-Sayed et al., 1979), up to 4.1 µg/g DW on the Italian coast (Baldi & Bargagli, 1984) and up to 350 µg/g DW in a semi-enclosed bay in Norway (Skei, 1978). The highest concentrations have been observed near a pollution source plant at Minamata (2,010 µg/g wet weight) (Takizawa, 1979), but very high levels were also found in the S. Gilla lagoon near Cagliari (1,300 µg/g DW) (Porcu & Masala, 1983).

In a marine region affected by drainage from cinnabar deposits (Mount Amiata), a maximum concentration of 4 µg/g DW was recorded (Baldi & Bargagli, 1984) and the adjacent coastal zone had above background levels. In another area (Gulf of Trieste) which receives wastes from the Idrija Hg mining area through the Isonzio river, levels around 40 µg/g DW have been reported (Majori et al.; 1967, Kosta et al., 1978).

1.5.4 Mercury concentrations in marine biota

Phytoplankton and seaweeds

Phytoplankton and seaweeds constitute the first level of the food chain. Knauer & Martin (1972) have published the only intercalibrated data on natural population of phytoplankton. The total Hg concentrations of plankton (mainly diatoms) collected with a 76 µ pore-size net over the Monterey submarine canyon averaged 11 µg total Hg/kg FW (n= 29) with a range from 5 to 30 µg total Hg/kg FW. On the Hawaii-Monterey transect (n= 7) the mean was 20 µg total Hg/kg FW and the range 10 to 52 µg total Hg/kg FW. A few preliminary measurements of the proportion "organic" Hg ranged from 0 to 67 % with a mean of 40 % (n = 4). The same authors determined in the planktonfage anchovy 76 to 100 % organic Hg. Assuming an open-ocean sea water concentration of 1 ng Hg/litre the concentration factor from sea water to phytoplankton is about 10,000. Comparing the plankton concentration with that of higher organisms shows that the enrichment from sea water to the first trophic level is much higher than the gain in concentration between the following trophic levels (see below).

Data on seaweeds are few and without quality control. In Chlorophyceae, Rhodophyceae and Phaeophyceae at Broughty Ferry, in the Tay estuary region of Scotland, the reported Hg concentrations (128 to 506 µg total Hg/kg FW) were higher than in the marine environment near Arbroath (1 µg total Hg/kg FW) (Jones et al., 1972). Ulva lactuca had the highest concentration at Broughty

Ferry with 6200 µg total Hg/kg FW. In the Solent region Hg levels in Phaeophyceae ranged from 16.3 to 35.7 µg total Hg/kg FW and in Rhodophyceae from 7.7 to 13.3 µg total Hg/kg FW (Leatherland & Burton, 1974). Similar concentration ranges were found for Sargassum and Spartina alterniflora in the NW Atlantic and in Macrocystis pyrifera near La Jolla and Pacific Grove (Kishore and Guinn, 1972; Robertson et al., 1972; Windom, 1972). The range of total Hg concentrations in Chlorophyceae, Phaeophyceae and Rhodophyceae from the Island of Penang (Malaysia) range from below detection limits to about 35 µg total Hg/kg FW. The discharge from a chlor-alkali plant raised the concentrations in Codium tomentosum from below detection limit to 23 µg total Hg/kg FW (Hornung et al., 1981).

Zooplankton

Data on zooplankton are scarce and only Knauer & Martin (1972) have published quality control data. On the Hawaii-Monterey transsect values similar to the phytoplankton concentrations were determined in the samples collected with a 360 µg-net: mean 10 µg total Hg/kg FW; range 4-35 µg total Hg/kg FW (Knauer & Martin, 1972). Similar levels were observed in plankton (330 µ net) off Pacific Grove, in the NW-Atlantic, in the Caribbean and Mediterranean (Martin & Knauer, 1972; Robertson et al., 1972; Windom, 1972; Fowler et al., 1976; Kosta et al., 1978). As expected in Minamata Bay and the adjacent Yatsushiro Sea, the Hg levels in plankton were 30 times and 10 times higher than in the open Pacific (Kumagai & Nishimura, 1978). The total Hg concentrations in netplankton (pore size 95 µ and 328 µ) from the Minamata bay decrease with distance from the pollution source (Kumagai & Nishimura, 1978). Very few data are published on single zooplankton species: Euphausids Hg levels range from 20 to 240 µg total Hg/kg DW (= 5 to 50 µg total Hg/kg FW); Arcatia clausi: 160 to 440 µg total Hg/kg DW (= 30 to 90 µg total Hg/Kg FW) (Leatherland et al., 1973; Zafiropoulos & Grimanis, 1977; Belloni et al., 1978; Fowler, 1985).

Molluscs

The International Council for the Exploration of the Sea (ICES) has carried out studies in the North Atlantic, North Sea, Baltic Sea and in these studies and in the Mediterranean MED POL projects the mussel (Mytilus spp) has been used to monitor pollutants. These collaborative studies included intercalibration. In the ICES area the average was 50 µg total Hg/kg FW with a range from 20 to 130 µg total Hg/kg FW (ICES 1977a,b,c, 1980). The area coverage in the Meditteranean was not complete as very little data referred to the southern coast of the Mediterranean. The mean value was 230 µg total Hg/kg FW with a range from 4 to 7,000 µg total Hg/kg FW (UNEP, 1983). In addition to these data bases there are numerous reports of individual authors on molluscs in which no quality control has been used (Eisler, 1981) and the accuracy of their data cannot be ascertained. Levels for various oyster species (edible parts) range from 1 to 370 µg total Hg/kg FW. For octopus the reported levels are from 50 to 370 µg total Hg/kg FW and in the pelagic squid from 10 to 220 µg total Hg/kg FW.

Davies et al. (1979) found that 50 to 100 % of the total Hg was MeHg in M. edulis in clean waters, but in polluted waters only 21 % was MeHg. Langton (1982) found that 14 to 35 % of the total Hg was MeHg in the deposit feeder bivalve Scrobicularia plana.

Crustaceans

In the North Atlantic, North Sea and Baltic Sea (ICES area) the brown shrimp has been monitored for several years in a collaborative study. The Hg levels ranged from 30 to 400 µg total Hg/kg FW in the North Sea (ICES, 1974, 1977b, 1977c). A typical average level should be 110 µg total Hg/kg FW. The range for the deep sea prawn sampled near West Greenland was 20 to 30 µg total Hg/kg FW. By comparison, the Norway lobster caught in Mediterranean areas influenced by natural Hg anomalies of Mt. Amiata range from 60 to 3,000 µg total Hg/kg FW with a mean of 920 µg total Hg/kg FW. The specimens from the Mediterranean showed positive correlations between Hg concentration and lobster size (ICES,1974, 1977b, 1977c).

Fish

In many areas of the world Hg is monitored in fish. Unfortunately, most of this data is published in reports of specialized government agencies and not in the open literature. Often these are collaborative studies which include some quality control and intercalibration. Four large data bases may serve as examples of government agency data: ICES data (ICES, 1974, 1977a,b,c), the MED POL data (UNEP, 1983), the data reviewed by the Australian Working Group on Hg in Fish (WGMF, 1980) and the data from US-NOAA (NMFS, 1978b). Although they cover large areas of the marine environment, many important fishing areas are not covered. The ICES data for the North Atlantic show that the plankton feeders (mainly herring and sardines) do not exceed 240 µg total Hg/kg FW (Table 1). Fish feeding on other marine organisms had slightly higher Hg levels. For comparison, data for the same species from the MED POL project are also included in the table. Mercury levels in sardines and hake from the Mediterranean are considerably higher. Since Hg concentrations in fish increase with age (size), for a precise comparison specimens of the same age should be compared. In fact large hakes from the North Sea have very much lower Hg concentrations than considerably smaller hakes from the Mediterranean (Bernhard, 1986). Hg levels in fish from the Mediterranean MED POL project (intercalibrated) are shown in Table 2.

A third large data base exists in the report on the chance of US seafood consumers exceeding the current acceptable daily intake of Hg (NMFS, 1978a). Table 3 gives the reported averages and the maximum levels in US seafoods. Unfortunately ranges or other parameters are not given in this publication. As can be seen from this table maximum values above 1 µg total Hg/g FW are quite frequent.

The large diversity of fish consumed in Australia made it necessary to analyse many different species. In order to obtain information about 70 % of the species consumed by Australians, 20 species have to be considered. Samples of 142 local and imported fish species (a total of 6,678 samples) were analysed, mainly by government laboratories which had participated in an intercalibration (WGMF, 1980). The data is grouped according to Hg levels (Table 4). As to be expected longlived and large fishes such as the ling reached high mean and maximum Hg concentrations. The marlins had the highest mean level (7.27 µg/g) and highest maximum 16.5 µg total Hg/g FW). Similar results for marlins have been published previously (MacKay et al., 1975; Shultz et al., 1976). Marlins are exceptional in having an unusually low percentage of organic Hg (10 % of total Hg) in the muscle tissue.

Table 1. Mercury (µg/kg FW) in some fish (muscle from the northern Atlantic (selected data from ICES and UNEP)

	Median of means	range of means	ref.	
plankton feeder				
herring	40	20-240	N.Sea	ICES 1974
herring	20	10-35	N.Atl.	ICES 1977a
herring	40	10-23	Irish coast	ICES 1980
"typical"	40			
sardine	60	6-80	N.Atl.	ICES 1977a
sardine	250	150-390	Medit.	Nauen et al. 1980
sprat	65	60-140	Irish c.	ICES 1980
capelin	10	10-30	N.Atl.	ICES 1977a
feed on invertebrates				
cod	100	30-480	N.Sea	ICES 1974
cod	100	60-300	N.Sea	ICES 1977a
cod	40	40-50	N.Atlantic	ICES 1977a
cod	260		Irish Sea	ICES 1977a
cod	140	70-370	Irish c.	ICES 1980
cod	70	50-140	NW-Atlantic	ICES 1977a
cod	80	70-90	NW Atlantic	ICES 1980
"typical"	100			
feed on crustaceans and fish				
hake	90	30-130	N.Atlantic	ICES 1977a
hake	230	30-850(!)	Mediter.	Nauen et al. 1980
haddock	50	20-60	Irish coast	ICES 1980
haddock	50		NW Atlantic	ICES 1980
whiting	80	30-90	Irish coast	ICES 1980
Greenl.halibut	40	30-50	N.Atlantic	ICES 1977a
plaice	90	20-260	N.Sea	ICES 1974
plaice	120	20-500(!)	N.Atlantic	ICES 1977a
plaice	25	10-80	Irish coast	ICES 1980
"typical"	90			
sole	150	50-320	N.Atlantic	ICES 1977a

(!) levels exceeding 500 ug Hg-T/kg

In general, longlived fish such as tuna, swordfish (up to 20 years) and sharks (up to 40 years) have the highest concentrations. These high concentrations result from Hg being an accumulative contaminant i.e. its concentration increases with the age of the host organism. Mercury concentrations in a selection of tuna species averaged 0.2 to 0.6 µg/g FW in the coastal waters of Hawaii, 0.1 to 0.8 µg/g FW in the Atlantic, 0.3 µg/g FW in the Pacific and 0.9 to 4 µg/g FW in the Mediterranean (Renzoni et al., 1979; Greig & Krzynowek, 1979; Nauen et al., 1980; Piotrowski & Inskip, 1981). The

Table 2. Mercury in Mediterranean Fish
(Source: UNEP, 1983)

Species	No. of samples	Mean + S.D. (ug/g FW)	Range (ug/g FW)
Solea vulgaris (sole)	10	0.07	0.01 - 0.22
Mullus surmeletus (red mullet)	234	0.09	0 - 0.51
Trachurus mediterraneus (mackerel)	54	0.12	0.008 - 0.96
Boops boops (bogue)	15	0.13	0.20 - 0.43
Merluccius merluccius	16	0.13	0.03 - 0.26
Dentex gibbosus (dentex)	12	0.14	0.10 - 0.18
Saurida undosquamis	143	0.14	0.04 - 0.65
Pagellus acarne (bream)	12	0.16	0.03 - 0.34
Engraulis encrasicolus	254	0.17	0.02 - 0.58
Mugil auratus	39	0.17	0.001 - 5.60
Pagellus erthrinus (bream)	112	0.20	0.05 - 0.81
Sardinella aurita (sardine)	47	0.25	0.12 - 0.39
Thunnus alalunga (albacore tuna)	38	0.26	0.06 - 0.40
Scomber scombrus (mackerel)	16	0.34	0.13 - 0.51
Upeneus molluccensis (goatfish)	127	0.43	0.04 - 1.11
Mullus barbatus (striped mullet)	1265	0.69	0.002 - 7.90
Thunnus thynnus thynnus (bluefin tuna)	325	1.05	0.02 - 6.30
Sarda sarda (Atlantic bonita)	11	1.15	0.29 - 2.30

highest tuna Hg concentrations have been observed in the Mediterranean. The influence of the Hg anomalies in the western Mediterranean results in two distinct Thunnus thynnus populations (Renzoni et al., 1979). The largest tunas in the 'Mediterranean population' of tunas had an Hg concentration up to about 4,000 µg total Hg/kg FW. An 'Atlantic population' which comes into the Mediterranean only for spawning had concentrations up to about 1,500 µg total Hg/kg FW in the largest tunas. The principal food organisms of these tunas (anchovy, sardine, mackerel) also had much higher (5 times) Hg concentrations in the Mediterranean. A similar range of concentrations has been reported for swordfish (0.3 to 1.3 µg/g FW in the Atlantic, 0.3 µg/g FW in the Pacific and 0.6 to 1.8 µg/g FW in the Mediterranean) (Freeman et al., 1978; Nauen et al., 1980; Piotrowski & Inskip, 1981). Mercury concentrations in the muscle of shark species from Mediterranean, South African and Australian coastal waters ranged from 0.6 to 2.6 µg/g FW (Glover, 1979; Nauen et al., 1980; Watling et al., 1982).

Table 3. Summary of mercury concentrations of seafoods sampled in the USA used to estimate the chance of consumers exceeding current acceptable daily intakes of mercury (NMFS, 1978a)

	average weight (g)	Hg concentration (ug/kg FW) average	maximum
molluscs:			
abalone	1092	18	120
oysters	104	27	450
clams	250	49	260
scallops	132	58	220
squid + octopus	356	31	400
crustaceans:			
northern lobster	1423	509	2310
spiny lobster	415	113	370
shrimp	15	46	330
northern lobster	718	339	1603
king crap	2724	70	240
crab	704	140	610
fish:			
sharks	8320	1244	4528
anchovy	23	39	210
herring	429	23	260
hake	550	100	1100
sea bass	346	157	575
stripped bass	4378	752	2000
red snapper	822	454	2170
snapper species	1406	362	1840
mullet	566	16	280
cod	1847	125	590
haddock	1277	109	368
flounder	606	96	880
halibut 4	32466	187	1000
halibut 3	43901	284	1280
halibut 2N	28048	440	1480
halibut	17163	534	1430
marine trout	676	242	1190
salmon	4077	48	210
Atlantic mackerel	391	48	190
jack mackerel	959	267	510
king mackerel, gulf	3932	823	2730
king mackerel	6799	1128	2900
Spanish mackerel, gulf	908	542	2470
Spanish mackerel	613	225	1605
bonito <3197 g	2206	302	470
bonito >3197 g	4407	382	740
skipjack tuna	3412	144	385
yellowfin tuna	29051	271	870
white tuna	9532	350	904
swordfish	47639	1208	2720
skipjack tuna	3412	144	385
yellowfin tuna	29051	271	870
tuna, white	9532	350	904

Note: the number of digits given do not reflect the accuracy of the data. Common names are used because these may include several taxonomic species.

Table 4. Mean mercury levels and ranges of Australian fish and seafood (grouped by level) (WGMF, 1980)

Common Name	Mean Mercury Level (mg/kg)	Range	No. in Sample (mg/kg)
Species with a mean mercury level between 0.01 and 0.10 mg/kg			
Cobbler	0.03	0.01-0.07	27
Cuttlefish	0.03	0.02-0.04	2
Flounder, Greenback	0.04	0.02-0.05	54
Luderick	0.05	0.02-0.12	88
Mackerel Trevalla	0.03	0.01-0.05	28
Morwong, Blue	0.10	0.05-0.16	15
Mullet, Sea	0.02	0.01-0.20	84
Mullet, Yellow-eye	0.07	0.01-0.45	50
Oysters	0.03	0.01-0.10	149
Sea Carp	0.01	0.01-0.02	8
Squid	0.05	0.02-0.27	13
Trumpeter, Bastare	0.05	0.01-0.28	28
Warehou	0.05	0.01-0.08	15
Whiting, King George	0.06	0.01-0.34	96
Species with a mean mercury level between 0.11 and 0.20 mg/kg			
Australian Herring	0.14	0.05-0.74	121
Australian Salmon	0.17	0.01-0.62	82
Cod, Southern Rock	0.13	0.04-0.45	9
Cucumber Fish	0.12	0.02-0.36	17
Dory, Mirror	0.16	0.05-0.36	30
Dory, Silver	0.12	0.01-0.50	77
Flathead, Dusky	0.15	0.05-0.35	44
Kingfish, Yellowtail	0.18	0.06-0.70	20
Knifejaw	0.11	0.04-0.24	50
Leatherjacket	0.19	0.02-0.64	80
Mackerel, Blue	0.15	0.05-0.37	50
Mackerel, Jack	0.11	0.04-0.25	77
Morwong, Brown Banded	0.18	0.02-0.59	10
Ruby Fish	0.11	0.03-0.22	50
Sandpaper Fish	0.12	0.03-0.50	20
Skippy	0.19	0.01-0.57	46
Snapper (Western)	0.20	0.02-1.25	72
Snapper, Queen	0.20	0.05-0.61	51
Snoek	0.11	0.01-0.54	183
Stingaree, White Spot	0.20	0.11-0.35	9
Trevally	0.16	0.09-0.39	50
Tuna, Skipjack	0.15	0.11-0.17	20

Table 4 continued

Species with a mean mercury level between 0.21 and 0.30 mg/kg

Anchovy	0.25	0.22-0.30	3
Australian Tusk	0.22	0.07-0.42	50
Barramundi	0.21	0.10-0.60	34
Bream, Black	0.23	0.03-1.17	69
Bream, Yellowfin	0.22	0.03-0.81	34
Boarfish, Black Spot	0.21	0.05-0.39	24
Flathead, Tiger	0.29	0.04-1.81	51
Morwong, Jackass	0.21	0.05-0.74	194
Morwong, Rubberlip	0.22	0.03-0.49	30
Mulloway	0.22	0.04-0.82	49
Parrot Fish	0.21	0.15-0.31	4
Perch, Gulf	0.27	0.11-0.61	9
Perch, Ocean	0.27	0.19-0.36	2
Ray, Fiddler	0.21	0.01-0.52	32
Shark, Elephant	0.26	0.02-0.97	139
Shark, Varied Cat-	0.25	0.11-0.38	4
Skate, White Spotted	0.26	0.03-0.82	32
Stargazer	0.22	0.09-0.55	11
Tailor	0.22	0.04-0.55	32
Tuna, Southern Bluefin	0.22	0.06-0.63	219
Whiptail	0.24	0.05-0.43	9
Whiting, Sand	0.30	0.09-0.66	24

Species with a mean mercury level between 0.31 and 0.40 mg/kg

Blue Grenadier	0.31	0.08-1.55	44
Boarfish, Long Snout	0.34	-	1
Boarfish, Yellow Spot	0.34	0.02-0.87	50
Dory, John	0.31	0.06-0.84	54
Flathead, Deepwater	0.37	0.12-0.87	116
Flathead, Sand	0.38	0.01-1.12	313
Nannygai	0.38	0.20-0.56	20
Shark, Angel	0.36	0.06-1.60	36
Shark, Ornate Angel	0.35	0.15-0.84	50
Snook	0.32	0.06-0.63	33
Southern Frost Fish	0.38	0.25-0.58	5
Tuna, Yellowfin	0.38	0.11-0.66	20

Species with a mean mercury level between 0.41 and 0.50 mg/kg

Hapuku	0.48	0.01-0.95	54
Shark, Blue Whaler	0.41	0.25-0.57	2
Shark, Common Saw	0.47	0.07-1.32	93
Shark, Graceful	0.43	0.34-0.56	5
Shark, Gummy	0.44	0.03-3.04	507
Shark, Port Jackson	0.46	0.12-3.07	71
Shark, Southern Saw	0.49	0.01-2.92	62
Snapper (Eastern)	0.43	0.04-1.94	104
Stingaree, Common	0.48	0.39-0.58	3
Trevalla, Deep Sea	0.45	0.04-1.23	53

Table 4 continued

Species with a mean mercury level between 0.51 and 0.60 mg/kg			
Bight Redfish	0.52	0.20-1.32	50
Latchet	0.57	0.01-5.71	180
Ling	0.52	0.20-1.10	10
Shark, Rusty Cat-	0.54	0.26-1.26	16
Shark, Whiskery	0.59	0.10-1.70	171
Species with a mean mercury level between 0.61 and 0.70 mg/kg			
Gemfish	0.66	0.07-3.07	237
Shark, Gulf Cat-	0.64	0.55-0.73	2
Species with a mean mercury level between 0.71 and 0.80 mg/kg			
Perch, Butterfly	0.75	-	1
Shark, Bronze Whaler	0.72	0.10-2.60	159
Shark, School	0.75	0.01-3.30	361
Shark, Thickskin	0.73	0.30-1.60	84
Species with a mean mercury level between 0.81 and 0.90 mg/kg			
Shark, White Spotted Spurdong	0.86	0.28-2.28	77
Species with a mean mercury level between 0.91 and 1.00 mg/kg			
Shark, Hammerhead	0.92	0.42-1.72	15
Species with a mean mercury level greater than 1.00 mg/kg			
Marlin Black	7.27	0.50-16.50	42
Shark, Blacktip Whaler	1.48	0.61-2.10	8
Shark, Blue Pointer	1.93	0.71-3.15	2
Shark, Carpet	1.02	0.10-3.40	76
Shark, Draughtboard	1.93	0.34-10.50	15
Shark, Grey Nurse	1.69	0.07-6.73	18
Shark, One finned	1.48	-	1
Shark, Pencil	2.44	0.40-9.65	5
Shark, Piket Spurdog	1.91	0.01-4.20	94
Shark, Seven Gilled	1.39	0.39-2.06	22
Shark, White Pointer	1.29	-	1

The majority of Hg in fish occurs as MeHg. In the muscle of cod from coastal waters off the Netherlands, approximately 97 % occurred as MeHg (Luten et al., 1980). Virtually all the Hg in large predatory fish such as swordfish is also present as MeHg (Freeman et al., 1978), although marlin is an exception with only 10 % of the total Hg as MeHg (Shultz et al., 1976). Recently Halim et al., (1985) has shown that, similar to marine mammals, the organic Hg in pilchards, Euthynnus alleteratus and Rhinobatus hallavi from the Egyptian coast is low: 7 to 23 % of total Hg. Eganhouse & Young, (1978) found on average only 9.6 % MeHg in the liver of the Dover sole (Microstomus pacificus). As this is a tissue where inorganic Hg accumulates preferentially (Friberg & Vostal, 1972) the low percentage MeHg in the liver may not reflect the situation for the fish muscle.

Marine birds

Pelagic seabirds contain higher levels of Hg than most other avian species. The greatest concentrations occur in the liver, kidney and feathers (Fimreite, 1979). Liver concentrations in a variety of Antarctic birds fall into three groups: 0.48 to 0.87 µg/g FW (chinstrap penguin Pygoscelis antarctica, macaroni penguin Eudyptes chrysolophus, snow petrel Pagodroma nivea), 1.3 to 2.7 µg/g FW (Cape pigeon Daptoir capense, southern fulmar Fulmarus glacialoides, southern polar skua Catharcta maccormicki) and 7.5 µg/g FW (brown skua C. lonnbergi) (Norheim et al., 1982). Lower concentrations occur in the livers of Arctic birds, for example, little auk Alle alle (0.49 µg/g FW), Brinich's guillemot Uria lomvia (0.63 µg/g FW), eider Somateria millissima (1.03 µg/g FW), glaucous gull Larus hyperboreus (1.6 µg/g FW) and fulmar Fulmarus glacialis (2.1 µg/g FW) (Norheim & Kjos-Hanssen, 1984).

The percentage of total Hg as MeHg shows considerable variation. Herring gulls (Larus argentatus) from three Danish locations, had average liver concentrations of 0.63 µg/g FW (range 0.36 to 0.87), with 42 % (range 32 to 57) of this as MeHg (Karlog & Clausen 1983). In three pelagic species from the U.K. puffin (Fratercula arctica), manx shearwater (Puffinus puffinus) and fulmar (Fulmarus glacialis), 83 to 97 % of total Hg in the liver (4.5 to 29.4 µg/g FW) occurred as MeHg (Osborn et al., 1979).

Marine mammals

Marine mammals (seals, porpoises, whales) have the highest Hg concentrations of all marine organisms. The levels in liver are particularly high, and the concentrations increase with age. The importance of age is especially evident at high Hg levels. For example in Stenella caeruleo-alba, a foetus contained 0.95 mg total Hg/kg FW in muscle tissue while 20 to 30 year old adults have levels between 5.7 to 9.4 mg total Hg/kg FW. The highest total Hg concentration was found in the muscle tissue of a 22-year-old dolphin (Tursiops gilli): 51.8 mg total Hg/kg FW (MeHg: 17 %) (Arima & Nagakura 1979). More recently Ronald et al., (1984) determined much lower total Hg levels in the harp seal from the northwest Atlantic. These authors observed in the muscle of adults (63 females and 22 males without age data) mean values from 0.27 to 0.49 mg total Hg/kg FW and in pups (23 females and 37 males) means of 0.12 to 0.27 mg total Hg/kg FW. Also the mean levels in the liver were lower: in pups from 0.7 to 2.82 mg total Hg/kg FW and in adults from 8.62 to 13.3 mg total Hg/kg FW. These lower levels in the harp seal from inshore Newfoundland were confirmed by Botta et al. (1983). They are the

only authors who report intercalibration data. In the muscles of pups (beaters) they observed even lower levels of 0.02 to 0.1 mg total Hg/kg FW, while in the liver the range was from 0.26 to 0.9 mg total Hg/kg FW. By comparison the muscles of adult harp seals (age four years) contained 0.21 to 0.3 mg total Hg/kg FW and the liver 0.76 to 4.65 mg total Hg/kg FW. Much of the other data is published without information on quality control and it is very difficult to say what the true levels are.

The position in the foodchain also affects Hg concentrations. Large plankton feeding fin whales (17 to 22 m) and sei whales (12 to 16 m) have lower Hg levels (0.01 to 0.07 mg total Hg/kg FW) in muscle tissue than sperm whales (9 to 14 m) (0.54 to 1.57 mg total Hg/kg FW) which feeds on cephalopods, or seal and porpoises which feed on crustaceans and fish. Again the highest Hg concentrations are always in the liver, and comparisons are not easy because ages or sizes are not always published together with the Hg concentrations. Smith & Armstrong (1978) found in the liver of 80 ringed seals, an important food in the diet of Eskimos, a mean FW concentration of 27.5 mg total Hg/kg. The highest value of total Hg was obtained from the liver of a 17 yr old female; 184 mg total Hg/kg FW. However, this is not the highest value published. Koeman et al. (1973) found maxima in Phoca vitulina ranging from 700 to 935 mg total Hg/kg FW near the Netherlands coast. Similar high levels have been reported for marine mammals from the French Mediterranean coast (review: Bernhard & Renzoni, 1977). The only comparisons made on an age/Hg relationship are from Roberts et al. (1976). Plotting the Hg concentration versus age in livers of common seals shows that the livers of seals from the Netherlands have consistently higher Hg levels than the livers of seals from East Anglia, while those from East Anglia have higher levels than those of West Scotland.

The small amount of data on MeHg levels in marine mammals show low MeHg percentage in the liver and medium to high levels in the muscle. For example, in the meat of the sperm whale the MeHg percentage ranged from 65 to 82 % (Nagakura et al., 1974). In the muscle of blue-white dolphins the MeHg proportion ranged from 32 to 86 % of total Hg (Arima and Nagakura, 1979). Pilot whale muscle contained 42 to 60 % MeHg while the liver had only about 2 %. Likewise in the ringed seal and the long-snouted dolphin muscle tissue had 60 to 100 % MeHg and again the MeHg percentage in the liver was 1 to 5 % (Gaskin et al., 1974; Smith & Armstrong, 1975). Falconer et al. (1983) found that the proportion of the MeHg in the liver of the seal Phocoena phocoena varied from 9 to 57 % of total Hg. The highest proportion were found in samples containing low total Hg. These mammals consume organisms with a high MeHg percentage as food, but they also consume inorganic Hg from this food and from sea water. As mentioned previously, the liver accumulates inorganic Hg more than other tissues.

Martoja & Berry (1980) have shown that in the liver of marine mammals Hg is bound with selenium in small granules of pure tiemannite which are chemically unreactive. Possibly, the Hg bound in these granules will not be easily absorbed when the liver of marine mammals is used as human food.

The Se/Hg relationships are discussed in the chapter on selenium.

2. EFFECTS ON MARINE BIOTA

2.1 Reference documentation

Reviews considered in preparing this section are those of Davies (1978) and Taylor (1979) which include publications up to about 1976/1977.

2.2 Methodology for studying effects

From the point of view of fishery management the effects of pollutants on marine organisms and their habitat must allow an acceptable level of productivity. From the point of view of environmental protection major alterations of the marine environment cannot be accepted. Not mere survival of important organisms but the maintenance of truly viable populations is required. This can only be guaranteed if successful reproduction can be achieved (Perkins, 1979). This means that in order to assess the hazards of pollutants information on their effects not only on adults but also on reproduction and development is needed. Many biological effects of pollution may not show up in the bioassay test for acute toxicity since the effects develop slowly without increasing acute mortality. Long-term experimental exposure to sublethal concentrations is necessary to estimate the reproductive success, growth, alterations in the life span, adaptations to environmental stresses, feeding habits, migration pattern, changes in physiological and biochemical functions, predisposition to diseases etc. (Water Quality Criteria, 1972; Perkins, 1979). The estimation of long-term effects from short term acute exposure (LD-50 bioassay) applying an "application factor" is questionable as short term acute effects may have a different biochemistry and physiology than long-term chronic effects. Moreover, in LD-50 bioassays for marine biota the organism is exposed only to one route of entry, namely the direct pathway from water, and the effects of pollutants present in food are completely neglected.

However, even if appropriate data on single species reactions to pollutants during a life cycle are available, the effects of these pollutants on ecosystems cannot be easily predicted. Natural changes of ecosystems are not well enough understood to distinguish between the effects of specific pollutants and changes occurring naturally. Only in certain circumstances can specific pollutant effects on natural ecosystems be identified. The effects in large enclosed ecosystems can help to understand the possible effects of pollutants but their application has so far been restricted to pelagic environments. At present the data available do not seem adequate to assess the general risk of Hg on marine biota and ecosystems.

Evidence presented in section 1.4.3 shows that the uptake of Hg in marine organisms depends both on the chemical species of Hg and on the route of entry into the organism. Organisms which belong to the first trophic level such as algae and aquatic plants take up inorganic and organic Hg directly from the surrounding sea water. Since the first trophic level enriches Hg by a concentration factor of about 5,000 over the concentration in sea water the uptake at higher trophic levels should occur primarily through the foodchain. MeHg is an accumulative pollutant with a near 100 % uptake efficiency (absorption) and very long retention times (Tb of years) while the uptake efficiency of inorganic Hg is less than 10 % and its Tb in the order of tens of days. This means that the older an organism becomes the more Hg will be present as MeHg. Since organisms belonging to high trophic levels generally feed on larger fish than organisms in lower trophic levels, the distribution between inorganic Hg

and MeHg in the organism's tissues will shift from inorganic Hg towards MeHg, making the intake of MeHg through the foodchain of increasing importance in higher levels of the foodchain. In reviewing the data available it is unfortunate that with the exception of one feeding experiment with marine mammals no data seems to have been published on the effect of inorganic Hg and MeHg supplied to marine organisms via the foodchain. Therefore only the data on algae and bacteria consider dominant routes of exposure. In these cases the exposure levels are usually only nominal because effective Hg concentrations in the sea water have rarely been determined. Depending on the type of sea water used marked reduction in the exposure level due to sorption to organic matter and the test organisms has been observed (e.g. in phytoplankton batch experiment discussed in section 2.3).

2.3 Effects on marine biota

Phyto- and Zooplankton

Davies (1978) reviewed the effects of heavy metals on phyto- and zooplankton organisms. If the phytoplankton organisms were tested in batch culture the nominal concentration of Hg in the sea water was reduced markedly during the first days and most of the Hg was associated with the algae (Smith, 1983). Later during growth the cell number increased and the Hg concentration per cell decreased rapidly, reducing the internal and external exposure level. Without chelators the lowest concentrations reported to give effects range from 0.02 to 0.35 µg Hg/litre. However, some algae can withstand much higher Hg concentrations: 1 to 10 µg Hg/litre. The greater tolerance is due to a reduced uptake of Hg (Davies, 1976).

Apparently different strains of the same species have different tolerances. Dunaliella tertiolecta tested by Davies (1976) was 1,000 times less sensitive than the same species examined by Sick and Windom (1975). A comparison of the effects of $HgCl_2$ and MeHg showed that an inhibition of C-14 uptake of natural phytoplankton populations began at less than 0.1g Hg/litre for MeHg and at 1 µg Hg/litre for $HgCl_2$ (Knauer and Martin, 1972). For comparison Holderness et al., (1975) observed that growth of the freshwater green algae Coelastrum microporum was not inhibited at concentrations of 0.8 µg MeHg/litre. Inhibition started at 3 µg MeHg/litre. In zooplankton organisms 2 µg Hg/litre decreased the faecal pellet production during the first 2 days but not in successive days (Reeve et al., 1977), probably because the effective Hg concentration had decreased.

Macrophytes

Fucales (seaweeds) exposed in a continuous flow system to concentrations of Hg ranging from 0.9 to 1250 µg Hg/litre (as $HgCl_2$) showed that at the lowest concentrations tested no effects could be detected in the growth of vegetative apices. A small reduction of growth as compared with controls occurred at concentrations greater than about 10 µg Hg/litre. (Stromgren, 1980).

Bacteria

Very few data exist on toxicity of Hg compounds on marine bacteria. Jonas et al. (1984) observed that natural populations from Chesapeake Bay showed 40 to 60 % growth inhibition at 1 µg inorganic Hg/litre. A similar

inhibition by MeHg was observed at 1 µg Hg/litre. Some toxic effects of MeHg were observed already at 0.1 µg Hg/litre. Unfortunately, the authors did not test lower concentrations of inorganic Hg so that the onset of the inhibition by inorganic Hg is not known. Their data seemed to indicate that inorganic and organic Hg have the same toxicity to marine bacteria. Pan-Hou & Imura (1981) found differences in the minimal inhibitory concentrations of $HgCl_2$ and CH_3HgCl on pure bacteria strains isolated from the intestines of yellow fin tunas. Of the 14 strains tested 9 showed effects at lower concentrations, 800 to 1,600 µg CH_3HgCl and 4,000 to 8,000 µg $HgCl_2$/litre. Five strains were more resistant and showed effects only at 6,400 to 12,800 µg CH_3HgCl/litre and 1,600 to 32,000 µg $HgCl_2$/litre. It is not clear why the strains examined by Pan-Hou & Imura (1981) were about 1,000 times more resistant than the natural populations studied by Jonas et al., (1984). The strains of Pan-Hou & Imura were obtained from another author and could have been isolated on a medium which was selective for Hg-resistant strains.

Crustaceans

The LC-50 at 96 hours for newly hatched zoeae of Palaemonetes vulgaris (shrimp) were 10 µg $HgCl_2$/litre for unfed larvae and 15 µg $HgCl_2$ for larvae fed with Artemia salina. The effect levels after 48 h exposure were estimated at 5 µg $HgCl_2$ for fed and 3 µg $HgCl_2$ for unfed larvae. Transferring larvae into clean sea water after a 48 h exposure to study delayed effects showed that none of the larvae exposed to 32 µg $HgCl_2$ survived more than one day. Forty-five hours exposure to 10 and 18 µg $HgCl_2$/litre markedly delayed the first molting and caused deformations. The growth of young Penaeus indicus was not significantly reduced at exposures up to 6 µg Hg/litre (McClurg, 1984).

Vernberg & Vernberg (1972) and DeCoursey & Vernberg (1972) showed that U. pugilator adults (fiddler crab) could survive for months in seawater containing 180 µg Hg/litre while all stage I zoeae died after only 48 h when exposed to the same concentration. In three species of fiddler crabs 100 µg MeHg/litre had no effects on regeneration of limbs and molting (Weis 1977). However, this concentration caused a complete inhibition of melanogensis in U. thayeri, partial inhibition of U. pugilator but no inhibition in U. rapax. Five hundred µg MeHg/litre inhibited U. thayeri the most and U. rapax the least. Inorganic Hg inhibited limb generation at 1,000 µg Hg/litre but had no effect at 100 µg Hg/litre. Pre-exposure of U. pugliator to 60 to 100 µg MeHg/litre did not reduce the inhibitory effect of 500 µg MeHg/litre, although differences in the inhibitory effect could be observed when three populations from an unpolluted site, a slightly polluted site and a chronic polluted site were compared. The inhibitory effect was smaller in the population from the chronic polluted site (Callahan & Weis, 1983). This may indicate that Hg does not induce metallothionein although it is induced by other pollutants. Similar results were obtained by Green et al., (1976) who found that pre-exposing postlarval shrimps (Penaeus setiferus) to 0.1 and 0.5 µg Hg/litre for 59 days did not increase the LC-50 value at 96 hours obtained for non pre-exposed shrimps. Chronic exposure of the shrimp to 0.5 and 1 µg Hg/litre did not affect respiration rate, growth, or molting frequency. Higher concentrations were not tested.

The brine shrimp is not a marine organism, but the effects seen in this shrimp may apply also to marine crustaceans. Experiments with the brine

shrimp exposed during an entire life cycle to inorganic and MeHg in water showed a significant reduction in adult lifespan at 10 µg $HgCl_2$ and 5 µg MeHg (Cunningham & Grosch, 1978). The survival of nauplii from treated parents was not reduced at 10 µg $HgCl_2$/litre but reduced at 1 µg MeHg/litre. Pairs exposed to 10 µg $HgCl_2$/litre exhibited only a slight reduction in brood production while pairs exposed to 5 µg MeHg/litre and higher concentrtions did not produce any nauplii.

Molluscs

Very few data exist on molluscs. The 7d-LC_{50} for mussels (M. edulis) is 150 µg Hg/litre (Martin et al., 1975). Growth of the shell is reduced to 50 % after expsure to only 0.3 µg Hg/litre (Stromgren, 1982). At concentrations above 1.6 µg Hg/litre growth stopped within 3 days. This does not seem to agree with the 7d-LC_{50} referred to above.

Fish

The killifish Fundulus heteroclitus, because it is easy to culture, was used for several studies on toxicity of inorganic Hg and MeHg. Sharp & Neff (1985) exposed embryos of F. heteroclitus at different times after hatching to various concentrations (0 to 100 µg Hg/litre) of $HgCl_2$ and MeHg. Comparing times after hatching, mortality and abnormal development showed that embryos exposed immediately after fertilization were more sensitive to both $HgCl_2$ and MeHg than older (up to 5 days) embryos. In general MeHg was more toxic but the relative toxicity to $HgCl_2$ varied widely from about half as toxic to several times more toxic. Embryos of the killifish Fundulus heteroclitus from polluted and unpolluted sites exposed for one week to 30 µg MeHg/litre exhibited different degrees of malformation. The embryos from polluted sites had virtually no anomalies while those from the other site showed a range of malformations from unaffected to rather severely affected (Weis et al., 1981b). When exposed to 50 µg MeHg/litre 55 % of the embryos from the polluted site had no malformations, while most batches of the embryos from the unpolluted site showed marked malformations. As a compari- son, the 96h-LC_{50} in adult F. heteroclitus for inorganic Hg ranges from 230 to 2,010 µg Hg/litre, which is about 8 to 70 times greater than the terato- genic dose for this species (Jackim et al., 1970; Klaung et al., 1975). For MeHg the 96h-LD_{50} in F. heteroclitus larvae is 5,320 µg MeHg/litre (Weis et al., 1985). When Fundulus heteroclitus adults were maintained in only 5 µg MeHg/litre they failed to produce additional clutches of eggs (Weis et al., 1985).

In order to investigate if pretreatment with MeHg could increase the tolerance against to later exposure to MeHg Weis et al., (1982) investigated pretreatment of embryos and adults of F. heteroclitus. They observed that embryos from an unpolluted site showed more malformations after exposure to MeHg than after exposure to $HgCl_2$. Embryos from a polluted site, however, had a lower tolerance to $HgCl_2$ than to MeHg. Metallothionein was found in some batches of unfertilized eggs but at very low concentrations and probably too low to have any influence on toxicity. After exposing adult fish to pretreatment with 10 µg MeHg and a subsequent exposure to 10 to 50 mg MeHg/litre, the caudal fins were regenerated more slowly than in control fish. The exposure of embryos to MeHg did not increase the level of metallothionein over controls (Weis, 1984). Therefore, it seems that the acquired greater tolerance of embryos from a polluted site must have been induced by trace

metal compounds other than MeHg. Weis (1984) observed that eggs had very little metallothionein (MT) but untreated embryos of tolerable clutches had twice as much MT as non-tolerable clutches at the time of hatching. This suggests that MT is produced during embryo development. Treatment of embryos with either Hg++ or MeHg did not produce any MT.

An interesting experiment on fresh water fish may be mentioned here because it involves several generations (McKim et al., 1976). Exposure of three generations of brook trout to MeHg in freshwater only (food was not contaminated) showed that MeHg concentrations up to 0.3 µg Hg/litre had no effects on survival, growth and spawning success in any of the three generations. The authors estimated that no effect would occur at concentrations below 0.93 µg Hg(as MeHg)/litre (hardness 45 mg/litre pH 7.5). On the other hand the mean 96h-LC_{50} for 20-wk-old (12g) fry and yearlings was 75 µg Hg/litre (as MeHg). This would result in an application factor between 0.004 and 0.013. A follow-up on the toxicity studies showed that concentration factors (CF) between water and tissue ranged from 1,000 to 63,000. Blood, spleen and kidney had the highest Hg levels, followed by liver, gill, brain, gonad and muscle. Of the total MeHg body burden 90-95 % was located in muscle. Mean muscle concentration in first generation trout dying after exposure to 930 ng Hg (MeHg)/litre was 23.5 mg Hg/kg FW. In the second generation dying after exposure to 930 ng Hg/litre the mean muscle level was 9.5 mg Hg/kg FW. Relating toxicity to Hg concentrations in the body tissues showed that body levels of 2.7 mg Hg/kg FW had no effect but at body levels of 5-7 mg Hg/kg FW effects could be detected. No appreciable elimination of Hg was observed after 12-16 weeks.

Marine mammals

Ronald et al. (1977) exposed seals to MeHg via food. Two seals given very high daily doses of 25 mg Hg/kg body weight died after 20 and 26 days of exposure after showing severe symptoms of poisoning. The symptoms of lassitude, lack of coordination and partial paralysis indicated that possibly central nervous system damage had occured. Two other seals exposed to daily dosage of 250 µg MeHg/kg body weight showed a reduction in activity and body weight, but in standard blood chemistry tests no abnormal values were seen.

Enclosed pelagic ecosystems

Pulse addition of 5 µg Hg/litre to large plastic containers (1.5 m^3 and 15 m^3) showed that the Hg concentration decreases rapidly in the bulk of seawater and inhibited the relative assimilation rate in the bag without nutrient addition during the whole experiment over 15 days (Kniper et al., 1983). In nutrient-enriched enclosures the phytoplankton growth was inhibited by concentrations above 2 to 2.5 µg Hg/litre in the bulk. Similar observations were made by other authors (e.g. Grice & Menzel, 1978). Pulse additions of 5 µg Hg/litre decreased phytoplankton productivity for 12 days, influenced the distribution of phytoplankton and mesozooplankton species, and reduced the number of copepod nauplii for 34 days. Copepods (Pseudocalanus) taken from the enclosure failed to molt until the concentration of Hg in the enclosure had dropped below 2 µg Hg/litre. Pulse additions of 1 µg Hg/litre on the other hand had no observable effects. Studying the biochemistry and toxicity of Hg in a controlled experimental ecosystem Wallace et al. (1982) found that the high affinity of Hg to the organic matter present in the system was the most important parameter governing the distribution

of the chemical species of Hg. Ninety percent of the Hg was present in particulate, colloidal and high molecular weight dissolved forms of Hg. If these fractions of organic matter were removed from the sea water by ultrafiltration from the sea water bioassays showed that 1 µg Hg/litre was toxic to natural phytoplankton populations.

3. HUMAN HEALTH ASPECTS

3.1 Reference documentation

There have been many reviews of human health effects of Hg exposures. Mercury compounds were the subject of the first Environmental Health Criteria Document by WHO (1976a). A re-evaluation was later carried out (WHO, 1980) and an updated Environmental Health Criteria Document for MeHg is currently being prepared by WHO, within the framework of the International Programme on Chemical Safety. Several other publications (e.g. Clarkson, 1984; Berlin, 1986) give good summaries of human exposures and effects and in section 1.1 reviews of Hg occurrence in the environment were referred to. The reports on exposed populations in Iraq (e.g. Bakir et al., 1973; WHO, 1976b; Marsh et al., 1980), in Japan (Swedish Expert Group, 1971; Tsubaki & Irukayama, 1977), and recent data from Canada (Methylmercury Study Group, 1980) have been useful in establishing the dose-effect and dose-response relationships.

3.2 Toxicokinetics

Mercury has no known metabolic function in human beings and, therefore, even low concentrations in the body may be considered potentially harmful. Different chemical forms of Hg have different toxic properties. Mercury in fish and seafood occurs mainly as MeHg (the most toxic form) and partly as inorganic Hg bound to organic molecules (section 1.5). MeHg is mostly absorbed by ingestion and this can lead to exposure levels sufficiently high to be of health concern. This type of exposure will therefore be discussed more in detail.

Swallowed MeHg is absorbed almost completely (95-100 %) in the intestines, also when it is bound to protein as in naturally occurring forms in fish. It is rapidly distributed throughout body tissues, the distribution phase being complete in approximately 3 to 4 days (Clarkson, 1984). Animal experiments indicate that the distribution of Hg among the different tissues after the administration of MeHg is more uniform than after the administration of inorganic Hg salts. MeHg passes the blood brain "barrier" into the central nervous system and the placental "barrier" into the fetus (Berlin, 1963). About 10 % of ingested MeHg ends up in the brain and 7 % in the blood. A rough rule of thumb is that in a 70 kg person approximately 1.0 % of the body burden of MeHg is found in one litre of blood. The brain to blood concentration ratio in humans is about 5 to 1 (Clarkson, 1984). There is also a relatively constant ratio between concentrations in blood and hair, making hair a convenient indicator of body levels. The concentration of Hg in hair near the scalp is 200 to 300 times higher than in blood. Segmental analysis of Hg in hair can be used to evaluate blood levels (and body burden) at the different times when the hair segments were formed (Bakir et al., 1973).

After experimental ingestion of MeHg in humans two phases of clearance from blood have been identified with half-times of about 8 hours, representing distribution to the tissues, and about 50 days, representing excretion from the body (Miettinen, 1973; Kershaw et al., 1980). Other studies have confirmed the slow component half-time in blood and the whole body half-time has been estimated at 70 days (WHO, 1976a). Data derived from human experimental studies indicate that the blood level is proportional to the long-term intake when it is in the non-toxic range.

About 80 % of the total excretion of MeHg in human beings is via faeces. There is a considerable secretion of Hg to the intestines via bile and much of this is re-absorbed to create an "entero-hepatic" system. (Ballatori & Clarkson, 1985). The total daily excretion via urine and faeces is about 1 % of the body burden (Clarkson, 1984). Virtually all Hg excreted is in the form of inorganic Hg even after MeHg exposure. It is known that the intestinal microflora can demethylate MeHg (Rowland et al., 1983, and Section 1.4.2). There is also evidence of a slow demethylation in body tissues, because inorganic Hg is secreted via bile after MeHg exposure (Berlin et al., 1975), but the site of demethylation is not known.

3.3 Health effects

The toxic effects of MeHg primarily occur in damage of the sensory part of the nervous system. According to (WHO, 1976a) it is expected that 5 % of an adult population will have overt symptoms when the blood concentration of total Hg is between 0.2 and 0.5 mg/litre. This corresponds to 50 - 125 mg Hg/kg hair or to a long-term daily intake of 3-7 µg Hg/kg body weight in the form of MeHg. The foetus and infants are more sensitive than adults to the toxic effects of MeHg. Later reviews of MeHg toxicity have confirmed these conclusions (e.g. WHO, 1980).

The earliest clinical signs and symptoms of MeHg poisoning is abnormal sensation or numbness (paresthesia) in hands, feet and around the mouth. Increased exposure may result in lack of coordination of movements (ataxia), constriction of the visual field, slurred speech, and hearing difficulties. In the most severe poisoning, the patients may develop blindness, deafness, involuntary muscle spasms, paralysis and general physical and mental debilitation (WHO, 1976a). Many patients died in the poisoning outbreak in Iraq (WHO, 1976b). The nervous system is irreversibly damaged by MeHg, but some clinical improvement may occur because the function of some dead neurons are taken over by others and slightly damaged neurons regenerate (Clarkson, 1984). On the other hand, when the exposure is short there may be a latent period between the end of exposure and the onset of the intoxication, because both MeHg accumulation and neurological damage take time (Magos et al., 1978).

MeHg exposure in early childhood and before birth (via the placenta of an exposed pregnant woman) also causes central nervous system damage. In severe cases in Minamata (Tsubaki & Irukayama, 1977) the children had cerebral palsy and some of them died. Other authors have reported faulty brain structure (Choi et al, 1978), lower than normal brain size, blindness and severe motoric disorders (Gerstner and Huff, 1977) after intra-uterine exposure. In Minamata a higher Hg concentration in umbilical cords of children with mental retardation than in control children was found (Harada et al., 1977).

In the poisoning incident in Iraq less severe symptoms of brain damage were studied. Marsh et al. (1980) found a significant increase in the occurrence of developmental retardation, neurological signs and seizures in children exposed prenatally.

Apart from the nervous system effects MeHg has no known other effects of relevance to the marine food chain. IARC has not included MeHg or any other Hg compound in its latest list (IARC, 1985) of potentially carcinogenic substances.

In animal experiments and in cell culture selenium and vitamin E can delay the onset of MeHg intoxication, but whether selenium or Vitamin E at realistic doses can interfere with the toxic effects of MeHg in human beings is unclear.

3.4 Total exposure to mercury compounds

Most of the data on Hg in foodstuffs only report the total Hg content and do not distinguish between MeHg and other Hg compounds (WHO, 1976a). However, as stated in Section 1.5.6 to 1.5.10, most Hg in fish and other seafood is in the form of MeHg. The total Hg intake via food in some countries has been reported at 20 µg/day or lower (WHO, 1976a). Cigna-Rossi et al. (1967) estimated for the average Italian an intake of 7 to 12 µg total Hg/day. Schelenz & Diehl (1973) reported 10 µg/day for the Federal Republic of Germany and Cohen (1974) reported 5 to 10 µg Hg/day for England. For Sweden, it was estimated (Swedish Expert Group, 1971) that about 5 µg Hg/day came from sources other than fish and marine food (i.e. drinking water and terrestrial food in Sweden.). Bread and cereals contributed more than 50 % to the Hg intake from terrestrial food and about the same amount came from fish and marine food. As Hg pesticides are no longer used for treating seeds the Hg intake from terrestrial sources may have decreased since the 1960s. A large general population survey in Australia (WGMF, 1980) estimated that terrestrial food contributed on average 4 µg Hg/day to the total intake. Most studies of Hg content in food suggest that the contribution of MeHg from terrestrial food is negligible from the toxicological viewpoint. (Swedish Expert Group, 1971). However, the MeHg intake from fish in the Swedish population varied between 0 and 800 µg/day. As will be seen in the next section, even higher intakes have been recorded in groups with extreme fish intake.

3.5 Contribution of mercury from marine food

3.5.1 Primary route - descriptive data

A high percentage of the total Hg in the muscle tissue of fish and marine mammals is in the form of MeHg (Section 1.5) and in many studies total Hg is analysed and all of it is assumed to be MeHg. By contrast, the majority of Hg in the liver of fish and aquatic mammals has been reported to be in the inorganic form (Section 1.5.8 and 1.5.10).

The predominant source of human exposure to MeHg is via the ingestion of fish although terrestrial food items can also contain Hg. Most commercially important fish have concentrations of MeHg in muscle tissue of less than 0.2 to 0.3 mg/kg FW. Much higher levels (up to 100 mg Hg/kg) were found in the polluted Minamata and Niigata areas (WHO, 1976a). Elevated levels (0.5 to

16 mg Hg/kg FW) are also found in predatory species even in open waters remote from pollution sources. These species include mainly large, old tuna, swordfish, marlin and shark, but also large striped mullets, king mackerel, halibut and shrimps.

The high concentrations of MeHg are also found in aquatic mammals such as seals, porpoises, whales and walruses which form a large and regular part of the diet in some populations, as for example Inuits (Inughuits) and possibly whalers (see Chapter I on food consumption pattern).

Estimates of MeHg intake from fish are often compared with the PTWI or Provisional Tolerable Weekly Intake set by a FAO/WHO Expert Group (FAO/WHO, 1972) at 200 µg MeHg per week or 300 µg total Hg per week for a 70 kg person. It incorporated an assumed "safety factor" of 10 (Section 3.6). Even though it is not specifically stated in FAO/WHO (1972), the calculations of equivalent PTWI's per kg body weight shows that the PTWI was based on an adult person weighing 60 kg. Many other "adult" risk estimates are based on a 70 kg person (e.g. Swedish Expert Group, 1971; WHO, 1976a).

As mentioned in the Introduction (section 2 on dietary intake considerations), there are great variations in seafood consumption within a population. People in "critical groups" with particularly high seafood consumption can often be identified; e.g. the families of workers in the fishing and other seafood industry (harvesting, processing and trading seafoods), ethnic groups subsiding on seafood (e.g. Inuits) and people on special diets high in seafood.

The distribution of MeHg intake via seafood in a population is not necessarily the same as the distribution of seafood intake. Different seafood species have different common concentration ranges (Section 1.5) and for the same species there are variations with size and geographic location.

A number of studies of Hg consumption via fish have been carried out. A full review will not be presented here. Instead we will exemplify the type of data available by referring to some large scale surveys and some more local investigations.

Studies of the MeHg intake in "general population" groups have been carried out in some countries, using either calculated intakes from fish consumption data and data on Hg concentrations in fish or Hg concentrations in hair or blood as indicators of intake (Section 3.2). A study calculating the intake from fish consumption data (NMFS, 1978a) was carried out on 24,650 people in the USA. They filled in diaries about the amounts and types of fish consumed during a month (8 % of them did this for a year). Average Hg concentrations in 135 different types of freshwater and marine fish and shellfish were evaluated, based on analysis of 18,900 specimens. Based on the distribution of individual intakes and the estimated Hg concentrations in the species consumed, it was calculated (NMFS, 1978b) that "with no regulatory control of the Hg content of marketed fish 99.81 % of all respondents had an upper limit Hg intake lower than their personal allowable daily intake (based on 30 µg Hg per day for a 70 kg person) at a 95 % confidence level. An action level of 1.0 mg Hg/kg in fish for regulatory control would increase this percentage to 99.87 % and an action level of 0.5 mg Hg/kg would increase it to 99.89 %".

In Australia (WGMF, 1980) a general population survey of the fish consumption during one week by 19,620 people ("Household survey" - probably by mailed questionnaires. No description of the methods in the report) found that 4.7 % never ate fish at home and 40 % never ate fish out of the home. At the other end of the consumption scale 6.4 % ate more than 500 g fish in the survey week and 0.9 % (177 persons) ate more than 1,000 g/week. Based on a weighted average Hg concentration in fish and seafood of 0.15 mg/kg (WGMF, 1980) these intakes would respond to 75 µg Hg/week and 150 µg Hg/week. It is pointed out in the report that almost all fish and shellfish consumed in Australia is of marine origin. As mentioned in Section 3.4, in Australia terrestrial food contributed on average 4 µg total Hg/day (28 µg total Hg/week) to the total Hg intake.

In two separate surveys a total of 311 high fish consumers, mainly consuming four or more fish meals per week, were selected for a more detailed Hg intake evaluation. Such a fish intake is reached by about 3 % of the general population surveyed earlier (WGMF, 1980). Within this high fish consumer group 53 % consumed more than 500 g fish/week, 13 % consumed more than 1,000 g/week and 3.6 % more than 2,000 g/week. This latter small group of the extreme consumers (estimated intakes of more than 300 µg total Hg/week) was identified in the majority as people connected to the fishing industry. The data from the household survey and this latter survey do not match exactly. The group consuming more than 1,000 g/week can be estimated as 13 % times 3 % or 0.39 % (0.13 x 0.03 x 100), which is less than the 0.9 % referred to earlier. Some people with infrequent fish consumption, and therefore excluded from the detailed study, could have consumed more than 1,500 g/week, but a more likely explanation is a systematic difference in the results of the two study methods. The detailed study is likely to be more accurate. Based on that study it can be calculated that the extreme group consuming more than 2,000 g/week comprises only 0.1 % (3.6 % of 3 %) of the whole population. This is similar to the figure estimated for USA (NMFS, 1978b). In the whole of Australia this group may amount to about 15,000 people.

Several studies of high fish consumption groups have been carried out in Italy. Nauen et al. (1983) conducted a survey in three Italian locations. Special attention was given to fishermen and their families. The authors found that a high percentage of the persons interviewed exceeded their daily allowance, among them many children. In fact the maximum average intake for an individual was estimated for a 3-year old child which reached 8.6 times the daily tolerable allowance. Paccagnella et al. (1973) selected the population of Carloforte (Sardinia) for an epidemiological study, because its average consumption of seafoods was about 4 times the national Italian average and because, during the summer months, fresh tuna meat was consumed. From 6,200 residents 195 persons chosen at random agreed to give information about their food habits, take a medical examination and allow a blood and hair analysis. Based on Hg analysis of tuna and other seafoods and the seasonal seafood consumption patterns it was estimated that the average weekly intake of Hg in the summer was 150 µg and in the winter 100 µg. The group with the highest consumption (14 seafood meals per week) had an estimated weekly Hg intake of 700 µg in the summer and 460 µg in the winter. Their average hair level was 11 mg Hg/kg (range: "not detected" to 60 mg/kg), which fits well with the (WHO, 1976a) estimate that of an average intake of 300 µg Hg/week the hair level would be about 5-6 mg total Hg/kg.

Riolfatti (1977) compared hair Hg levels in an inland town with a coastal town, where 13 % of the 52 persons examined had consumed more than four fish meals per week. One man in the coastal town had hair levels which fell within the range of possible earliest effects (WHO, 1976a), i.e. his hair concentration was about 45 mg Hg/kg and six others reached hair concentrations between 16 and 20 mg/kg. In the inland town relatively high hair concentrations were also observed. One woman had about 30 mg/kg and three had levels between 16 and 25 mg/kg, despite the fact that none of the persons examined in the inland town had consumed more than 2 fish meals per week. As no quality assurance of the analytical method was carried out and the results are unusually high no conclusions can be drawn.

Bacci et al. (1976) studied the total Hg and MeHg concentration in blood, urine, hair and nails of 16 persons from the town of Vada, who consumed from 0 to more than 6 meals of seafood per week. The fish came from the banks of the Vada river about 10 km west of the Solvay chlor-alkaline plant. As expected, the Hg concentrations increased with the amount of seafood meals consumed. The concentration in the hair ranged from 4 to 110 mg Hg/kg.

High fish consumers have been studied also in Japan (Doi & Ui, 1975). Of 34 tuna fish retailers 22 ate 100 to 200 g tuna meat daily besides 70 to 300 g shellfish and other fish meat. One person consumed daily 200 g FW of tuna meat in addition to 1,000 g of other seafoods. The daily tuna consumption of tuna fishermen aboard the ship ranged from 50 to 400 g during seasonal periods of between 130 and 180 days. Assuming an average concentration of 0.5 mg Hg/kg FW in tuna and the average daily consumption rate, the weekly intake from tuna for these fishermen is about 500 µg Hg. The retailers ingested an additional Hg intake of about 140 µg Hg per week from other seafoods which contained on an average 0.1 mg Hg/kg. This high Hg intake, in particular from tuna, was reflected in high hair and blood concentrations. The hair of these tuna fishermen contained from 25 to 46 mg Hg/kg. The mean Hg concentration in the hair of the retailers was 26 mg/kg (range 6.4 - 44 mg Hg/kg) while blood levels averaged 100 µg Hg/litre (range 45 - 175 µg Hg/litre). One individual had at one time 65 mg Hg/kg hair.

The high levels in marine mammals eaten by Inuits raised their Hg levels. Examining Hg levels in cord blood, placenta hair and milk from 38 maternal-infant pairs from Anchorage and the Yukon-Kushokwin delta showed that women eating high amounts of seafood had much higher Hg levels than women eating little seafood (Canadian Government Report, 1984). Studies in Greenland found average hair mercury levels of about 15 mg/kg in people eating seal six or more times per week (Hansen, 1981). Based on the dietary habits it was calculated that the average daily intake of total Hg could be 2,400 µg/week (97.5 percentile = 13,700 µg/week). The average weekly MeHg intake in this high exposure group was estimated at 500 µg, based on blood analysis Hansen, 1981). This agrees with the reports of a high consumption of Hg via liver of marine fish and mammals among Inuits (Section 3.5.1) and such livers have a low relative MeHg content (Section 1.4.4).

Studies have also been carried out in Canada, Sweden, Japan, Peru, Seychelles, Samoa, Papua New Guinea and New Zealand. In the interest of brevity they have been excluded here, but it should be pointed out that in many places people have been found with a MeHg intake above the PTWI.

3.5.2 Primary route - calculation of intake

The data referred to in previous reviews and the additional data cited above indicate the dominant influence fish and marine food consumption has on daily MeHg intake. In order to illustrate the effect on MeHg intake of different fish consumption patterns and Hg concentrations in fish, Table 5 was prepared. More categories than those referred to in Chapter II were included so that the intakes for different population groups can more easily be estimated.

One typical fish meal has been estimated at 150 g (Chapter II), so an intake of 20 g fish per day is equivalent to about one meal per week. The Hg concentrations in fish are representative for sardine and cod (0.1 mg/kg), tunafish (0.5 mg/kg) and shark (1.0 mg/kg) (Section 1.5).

For a 70 kg person a long-term daily intake of 30 µg MeHg can be expected to lead to a hair level of 6 mg Hg/kg. Smaller people will have a proportionately higher hair Hg value at the same intake level (e.g. 8.4 mg/kg hair for a 50 kg person). Some of the high fish consumers referred to in the studies mentioned above consumed 150 g fish/day or more and had hair Hg levels in the range 30-40 mg/kg, corresponding to daily MeHg intakes of 70-100 µg/day. As seen from Table 5 high fish consumers will exceed the PTWI unless they only consume fish with very low MeHg levels.

On the other hand a person who eats fish with low Hg level (0.1 mg/kg, e.g. sardines and cod) no more than once a week will not exceed a daily MeHg intake of 2 µg (Table 5).

Table 5. Daily intake (in µg/day) of methylmercury via fish resulting from different combinations of fish consumption and methylmercury concentration in fish

Concentration in fish mg MeHg/kg	Intake, g fish/day						
	20	40	60	100	150	300	1000
0.1	2	4	6	10	15	30	100
0.25	5	10	15	25	38	75	250
0.5	10	20	30	50	75	150	500
0.75	15	30	45	75	113	225	750
1.0	20	40	60	100	150	300	1000
1.25	25	50	75	125	188	375	1250
1.5	30	60	90	150	225	450	1500

3.5.3 Secondary routes

A sizable proportion of the marine fish caught is used to produce fish-meal, which is used partly to feed poultry, pigs and other farm animals. Other uses are as petfood and raw materials. MeHg from fish may therefore lead to human intakes via food other than fish and in assessing the hazards of

marine pollution due to Hg it is necessary to take this secondary route into account.

The total Hg and the MeHg concentrations in fish meals were higher than in meat and bone meals (Szprengler, 1975). For example, chickens fed herring meals containing 0.014 and 0.018 mg total Hg/kg DW raised their body levels (March et al., 1974). According to unpublished FAO statistics (Crispoldi, pers. com.) in 1983 about 5,200,000 metric tonnes of meals and other animal feeding stuffs were producted world-wide. Assuming a fresh weight/dry ratio of 5 this corresponds to about 26,000,000 tons or about 23 % of the total world catch of 76,470,600 tons. Major producers are Japan (21.7 %), Chile (15 %), USSR (11.5 %), USA (9 %), Norway (6.6 %), Denmark (6 %), Thailand (4 %), Peru (4 %) and S. Africa (3 %). The species used for the production of fishmeal vary with regions. In Europe fish meal is produced mainly from capelin and herring, in North America from menhaden species, in Middle and South America from anchovy and clupeoid species. Wastes from tuna, herring, mackerel, lobster, crab, shrimp and various other species are also used. In some countries meal is still produced from whales. Of course, not all fishmeal produced is used in the country of origin. For example, Peru exports 96 %, Chile 91 % and Norway 81 %, while large amounts are imported by many European countries.

The Hg content of these fishmeals can be estimated from the Hg concentration in fresh species (see section 1.5.8) and a DW/FW ratio of 5. Direct determination of herring meals from British Colombia, Canada, Newfoundland, Denmark and Norway range from 0.09 to 0.29 mg total Hg/kg DW (Anonymous, 1971). White fish meals from Britain, Canada, Denmark, Iceland and South Africa also had similar ranges (0.04 to 0.29). Beasley (1971) found a mean concentration of 0.44 mg total Hg/kg DW in Engraulis mordax from the Californian coast, 0.6 mg total Hg/kg DW in Clupea harengus from the Massachusetts coast, 0.5 mg total Hg/kg DW in menhaden (Brevoortia patrona) from the coast of the state of Mississippi, and 0.34 mg total Hg/kg DW in the menhaden (B. tyrannus) from Chesapeake Bay. Taking into consideration that these are dry weight levels and applying a DW/FW ratio of 0.2 will reduce these levels to one-fifth on a fresh weight basis.

3.6 Evaluation of potential health effects

The high Hg concentrations observed in edible marine organisms and the high intakes reached by some population groups raise the question of possible health risks caused by these intakes. WHO (1976a) estimated that the earliest poisoning symptoms in the most sensitive group of an adult population may appear following a long-term daily ingestion of 200 to 500 µg Hg (as MeHg) for a 70 kg person. This long-term intake is associated with a blood level approximately in the range of 200 to 500 µg/l and a hair concentration of between 50 to 125 mg Hg/kg. FAO/WHO (1972) suggested a Provisional Tolerable Weekly Intake (PTWI) of 200 µg Hg (as MeHg) or 300 µg (as total Hg) for a 70 kg person or 3.3 µg MeHg per kg body weight per week and 5 µg total Hg per kg body weight per week. These PTWI for Hg have been reconfirmed (UNEP, 1980), but it was pointed out that "there are indications that pre-natal life is more sensitive to MeHg" and it was "strongly recommended that further research be directed towards the recognition of any early health effects associated with low levels of exposure". It was also recommended (UNEP, 1980) that "the total intake of MeHg through seafood should be limited to protect groups believed to exceed the WHO PTWI". It was suggested that such a limitation of intake could be achieved by:

a. advice on dietary intake
b. establishment of maximum limits for Hg in seafood
c. restrictions on the size of fish allowed for consumption
d. banning or limiting of fishing in certain areas
e. limitation of anthropogenic discharges.

The reevaluation of the WHO Environmental Health Criteria for Mercury (WHO, 1980) emphasized the sensitivity to MeHg of the growing fetus. According to the review by Clarkson et al. (1985) the fetus may be as much as 10 times more sensitive than the adult. It should be pointed out that the response estimates for the prenatal effects have wide confidence limits due to the small number of children studied. Another factor of importance in this comparison is the difference in type of effect. The risk estimates which lead to the establishment of the adult PTWI (FAO/WHO, 1972) were based on the occurrence of symptoms in exposed people, whereas for the foetus, effects on development in early childhood were considered. No practically measurable early biochemical, physiological or pathological indicator of MeHg damage to the nervous system has so far been discovered.

Table 5 showed the daily intakes of MeHg that could be reached by different combinations of fish consumption and MeHg concentration in fish. The PTWI for adults in general (70 kg body weight) is equivalent to a MeHg intake of 30 µg/day.

As has been discussed in Section 3.5 and in Chapter I a significant proportion of some populations hardly ever eat fish or other seafood. For those who do, one seafood meal per week (about 20 g fish per day) is the most commonly reported. Such a fish intake will not lead to the PTWI for adults being exceeded even if the average MeHg concentration in fish is very high (Table 5).

It has been estimated that the world average fish consumption is approximately 16 g/day (Chapter I). This is similar to the average consumption of many individual countries. For those populations where the fish consumption distributions have been studied it has been estimated that the 97.5 percentile may be about 3 times the average. (WHO, 1985). If this applies on a world basis it means that 2.5 % of the population would consume more than 48 g/day. From Table 5 it can be seen that at this intake level the PTWI can be reached by MeHg concentrations in fish lower than 0.75 mg/kg.

Population groups consuming one "normal" fish meal per day (150 g fish/day) will reach either of the two PTWI at very low MeHg concentration in fish. As stated in section 3.5 these high fish consumers are the "critical group" and further efforts should be made to ascertain whether health effects of MeHg do occur. This is of particular importance for children exposed prenatally and postnatally. The studies of high fish consumers in Italy referred to in Section 3.5 included health examinations, but none of the studies could find obvious clinical effects.

It should be pointed out that the PTWI incorporates an assumed "safety factor" of 10 from an intake that has caused a 5 % prevalence of symptomatic MeHg poisoning. It is compatible with the PTWI that relatively small scale studies fail to demonstrate an increased prevalence of health effects at intakes higher than the PTWI. Even at a 10 times higher intake one should expect only one affected among 20 studied.

In summary, there are population groups consuming MeHg via marine food (particularly fish) to the extent that they exceed the established PTWI. In populations where the average MeHg concentration in fish consumed is high, even rather modest fish consumptions can lead to the PTWI being exceeded. A quantitative estimate of the number of people who exceed PTWI has not been made here, but could be made on a national basis using surveys of Hg in fish and fish consumption and in-depth studies of "critical groups" with a particularly high MeHg intake.

4. CONCLUSIONS ON MERCURY

4.1 Potential harm to living resources

Problems associated with Hg in the marine environment are related to the methylation of inorganic Hg and the subsequent accumulation of MeHg by marine organisms. The MeHg in food is more easily absorbed by the higher organisms than inorganic Hg, and MeHg is also more toxic at the same dose.

Total Hg concentrations in seawater, sediments and biota are generally low, although due to insufficient quality control of the analytical procedures used to determine Hg in these matrices the validity of the data available is not easy to assess. This is especially relevant for the very low concentrations of Hg in seawater. Mercury compounds accumulate in the food chain. In the first trophic level (plankton) the Hg concentrations are about 1,000 - 10,000 times higher than in seawater. In the higher levels the concentrations increase further, but not to the same extent.

High concentration of MeHg are found in long-lived predatory fish at high trophic levels and in a variety of organisms near sources of natural or anthropogenic Hg discharge. High concentrations of Hg are also found in some marine mammals and this is related to their geographical location, longevity and trophic status. The proportion of total Hg which occurs as MeHg in the liver of fish and of marine mammals is lower than in muscle tissue. A possible demethylation in marine mammals is thought to be associated with the co-accumulation of selenium but experimental evidence is lacking.

There exist two belts on the earth in which natural Hg concentrations in these environment are high. In the areas of Hg geochemical anomalies where the higher than average Hg concentrations have been studied, elevated Hg levels are also found in marine biota. Discharges from chlor-alkali and petrochemical plants have locally increased the Hg concentration in the biota. Organisms living within a range of 10 to 20 km distance from the discharges have Hg levels up to 1,000 times above background levels, although any adverse effects on marine biota observed were not attributed to the higher Hg concentrations but to the release of other wastes discharged simultaneously. In Minamata Bay the discharge of MeHg and inorganic Hg from a chemical plant raised Hg levels in marine organisms to 10 to 25 mg Hg/kg FW. It did not result in massive kills of marine organisms, but other effects on marine biota were not studied.

Increasingly stringent controls and changes in process technology are likely to have led to a reduction in industrial Hg discharges from developed countries. On the other hand, the industrialization in developing countries may lead to an increase in the discharge of Hg there.

Mercury is highly toxic to marine organisms (bacteria and algae) in tests relating water concentrations to effects. Since direct uptake of Hg from water dominates only in the first trophic levels (autotrophs and herbivores) the experimental set-up of the tests so far conducted have only validity for the first trophic levels. Toxicity tests which also consider the uptake through the food chain are needed to evaluate MeHg toxicity to marine organisms of higher trophic levels (crustaceans, fish and mammals) in natural habitats. One such study of seals exposed to MeHg produced severe symptoms, possibly due to central nervous system damage.

4.2 Potential hazards to human health

Fish and marine seafood are the main sources of MeHg intake by human beings. Data from studies of large populations indicate that the daily intake of total Hg from other sources than fish and seafood is about 4-5 µg and that probably most of this Hg is inorganic. In the general population fish and seafood may on average contribute another 5-10 µg/day as MeHg. Whether this is of marine origin or freshwater origin depends very much on the geographic area. Individuals belonging to "critical groups" such as workers in the fishing industry (harvesting, processing and trading), ethnic groups fishing for subsistence (e.g. Inuits, American Indians, Ocean Island people) and people on special diets, have been shown to have much higher daily MeHg intakes (up to several hundred µg/day).

In 1972 an Expert Committee of the FAO and WHO established a Provisional Tolerable Weekly Intake (PTWI) for adults at 0.3 mg of total Hg per person, of which no more than 0.2 mg should be present as MeHg. This apparently referred to a 60 kg adult, so it was explained that the intakes could also be set at 5 µg/week per kg body weight for total Hg and 3.3. µg/week per kg body weight for MeHg. These figures can be used to calculate the PTWI for smaller people. (Some other risk estimates are based on a 70 kg adult). These intake values were about 10 times lower than those at which effects have been reported.

The FAO/WHO Committee (1972) pointed out that the levels of MeHg in food of some fish-eating populations will lead to an intake in excess of the PTWI, and recommended that "in these circumstances suitable investigations should be instituted in exposed populations and all possible steps should be taken to keep the levels of MeHg in food as low as possible".

In 1976 WHO reviewed the available data in an Environmental Health Criteria document. It was concluded that a 5 % prevalence of the earliest symptoms of poisoning would occur at a blood Hg concentration of 200-500 µg Hg/l, equivalent to 50-125 mg Hg/kg in hair or a daily long-term MeHg intake of 3-7 µg/kg body weight (for a 70 kg person this means 200-500 µg MeHg/day). These dose-response estimates have been confirmed by the WHO in 1980 and for adults in general no change in the PTWI have been suggested, as the PTWI is about 10 times lower than the intakes mentioned above. However, studies of prenatally exposed children and animal experiments have shown that the developing brain is more sensitive than the adult brain. Therefore in 1980 a WHO Committee strongly recommended that further research should be carried out on this issue. The WHO is currently preparing a revised version of the Environmental Health Criteria for MeHg.

Individuals in the "critical groups" referred to above have been shown to have daily MeHg intakes exceeding the PTWI. Their high intakes are reflected in elevated Hg levels in blood and hair, but health examinations have generally failed to demonstrate an increased prevalence of symptoms of MeHg poisoning. This is compatible with the "safety factor" of 10 incorporated into the PTWI, there being an estimated 5 % chance of effects at an intake 10 times higher than the PTWI. Very large detailed studies are likely to be needed to confirm or rule out effects occurring at intakes in the range between the PTWI and 10 times the PTWI. On the other hand, the studies so far show that "epidemics" of Minamata Disease have not occurred in these "critical groups".

The evaluation of potential human health effects show that groups with a high fish intake, or consuming fish with a high MeHg content, can easily exceed the PTWI. Some "critical groups" can reach MeHg intakes several times the PTWI. Only a few epidemiological studies on the possible effects of extreme intakes have been carried out and most of them have studied adults only, even though the developing brain of the young child or the prenatally exposed fetus is more sensitive to MeHg than the adult brain.

There is a need to carry out national evaluations of fish consumption patterns and Hg levels in commonly consumed fish as well as investigations of "critical groups", in order to assess the public health impact of MeHg in marine foods. Fish surveys will assist in identifying pollution sources and these can then be controlled. However, most of the fish with high MeHg levels live constantly in the open sea and the sources and pathways of this MeHg is unknown. An international coordinated research approach is needed to get the information necessary for governments and international organizations to develop valid guidelines to deal with the problem of MeHg in marine food. Such an international approach should include epidemiological studies of prenatally and postnatally exposed young children, using agreed protocols for measuring effects on mental and physical development.

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IV. SELENIUM

1. SELENIUM IN THE MARINE ENVIRONMENT

1.1 Reference documentation

Apart from reviews by Johnson (1976) and Lakin (1973) dealing with the chemistry and occurrence of selenium in the environment, by Bem (1981) and Gunn (1981) on analytical techniques, and the WHO review of environmental health criteria for selenium (WHO, 1986), most of the information has been obtained from the many individual papers by research workers listed in the reference section.

1.2 General facts

Selenium (Se): atomic weight 78.96, is an element having both metallic and non-metallic properties and is chemically related to sulphur and tellurium. It is released into the environment from volcanic sources, from the combustion of fossil fuels, and in wastes from industrial processes using the element, or disposing of by-products containing it (e.g. copper refining). Selenium occurs occasionally in native sulphur, and as metallic selenides of Pb, Cu, Ag and Hg. It is common in iron and copper pyrites, and is extracted commercially from the anode-slime in electrolytic copper refining.

The element is ubiquitous, but unevenly distributed over the globe. It is now known to be essential in animal nutrition, and there is some evidence that selenium is a nutritional factor in the growth of certain freshwater algae (Lindström, 1984). Some crop plants can absorb high concentrations and be acutely toxic to grazing animals. Its availability to plants depends on the parent material (primarily sedimentary rocks such as shales), the pH and oxidation potential of the soil, alkaline soils favouring the formation of selenate, which is easily absorbed by plants but also easily leached from the surface soils.

Selenium exists in nature in several states of oxidation, and some chemical forms are volatile. The oxidation states are (-II) selenides, (0) elemental, (+IV) selenites amd (+VI) selenates. Despite the similarity to sulphur, the properties of the analogous compounds are usually quite different. Selenate in alkaline and oxidising conditions (e.g. in soils) is very soluble and stable, and available to plants. Both it and selenite are in anoxic conditions readily reduced to elemental selenium. Selenite is slowly oxidised to selenate in the presence of oxygen under alkaline conditions, but is stable in an acid medium. Selenite is also strongly adsorbed to hydrous ferric oxides, and is more biologically reactive than selenate.

Elemental selenium can exist in several allotropic forms and is very stable. It can be formed by natural processes, and may thus be inactivated in the environment. In fossil fuel combustion, selenium is mobilised to a lesser extent than sulphur. Unlike SO_2, SeO_2 is a solid, but is unstable and is reduced in air to selenium. Elemental selenium has been oxidised experimentally to selenite by a soil bacterium (Sarathchandra and Watkinson, 1981).

Selenides of several heavy metals occur naturally in many minerals, and are very insoluble. Iron selenide may be present in soils.

Selenium is bound into proteins and amino-acids, a number of which are analogues of sulphur-containing compounds. The biological degradation of organic selenium-containing compounds does not necessarily follow that of the sulphur-containing analogues.

In the aquatic environment selenium is adsorbed to particulate and colloidal material, but measurable levels of selenite, selenate and organic selenium have been recorded.

1.3 Sources

Selenium occurs in trace amounts in most crustal materials of the earth, but its concentration rarely exceeds 500 µg/g. The average concentration is estimated to be 0.09 µg/g, and the ratio of S to Se in igneous rocks is given as 6,000:1. Volcanic eruptions discharge volatile selenium which is subsequently deposited over the surface of the earth. Sedimentary rocks usually contain concentrations of selenium much higher than the earth's average, values of 0.24-277 µg/g being reported for shales, 0.05-1.12 µg/g for sandstones and 0.0-30 µg/g for carbonate rocks (Lakin and Davidson, 1967). Igneous rocks are impoverished as the result of volcanism.

Shales are the parent materials of about 40% of the earth's soils. Selenium concentrations in soils range from less than 0.1 µg/g in a Se-deficient area of New Zealand to as high as 1,200 µg/g in an organic soil in Ireland (Johnson, 1976). Soils derived from sedimentary rocks are alkaline, favouring the formation of selenates which can be leached by rainfall to lower soil horizons. Selenium as selenite is detectable in fresh and sea waters, but selenite is strongly adsorbed to hydrous ferric oxides (Howard, 1971) much more so than to clays, and at all pH levels from 1 to 8. Transport of selenium in the aqueous environment may thus be largely by sediment transfer.

Selenium is recovered commercially from copper refinery wastes, which also form a source of discharge to the environment. The element is a semiconductor, and is widely used in photoelectric cells and rectifiers. In glass manufacture, small amounts decolorise the green tint caused by ferrous silicate, while larger amounts produce coloured glass. In metallurgy, selenium improves machinability and controls porosity of steels and alloys. It is a catalyst for the petroleum industry and an anti-oxidant in the rubber industry. It has also been used (as selenates and a selenosulphide) for pest control in greenhouses, but is restricted. It has limited use in the form of an anti-dandruff shampoo (selenium sulphide), but has been administered in the clinical treatment of Keshan disease.

Most of the above uses will lead to low levels of environmental contamination of the atmosphere (e.g. from the burning of wastes or refuse) or aquatic environment (discharge to drains, sewers or water courses). Large quantities of selenium enter the atmosphere from fossil fuel combustion, however. Concentrations of selenium in U.S. coal average 3.3 µg/g (range 0.46-10.65), and in crude oil values range from 0.026 to 1.21 µg/g (Lakin, 1973). Mackenzie et al. (1979) have calculated from various published data that emissions of selenium to the atmosphere from combustion of fossil fuels (10,000t) and roasting of sulphide ores (2,000t) total 12,000 tonnes annually. The same authors also estimated that 11,000 tonnes entered the oceans in the suspended load, and 7,200 tonnes in solution, by discharge from rivers.

Concentrations of selenium in the atmosphere are very low, but measurement of atmospheric particulates by Duce et al., (1974) over the North Atlantic and Zoller et al., (1974) in the Antarctic both indicated enrichment factors (relative to the proportion of selenium in crustal materials) of 16 to 18 x 10^3 using a common element such as iron as a reference. The atmospheric selenium is thus of anthropogenic or natural origin, but not crustal (soil). The enrichment factor measured at an active volcano in Hawaii was 56 x 10^3 (Duce et al., 1976). Evidence from the analysis of ice cores in Greenland indicates that, unlike sulphur, there has been no increase in selenium deposition over the past two and half centuries (Weiss et al., 1971). While the increase in sulphur deposition is attributed to fossil fuel combustion, the authors suggest that at high temperatures (whether man-made or volcanic) Se is oxidised to the solid SeO_2 which is readily reduced to elemental particulate Se. Sulphur is oxidised to the relatively stable gaseous SO_2, which is more easily transported for long distances.

Examples of the ranges of concentrations of selenium measured in the atmospheric, freshwater and marine environments are summarised in Table 1. Fresh waters average 0.2 µg/litre, with much higher concentrations in snow in industrial areas, and in Greenland ice. The mean value in sea water is 0.09 µg/litre.

1.4 Transport, transformation and bioaccumulation

1.4.1 Transport

Selenium exists in the earth's crust mainly as selenides, which are relatively slow-weathering, and in sedimentary materials, particularly shales. It is discharged to the atmosphere from volcanic sources, with sulphur, but the properties of the two elements differ substantially, leading to their separation in environmental pathways. Selenium is less volatile than sulphur, and hydrogen selenide is readily oxidised to elemental selenium. Selenium oxide is a solid at ambient temperatures and less easily transported in the atmosphere than sulphur dioxide. Moreover, it is easily reduced to elemental selenium, which is thus a very stable form. Selenium is likely to be deposited fairly near to sources of emission, unlike sulphur. This is confirmed by reported values of the ratios (all x10^6) of selenium to sulphur in volcanoes (1.6-1,040) and in sedimentary sulphur (0.1-10).

In an oxidising environment, sulphur is oxidised to the relatively mobile sulphate, but selenium is oxidised to the selenite form, which is strongly bound to aqueous ferric oxides or forms a basic ferric selenite and is rendered immobile. However, at pH levels over 7.5 selenite can be further oxidised to selenate, which is water soluble and is readily available to plants.

Quantitative estimates of these transport mechanisms are minimal. Three separate estimates of the rate of deposition of selenium derived from analyses of air and rain at Bermuda, using different methods of calculation, gave values of 0.06, 0.08 and 0.20 x 10^{-15} g/cm^2.sec (Duce et al., 1976). The atmospheric deposition rate estimated from these data, if assumed to be typical of oceanic areas, is equivalent to about 4,000 tonnes Se per annum in the northern hemisphere oceans. The mean air concentration of 130 pg/m^3 was very much larger than Zoller et al. (1974) found at the South Pole (5.6 pg/m^3), suggesting that wide variations may exist between different locations. More recently Buat-Menard (1983) estimated the selenium input from

Table 1. Examples of total selenium concentrations in air and water

Type of sample	Source		Number of samples	Mean	Range	Reference
AIR	Antarctic		10	5.6 pg/m^3	4.2 - 8.2 pg/m^3	Zoller et al., (1974)
	Bermuda		29	130 pg/m^3	<20 - 620 pg/m^3	Duce et al., (1976)
	Massachusetts		7	900 pg/m^3		Hashimoto and Winchester (1967)
SNOW	Massachusetts		16		90 - 270 µg/l	Hashimoto and Winchester (1967)
ICE	Greenland		12		5 - 22 µg/kg	Weiss et al., (1971)
FRESH WATER	Various, USA		7	0.2 µg/l	0.11 - 0.25 µg/l	Kharkar et al., (1968)
	Colorado, USA (pH 7.8-8.2)		9		1 - 400 µg/l	Scott and Voegeli (1961)
	England		8		0.07 - 0.39 µg/l	Measures and Burton (1978)
SEA WATER	North Atlantic	surface	4		0.08 - 0.11 µg/l	Schutz and Turekian (1965)
		deep	4		0.07 - 0.10 µg/l	Schutz and Turekian (1965)
	Pacific and Indian Oceans	surface			(IV) 0.004-0.061 µg/l	Measures et al., (1983)
		deep			(VI) 0.039-0.108 µg/l	Measures et al., (1983)
	North Pacific Ocean	surface			0.08 - 0.10 µg/l	Cutter and Bruland (1984)
		deep			0.16 - 0.19 µg/l	Cutter and Bruland (1984)
SEWAGE	Raw				15 - 60 µg/l	Baird et al., (1972)
	Primary effluent				30 - 45 µg/l	Baird et al., (1972)
	Secondary effluent				10 - 50 µg/l	Baird et al., (1972)
	Industrial trunk line				280 µg/l	Baird et al., (1972)
	Municipal waste discharges				100, 170, 450 µg/l	MacDermott (1974)

the atmosphere to the North Pacific to be 0.13 nmol/cm^2/year, or 0.3×10^{-15} g/cm^2.sec, about three times that derived from the Bermuda data of Duce et al., (1976).

Mackenzie et al., (1979) calculated that the annual transfer of selenium from the atmosphere via rain to the total ocean surface amounted to 12,200 tonnes, with a further 2,000 tonnes falling as dust.

The transport of selenium to the oceans is thus partly by atmospheric deposition (probably in elemental form) following volcanic emission or fossil fuel combustion, although the major part of the selenium emitted is likely to be deposited relatively near to the source. The other major transport route is by sediment transfer from rivers, the selenium being strongly bound to particulate matter. The discharge of selenium to the oceans from rivers was estimated by Mackenzie et al. (1979) to be about 18,000 tonnes, of which 11,000 tonnes were in the suspended load and 7,200 tonnes in solution. It seems likely, however, that except in the more alkaline waters the insoluble selenite bound to particulate material will predominate. On entering the estuarine environment, selenite might be desorbed from particulates (Kharkar et al., 1968) and at the pH of sea water some oxidation to selenate could occur, but no firm evidence is currently available.

The residence time was estimated by Goldberg et al. (1971) to be 2×10^4 years, but Mackenzie et al. (1979) calculated a shorter time of 2,300 years for dissolved selenium. A more detailed consideration by Cutter and Bruland (1984), allowing for mixing by gyres and upwelling, gave a value for total selenium of 39 years in a 50 m water column in the North Pacific, while Se (IV) and Se(VI) have much shorter residence times of 2 and 5 years respectively. These authors observed that for the central Pacific gyres the atmospheric input of total selenium was 37% of that for vertical mixing.

1.4.2 Transformation

The transformation of selenium depends on pH, redox conditions and the role of microorganisms. Selenide Se (-II) is aerobically oxidised to selenium Se (0), selenite Se (+IV) at the pH of sea water can be oxidised to selenate Se (+VI), but in biological systems selenite can also be reduced by thiol groups to selenide Se (-II) to form a selenotrisulphide complex. Selenium-protein binding can occur, as in the synthesis of seleno-amino acids from inorganic selenium and their subsequent incorporation into peptide chains. This process is biologically mediated, involving carbon-selenium bonding. Wrench (1978) observed the assimilation of radioactive selenium (Se IV) by marine algae into cellular protein, and in amino acids, some being seleno-analogues of the sulphur amino acids.

Selenium methylation may also occur. Chau et al. (1976) observed the formation of both dimethyl selenide and dimethyl diselenide by micro-organisms in lake sediments, and by bacteria and fungi in pure culture. Both selenate and selenite were methylated, the rate of methylation being low at 4°C, significant at 10°C and rapid at 20°C. However, there is so far no clear evidence that methylation occurs in the marine environment under natural conditions. Methylated selenides are very volatile and localised concentration at the site of formation seems unlikely. Cutter (1978) was unable to detect free methyl selenides in sea water, but considered it possible that they could have been lost during sample storage. Jiang et al. (1983) measured both

dimethyl selenide and dimethyl diselenide in air above coastal and fresh waters, but no definite correlation was found between the selenium in air and that dissolved in the adjacent waters, or between particulate and volatile selenium in air.

Selenium (IV) displays nutrient-type behaviour and can be selectively assimilated or adsorbed by marine algae during phytoplankton blooms, and recycled back into solution when the blooms decay (Wrench and Measures, 1982). Selenium (VI) is largely unaffected, suggesting that selenium (IV) is the most bioavailable form. However, Cutter and Bruland (1984) found that selenium in the Pacific is transferred from surface to deep water primarily as organic selenide and not as selenite or selenate, implying that biological uptake involves a reduction process. They postulate that dissolved organic selenide is regenerated rapidly from biogenic particles in surface waters and is oxidised to selenite, which is rapidly depleted by biological uptake. In the suboxic zone (150-600 m) organic selenide is produced but its oxidation to selenite is inhibited by low oxygen levels. In the deeper waters, where biological incorporation is negligible, the oxidation of selenite to selenate is very slow, and the mean residence time of selenite is calculated to be 1,150 years. The average residence time of water in the deep ocean is 1,600 years, explaining the presence of selenite at these depths.

1.4.3 Bioaccumulation

Although there is a large amount of information on the selenium content of marine organisms, often obtained in conjunction with measurements of mercury concentrations, measurements of uptake and loss rates are few. Fowler and Benayoun (1976a) studied the uptake of radioactive selenium from both water and food by the euphausiid Meganyctiphanes norvegica. Accumulation was greatest from food into the viscera, and to a lesser degree into the eyes, muscle and exoskeleton. The half-life in tissue was 37 days. Faecal pellets accounted for 54% of the excretion and soluble processes 43% and it is considered that faecal material effectively redistributes selenium to the water and sediments. Fowler and Benayoun (1976b, c) also studied the flux of radioactive selenium through two other marine invertebrates, the mussel Mytilus galloprovincialis and the shrimp Lysmata seticaudata. Selenite (IV) was accumulated more rapidly by the mussel than selenate (VI) over an aqueous concentration range spanning three orders of magnitude, the process being dependent on temperature and mussel size. For both forms equilibrium was reached in 12 days, with tissue concentrations increased over that in water by factors of 80 for the selenite and 15 for selenate. Uptake was slightly reduced in the presence of mercury. The rates of loss were similar for both forms of selenium. The mussel had retained most selenium in the shell after 63 days, while in the shrimp much of the selenium was in the exoskeleton, and lost at the moult. The shrimp did not accumulate selenite to the same degree as mussels (an increase in tissue over water by a factor of 20), but biological half lives were similar for the two species, in the range 58 to 81 days. Dietary intake is of greater importance than uptake from water in the distribution of selenium within the organism, as well as on the selenium content of crustacean faeces.

The importance of diet as the main route of selenium uptake is stressed by Wrench (1979), who recorded concentration factors for selenium in the digestive gland of oysters (Ostrea edulis) of about 130, although factors for gill, labial palps and mantle were all less than 50.

There appear to be no data for the biological half-life of selenium in marine fish, but Sato et al., (1980) found a value of 28 days for carp in freshwater, while Gissel-Nielsen and Gissel-Nielsen (1973) reported values of 27 days for the eel and 13 days for guppies.

1.5 Selenium in sea water, sediments and marine biota

Reference is made in chapter I of this document to the general absence of verification of analytical data, or of intercalibration between analysts, for many elements, and this applies also to the data for selenium.

1.5.1 Sea water

The methods of analysis for the measurement of different forms of selenium are not yet adequately developed. Apart from the separation of particulate and soluble material, the latter probably including colloidal selenium, the analyst is required to separate and determine selenide (-II oxidation state), selenium (0), selenite (+IV) and selenate (+VI). Selenate, selenite and organic selenium appear to co-exist in sea water, although as yet there is no evidence for colloidal selenium, which in some studies would have been included in the organic selenium fraction (Cutter and Bruland, 1984). Takayanagi and Wong (1984a) measured organic and colloidal selenium, and found that colloidal inorganic selenium decreased to zero with increasing salinity from river to coastal water. Colloidal organic selenium (mostly in low molecular weight material) was found at all salinities, but in coastal waters formed only 35% of total dissolved organic selenium. They observed that other determinations of selenium which involve strong oxidising agents to convert the inorganic species to Se(VI) may have oxidised organic Se, which would enhance the measurements of Se(VI).

The same authors (1984b) found up to 0.20 µg/litre total selenium in Chesapeake Bay water (up to 0.05 µg/litre Se(IV); but in adjacent estuarine waters the total selenium concentration was in the range 0.016-0.36 µg/litre. They considered Se(IV) to be the dominant form in fresh waters, but thought that significant quantities were adsorbed to particulate material or oxidised to Se(VI). Howard (1971) found Se(IV) to be very strongly adsorbed to particulate material in fresh waters (more by hydrous ferric oxide than clay), and consequently transported by sediments.

Although there are uncertainties in the data reported for the various forms of selenium in sea water, there is a reasonable consistency in the levels reported for total selenium from samples taken in a wide range of marine waters. In surface sea waters total selenium concentrations appear to be in the range 22-130 ng/litre (Burton et al., 1980; Measures et al., 1983; Schutz and Turekian, 1965; Shimoishi and Toie, 1978; Suzuki et al., 1979; Takayanagi and Wong, 1984b; Uchida et al., 1980). Measures et al., (1983) made over 500 determinations each of Se(IV) and Se(VI) in the Atlantic, Pacific and Indian Oceans. The distribution of the two oxidation states was similar in the three oceans, the Pacific and Indian Oceans giving Se(IV) values of 4 ng/litre and Se(VI) of 39 ng/litre in surface waters, while below 2 km the concentrations were 62 ng/litre and 110 ng/litre respectively. Atlantic Ocean concentrations were 30-40% lower. Sugimura et al., (1978) state that up to 45% of the total selenium in sea water is in organic forms not completely characterised, while in another paper the same authors (1976) estimated that 20-60% of selenium in sea water was in the Se(VI) form. Cutter

and Bruland (1984) found that organic selenides represented about 80% of the total dissolved selenium in Pacific surface waters, but they were less significant below about 60m and undetectable below about 2,000m.

Selenium(VI) predominated below 60m, the concentration being fairly uniform below 125m, while Se(IV) increased steadily with depth from 60m to 2000m. Cutter and Bruland (1984) suggested that the method of Measures et al. (1983) could have included organic selenium in their Se(VI) data. In deep water samples, the data from both groups of workers were in agreement despite the different methods, as the organic fraction was insignificant.

The organic fraction of selenium, already referred to, requires further study. It could include organic forms resistant to mild oxidation pressures, and also elemental selenium, possibly from atmospheric deposition or as a stage in the transformation from selenide or selenite forms.

1.5.2 Sediments

Few data on selenium in sediments have been found in the literature, but Brewer (1975) reported about 0.15 µg/g dry weight in nearshore and shelf sediments. Turekian and Wedepohl (1961) recorded 0.17 µg/g dry weight in both deep sea clays and carbonate sediments. Young (1974) analysed an inshore sediment core taken near a municipal waste outfall, and found 4-6 µg/g dry weight in the upper 10cm, but only 0.25-0.40 µg/g at 30 cm.

1.5.3 Marine biota

Phytoplankton: Although experimental work on species of marine algae has indicated that selenium may be covalently bound to lipids (Gennity et al. 1984) or integrated into primary protein (Wrench, 1978), no data on natural levels in marine phytoplankton have been found.

Zooplankton: Concentrations of selenium in samples taken in the Adriatic Sea, with a 220 um mesh net through 0-50 m depth, were fairly constant, averaging 3.5±1.1 µg/g dry weight for 22 samples (Kosta et al., 1978). Fowler (1977) found 4.4 µg/g dry weight in euphausiids, and 6.6 µg/g dry weight in faecal pellets.

Molluscs: In the Adriatic Sea, Kosta et al. (1978) found 0.41 µg/g wet weight in mussels, and 3.51 µg/g in Ostrea sp. Wrench and Campbell (1981) reported 0.82 µg/g wet weight in the hepatopancreas of the clam Mya arenaria from the Arctic, 98.7% being bound to protein. Two species of molluscs analysed in South Australia contained 1.1 and 2.3 µg/g dry weight in muscle (Maher, 1983). (N.B. the ratio of dry weight to wet weight is approximately 1:6).

Crustacea: Five species of crustacea analysed by Maher (1983) contained 2.4 - 4.4 µg/g dry weight in muscle tissue. (Dry weight/wet weight ratio approximately 1:5). Greig and Jones (1976) reported 1.0-2.2 µg/g wet weight in the muscle of rock crab on a United States ocean dumping site, but slightly higher levels of 2.8-5.5 µg/g in Long Island Sound, U.S.A. Wrench and Campbell (1981) reported 0.18 µg/g wet weight in lobster muscle but 0.61 µg/g in the hepatopancreas. Only 61% of the selenium in the muscle was protein bound but over 99% in the hepatopancreas. Lobsters from a contaminated area in New Brunswick (Canada) contained 0.39 µg/g in muscle and

0.91 µg/g in the hepatopancreas, with a somewhat lower percentage incorporated in protein. Kosta et al., (1978) found 0.39-2.61 µg/g wet weight in Nephrops sp. in the Adriatic.

Fish: Itano (1983) found that skipjack tuna (Katsuwonus pelamis) liver contained the highest selenium concentration of all tissues examined, most being incorporated in proteins, peptides and oligopeptides, or amino acids. Cappon and Smith (1981) measured the fraction of Se (VI) in fish muscle, the values for 11 species of marine fish being 14-33% as Se (VI) for selenium concentrations over the range 0.21-0.82 µg/g fresh weight. Maher (1983) reported 0.49-1.5 µg/g dry weight in the muscle of 4 Australian fish species. (Dry weight/wet weight ratio approximately 1:6). Mackay et al. (1975) analysed 42 black marlin (Makaira indica Cuvier), finding 1.4-13.5 µg/g wet weight in liver and 0.4-4.3 µg/g in muscle. For both tissues the concentrations were significantly correlated with both weight and girth. Glover (1979) found 0.2-0.8 µg/g wet weight in the edible tissues of two species of Australian sharks. Cappon and Smith (1982) found 0.20-0.64 µg/g fresh weight in the edible tissues of samples of fresh fish, but 0.31-4.1 µg/g in canned fish. The highest levels (2.5 and 4.1 µg/g) were in canned Pacific blue marlin, but the percentage of Se(VI) in these samples was the lowest found (3.5-8.3%). Unprocessed fish averaged 26% of Se(VI). Luten et al., (1980) found 0.15-1.04 µg/g wet weight in the muscles of 7 species of marine fish, concentrations being higher in the liver (0.45-4.6 µg/g) in four of the species.

Concentrations in the muscle tissue of marine fish thus appear to fall mostly in the range 0.2-1.0 µg/g fresh weight. Liver concentrations are usually higher than in muscle, up to 13.5 µg/g being found in black marlin liver. The proportion of Se (VI) in fish appears generally to be below 50%.

Mammals: The selenium content of marine mammals has generally been determined in association with analyses for mercury, the inter-correlation of these elements being discussed later. Koeman et al., (1973) first observed the correlation, reporting selenium concentrations in the livers of five seals as 46 - 134 µg/g wet weight. Reijnders (1980) analysed 30 harbour seals found dead in the Wadden Sea, and measured levels of selenium in liver from a trace up to 409 µg/g, increasing with age but averaging 110 µg/g among 13 adults, while van der Ven et al. (1979) found 2.4 µg/g wet weight in the liver of seal pups and 6-29 µg/g in juveniles, with 1.0-2.5 µg/g in muscle tissue of seals of all ages. Smith and Armstrong (1978) examined livers of 390 ringed and 64 bearded seals from 7 locations in Arctic Canada. There were no significant differences between the localities. Selenium concentrations were very high, up to 420 µg/g wet weight but with mean values of 4.1-34.4 µg/g in samples of the two species from the seven locations. Muscle tissue contained only a little over 0.5 µg/g in mature animals. The selenium levels were correlated with age. The marked difference between muscle and liver contents was also noted by Kari and Kauranen (1978), who found 0.44 - 0.92 µg/g wet weight in the muscle of Baltic ringed seals (Pusida hispida) but 6.1-110 µg/g in the liver.

Analyses of whale tissues reveal similar concentrations. Stoneburner (1978) examined short finned pilot whales, which contained 23-62 µg/g wet weight in liver samples, and 3-10 µg/g wet weight in kidney, but only 0.8-1.3 µg/g wet weight in blubber. Koeman et al., (1972) found

3.2 - 79 µg/g in wet weight in liver of North Sea porpoises (Phocoena phocoena), 9.3-24 µg/g in livers of dolphins but only 0.6 and 3.2 µg/g in the livers of two Surinam dolphins.

In Table 2, examples are given of typical concentrations of selenium in marine species which are used for human consumption (based upon references listed above).

Table 2. Typical concentration ranges of selenium in tissues of various marine species

(µg/g fresh weight)

Molluscs	Various spp.	0.4 - 0.8	
Crustacea	Various spp.	0.4 - 5.5	
Fish	Shark spp.	0.2 - 0.8	(muscle)
		0.5 - 15	(liver)
	Various spp.	0.1 - 0.9	(muscle)
Seal	Various spp.	0.4 - 1.0	(muscle)
		4 - 400	(liver)
Whale	Various spp.	4 - 80	(liver)

(Information based on data referenced in Section 1.5.3)

1.5.4 Selenium/mercury correlation

Fish: In view of the strong correlation often observed between selenium and mercury concentrations in seals (see next sub-section), several studies have been made of the concentrations of these elements in fish, which are the food of several species of seals. There is general agreement that the molar concentration of selenium in muscle tissue exceeds that of mercury in most samples, but the Hg/Se ratio varies widely, Cappon and Smith (1981) finding values averaging 0.65 but varying from 0.08 to 1.71 for the muscle tissues of 11 species examined. Luten et al., (1980) similarly reported values of the Hg/Se ratio in muscle tissue of 7 species ranging from 0.03 to 1.02. The higher values appear to occur in larger or older specimens, the mercury concentrations increasing with age. Freeman et al. (1978) examined 47 swordfish Xiphias gladius and found that all fish with mercury concentrations up to 2 µg/g had a molar excess of selenium compared to mercury, few fish having more than 2 µg/g. With the exception of certain piscivorous species, most workers have not found any significant correlation between the elements in fish.

Ueda and Takeda (1983) reported a significant positive correlation in shark muscle between the Hg/Se ratio and the total mercury concentration. Kai et al. (1983) found a similar relationship for tuna muscle, the molar ratio approaching unity as the total mercury increased. Leonzio et al. (1982) have observed that in Mullus barbatus the molar total of Hg + Se is linearly related to the age of the fish (computed from the length), whether the mercury level is high or low, suggesting that some form of "compensation" occurs. They also recalculated the earlier data of Freeman et al. (1978) for swordfish and found a similar correlation between total Hg + Se and length.

Most of the selenium in fish muscle is water-soluble and does not form a stable complex with mercury, which is in the organic form. Blue marlin (Nakaira nigricans) is an exception. Schultz and Ito (1979) reported that 87% of mercury is in the inorganic form in the muscle of this species, and in liver 98% is inorganic, and there was a significant correlation between the concentrations of Hg and Se. Takeda and Ueda (1978) identified a similar relationship in tuna liver.

The data on mercury selenium relationships in different tissues, the significance of the proportion of methylmercury, and of the various forms of selenium in fish, render it impossible to obtain any clear understanding of the interaction, if any, between the two elements at the present time.

Mammals: Koeman et al. (1973) first observed that the molar ratio of mercury to selenium in the livers of marine mammals was approximately 1:1. In a further paper (Koeman et al., 1975) it was shown that the correlation between the concentrations of the two elements in the livers of nine species of seals and whales (56 individuals) from Europe, North and South America and New Zealand was strong and almost linear (correlation coefficient 0.991) with a slope of 2.50. The ratio of atomic weights of the two elements is 2.54. A similar correlation was found for the brain. The authors found that, in contrast to fish, only 2-14% of the mercury in liver and brain of adult common seals was in the form of methylmercury, but in juveniles (1 year or less) the proportion was 16-78%.

The equimolar ratio appears to be established only in some tissues of the marine mammals, and suggests a structural connection. The low methylmercury fraction (as compared with fish, the diet of many marine mammals) indicates that demethylation probably occurs within the mammals, but it is uncertain whether the selenium is involved in a protective action against the toxic activity of mercury. (Koeman et al. (1975) found neither a 1:1 molar ratio nor any mercury-selenium correlations in marine birds, although Cottiglia et al. (1983) reported linear relationships between the elements in livers and brains of cormorants Phalacrocorax carbo, that in the liver being equimolar).

Martin et al. (1976) confirmed the 1:1 molar ratio of mercury to selenium in the livers of California sea lions (Zalophus californianus), both females and full-term pups showing this ratio, although premature pups had low concentrations and showed a low Hg:Se ratio. Stoneburner (1978) found a 1:1 molar ratio in the liver of one male and a gravid female short-finned pilot whale, but a large excess of mercury in two non-gravid females, suggesting a breakdown of the "protective" selenium mechanism. Smith and Armstrong (1978) examined 390 ringed and 64 bearded seals in Arctic Canada and confirmed the 1:1 molar ratio of Hg:Se in the liver. They found less than 5% of the mercury

in ringed seal livers to be in the methyl form (less than 1% in bearded seals) but about 75% of mercury was methylated in muscle tissues. Both mercury and selenium were positively correlated with age.

Kari and Kauranen (1978) confirmed the strong correlation between the elements in the livers of ringed seals in the Baltic (molar ratio Hg/Se = 1.16), but found no significant correlation in muscle or kidney. Reijnders (1980) found a strong linear correlation between Hg and Se in the liver and brain of harbour seals from the Netherlands, German and Danish coasts, but despite the high mercury concentrations (up to 751 µg/g wet weight) did not consider the levels responsible for decreased reproduction in the seals from the Netherlands coast.

Van der Ven et al. (1979) treated grey seals (Halichoerus grypus) experimentally with methylmercury, and found increases in both mercury and selenium in the liver and kidney, but only of mercury in the brain, thyroid and blood. They could find no evidence of a biochemical demethylation process, not of any effect of selenium on demethylation, but suggested that selenium influenced the distribution of mercury, the process taking place mainly in the liver and kidney.

Martoja and Berry (1980) identified particles of tiemannite (HgSe) in the livers of two cetaceans, Ziphius and Tursiops sp, in the Mediterranean. These particles, which cannot be excreted, are not degradable but are believed to be the result of a detoxification pathway involving several unknown reactions. The elements at the last stage are not linked to proteins by means of sulphur.

The reduction of selenate or selenite to selenide is an essential step for the reaction of selenium with inorganic mercury (Hg^{2+}) or methylmercury. The reaction product of selenide with Hg^{2+}, as with Cd^{2+}, is a stable colloidal metal selenide (Gasiewitz and Smith, 1978). The formation of such an insoluble complex in brain ensures that after prolonged exposure to mercury vapour and adequate selenium intake, mercury selenide formed in the brain remains there for many years after the cessation of exposure, in an approximately 1:1 molar ratio (Kosta et al., 1975). When such a complex is formed in blood, it is mainly taken up by the reticuloendothelial system, mainly by liver and spleen, and stored there in a 1:1 molar ratio (Groth et al., 1976).

In contrast to mercuric selenide, bismethylmercury selenide, the complex formed between selenide and methylmercury (Magos et al., 1979), is not stable (Masukowa et al., 1982) and the concentrations of mercury and selenium are independent of each other as they follow their own kinetics. Thus, Ueda and Takeda (1983) found that in tuna the higher is the mercury concentration, the higher is the Hg:Se ratio. However, marine mammals apparently can demethylate methylmercury to the extent that, for example, the seal liver stores nearly all the mercury in the inorganic form where the molecular ratio of mercury to selenium is 1:1 (Koeman et al., 1973) irrespective of whether they were caught in Europe, North or South America or New Zealand (Koeman et al., 1975).

1.5.5 Other correlations

Bjerregaard (1982) studied the exposure of the shore crab Carcinus maenas to combinations of selenium (IV) and cadmium, and found that selenium augmented the cadmium concentrations in the gills. Selenium levels increased in

the gills and carapace but not in muscle or hepatopancreas. There appeared to be no causal relationship between the concentrations of the two elements, although for any given combination of selenium and cadmium in the water there was a positive Se/Cd correlation in the carapace. There was no increase of selenium in internal organs on exposure to higher ambient Se levels.

Mackay et al. (1975) found positive correlations between copper and selenium, and cadmium and selenium, in the liver of black marlin (Makaira indica Cuvier).

2. EFFECTS ON MARINE BIOTA

2.1 Reference documentation

Summaries of the data available on the effects of selenium on marine biota have been published by USEPA (1980) and Taylor (1982), but the information is relatively sparse. There is, however, consistency regarding the concentrations producing an adverse effect, as described in individual research papers.

2.2 Effects on marine biota

Algae: Hollibaugh et al. (1980) found selenium as selenite had no effect on the growth of a natural population of algae, up to a concentration of 79 µg/litre. This concentration was not toxic to a pure culture of Thalassiosira aestevalis, although the cell size was slightly greater and total biomass slightly less than at lower selenium concentrations. Wheeler et al., (1982) found that 10 mg Se/litre as selenate was toxic to marine unicellular algae, with effects on growth within the range 0.1 - 1.0 mg/litre, whereas selenite was not toxic at 100 mg Se/litre. Low concentrations of selenium reduced the growth inhibition caused by sulphate deficiency in sea water. There was considerable variation in response among the six species of algae tested, Platymonas sp being most sensitive and Chlorella and Porphridium the least.

Gotsis (1980) found that selenite affected the growth of Pavlova lutheri above 2 mg/litre and of Dunaliella minuta above 10 mg/litre. There was an antagonistic action between mercury and selenium when present in a mixture.

Fries (1982) found that selenium, as either selenite or selenate, increased the growth of the macro-algae Fucus spiralis L and Goniotrichum alsidii, there being two optimum concentrations at 26 µg/litre and 2.6 mg/litre. Organically bound selenium was ineffective.

Invertebrates: Ward et al. (1981) estimated the 96h LC_{50} for the shrimp Penaeus aztecus to be 1.2 mg Se/litre. Ahsanullah and Palmer (1980) found that for sodium selenite the 96h LC_{50} for the cumacean Cylaspis usitata was 6.1 mg Se/litre, for the amphipod Allorchestes compressa 4.8 - 6.2 mg/litre, and for the juvenile bivalve Notocallista sp. 2.9 mg/litre. Glickstein (1978) recorded a 96h LC_{50} of 1.04 mg Se/litre for the zoeal larvae of the crab Cancer magister exposed to selenite, but a 48h exposure to 10 mg/litre did not affect developing embryos of the bivalve Crassostrea gigas. With both species there was some evidence that low levels of selenium in the water reduced sensitivity to mercury, although at levels close to the lethal level sensitivity increased. The USEPA review (1980) records acute toxicity data for

copepods, shrimps and crab exposed to selenium at concentrations of 0.6 - 4.6 mg/litre, and a no effect concentration of 0.135 mg/litre in a life cycle test with the mysid Mysidopsis bahia.

Fish: Most data in the literature are for freshwater fish, Hodson et al. (1980) giving a 96h LC_{50} of 8.1 mg Se/litre as selenite for rainbow trout, and Sato et al., (1980) a value for the same period of 35 mg/litre for carp (using selenite). Ward et al., (1981) reported a value for the 96h LC_{50} of 7.4 mg/litre for juveniles of the estuarine sheepshead minnow Cyprinodon variegatus, and 4.4 mg/litre for sub-adult pinfish Lagodon rhomboides.

Selenium is an essential dietary mineral for humans (see section 3) and Heisinger and Dawson (1983) found that in the freshwater black bullhead only one form of glutathione peroxidase activity in the liver was selenium dependent. Hodson and Hilton (1983) considered that freshwater fish were unlikely to suffer a selenium deficiency, the uptake from the diet being very efficient and independent of dietary loading. Uptake from water was also very efficient at low concentrations, but at high concentrations efficiency was reduced by saturation of the gill absorption processes.

In summary, the aqueous concentrations of selenium likely to affect marine species adversely are usually well in excess of 0.1 mg/litre. This value is about three orders of magnitude greater than the average concentration found for total selenium in the marine environment generally, even in coastal waters, and indicates that no risk is likely to be presented to marine organisms from selenium in water, although no long-term studies have been made. However, the intake from food is likely to be more important than from water, and in areas of local pollution the dietary intake might give rise to abnormal concentrations in both invertebrates and fish.

3. HUMAN HEALTH ASPECTS

3.1 Introduction and reference documentation

Animal diseases caused by the deficiency or excess of dietary selenium have established this element as both essential and toxic. Recently the association between low level of dietary selenium intake and Se-responsive conditions affecting human health in large areas of the People's Republic of China (PRC), and between high selenium intake and an episode of intoxication reported to be selenosis in a small area of the PRC, has been critically analysed by a WHO Task Group. Their evaluation forms part of the comprehensive Review of Environmental Health Criteria: Selenium (WHO, 1986). This WHO document and the reviews of Robinson and Thomson (1983) and Robinson (1984), together with individual papers referred to in the text and listed in the reference section, were consulted in the preparation of this document.

3.2 Metabolism of selenium

3.2.1 Absorption

Depending on regional availability for incorporation into animal or human food, there are wide variations in the daily intake of this essential element. Some of the most extreme values for human intakes and blood selenium

concentrations have been reported from the People's Republic of China and are shown in Table 3. Estimates of human adult dietary intake thus range from 11 to 5000 µg Se/day. There are diseases at both ends of the nutritional spectrum, at the lower end Keshan disease, in the aetiology of which selenium deficiency plays a major part, and at the higher end clinical conditions attributed to selenotoxicosis.

Table 3. Daily selenium intakes and blood selenium concentrations of residents living in high-, medium- and low-selenium areas of the People's Republic of China

Place	n	Mean daily selenium intake (with range) in µg	n	Mean blood Se (with range or ± S.D.) in mg/l
High selenium area with history of suspected selenosis	6	4,990 (3,200-6,690)	65	3.2 (1.3-1.7)
High selenium area without selenosis	3	750 (240-1,510)	14	0.44 (0.35-0.58)
Selenium-adequate area	8	116 (42-232)	1745	0.095 (±0.091)
Low selenium area with Keshan disease	13	11 (3-22)	1478	0.021 (±0.010)

Adapted from Yang et al. (1983)

It might be considered that a homeostatic mechanism would favour the maintenance of selenium levels within the body at a "safe" level, but only excretion is influenced by the nutritional status for selenium, while absorption is unaffected. The average selenium intake is about 30 µg/day in New Zealand and 132 µg/day in the USA (Robinson and Thomson, 1983), and volunteers in both countries absorbed about 80% of selenium naturally present in food (Stewart et al., 1978; Young et al., 1982). Similar absorption values were reported from Germany (Heinrich et al., 1977) and Sweden (Swanson et al., 1983). Gastrointestinal absorption is influenced by the chemical form of selenium. Thus selenomethionine, which constitutes 50% of cereal selenium (Thomson et al., 1985) is nearly completely (95%) absorbed (Griffiths et al., 1976), while only 44-70% (median : 52%) of selenite selenium was absorbed (Thomson and Stewart, 1974; Janghorbani et al., 1984).

3.2.2 Excretion and clearance half times

In contrast to absorption, selenium excretion is influenced by the previous dietary intake of, and nutritional status for, selenium. Thus, in the first 24 hours the excretion of ingested ^{74}Se was 3.3, 7, 11 or 17% depending on whether the young American males participating in the study were provided with 11.4, 107, 170 or 270 µg Se/day (Janghorbani et al., 1982,

1984). New Zealand residents with selenium intakes below 30 µg/day and having 50-70 ng Se/ml plasma had 0.1-0.2 ml/min plasma clearance, while North Americans with 80 µg Se/day consumption and 120-140 ng Se/ml plasma had 0.2-0.3 ml/min (Robinson et al., 1985).

The total daily urinary excretion is approximately half of the daily intake (Thomson and Robinson, 1980), but there is a significant difference between the clearance half times of selenite and selenomethionine. One week after the ingestion of ^{75}Se-selenite or ^{75}Se-selenomethionine about 50% of selenite ^{75}Se and 90% of selenomethionine ^{75}Se were retained and these cleared with half times of 115±15 (Thomson and Stewart, 1974) and 245±19 days respectively (Griffiths et al., 1976). In another study 11% of the absorbed selenium cleared with a half time of 2.3 days and 86% with a half time of 162 days (Janghorbani et al., 1984).

The comparison of blood selenium concentrations presented in Table 3 and the predicted equilibrium blood concentrations calculated by using the metabolic model of Medinsky et al. (1985) also indicates that the retention of selenium decreases with increasing intake. The model gives the correct relationship between intake and blood selenium concentration on the assumption that 55% of the ingested selenium was absorbed in the low selenium areas, 30% in the adequate areas and 22% in the two high selenium areas. As absorption is not influenced by the selenium status, these differences must reflect differences in retention.

3.2.3 Metabolic pathways

The higher clearance half time of selenomethionine selenium is likely to be due to a delay in reaching the same stages in the metabolism of other selenium compounds. Selenate and selenite are reduced throuth enzymic and non-enzymic reactions to a labile intermediate selenide. The selenide is then methylated to form trimethylselenomium ion which is excreted in urine. When the intake is below the accutely toxic dose, this is the usual mode of excretion via the urinary tract. At higher levels of selenium intake the saturation of the final methylation step results in the exhalation of dimethylselenide. The selenide may also form -S-Se-S- bonds in proteins or stable complexes with heavy metals such as cadmium and mercury (see reviews of Diplock, 1976; Ganther, 1979, Magos and Webb, 1980). Selenoaminoacids, with the exception of selenomethionine, are rapidly oxidised to selenite, while the conversion of selenomethionine is limited and/or delayed by incorporation into the primary structure of proteins (Martin and Hurlbut, 1976) from which it may be liberated slowly.

3.2.4 Selenium in blood

The difference in the metabolism and retention half times of selenomethionineand selenite-selenium is consistent with the observation that selenomethionine is more effective than selenite in raising blood selenium levels (Thomson et al., 1982). Selenium naturally present in fish behaved as selenomethionine (Robinson et al., 1978). Likewise when Finnish men of low selenium status were given for 11 weeks 200 µg Se/day either as selenate or as Se-rich wheat or yeast, the plasma concentration of selenium was raised less by the inorganic selenium salt than by food selenium. Thus selenate increased plasma selenium from 70 to 100 µg/litre with little change in the last 7 weeks, while food selenium increased plasma selenium progressivley to

160 µg/litre. Ten weeks after the subjects were returned to normal diet, plama selenium concentration declined in both groups but in the food selenium groups it remained 30 µg/litre higher than in the selenate group or the base line level (Levander et al., 1983).

Plasma selenium concentration at low selenium status rises and falls more rapidly in response to change in Se intake than it does in erythrocytes (Robinson et al., 1978). At steady state blood selenium concentration is always higher in erythrocytes than in plasma and up to a whole blood level of 160 µg/litre they correlate with each other very well (Rea et al., 1979). When the whole blood selenium concentration is higher than 160 µg/litre, increased intake apparently increases erythrocyte selenium without a corresponding increase in plasma selenium (Thomson and Robinson, 1980).

3.2.5 Glutathione peroxidase

A proportion of the selenium in blood is a functional part of the selenium-dependent enzyme glutathione peroxidase (GSHPx). This enzyme has four sub-units and each sub-unit contains one selenium atom at the active site. Blood GSHPx activity is linearly correlated with erythrocyte selenium up to a certain limit, which is about 140 µg/litre whole blood (Rea et al., 1979). Thus a correlation between GSHPx and erythrocyte selenium can be expected only in areas of moderate or low selenium intake (Rea et al., 1979). In the USA (Valentine et al., 1980) and Germany (Schmidt and Heller, 1976) no such correlation was noted. Likewise selenium supplementation did not increase blood GSHPx in the USA (Schrauzer and White, 1978), but in New Zealand, when initial blood levels were low, supplementation of the diet with selenite, selenomethionine (Thomson et al., 1982) or high-Se wheat bread (Thomson et al., 1985) increased plasma GSHPx at once and, after a delay, erythrocyte GSHPx. The delay in the increase of erythrocyte GSHPx is presumed to be due to the dependence of erythrocyte GSHPx on the selenium that was available at the time of the formation of these cells (Cohen et al., 1985).

3.3 Health effects

The establishment of a reliable dose-response relationship is difficult, partly because analytical quality control data are missing for most of the period which has elapsed since the publication of the first epidemiological study nearly 50 years ago. There seem to be accurate analytical methods for selenium determination but valid quality assurance data for selenium analysis in food and biological index media is an exception and not a rule.

3.3.1 Selenium deficiency

In North America and most of Europe blood selenium concentrations lie between 150 and 250 µg/litre, in Finland and New Zealand between 50 to 100 µg/litre (Thomson and Robinson, 1980) and in the area of the People's Republic of China affected by Keshan Disease the average concentration was 21 µg/litre (Yang et al., 1983). Keshan Disease, a cardiomyopathy of children and young women, responded favourably to selenium (selenite) supplementation and was absent in areas where daily intake of selenium was at least 30 µg/day (Keshan Disease Research Group, 1979) which is the level of average intake in parts of New Zealand (Thomson and Robinson, 1980). In New Zealand or Scandinavia no consistent causal relationship has been found between low selenium status and any naturally occurring diseases (Robinson and Thomson, 1983).

3.3.2 Selenium toxicity

The source of toxic exposure to selenium can be occupational (through inhalation or dermal contact), the ingestion of selenium-containing tablets or dietary.

3.3.2.1 Occupational exposure

Fumes and dusts of selenium and its compounds are irritative and in the most severe cases exposure results in breathing difficulty and gastrointestinal distress. The observed persistent garlic odour of the breath is due to the respiratory excretion of dimethylselenide. There are no systematic epidemiolgical investigations of long term overexposure, data derived from accidental overexposure are poorly documented, and the pathway of exposure is quite different from that of overexposure by dietary means as are the selenium compounds likely to be encountered (WHO, 1986).

3.3.2.2 Self medication

The daily ingestion of one tablet containing 2 mg sodium selenite for more than two years elevated blood selenium level to 0.179 mg/litre and hair selenium to 830 ng/g and caused thickened, fragile nails and garlic odour (Yang et al., 1983). A dietary supplement containing 25 mg sodium selenite and 5 mg elemental selenium were ingested daily by a 57 year old woman for 77 days (Jensen et al., 1984). She started to lose hair after 11 days, became bald after two months and over the next 5 weeks white horizontal streaks occurred on fingernails, fingertips became inflammed and fingernail beds purulent. The patient also experienced periodic episodes of vomiting, sour-milk breath odour and increasing fatigue. A serum sample, taken 4 days after the last tablet, was reported to contain 0.528 mg Se/litre and the whole blood level was presumably considerably higher.

3.3.2.3 Dietary exposure in seleniferous areas

The public health consequences of high dietary intakes of selenium were first described in people living on farms and ranches of South Dakota, USA, where farm animals suffered from selenium poisoning caused by feeds grown locally on selenium-rich soil. Two health surveys were carried out, the first on the members of 111 families and the second on a sub-group of 100 residents with the highest urinary selenium levels ranging from 0.1 to 1.98 mg/litre (Smith and Westfall, 1937). The frequency of various signs and symptoms observed in the second study are shown in Table 4. The interpretation of the findings was hampered by the lack of a control population and the lack of correlation between abnormalities and urinary selenium excretion. Nevertheless the high incidence of gastrointestinal disorders and yellowish skin discoloration, thought to be due to hepatic dysfunction, raised the possibility that the cause of these abnormalities was selenosis. Based on the assumption that in chronic selenosis the urinary excretion of selenium of cats bears a rather definite relationship to the daily ingested dose, Smith and Westfall (1937) calculated that some members of the study group might have ingested daily (assuming 100% absorption) 10-100 µg Se/kg, and some may have ingested 200 µg Se/kg. For a 70 kg man, these rates of absorption would be equivalent to a dietary selenium intake of 700 to 14000 µg/day.

Table 4. Signs and symptoms observed in humans excreting high levels of urinary selenium

Signs and symptoms	Frequency of occurrence
None	24
Gastrointestinal disturbances	31
Icteroid discoloration of the skin	28
Bad teeth	27
Sallow and pallid colour, especially in younger individuals	17
History of recurrent jaundice	5
Dermatitis	5
Pigmentation of the skin (chloasma?)	3
Pathological nails	3
Rheumatoid arthritis	3
Cardiorenal disease	2
Vitiligo	2

Adapted from Smith and Westfall (1937)

Some years later a field study was conducted in Venezuela in order to compare the health of two groups of children, one group living in a seleniferous area (Villa Bruzual) and the other in Caracas (Jaffe et al., 1972, 1976). The mean blood selenium level in Villa Bruzual was 0.81 mg/litre with a maximum of 1.8 mg/litre and the mean urinary concentration 0.365 mg/litre with a maximum of 3.9 mg/litre. Only the report of nausea and pathological nails correlated with blood and urinary selenium levels. The lower haemoglobin concentration in Villa Bruzual was explained by the inadequate nutrition of the subjects and their parasitic infections.

An outbreak of disease in a seleniferous area of the People's Republic of China in 1961 and its possible relationship with dietary selenium intoxication was reported by Yang et al. (1983). The most common signs were brittle nails with white spots an longitudinal streaks, loss of hair and nails, skin lesions and in the more severe cases paraesthesia. In the five most severely affected villages 49.2% of the 248 inhabitants were affected. The incidence of the disease reached a peak between 1961 and 1964, but as the aetiological role of selenium was suspected only later, the first analysis of food and bio- logical samples were carried out on samples collected in 1966. At this time selenium intakes ranged from 3,200 to 6,700 µg/day (mean: 4,990 µg/day), hair concentration from 4.1 to 100 µg/g (mean: 32.2 µg/g), blood selenium concentration from 1.3 mg/litre to 7.5 mg/litre (mean: 3.2 mg/litre) and urinary concentration from 0.88 mg/litre to 6.63 mg/litre (mean: 2.68 mg/litre). Though, after a temporary evacuation from their villages at the time of the peak incidence, there was a recovery as soon as their diets were changed, the interaction of selenium with other nutritional and toxic agents could not be ruled out (Yang et al., 1983). No adverse effects were observed on nail or hair in a high selenium area without selenosis, where blood conentration ranged from 0.35 to 0.58 mg/litre (mean: 0.44 mg/litre). The WHO Selenium Task Group (WHO, 1986) recognised the difficulty of establishing an exact dose-response relationship with respect to selenium in this study.

3.4 Total exposure to selenium

In the absence of selenium-emitting industries, the atmospheric concentration of selenium is so low that this source of exposure is expected to contribute less than 1.0 µg to the daily intake. Likewise, with the exception of seleniferous regions, the selenium content of drinking-water is so low that it can contribute only a few µg to the total daily intake. In the chronic selenosis area of China where the average selenium concentration of 11 drinking-water samples was 54 µg/litre and in a heavily affected village where four surface water samples contained 117-159 µg/litre selenium, the drinking-water increased the daily dietary intake by only a few percent (Yang et al., 1983). Based on these considerations it was concluded that for the general population the primary route of exposure to selenium is via the diet (WHO, 1986).

There are great differences in the contribution of different food products to the dietary intake of selenium and in the level of daily intakes reported from various countries of the world (Table 5). Individual selenium contents in foods (on the wet weight basis, before cooking) in mg/kg are (WHO, 1986): liver, kidney and seafoods: 0.4-1.5; muscle meats: 0.1-0.4; cereal and cereal products: <0.1->0.8 (in extreme cases 8.0); dairy products: 0.1-0.3; fruits and vegetables: <0.1. In a high selenium area of China the daily average selenium intake of 4.99 mg/kg was nearly equally distributed between vegetables and cereals. The peak observed in the endemic disease corresponded to a period of drought resulting in the failure of the rice crop. This forced villagers to eat more vegetables and also maize which had a selenium content higher than rice (Yang et al., 1983)

Table 5. Estimated human daily intake of selenium from dietary sources (µg/day)

Country	Plants		Animals		Totals
	vegetables fruits, sugars	cereals	dairy products	meat fish	
New Zealand	1	4	11	12	28
Britain	4	30	4	22	60
Canada (Toronto)	5.1	62.0	6.5	24.7	98.3
Canada (Halifax)	7.4	105.0	21.8	90.0	224.2
USA (Maryland)	5.4	44.5	13.5	68.6	132.0
USA (Dakota)	10.4	57.0	47.6	101.4	216.4
Japan	34.8	72.7	5.7	94.6	207.8
Venezuela	14.6	88.2	70.4	152.6	325.8

Adapted from WHO (1986)

3.5 Contribution of selenium from marine food

Although there are variations in the levels of selenium reported by different countries and laboratories for various finfish and shellfish species (see Table 6), data can be divided into two groups. Concentrations can be clustered either around 0.5 mg/kg (cod, tuna, flounder, salmon, clam, oyster) or 1.5 mg/kg (marlin, halibut, lobster, shrimp). The highest selenium concentration was found in marlin, probably because this fish retains about 80% of muscle mercury in the inorganic form (Rivers et al., 1972; Shultz and Ito, 1979) and therefore a significant part of tissue selenium is probably HgSe. Mercury concentrations are also high and in 1971 the Hawaii State Department of Public Health declared a moratorium on the use of Pacific blue marlin for human consumption (Rivers et al., 1972). In the flesh of 42 black marlins (Makaira indica) the average mercury concentration was 7.3 mg/kg (Mackay et al., 1975), in 29 Pacific marlin (Makaria ampla) 4.78 mg/kg (Rivers et al., 1972) and 46 blue marlins (Makaira nigricans) 3.12 mg/kg (Shultz and Ito, 1979).

In the calculation of selenium intake from marine foods an uncertainty is introduced by variation in dietary habits and perhaps by the lack of correction for waste, that is the difference between purchased weight and consumed weight. A commonly used figure of 20 g/day is thought to be representative of the worldwide average fish consumption (FAO, 1977; NAS, 1978) and equivalent to one fish meal per week. The average per capita fish consumption ranges from 4 g to 60 g/day in countries along the Mediterranean coastline (Bernhard and Renzoni, 1977) and it is nearly 200 g in the Faroes Islands (Bernhard and Andreae, 1984). However certain individuals are reported to consume much greater amounts than the national average, such as the 800 g fish/day consumption by the crew of fishing boats in Southern Italy (Bernhard and Andreae, 1984). In Australia, where the average fish consumption is 17 g/day, 6.4 % of the population consumed more than 62 g day and 0.9 % more than that 143 g/day (WGMF, 1980). In Japan some fish retailers ate 200 to 500 g seafood daily and one consumed 1,200 g (Doi and Ui, 1975).

Table 7 gives the selenium intakes from marine foods for consumption at four levels, 20 g/day for the worldwide average consumption, 60 g/day for the average consumer in some Western countries, 150 g/day which may be typical for countries where fish is a major component of the diet, and 1,000 g for extreme cases, and for two levels of concentration, 0.5 and 1.5 µg/g.

Certain fish meals are ingredients of animal feeds. These could represent a secondary route of exposure of humans to dietary selenium from marine products, but the difficulties are too great to allow the calculation of daily intake by this route. However, it is considered highly improbable that appreciable selenium intake could be obtained by this way.

3.6 Evaluation of potential health effects

3.6.1 Seafood as a source of nutritionally desirable selenium levels

Health effects observed at 10 ng Se/ml blood concentrations and the observed protection by selenium supplementation (Yang et al., 1984) indicate that abnormally low selenium status can be associated with both biochemical and clinical disorders. The WHO review on selenium (1986) indicates that when the daily selenium intake is less than 30 µg, the selenium concentration in

Table 6. Selenium concentration of muscle tissues of various species of marine foods. (Means of reported values)

Seafood	Country	mg Se/kg wet weight (mean value)	Reference
Cod	Canada	0.86	WHO, 1986
	USA	0.43; 0.44	" "
	New Zealand	0.31; 0.36	Thomson and Robinson, 1980
Tuna	Australia	0.5	IRPTC, 1978
	Japan	0.54	Takeda and Ueda, 1978
	USA	0.60	Cappon and Smith, 1981
Black marlin	Australia	2.2	Mackay et al., 1975
Blue marlin	USA	1.98	Schultz and Ito, 1979
Flounder	New Zealand	0.27	Thomson and Robinson, 1980
	USA	0.34	WHO, 1986
Halibut	Canada	1.36	" "
Salmon	Japan	0.25	" "
	Canada	0.75	" "
Clam	Japan	0.18	" "
	USA	0.30; 0.65	IRPTC, 1978; WHO, 1986
Oyster	New Zealand	0.46; 0.82	Thomson and Robinson, 1980
	USA	0.49; 0.65	WHO, 1986
Lobster	Canada	1.22	" "
	USA	0.66; 1.04	" "
Shrimp	Canada	1.61	" "
	USA	0.59; 1.88	" "

blood may be lower than 0.050-0.070 mg/litre. This blood concentration is usually associated with a 50% reduction in erythrocyte glutathione peroxidase activity (Rea et al., 1979). The US Food and Nutrition Board (1980) has estimated that the safe and adequate range of dietary selenium intake is 50-200 µg/day.

Any discussion about the nutritional value of selenium in seafood has to take into account the bioavailability of selenium. Bioavailability estimations based on the restoration of hepatic glutathione peroxidase activity in selenium-depleted rats indicated that whole wheat bread is one and a half

Table 7. Daily selenium intake from seafood in µg/day at two concentrations and four consumption levels

Mean Se concentration µg/g	average daily consumption of seafood in g per day			
	20	60	150	1,000
0.5	10	30	75	500
1.5	30	90	225	1,500

times and tuna only half as effective as selenite (Levander, 1983). The reason for this low bioavailability is not known, but it is not associated with lowered hepatic selenium levels. Moreover tuna is not representative of all seafood products since selenium in fish protein concentrate prepared from shark flesh was more active than selenite in increasing glutathione peroxidase in rat tissue (see Levander, 1983).

3.6.2 Seafood as a source of potentially hazardous levels of selenium intake

The WHO review on selenium (1986) considered the epidemiological data from the seleniferous area of China and points out that the role of additional factors could not be ruled out and no intervention studies were carried out. However, in view of frank toxicity in cattle with blood selenium concentration greater than 2.0 mg/litre and possible borderline toxicity between 1.0 and 2.0 mg/litre, the suggested association between selenium and human health effects observed in the seleniferous area of China cannot be dismissed. In the affected villages the average blood concentration of 3.2 mg/litre (range 1.3-7.5 mg/litre) was associated with a selenium intake of 5,000 µg/day while in another area, where no similar effects were observed, the average blood concentration was 0.44 mg/litre and intake 750 µg/day (Yang et al., 1983). Based on these considerations, the WHO Selenium Task Group (WHO, 1986) expressed the view that the threshold limit for adverse health effects may be between 700 and 7,000 µg Se/day. According to Table 7 even exceptionally excessive consumption of seafood with 0.5 mg Se/kg concentration does not bring intake into this region, and when the concentration is 1.5 mg Se/kg the same excessive dietary habit brings selenium intake to twice the lower end of the 700 and 7,000 µg/day range. Thus it seems very unlikely that even excessive seafood consumption can increase selenium intake to a dangerous level. Moreover, seafood containing high levels of selenium may have high levels of mercury, so that an individual consuming seafood would probably suffer from methylmercury intoxication before any signs or symptoms of selenium over-exposure appeared.

3.6.3 Carcinogenicity and human reproduction

The critical evaluation of evidence for the involvement of hyperand hypo-selenosis in the aetiology of cancer leads to the conclusion that high selenium intake is not associated with a high risk of cancer, but low selenium intake may be associated with an increased incidence of certain forms of

cancer (Diplock, 1984). According to the WHO Selenium Task Group (WHO, 1986) at present there is no evidence that selenium has significant effects on human reproduction.

3.6.4 Interaction of selenium with other elements

The interaction between Hg^{2+} or Cd^{2+} and selenium is based on the formation of stable HgSe or CdSe complexes (Gasiewicz and Smith, 1978). When such a complex is formed in blood, it is mainly taken up by the reticuloendothelial system, principally the liver and spleen, and stored there with a 1:1 molar ratio (Groth et al., 1976), as in marine mammals (Koeman et al., 1973). The formation of such a complex may be a factor by which cadmium (Ganther and Baumann (1962) or inorganic mercury (Levander and Argrett, 1969) decreased the exhalation of dimethylselenide in selenite-treated rats. The timing of selenite and mercury administration is important. Selenite given in equimolar doses with Hg gave complete protection against the toxicity of $HgCl_2$ (Parizek and Ostadalova, 1967) but the protective effect decreased with increasing delay in selenium administration (Naganuma et al., 1984). When the administration of $HgCl_2$ was delayed, the interaction of Hg^{2+} with the already formed dimethylselenide resulted in severe, frequently lethal intoxication (Parizek et al., 1971).

Unlike HgSe, bismethylmercury selenide, the complex formed between selenide and methylmercury (Magos et al., 1979) is not stable (Masukowa et al., 1982), and methylmercury potentiated the exhalation of dimethylselenide (Yonemoto et al., 1985). Dietary selenium only delayed the onset of methylmercury intoxication (Chang et al., 1977), and the timing of selenium administration, judged from the shift in methylmercury distribution, was not as important as in the case of inorganic mercury (Magos and Webb, 1977). Such a change in the distribution of methylmercury was not associated with the increased demethylation of methylmercury as shown by the unchanged organic to inorganic mercury ratio (Komsta-Szumska et al., 1983). Interaction may be influenced by the form of selenium present in food. Using HgSe formation as an indicator of reactive Se^{2+}, it has been found that in rats the bioavailability of selenium for this reaction was less after the administration of selenomethionine or food selenium than after selenite (Magos et al., 1984).

Animal experiments indicated that arsenic and selenium increased the biliary excretion of each other and arsenic decreased the pulmonary excretion of selenium without increasing the selenium deficiency of animals fed on low selenium diet (Levander, 1977).

4. CONCLUSIONS ON SELENIUM

4.1 Potential harm to living resources

In view of the extremely low concentrations of selenium measured in coastal and oceanic waters, of the order of 0.1 µg/litre, and the absence of any evidence for elevated levels due to local discharges, it is not considered that any risk is presented to marine biota from aqueous concentrations of selenium. The minimum concentration likely to cause any adverse effect is at least 10^3 times larger than those found in the environment. It is almost inconceivable that, given the normal usage of selenium, concentrations could reach such levels even close to sources related to industrial usage.

Concentrations of selenium in marine species seem to be entirely of natural origin, although slightly enhanced levels might occur in the immediate vicinity of a discharge of selenium from an industrial or natural source (such enhanced levels have been reported only from the freshwater environment).

Particularly high concentrations of selenium are found in marine mammals, primarily in the liver, where equimolar amounts of mercury also occur. The mercury in liver tissue is very largely demethylated, but it is unclear whether this or the association with selenium renders the mercury non-toxic. No evidence has been reported of any toxic effects in marine mammals resulting from the high selenium and mercury levels.

4.2 Potential hazards to human health

Examples of disease states in humans associated with both high and low levels of dietary selenium intake have been reported. Diet is considered to be the main source of selenium exposure of the general population. Marine food products, both finfish and shellfish, contain substantial amounts of selenium and could be beneficial in areas deficient in selenium. However, the consumption of seafood resulting in selenium intake in excess of 700 µg/day, which is needed to elevate the blood selenium concentration to an undesirably high level (WHO, 1986), is unlikely. Even the uninterrupted daily consumption of 1,000 g seafood in the upper concentration range, around 1.5 µg/g, would bring daily consumption of selenium only marginally higher than that considered to be the lower limit of undesirable exposure. Moreover the associated presence of other elements may decrease its toxicity, and the toxicity of methylmercury in marine products presents a greater health risk. The equimolar occurrence of high concentrations of selenium and mercury in the livers of marine mammals is caused by the formation of biologically inert mercuric selenide and therefore such concentrations are unlikely to be toxic either to mammals or to human consumers.

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ANNEX 1

SECOND SESSION OF THE GESAMP WORKING GROUP
ON THE REVIEW OF POTENTIAL HARMFUL SUBSTANCES,
LONDON, 30 JANUARY - 3 FEBRUARY, 1984

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ANNEX 2

FIFTH SESSION OF THE GESAMP WORKING GROUP ON THE REVIEW OF POTENTIALLY HARMFUL SUBSTANCES, GENEVA, 4-8 NOVEMBER 1985

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ANNEX 3

GESAMP REPORTS AND STUDIES PUBLICATIONS

These Reports may be obtained from any of the Sponsoring Agencies in the Language(s) indicated: English, French, Russian, Spanish

Rept. & Stud. No.	Title	Date	Language
1.	Report of the Seventh Session	1975	E,F,R,S
2.	Review of Harmful Substances	1976	E
3.	Scientific Criteria for the Selection of Sites for Dumping Wastes into the Sea	1975	E,F,R,S
4.	Report of the Eighth Session	1976	E,F,R
5.	Principles for Developing Coastal Water Quality Criteria	1976	E
6.	Impact of Oil on the Marine Environment	1977	E
7.	Scientific Aspects of Pollution Arising from the Exploration and Exploitation of the Sea-bed	1977	E
8.	Report of the Ninth Session	1977	E,F,R
9.	Report of the Tenth Session	1978	E,F,R,S
10.	Report of the Eleventh Session	1980	E,F,S
11.	Marine Pollution Implications of Coastal Area Development	1980	E
12.	Monitoring Biological Variables related to Marine Pollution	1980	E,R
13.	Interchange of Pollutants between the Atmosphere and the Oceans	1980	E
14.	Report of the Twelfth Session	1981	E,F,R
15.	The Review of the Health of the Oceans	1982	E
16.	Scientific Criteria for the Selection of Waste Disposal Sites at Sea	1982	E
17.	The Evaluation of Hazards of Harmful Substances Carried by Ships	1982	E
18.	Report of the Thirteenth Session	1983	E,F,S
19.	An Oceanographic Model for the Dispersion of Wastes Disposed of in the Deep Sea	1983	E
20.	Marine Pollution Implications of Ocean Energy Development	1984	E

21.	Report of the Fourteenth Session	1984	E,F,S
22.	Review of Potentially Harmful Substances - Cadmium, Lead and Tin	1985	E
23.	Interchange of Pollutants Between the Atmosphere and the Oceans	1985	E
24.	Thermal Discharges in the Marine Environment	1984	E
25.	Report of the Fifteenth Session	1985	E,F
26.	Atmospheric Transport of Contaminants into the Mediterranean Region (in preparation)		
27.	Report of the Sixteenth Session	1986	E,F,S
28.	Review of Potentially Harmful Substances: Arsenic, Mercury and Selenium	1986	E
29.	Review of Potentially Harmful Substances: Organosilicon Compounds (Silanes and Siloxanes)	1986	E
30.	Environmental Capacity - An Approach to Marine Pollution Prevention	In preparation	