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Environmental Health Criteria 224

ARSENIC AND ARSENIC COMPOUNDS

Second edition

The first and second drafts of this monograph were prepared, under the coordination of Dr J. Ng, by the authors A. Gomez-Caminero, P. Howe, M. Hughes, E. Kenyon, D.R. Lewis, M. Moore, J. Ng, and by A. Aitio and G. Becking.

Please note that the layout and pagination of this pdf file are not identical to those of the printed document

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World Health Organization
Geneva, 2001

The **International Programme on Chemical Safety (IPCS)**, established in 1980, is a joint venture of the United Nations Environment Programme (UNEP), the International Labour Organization (ILO), and the World Health Organization (WHO). The overall objectives of the IPCS are to establish the scientific basis for assessment of the risk to human health and the environment from exposure to chemicals, through international peer-review processes, as a prerequisite for the promotion of chemical safety, and to provide technical assistance in strengthening national capacities for the sound management of chemicals.

The **Inter-Organization Programme for the Sound Management of Chemicals (IOMC)** was established in 1995 by UNEP, ILO, the Food and Agriculture Organization of the United Nations, WHO, the United Nations Industrial Development Organization, the United Nations Institute for Training and Research, and the Organisation for Economic Co-operation and Development (Participating Organizations), following recommendations made by the 1992 UN Conference on Environment and Development to strengthen cooperation and increase coordination in the field of chemical safety. The purpose of the IOMC is to promote coordination of the policies and activities pursued by the Participating Organizations, jointly or separately, to achieve the sound management of chemicals in relation to human health and the environment.

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NOTE TO READERS OF THE CRITERIA MONOGRAPHS

Every effort has been made to present information in the criteria monographs as accurately as possible without unduly delaying their publication. In the interest of all users of the Environmental Health Criteria monographs, readers are requested to communicate any errors that may have occurred to the Director of the International Programme on Chemical Safety, World Health Organization, Geneva, Switzerland, in order that they may be included in corrigenda.

* * *

A detailed data profile and a legal file can be obtained from the International Register of Potentially Toxic Chemicals, Case postale 356, 1219 Châtelaine, Geneva, Switzerland (telephone no. + 41 22 - 9799111, fax no. + 41 22 - 7973460, E-mail irptc@unep.ch).

* * *

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The Commonwealth Department of Health and Aged Care, Australia, contributed financially to the preparation of this Environmental Health Criteria monograph. The Task Group meeting was arranged by the National Research Centre for Environmental Toxicology, Australia.

Environmental Health Criteria

PREAMBLE

Objectives

In 1973 the WHO Environmental Health Criteria Programme was initiated with the following objectives:

(i) to assess information on the relationship between exposure to environmental pollutants and human health, and to provide guidelines for setting exposure limits;

(ii) to identify new or potential pollutants;

(iii) to identify gaps in knowledge concerning the health effects of pollutants;

(iv) to promote the harmonization of toxicological and epidemiological methods in order to have internationally comparable results.

The first Environmental Health Criteria (EHC) monograph, on mercury, was published in 1976 and since that time an ever-increasing number of assessments of chemicals and of physical effects have been produced. In addition, many EHC monographs have been devoted to evaluating toxicological methodology, e.g. for genetic, neurotoxic, teratogenic and nephrotoxic effects. Other publications have been concerned with epidemiological guidelines, evaluation of short-term tests for carcinogens, biomarkers, effects on the elderly and so forth.

Since its inauguration the EHC Programme has widened its scope, and the importance of environmental effects, in addition to health effects, has been increasingly emphasized in the total evaluation of chemicals.

The original impetus for the Programme came from World Health Assembly resolutions and the recommendations of the 1972 UN Conference on the Human Environment. Subsequently the work became an integral part of the International Programme on Chemical Safety (IPCS), a cooperative programme of UNEP, ILO and WHO. In this manner, with the strong support of the new partners, the

importance of occupational health and environmental effects was fully recognized. The EHC monographs have become widely established, used and recognized throughout the world.

The recommendations of the 1992 UN Conference on Environment and Development and the subsequent establishment of the Intergovernmental Forum on Chemical Safety with the priorities for action in the six programme areas of Chapter 19, Agenda 21, all lend further weight to the need for EHC assessments of the risks of chemicals.

Scope

The criteria monographs are intended to provide critical reviews on the effect on human health and the environment of chemicals and of combinations of chemicals and physical and biological agents. As such, they include and review studies that are of direct relevance for the evaluation. However, they do not describe *every* study carried out. Worldwide data are used and are quoted from original studies, not from abstracts or reviews. Both published and unpublished reports are considered and it is incumbent on the authors to assess all the articles cited in the references. Preference is always given to published data. Unpublished data are used only when relevant published data are absent or when they are pivotal to the risk assessment. A detailed policy statement is available that describes the procedures used for unpublished proprietary data so that this information can be used in the evaluation without compromising its confidential nature (WHO (1990) Revised Guidelines for the Preparation of Environmental Health Criteria Monographs. PCS/90.69, Geneva, World Health Organization).

In the evaluation of human health risks, sound human data, whenever available, are preferred to animal data. Animal and *in vitro* studies provide support and are used mainly to supply evidence missing from human studies. It is mandatory that research on human subjects is conducted in full accord with ethical principles, including the provisions of the Helsinki Declaration.

The EHC monographs are intended to assist national and international authorities in making risk assessments and subsequent risk management decisions. They represent a thorough evaluation of risks and are not, in any sense, recommendations for regulation or standard setting. These latter are the exclusive purview of national

and regional governments.

Content

The layout of EHC monographs for chemicals is outlined below.

- Summary – a review of the salient facts and the risk evaluation of the chemical
- Identity – physical and chemical properties, analytical methods
- Sources of exposure
- Environmental transport, distribution and transformation
- Environmental levels and human exposure
- Kinetics and metabolism in laboratory animals and humans
- Effects on laboratory mammals and *in vitro* test systems
- Effects on humans
- Effects on other organisms in the laboratory and field
- Evaluation of human health risks and effects on the environment
- Conclusions and recommendations for protection of human health and the environment
- Further research
- Previous evaluations by international bodies, e.g. IARC, JECFA, JMPR

Selection of chemicals

Since the inception of the EHC Programme, the IPCS has organized meetings of scientists to establish lists of priority chemicals for subsequent evaluation. Such meetings have been held in Ispra, Italy, 1980; Oxford, United Kingdom, 1984; Berlin, Germany, 1987; and North Carolina, USA, 1995. The selection of chemicals has been based on the following criteria: the existence of scientific evidence that the substance presents a hazard to human health and/or the environment; the possible use, persistence, accumulation or degradation of the substance shows that there may be significant human or environmental exposure; the size and nature of populations at risk (both human and other species) and risks for environment; international concern, i.e. the substance is of major interest to several countries; adequate data on the hazards are available.

If an EHC monograph is proposed for a chemical not on the priority list, the IPCS Secretariat consults with the Cooperating Organizations and all the Participating Institutions before embarking

on the preparation of the monograph.

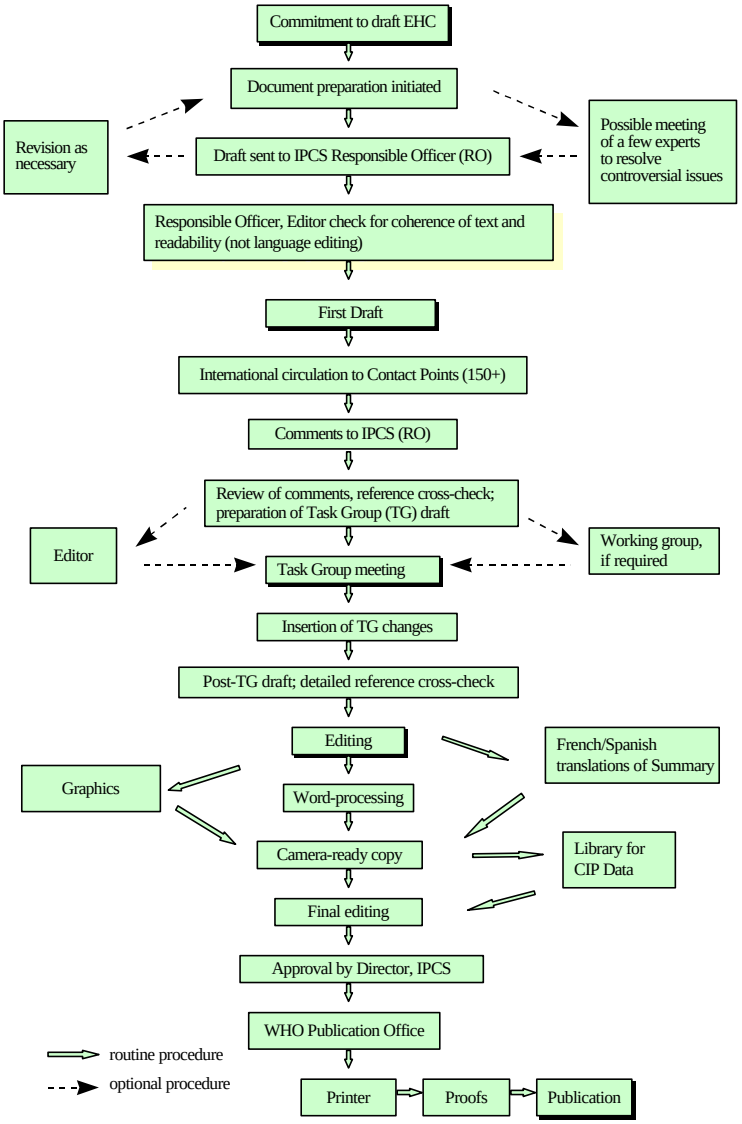
Procedures

The order of procedures that result in the publication of an EHC monograph is shown in the flow chart on p. xvii. A designated staff member of IPCS, responsible for the scientific quality of the document, serves as Responsible Officer (RO). The IPCS Editor is responsible for layout and language. The first draft, prepared by consultants or, more usually, staff from an IPCS Participating Institution, is based initially on data provided from the International Register of Potentially Toxic Chemicals, and reference data bases such as Medline and Toxline.

The draft document, when received by the RO, may require an initial review by a small panel of experts to determine its scientific quality and objectivity. Once the RO finds the document acceptable as a first draft, it is distributed, in its unedited form, to well over 150 EHC contact points throughout the world who are asked to comment on its completeness and accuracy and, where necessary, provide additional material. The contact points, usually designated by governments, may be Participating Institutions, IPCS Focal Points, or individual scientists known for their particular expertise. Generally some four months are allowed before the comments are considered by the RO and author(s). A second draft incorporating comments received and approved by the Director, IPCS, is then distributed to Task Group members, who carry out the peer review, at least six weeks before their meeting.

The Task Group members serve as individual scientists, not as representatives of any organization, government or industry. Their function is to evaluate the accuracy, significance and relevance of the information in the document and to assess the health and environmental risks from exposure to the chemical. A summary and recommendations for further research and improved safety aspects are also required. The composition of the Task Group is dictated by the range of expertise required for the subject of the meeting and by the need for a balanced geographical distribution.

EHC PREPARATION FLOW CHART



The three cooperating organizations of the IPCS recognize the important role played by nongovernmental organizations. Representatives from relevant national and international associations may be invited to join the Task Group as observers. Although observers may provide a valuable contribution to the process, they can only speak at the invitation of the Chairperson. Observers do not participate in the final evaluation of the chemical; this is the sole responsibility of the Task Group members. When the Task Group considers it to be appropriate, it may meet *in camera*.

All individuals who as authors, consultants or advisers participate in the preparation of the EHC monograph must, in addition to serving in their personal capacity as scientists, inform the RO if at any time a conflict of interest, whether actual or potential, could be perceived in their work. They are required to sign a conflict of interest statement. Such a procedure ensures the transparency and probity of the process.

When the Task Group has completed its review and the RO is satisfied as to the scientific correctness and completeness of the document, it then goes for language editing, reference checking and preparation of camera-ready copy. After approval by the Director, IPCS, the monograph is submitted to the WHO Office of Publications for printing. At this time a copy of the final draft is sent to the Chairperson and Rapporteur of the Task Group to check for any errors.

It is accepted that the following criteria should initiate the updating of an EHC monograph: new data are available that would substantially change the evaluation; there is public concern for health or environmental effects of the agent because of greater exposure; an appreciable time period has elapsed since the last evaluation.

All Participating Institutions are informed, through the EHC progress report, of the authors and institutions proposed for the drafting of the documents. A comprehensive file of all comments received on drafts of each EHC monograph is maintained and is available on request. The Chairpersons of Task Groups are briefed before each meeting on their role and responsibility in ensuring that these rules are followed.

WHO TASK GROUP ON ENVIRONMENTAL HEALTH CRITERIA FOR ARSENIC AND ARSENIC COMPOUNDS

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ENVIRONMENTAL HEALTH CRITERIA FOR ARSENIC AND ARSENIC COMPOUNDS

The first and second drafts of this monograph were prepared, under the coordination of Dr J. Ng, by the authors A. Gomez-Caminero, P. Howe, M. Hughes, E. Kenyon, D.R. Lewis, M. Moore, J. Ng, and by A. Aitio and G. Becking. The group of authors met at National Health and Environmental Effects Research Laboratory, US. EPA, North Carolina, on 20–22 July 1998.

A WHO Task Group on Environmental Health Criteria for Arsenic and Arsenic Compounds met at the National Research Centre for Environmental Toxicology, Brisbane, Australia, on 15–19 November 1999. The group reviewed the draft and the peer review comments, revised the draft and made an evaluation of the risks for human health and environment from exposure to arsenic and arsenic compounds.

After the meeting, and based on the peer reviewer comments and Task Group advice, Drs Gibb, Hopenhayn-Rich, Järup, Sim, and Aitio revised and updated the section on Effects on Human Health. This section was then sent for review to a selected group of experts.

The document was revised on the basis of the peer review comments received, these revisions were verified, and the document was finalized by a Review Board, consisting of Drs D. Anderson, H. Gibb, L. Järup, M. Sim and A. Aitio, in TNO BIBRA, Carshalton, UK. The document was finally approved by the Task Group in a mail ballot.

The cut-off date for the literature searches for the document was the Task Group meeting, i.e. November 1999, with the exception of the section on effects on human health, for which the last literature searches were performed in November 2000.

Peer review comments at the first stage international review were received from:

Dr J. Ahlers, Umwelt Bundes Amt, Germany
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Comments on the revised section on effects on human health were received from members of the Task Group, and from:

Dr D Anderson, TNO BIBRA International, UK
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Professor PE Enterline, University of Pittsburgh, USA
Dr A. Gomez-Caminero, National Health and Environmental Effects Research Laboratory, US. EPA
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Professor AH Smith, University of California, USA

Dr A. Aitio of the IPCS central unit was responsible for the scientific aspects of the monograph, and Kathleen Lyle for the technical editing.

The efforts of all, especially Queensland Health and the Natinal Research Centre for Environmental Toxicology, Australia, who helped in the preparation and finalization of the monograph are gratefully acknowledged.

ABBREVIATIONS

AAS	atomic absorption spectrometry
ABI	ankle–brachial index
AFS	atomic fluorescence spectrometry
AgDDTC	silver diethyldithiocarbamate
ALA	aminolaevulinic acid
ASV	anodic stripping voltammetry
ATPase	adenosine triphosphatase
AUC	area under the curve
BAL	dimercaprol
BCF	bioconcentration factor
BFD	blackfoot disease
BFD-endemic area	Geographic area in south-western Taiwan, where arsenic-contaminated artesian well water has been used as drinking water, and where BFD is endemic; the area has been also called the "arseniasis" area, or "hyperendemic" area. In this document it is called BFD-endemic area, to differentiate it from other areas e.g. in Taiwan, where high arsenic concentrations in drinking water have been reported
BMI	body mass index
BSO	*L*-buthionine-(RS)-sulfoximine
CA	chromosome aberrations
CAS	Chemical Abstract Service
CCA	copper chrome arsenate
CCGG	cytosine-cytosine-guanine-guanine
cDNA	complementary DNA
CE	capillary electrophoresis
CI	confidence interval; unless otherwise stated, the 95% CI is given. Accordingly, the term statistically significant in this documents denotes significance at 95% level
CVD	cardiovascular disease
DBDTC	sodium dibenzyldithiocarbamate

DD	duplicate diet study
DMA	dimethylarsinic acid
DMA^{3+}	dimethylarsinous acid
DMA-TGM	dimethylarsinic acid thioglycolic acid methyl ester
DMSA	dimercaptosuccinic acid
DPSCV	differential pulse cathodic stripping voltammetry
DSA	disodium arsenate heptahydrate
Eh	redox potential
EMG	electromyography
ETAAS	electrothermal atomic absorption spectrometry
FAAS	flame atomic absorption spectrometry
FAFS	flame atomic fluorescence spectrometry
FR	frequency ratio
GC	gas chromatography
GM-CSF	granulocyte macrophage-colony stimulating factor
GSH	glutathione
GSSG	oxidized glutathione
GTP	guanosine triphosphate
HFC	high frequency cell
HGAAS	hydride generation atomic absorption spectrometry
HMDE	hanging mercury drop electrode
HPLC	high pressure liquid chromatography
HPRT	hypoxanthine phosphoribosyltransferase
HSDB	Hazardous Substances Data Bank
ICP-AES	inductively coupled plasma atomic emission spectrometry
ICP-MS	inductively coupled plasma mass spectrometry
Ig	immunoglobulin
IHD	ischaemic heart disease
LC	liquid chromatography
LC_{50}	median lethal concentration
MB	market basket survey
MLC	micellar liquid chromatography
MMA	monomethylarsonic acid
MMA^{3+}	monomethylarsonous acid

MMA-TGM	monomethylarsonic acid thioglycolic acid methyl ester
MN	Micronucleus/i
mRNA	messenger RNA
MSMA	monosodium methanearsonate
MTHFR	5,10-methylene-tetradrofolate reductase
NAA	neutron activation analysis
NaFDDC	sodium (bistrifluoroethyl) dithiocarbamate
NER	nucleotide excision repair
4-NQO	4-nitroquinoline oxide
OR	odds ratio
PAD	periodate-oxidized adenosine
PIXES	particle-induced X-ray emission spectrometry
POR	prevalence odds ratio
PVD	peripheral vascular disease
RI	replication index
RPLC	reversed phase liquid chromatography
RTECS	Registry of Toxic Effects of Chemicals
SAH	*S*-adenosylhomocysteine
SAM	*S*-adenosyl methionine
SCE	sister chromatid exchange
SD	standard deviation
SE	standard error of mean
SEM	scanning electron microscopy
SFC	supercritical fluid chromatography
SFE	supercritical fluid extraction
SIR	standardized incidence ratio
SMR	standardized mortality ratio
SRBC	sheep red blood cell
TDT	toluene-3,4-dithiol
TGF	transforming growth factor
TGM	thioglycolic acid methylester
TMA	trimethylarsine
TMAO	trimethylarsine oxide
TWA	time-weighted average

UN	United Nations
UV	ultraviolet
XAFS	X-ray absorption fine structure spectroscopy
XRF	X-ray fluorescence

1. SUMMARY

1.1 Properties and analytical procedures

Arsenic is a metalloid widely distributed in the earth's crust and present at an average concentration of 2 mg/kg. It occurs in trace quantities in all rock, soil, water and air. Arsenic can exist in four valency states: –3, 0, +3 and +5. Under reducing conditions, arsenite (As(III)) is the dominant form; arsenate (As(V)) is generally the stable form in oxygenated environments. Elemental arsenic is not soluble in water. Arsenic salts exhibit a wide range of solubilities depending on pH and the ionic environment.

There is a variety of instrumental techniques for the determination of arsenic. These include AAS, AFS, ICP-AES, ICP-MS and voltammetry. Some of these (e.g. ICP-MS) can serve as element-specific detectors when coupled to chromatographic separation techniques (e.g. HPLC and GC). These so-called "hyphenated" methods are used for determining individual arsenic species. Additional sensitivity for a limited range of arsenic compounds can often be achieved by the use of hydride generation techniques. A test kit based on the colour reaction of arsine with mercuric bromide is currently used for groundwater testing in Bangladesh and has a detection limit of 50–100 µg/litre under field conditions.

1.2 Sources and occurrence of arsenic in the environment

Arsenic is present in more than 200 mineral species, the most common of which is arsenopyrite.

It has been estimated that about one-third of the atmospheric flux of arsenic is of natural origin. Volcanic action is the most important natural source of arsenic, followed by low-temperature volatilization.

Inorganic arsenic of geological origin is found in groundwater used as drinking-water in several parts of the world, for example Bangladesh.

Organic arsenic compounds such as arsenobetaine, arsenocholine, tetramethylarsonium salts, arsenosugars and arsenic-containing lipids are mainly found in marine organisms although some of these compounds have also been found in terrestrial species.

Elemental arsenic is produced by reduction of arsenic trioxide (As_2O_3) with charcoal. As_2O_3 is produced as a by-product of metal smelting operations. It has been estimated that 70% of the world arsenic production is used in timber treatment as copper chrome arsenate (CCA), 22% in agricultural chemicals, and the remainder in glass, pharmaceuticals and non-ferrous alloys.

Mining, smelting of non-ferrous metals and burning of fossil fuels are the major industrial processes that contribute to anthropogenic arsenic contamination of air, water and soil. Historically, use of arsenic-containing pesticides has left large tracts of agricultural land contaminated. The use of arsenic in the preservation of timber has also led to contamination of the environment.

1.3 Environmental transport and distribution

Arsenic is emitted into the atmosphere by high-temperature processes such as coal-fired power generation plants, burning vegetation and volcanism. Natural low-temperature biomethylation and reduction to arsines also releases arsenic into the atmosphere. Arsenic is released into the atmosphere primarily as As_2O_3 and exists mainly adsorbed on particulate matter. These particles are dispersed by the wind and are returned to the earth by wet or dry deposition. Arsines released from microbial sources in soils or sediments undergo oxidation in the air, reconverting the arsenic to non-volatile forms, which settle back to the ground. Dissolved forms of arsenic in the water column include arsenate, arsenite, methylarsonic acid (MMA) and dimethylarsinic acid (DMA). In well-oxygenated water and sediments, nearly all arsenic is present in the thermodynamically more stable pentavalent state (arsenate). Some arsenite and arsenate species can interchange oxidation state depending on redox potential (Eh), pH and biological processes. Some arsenic species have an affinity for clay mineral surfaces and organic matter and this can affect their environmental behaviour. There is potential for arsenic release when there is fluctuation in Eh, pH, soluble arsenic concentration and sediment organic content. Weathered rock and soil

may be transported by wind or water erosion. Many arsenic compounds tend to adsorb to soils, and leaching usually results in transportation over only short distances in soil.

Three major modes of arsenic biotransformation have been found to occur in the environment: redox transformation between arsenite and arsenate, the reduction and methylation of arsenic, and the biosynthesis of organoarsenic compounds. There is biogeochemical cycling of compounds formed from these processes.

1.4 Environmental levels and human exposure

Mean total arsenic concentrations in air from remote and rural areas range from 0.02 to 4 ng/m^3. Mean total arsenic concentrations in urban areas range from 3 to about 200 ng/m^3; much higher concentrations (> 1000 ng/m^3) have been measured in the vicinity of industrial sources, although in some areas this is decreasing because of pollution abatement measures. Concentrations of arsenic in open ocean seawater are typically 1–2 µg/litre. Arsenic is widely distributed in surface freshwaters, and concentrations in rivers and lakes are generally below 10 µg/litre, although individual samples may range up to 5 mg/litre near anthropogenic sources. Arsenic levels in groundwater average about 1–2 µg/litre except in areas with volcanic rock and sulfide mineral deposits where arsenic levels can range up to 3 mg/litre. Mean sediment arsenic concentrations range from 5 to 3000 mg/kg, with the higher levels occurring in areas of contamination. Background concentrations in soil range from 1 to 40 mg/kg, with mean values often around 5 mg/kg. Naturally elevated levels of arsenic in soils may be associated with geological substrata such as sulfide ores. Anthropogenically contaminated soils can have concentrations of arsenic up to several grams per 100 ml.

Marine organisms normally contain arsenic residues ranging from < 1 to more than 100 mg/kg, predominantly as organic arsenic species such as arsenosugars (macroalgae) and arsenobetaine (invertebrates and fish). Bioaccumulation of organic arsenic compounds, after their biogenesis from inorganic forms, occurs in aquatic organisms. Bioconcentration factors (BCFs) in freshwater invertebrates and fish for arsenic compounds are lower than for marine organisms. Biomagnification in aquatic food chains has not been observed. Background arsenic concentrations in freshwater and

terrestrial biota are usually less than 1 mg/kg (fresh weight). Terrestrial plants may accumulate arsenic by root uptake from the soil or by adsorption of airborne arsenic deposited on the leaves. Arsenic levels are higher in biota collected near anthropogenic sources or in areas with geothermal activity. Some species accumulate substantial levels, with mean concentrations of up to 3000 mg/kg at arsenical mine sites.

Non-occupational human exposure to arsenic in the environment is primarily through the ingestion of food and water. Of these, food is generally the principal contributor to the daily intake of total arsenic. In some areas arsenic in drinking-water is a significant source of exposure to inorganic arsenic. In these cases, arsenic in drinking-water often constitutes the principal contributor to the daily arsenic intake. Contaminated soils such as mine tailings are also a potential source of arsenic exposure. The daily intake of total arsenic from food and beverages is generally between 20 and 300 µg/day. Limited data indicate that approximately 25% of the arsenic present in food is inorganic, but this depends highly on the type of food ingested. Inorganic arsenic levels in fish and shellfish are low (< 1%). Foodstuffs such as meat, poultry, dairy products and cereals have higher levels of inorganic arsenic. Pulmonary exposure may contribute up to approximately 10 µg/day in a smoker and about 1 µg/day in a non-smoker, and more in polluted areas. The concentration of metabolites of inorganic arsenic in urine (inorganic arsenic, MMA and DMA) reflects the absorbed dose of inorganic arsenic on an individual level. Generally, it ranges from 5 to 20 µg As/litre, but may even exceed 1000 µg/litre.

In workplaces with up-to-date occupational hygiene practices, exposure generally does not exceed 10 $\mu g/m^3$ (8-h time-weighted average [TWA]). However, in some places workroom atmospheric arsenic concentrations as high as several milligrams per cubic metre have been reported.

1.5 Kinetics and metabolism

Absorption of arsenic in inhaled airborne particles is highly dependent on the solubility and the size of particles. Both pentavalent and trivalent soluble arsenic compounds are rapidly and extensively absorbed from the gastrointestinal tract. In many species

arsenic metabolism is characterized by two main types of reactions: (1) reduction reactions of pentavalent to trivalent arsenic, and (2) oxidative methylation reactions in which trivalent forms of arsenic are sequentially methylated to form mono-, di- and trimethylated products using *S*-adenosyl methionine (SAM) as the methyl donor and glutathione (GSH) as an essential co-factor. Methylation of inorganic arsenic facilitates the excretion of inorganic arsenic from the body, as the end-products MMA and DMA are readily excreted in urine. There are major qualitative and quantitative interspecies differences in methylation, to the extent that some species exhibit minimal or no arsenic methylation (e.g. marmoset monkey, guinea-pig, chimpanzee). However, in humans and most common laboratory animals, inorganic arsenic is extensively methylated and the metabolites are excreted primarily in the urine. Factors such as dose, age, gender and smoking contribute only minimally to the large inter-individual variation in arsenic methylation observed in humans. However, lower methylation efficiency in children has been observed in only one study out of three. Studies in humans suggest the existence of a wide difference in the activity of methyl-transferases, and the existence of polymorphism has been hypothesized. Animal and human studies suggest that arsenic methylation may be inhibited at high acute exposures. The metabolism and disposition of inorganic arsenic may be influenced by its valence state, particularly at high dose levels. Studies in laboratory animals indicate that administration of trivalent inorganic arsenic such as As_2O_3 and arsenite initially results in higher levels in most tissues than does the administration of pentavalent arsenic. However, the trivalent form is more extensively methylated, leading to similar long-term excretion. Ingested organoarsenicals such as MMA, DMA and arsenobetaine are much less extensively metabolized and more rapidly eliminated in urine than inorganic arsenic in both laboratory animals and humans.

Levels of arsenic or its metabolites in blood, hair, nails and urine are used as biomarkers of arsenic exposure. Blood arsenic is a useful biomarker only in the case of acute arsenic poisoning or stable chronic high-level exposure. Arsenic is rapidly cleared from blood, and speciation of its chemical forms in blood is difficult. Arsenic in hair and nails can be indicators of past arsenic exposure, provided care is taken to prevent external arsenic contamination of the samples. Arsenic in hair may also be used to estimate relative length

of time since an acute exposure. Speciated metabolites in urine expressed either as inorganic arsenic or as the sum of metabolites (inorganic arsenic + MMA + DMA) provide the best quantitative estimate of recently absorbed dose of arsenic. However, consumption of certain seafood, mainly seaweed and some bivalves, may confound estimation of inorganic arsenic exposure because of metabolism of arsenosugars to DMA in the body or the presence of DMA in the seafood. Such food should be avoided for 2–3 days before urine sampling for monitoring of exposure to inorganic arsenic.

1.6 Effects on laboratory animals and *in vitro* systems

Both inorganic and organic forms of arsenic may cause adverse effects in laboratory animals. The effects induced by arsenic range from acute lethality to chronic effects such as cancer. The degree of toxicity of arsenic is basically dependent on the form (e.g. inorganic or organic) and the oxidation state of the arsenical. It is generally considered that inorganic arsenicals are more toxic than organic arsenicals, and within these two classes, the trivalent forms are more toxic than the pentavalent forms, at least at high doses. Several different organ systems are affected by arsenic, including skin, respiratory, cardiovascular, immune, genitourinary, reproductive, gastrointestinal and nervous systems.

Several animal carcinogenicity studies on arsenic have been carried out, but limitations such as high dose levels, limited time of exposure and limited number of animals make these inconclusive. However, a recently reported animal model may be a useful tool for future carcinogenicity studies. In that study, female C57B1/6J mice exposed to arsenic in drinking-water containing 500 µg As(V)/litre over 2 years was associated with increased incidence in tumours involving mainly lung, liver, gastrointestinal tract and skin. Inorganic arsenic does not induce point mutations. However, arsenic can produce chromosomal aberrations *in vitro*, affect methylation and repair of DNA, induce cell proliferation, transform cells and promote tumours. One study has indicated that DMA may cause cancer of the urinary bladder in male rats at high doses.

1.7 Effects on human health

Soluble inorganic arsenic is acutely toxic, and ingestion of large doses leads to gastrointestinal symptoms, disturbances of cardiovascular and nervous system functions, and eventually death. In survivors, bone marrow depression, haemolysis, hepatomegaly, melanosis, polyneuropathy and encephalopathy may be observed.

Long-term exposure to arsenic in drinking-water is causally related to increased risks of cancer in the skin, lungs, bladder and kidney, as well as other skin changes such as hyperkeratosis and pigmentation changes. These effects have been demonstrated in many studies using different study designs. Exposure–response relationships and high risks have been observed for each of these end-points. The effects have been most thoroughly studied in Taiwan but there is considerable evidence from studies on populations in other countries as well. Increased risks of lung and bladder cancer and of arsenic-associated skin lesions have been reported to be associated with ingestion of drinking-water at concentrations ≤50 µg arsenic/litre.

Occupational exposure to arsenic, primarily by inhalation, is causally associated with lung cancer. Exposure–response relationships and high risks have been observed. Increased risks have been observed at cumulative exposure levels ≥ 0.75 $(mg/m^3) \cdot$ year (e.g. 15 years of exposure to a workroom air concentration of 50 $\mu g/m^3$). Tobacco smoking has been investigated in two of the three main smelter cohorts and was not found to be the cause of the increased lung cancer risk attributed to arsenic; however, it was found to be interactive with arsenic in increasing the lung cancer risk.

Even with some negative findings, the overall weight of evidence indicates that arsenic can cause clastogenic damage in different cell types with different end-points in exposed individuals and in cancer patients. For point mutations, the results are largely negative.

Chronic arsenic exposure in Taiwan has been shown to cause blackfoot disease (BFD), a severe form of peripheral vascular disease (PVD) which leads to gangrenous changes. This disease has

not been documented in other parts of the world, and the findings in Taiwan may depend upon other contributing factors. However, there is good evidence from studies in several countries that arsenic exposure causes other forms of PVD.

Conclusions on the causality of the relationship between arsenic exposure and other health effects are less clear-cut. The evidence is strongest for hypertension and cardiovascular disease, suggestive for diabetes and reproductive effects and weak for cerebrovascular disease, long-term neurological effects, and cancer at sites other than lung, bladder, kidney and skin.

1.8 Effects on other organisms in the environment

Aquatic and terrestrial biota show a wide range of sensitivities to different arsenic species. Their sensitivity is modified by biological and abiotic factors. In general, inorganic arsenicals are more toxic than organoarsenicals and arsenite is more toxic than arsenate. The mode of toxicity and mechanism of uptake of arsenate by organisms differ considerably. This may explain why there are interspecies differences in organism response to arsenate and arsenite. The primary mechanism of arsenite toxicity is considered to result from its binding to protein sulfhydryl groups. Arsenate is known to affect oxidative phosphorylation by competition with phosphate. In environments where phosphate concentrations are high, arsenate toxicity to biota is generally reduced. As arsenate is a phosphate analogue, organisms living in elevated arsenate environments must acquire the nutrient phosphorous yet avoid arsenic toxicity.

Arsenic compounds cause acute and chronic effects in individuals, populations and communities at concentrations ranging from a few micrograms to milligrams per litre, depending on species, time of exposure and end-points measured. These effects include lethality, inhibition of growth, photosynthesis and reproduction, and behavioural effects. Arsenic-contaminated environments are characterized by limited species abundance and diversity. If levels of arsenate are high enough, only species which exhibit resistance may be present.

RESUME

1. Propriétés et méthodes d'analyse

L’arsenic est un métalloïde largement répandu dans l’écorce terrestre où sa concentration moyenne est de 2 mg/kg. Il est présent à l’état de traces dans l’ensemble des roches, des sols et des eaux ainsi que dans l’air. L’arsenic présente quatre degrés d’oxydation : –3, 0, +3 et +5. En milieu réducteur, c’est l’arsénite qui constitue la forme prédominante (As III); en milieu oxygéné, c’est généralement l’arséniate (As V) qui constitue la forme stable. L’arsenic élémentaire n’est pas soluble dans l’eau. La solubilité des sels d’arsenic varie dans de larges proportions selon la valeur du pH et la nature des ions présents.

Il existe diverses méthodes instrumentales pour la recherche et le dosage de l'arsenic, notamment la spectrométrie d'absorption atomique (AAS), la spectrométrie de fluorescence atomique (AFS), la spectrométrie d'émission atomique à source plasma à couplage inductif, la spectrométrie d'émission Auger à source plasma à couplage inductif (ICP-AES) la spectrométrie de masse à source plasma à couplage inductif (ICP-MS) et la voltamétrie. Certaines d'entre elles peuvent être utilisées pour la détection spécifique d'un élément donné (par ex. l'ICP-MS) en les couplant avec à une technique de séparation chromatographique (chromatographie en phase liquide à haute performance ou chromatographie en phase gazeuse). Ces méthodes couplées sont utilisées pour le dosage des différentes espèces arsenicales. Pour quelques dérivés l'arsenic, on peut souvent accroître la sensibilité des dosages en utilisant des techniques basées sur la génération d'hydrure. Au Bangladesh, on utilise actuellement une trousse d'analyse basée sur la réaction colorée que l'arsine donne avec le bromure mercurique pour contrôler les eaux souterraines. Sur le terrain, la limite de détection autorisée par cette technique est de 50-100 μg/litre.

2. Etat naturel et sources environnementales diverses

L'arsenic est présent dans plus de 200 minéraux différents, dont le plus courant est l'arsénopyrite.

On estime qu'environ un tiers du flux arsenical atmosphérique est d'origine naturelle. La source naturelle la plus importante d'arsenic est l'activité volcanique, la volatilisation à basse température venant au second rang.

Dans de nombreuses régions du monde, et notamment au Bangladesh, les eaux souterraines utilisées pour la consommation contiennent de l'arsenic minéral géogène.

Des organoarsenicaux comme d'arsénobétaïne, l'arsénocholine, les sels de tétraméthylarsonium ou encore des arsénoglucides ou arsénolipides sont surtout présents dans les organismes marins mais on trouve également certains d'entre eux dans quelques espèces terrestres.

On prépare l'arsenic élémentaire en réduisant le trioxyde d'arsenic (As_2O_3) par le charbon de bois. Le trioxyde d'arsenic est un sous-produit des opérations de fonderie. On estime que 70% de la production mondiale d'arsenic est utilisée pour le traitement du bois d'oeuvre par l'arséniate double de cuivre et de chrome, 22% pour la préparation de produits chimiques à usage agricole, le reste entrant dans la composition de certains verres, produits pharmceutiques et alliages non ferreux.

Les exploitations minières, la fusion des métaux non ferreux et l'utilisation des combustibles fossiles sont les principaux processus industriels qui contribuent à la pollution anthropogénique de l'air, des sols et de l'eau par l'arsenic. De grandes étendues de terres agricoles sont depuis toujours contaminées par l'épandage de pesticides à base d'arsenic. La protection du bois d'oeuvre au moyen de dérivés arsenicaux contribue également à la pollution de l'environnement.

3. Transport et distribution dans l'environnement

De l'arsenic est libéré dans l'atmosphère lors de processus impliquant des températures élevées comme la combustion du charbon dans les centrales thermiques, le brûlage de la végétation et le volcanisme. La biométhylation a basse température et la réduction naturelles en arsines libère également de l'arsenic dans l'atmosphère. C'est essentiellement sous forme d'As_2O_3 que l'arsenic est libéré dans

l'atmosphère où il est en majeure partie adsorbé à des particules en suspension. Ces particules sont dispersées par le vent et regagnent sol par en se déposant par voie humide ou par voie sèche. Les arsines qui résultent de l'activité microbienne se dégagent du sol ou des sédiments où elles ont pris naissance pour s'oxyder à l'air en composés arsenicaux non volatils qui se redéposent. L'arsenic est présent dans la colonne d'eau sous la forme d'arséniates, d' arsénites ou encore d'acide méthylarsonique ou diméthylarsinique. Dans le sol et les sédiments bien oxygénés, la presque totalité de l'arsenic est présent sous sa forme pentavalente (arséniate), qui est la plus stable thermodynamiquement. Certains arsénites ou arséniates peuvent alterner leur degré d'oxydation sous l'influence du potentiel rédox (Eh), du pH et de certains processus biologiques. Quelques dérivés de l'arsenic présentent une certaine affinité pour les surfaces argileuses et les matières organiques, ce qui peut influer sur leur comportement dans l'environnement. Les fluctuations du potentiel rédox, du pH, de la concentration en dérivés arsenicaux solubles et de la teneur en matières organiques en suspension peuvent avoir pour conséquence la libération d'arsenic. Des débris résultant de l'érosion des rochers et des sols peuvent être transportés par le vent ou entraînés par les eaux. De nombreux dérivés de l'arsenic sont adorbés sur les particules de terre et le lessivage des sols ne les transporte habituellement que sur de courtes distances.

On observe trois types principaux de biotransformation de l'arsenic dans l'environnement : interconversion rédox entre arsénites et arséniates, réduction et méthylation de l'arsenic ou biosynthèse d'organoarsenicaux. Les composés formés selon ces divers processus participent à un cycle biogéochimique.

4. Concentrations dans l'environnement et exposition humaine

Dans les zones reculées et en milieu rural, la concentration moyenne de l'arsenic dans l'air va de 0,02 à 4 ng/m^3. Dans l'air des villes, la concentration moyenne va de 3 à environ 200 ng/m^3; des concentrations beaucoup plus fortes (> 1000 ng/m^3) ont été relevées à proximité de sources industrielles, encore que la tendance soit à la réduction de ces teneurs du fait des mesures de lutte contre la pollution. Les eaux pélagiques ont le plus souvent une teneur de

l'ordre de 1 à 2 µg/litre. L'arsenic est très répandu dans les eaux douces superficielles et sa concentration dans les cours d'eau et les lacs est généralement inférieure à 10 µg/litre, encore que l'on puisse trouver des valeurs pouvant aller jusqu'à 5 mg/litre dans des échantillons prélevés à proximité de sources anthropogéniques. Dans les eaux souterraines, la concentration de l'arsenic est d'environ 1 à 2 µg/litre sauf en présence de roches volcaniques ou de dépôts de sulfures, auquel cas la teneur en arsenic peut aller jusqu'à 3 mg/litre. La concentration moyenne dans les sédiments va de 5 à 3000 mg/kg, les valeurs les plus fortes se retrouvant dans les zones polluées. Dans le sol, la concentration de fond est comprise entre 1 et 40 mg/kg, la valeur moyenne se situant à 5 mg/kg. Une forte concentration naturelle d'arsenic peut être attribuée à certains substrats géologiques, par exemple à des minerais sulfurés. Dans les sols pollués par les activités humaines, la concentration de l'arsenic peut atteindre plusieurs grammes pour 100 ml.

Les résidus d'arsenic que l'on retrouve dans les organismes marins peuvent aller de < 1 à plus de 100 mg/kg, principalement sous la forme d'organoarsénicaux tels que les arsénoglucides (algues macroscopiques) et l'arsénobétaïne (invertébrés et poissons). Après biogénèse à partir des dérivés minéraux, les organoarsenicaux peuvent être accumulés par les organismes aquatiques. Le facteur de bioconcentration des dérivés arsenicaux est plus faible chez les invertébrés et les poissons d'eau douce que chez les organismes marins. On n'observe pas de bioamplification dans les différentes chaînes alimentaires aquatiques. La concentration de fond dans les biotes dulçaquicoles et terrestres est généralement inférieure à 1 mg/kg de poids frais. Les végétaux terrestres peuvent accumuler l'arsenic par captage radiculaire ou par adsorption foliaire des dépôts de particules aéroportées. Dans les échantillons biologiques prélevés à proximité de sources anthropogéniques où de zones d'activité géothermique, la concentration de l'arsenic est plus élevée. Certaines espèces en accumulent des quantités importantes, avec des concentrations qui peuvent atteindre 3000 mg/kg sur les sites miniers.

L'exposition à l'arsenic en dehors du cadre professionnel est essentiellement due à l'ingestion d'aliments et d'eau contaminés. La dose journalière totale est principalement constituée par l'apport alimentaire. Dans certaines régions, c'est l'eau de boisson qui constitue une source importante d'exposition à l'arsenic minéral. En

pareil cas, elle intervient souvent de façon prépondérante dans l'apport journalier d'arsenic. Les résidus des industries extractives constituent également une source potentielle d'exposition à l'arsenic. L'apport journalier d'arsenic provenant de l'alimentation et des boissons se situe en général entre 20 et 300 μg. Selon des données limitées, environ 25% de l'arsenic présent dans l'alimentation s'y trouve sous la forme de dérivés minéraux, mais cela dépend en grande partie de la nature des aliments ingérés. Les poissons et les fruits de mer ont une faible teneur en arsenic minéral (< 1%). En revanche, la concentration en arsenic minéral est plus élevée dans la viande, la volaille, les produits laitiers et les céréales. L'exposition par la voie respiratoire peut correspondre à 10 μg/jour environ chez un fumeur et à environ 1 μg/jour chez un non fumeur et même davantage dans les zones polluées. La concentration des métabolites de l'arsenic minéral dans les urines (arsenic minéral, acides monométhylarsonique et diméthylarsinique) est révélatrice de la dose d'arsenic minéral absorbée par un individu. En général, elle va de 5 à 20 μg d'arsenic par litre, mais elle peut dépasser 1000 μg par litre.

Sur les lieux de travail qui bénéficient d'une pratique de pointe en matière d'hygiène et sécurité, l'exposition ne dépasse généralement pas 10 $\mu g/m^3$ (en moyenne pondérée par rapport au temps calculée sur 8 h). Cependant, des concentrations pouvant atteindre plusieurs milligrammes par mètre cube ont été relevées dans l'air de certains lieux de travail.

5. Cinétique et métabolisme

Lorsque des particules aéroportées sont inhalées, l'absorption de l'arsenic qu'elles renferment dépend dans une très large mesure de la solubilité et de la taille des particules. Les dérivés solubles de l'arsenic III et de l'arsenic V sont rapidement et largement résorbés au niveau des voies digestives. Pour beaucoup de dérivés arsenicaux, le métabolisme se caractérise par deux types principaux de réactions: 1) des réactions de réduction de l'arsenic V en arsenic III et 2) des réactions de méthylation oxydatives dans lesquelles les dérivés de l'arsenic III subissent des méthylations successives qui les transforment en dérivés mono- di- et triméthylés, la *S*-adénosylméthionine (SAM) jouant le rôle de donneur de méthyle avec le glutathion(GSH)

comme co-facteur essentiel. La méthylation de l'arsenic minéral en facilite l'excrétion car les produits qui en résultent, à savoir l'acide monométhylarsonique et l'acide diméthylarsinique, sont facilement éliminés par la voie urinaire. Il existe des différences interspécifiques quantitatives et qualitatives très importantes en ce qui concerne ces réactions de méthylation, puisque chez certaines espèces, la méthylation de l'arsenic est minime, voire inexistante (par ex. chez le ouistiti, le cobaye ou le chimpanzé). Cependant, chez l'Homme comme chez la plupart des animaux de laboratoire, l'arsenic subit une méthylation importante conduisant à des métabolites qui sont essentiellement excrétés par voie urinaire. Des facteurs comme la dose, l'âge, le sexe ou le tabagisme ne jouent qu'un rôle minime dans les importantes variations interindividuelles observées chez l'Homme en ce qui concerne la méthylation métabolique de l'arsenic. Sur trois études consacrées à ce problème, une seule a révélé une moins bonne méthylation de l'arsenic chez l'enfant. Les études effectuées sur des sujets humains donnent à penser que l'activité des méthyltransférases varie dans d'importantes proportions et on a avancé l'hypothèse d'un polymorphisme pour expliquer cet état de choses. L'expérimentation humaine et animale indique que la méthylation de l'arsenic pourrait être inhibée en cas d'exposition aiguë. Le métabolisme et la distribution de l'arsenic minéral peuvent dépendre du degré d'oxydation de cet élément, notamment à forte dose. Les études effectuées sur des animaux de laboratoire indiquent que l'administration d'arsenic minéral trivalent sous la forme d'As_2O_3 ou d'arsénite, par exemple, peut initialement donner lieu à de plus fortes concentrations, dans la plupart des tissus, que l'administration d'arsenic pentavalent. Il reste que la forme trivalente est plus largement méthylée, ce qui conduit en définitive à une excrétion similaire à long terme. Chez l'Homme et chez l'animal, on constate qu'après ingestion, des organoarsenicaux tels que l'acide monométhylarsonique, l'acide diméthylarsinique ou la bétaïne subissent une moindre métabolisation et sont plus rapidement éliminés dans l'urine que les dérivés minéraux.

La concentration de l'arsenic ou de ses métabolites dans le sang, les cheveux, les ongles ou les urines sert de biomarqueur de l'exposition à l'arsenic. Le taux sanguin n'est utile comme marqueur qu'en cas d'intoxication aiguë ou d'exposition chronique constante et de longue durée. L'arsenic est rapidement éliminé du sang et il est difficile de déterminer sous quelle forme chimique il s'y trouve.

L'arsenic présent dans les cheveux et les ongles peut constituer un indicateur d'une exposition ancienne, dans la mesure où l'on prend la précaution d'éliminer toute contamination externe. On peut également se servir de la teneur des cheveux en arsenic pour évaluer le temps écoulé depuis une exposition à l'arsenic. Le dosage des divers métabolites urinaires exprimés soit en arsenic minéral, soit par la somme de ces métabolites (arsenic minéral, acide monométhylarsonique, acide diméthylarsinique) fournit la meilleure évaluation quantitative d'une dose d'arsenic récemment absorbée. Toutefois, la consommation de certains produits de la mer, notamment des algues et quelques bivalves, peut constituer un facteur de confusion dans l'estimation de l'exposition à l'arsenic, en raison de la métabolisation des arsénoglucides en acide diméthylarsinique par l'organisme du consommateur ou de la présence même de cet acide arsinique dans les produits consommés. Pendant les 2 à 3 jours précédant un prélèvement d'urine destiné à la recherche d'une exposition à des dérivés minéraux de l'arsenic, il faut s'abstenir de consommer tout produit de ce genre.

6. Effets sur les animaux de laboratoire et les systèmes d'épreuve *in vitro*

Qu'il soit sous forme minérale ou organique, l'arsenic peut provoquer des effets indésirables chez les animaux de laboratoire. Cette action toxique va de l'intoxication aiguë mortelle à des effets chroniques comme le cancer. Le degré de toxicité de l'arsenic dépend essentiellement de la forme (organique ou minérale) sous laquelle il se trouve et de son degré d'oxydation dans l'espèce chimique en cause. On estime en général que les dérivés minéraux de l'arsenic sont plus toxiques que les organoarsenicaux et qu'à l'intérieur de ces deux catégories de composés, les dérivés de l'arsenic III sont plus toxiques que ceux de l'arsenic V, tout au moins à forte dose. L'arsenic s'attaque à plusieurs systèmes et organes comme les téguments, l'appareil respiratoire, le système cardiovasculaire, le système immunitaire, l'appareil génito-urinaire, les gonades, les voies digestives et le système nerveux.

Plusieurs études de cancérogénicité ont été effectuées sur l'animal, mais un certain nombre de limites relatives à la dose administrée, à la durée d'exposition et au nombre d'animaux utilisés n'ont pas permis d'obtenir des résultats concluants. Toutefois, on a

récemment proposé un modèle animal qui pourrait se révéler interessant pour de futures études de cancérogénicité. On a ainsi effectué une étude sur des souris femelles C57B1/6J, qui a consisté à donner aux animaux pendant 2 ans une eau de boisson contenant 500 µg d'As V par litre. Les résultats obtenus montrent que l'incidence des tumeurs a augmenté, les localisations principales étant le poumon, le foie, les voies digestives, et la peau. L'arsenic minéral ne provoque pas de mutations ponctuelles. L'arsenic peut toutefois causer des aberrations chromosomiques ***in vitro***, affecter la méthylation et la réparation de l'ADN, provoquer la prolifération et la transformation des cellules et jouer le rôle de promoteur tumoral. Selon une étude, de fortes doses d'acide diméthylarsinique pourraient provoquer des cancers de la vessie chez le rat mâle.

7. Effets sur la santé humaine

Les dérivés minéraux solubles de l'arsenic sont fortement toxiques et l'ingestion de doses importantes entraîne l'apparition de symptômes gastrointestinaux et de troubles cardiovasculaires et neurologiques pouvant aboutir à la mort. Chez les survivants, on peut observer les anomalies suivantes: dépression médullaire, hémolyse, hépatomégalie, mélanose, polynévrite et encéphalopathie.

Il existe une relation de cause à effet entre une exposition de longue durée à l'arsenic présent dans l'eau de boisson et l'accroissement du risque de cancer de la peau, du poumon, de la vessie et du rein, ainsi que d'autres anomalies cutanées telles qu'une hyperkératose ou une modification de la pigmentation. Ces effets ont été mis en évidence dans de nombreuses études utilisant divers protocoles expérimentaux. Pour chacun de ces points d'aboutissement de l'action toxique de l'arsenic, on a pu établir une relation exposition-réponse et l'existence d'un risque important. C'est à Taiwan que ces effets ont été le plus étudiés, mais ils sont très largement attestés par des enquêtes menées dans les populations d'autres pays. On estime qu'il y a augmentation du risque de cancer du poumon ou de la vessie et de lésions cutanées d'origine arsenicale en cas de consommation d'eau de boisson dont la teneur est ≤ 50 µg/litre.

Il existe également une relation de cause à effet entre l'exposition, principalement respiratoire, à l'arsenic et le cancer du

poumon. Le risque est important et des relations exposition-réponse ont été établies. On observe une augmentation du risque lorsque l'exposition cumulée est ≥ 0.75 mg/m^3.année (c'est-à-dire, par exemple, 15 ans d'exposition à une concentration dans l'air de 50 μg/m^3). Dans deux des trois principales cohortes de fondeurs étudiées notamment sous l'angle du tabagisme, on a constaté que cette habitude n'était pas responsable de l'accroissement du risque de cancer du poumon attribué à l'arsenic; en revanche, il apparaît que l'interaction entre tabagisme et arsenic intervient dans l'augmentation de ce risque.

Malgré quelques résultats négatifs, tout semble indiquer que chez les individus et les cancéreux, l'arsenic a des effets clastogènes sur différents types de cellules et avec différents points d'aboutissement. En ce qui concerne les mutations ponctuelles, les résultats sont largement négatifs.

A Taiwan, on a montré qu'une exposition chronique à l'arsenic peut provoquer une affection appelée ‘‘maladie du pied noir’’. Il s'agit d'une vasculite périphérique grave pouvant aboutir à la gangrène. Cette affection n'est pas attestée dans d'autres régions du monde et il est possible que d'autres facteurs soient à prendre en compte dans la pathologie observée à Taiwan. Quoi qu'il en soit, on a de bonnes raisons de penser, compte tenu des études effectuées dans plusieurs pays, que l'exposition à l'arsenic peut être à l'origine d'autres formes de vasculite périphérique.

Les conclusions relatives à la relation de cause à effet entre l'exposition à l'arsenic et d'autres pathologies sont moins tranchées. C'est dans le cas de l'hypertension et des cardiopathies que les éléments d'appréciation sont les plus convaincants; ils sont révélateurs pour ce qui est du diabète et des effets sur la reproduction et faibles en ce qui concerne les accidents vasculaires cérébraux, les effets neurologiques à long terme et les cancers de localisation autre que le poumon, la vessie, le rein et la peau.

8. Effets sur les autres êtres vivants dans leur milieu naturel

La sensibilité des organismes aquatiques et terrestres aux divers dérivés de l'arsenic est très contrastée. Elle peut varier sous

l'influence de divers facteurs biologiques ou abiotiques. En général les composés minéraux sont plus toxiques que les organoarsenicaux et les arsénites davantage que les arséniates. Le type de toxicité et le mécanisme s'absorption des arséniates par les différents organismes varie considérablement. Ces considérations peuvent expliquer les différences de sensibilité aux arsénites et aux arséniates selon les espèces. On pense que la toxicité des arsénites résulte essentiellement de leur fixation aux groupements thiols des protéines. En ce qui concerne les arséniates, on sait qu'ils perturbent la phosphorylation oxydative par compétition avec les phosphates. Dans un environnement où la concentration en phosphates est élevée, les effets toxiques des arséniates se font généralement moins sentir. En raison de l'analogie entre phosphates et arséniates, les organismes qui vivent dans un milieu riche en arséniates, doivent fixer le phosphore tout en évitant l'action toxique de l'arsenic.

Les composés arsenicaux provoquent des intoxications aiguës et des effets chroniques au niveau individuel ou collectif à des concentrations allant de quelques microgrammes à plusieurs milligrammes par litre, selon la nature de l'espèce en cause, de la durée de l'exposition et du point d'aboutissement de l'effet toxique. Ces effets peuvent être mortels, se traduire par l'inhibition de la croissance ou de la photosynthèse, perturber la reproduction ou le comportement. Les milieux fortement pollués par l'arsenic se caractérisent par une limitation de l'abondance et de la diversité des espèces. Lorsque la concentration en arséniates atteint une valeur élevée, seules les espèces résistantes sont à même de survivre.

RESUMEN

1. Propiedades y procedimientos analíticos

El arsénico es un metaloide que se encuentra ampliamente distribuido en la corteza terrestre con una concentración media de 2 mg/kg. Está presente en cantidades ínfimas en todo tipo de rocas, suelos, agua y aire. El arsénico puede existir en cuatro estados de valencia: –3, 0, +3 y +5. En condiciones de reducción, la forma predominante es el arsenito (As(III)); el arseniato (As (V)) suele ser la forma estable en condiciones oxigenadas. El arsénico elemental no es soluble en agua. Las sales de arsénico tienen una amplia gama de solubilidades en función del pH y de las condiciones iónicas.

Hay diversas técnicas instrumentales para la determinación del arsénico. Entre ellas figuran la espectrometría de absorción atómica (AAS), la espectrometría de fluorescencia atómica (AFS), la espectrometría electrónica de Auger de plasma con acoplamiento inductivo (ICP-AES), la espectrometría de masas de plasma con acoplamiento inductivo (ICP-MS) y la voltametría. Algunas de éstas (por ejemplo, la espectrometría de masas de plasma con acoplamiento inductivo) pueden servir como detectores específicos del elemento cuando se combinan con técnicas de separación cromatográficas (por ejemplo, la cromatografía líquida de alto rendimiento y la cromatografía de gases). Estos métodos que combinan la cromatografía con la espectrometría se utilizan para determinar especies concretas de arsénico. Con frecuencia se puede conseguir una mayor sensibilidad para una serie limitada de compuestos de arsénico mediante el uso de técnicas de generación de hidruros. En Bangladesh se está utilizando actualmente un estuche de pruebas basado en la reacción coloreada de la arsina con el bromuro mercúrico para el análisis de las aguas freáticas, con un límite de detección de 50-100 µg/litro en las condiciones que se encuentran sobre el terreno.

2. Fuentes y presencia de arsénico en el medio ambiente

El arsénico está presente en más de 200 especies minerales, de las cuales la más común es la arsenopirita.

Se ha estimado que alrededor de un tercio del flujo atmosférico de arsénico es de origen natural. La actividad volcánica es la fuente natural más importante de arsénico, seguida de la volatilización a baja temperatura.

En el agua freática utilizada como agua de bebida en varias partes del mundo, por ejemplo en Bangladesh, hay arsénico inorgánico de origen geológico.

Los compuestos orgánicos de arsénico, por ejemplo la arsenobetaína, la arsenocolina, las sales de tetrametilarsonio, los arsenoazúcares y los lípidos con arsénico se encuentran fundamentalmente en organismos marinos, aunque también se han detectado algunos de estos compuestos en especies terrestres.

El arsénico elemental se obtiene por reacción del trióxido de arsénico (As_2O_3) con carbón vegetal. El As_2O_3 es un subproducto de las operaciones de fundición de metales. Se ha estimado que el 70% de la producción mundial de arsénico se utiliza en el tratamiento de la madera como arseniato de cobre y cromo, el 22% en productos químicos de uso agrícola y el resto en la obtención de vidrio, productos farmacéuticos y aleaciones no ferrosas.

La extracción y fundición de metales no ferrosos y la utilización de combustibles fósiles son los principales procesos industriales que contribuyen a la contaminación antropogénica del aire, el agua y el suelo con arsénico. El empleo de plaguicidas con arsénico a lo largo del tiempo ha dejado contaminadas amplias zonas de tierras agrícolas. El uso del arsénico en la conservación de la madera también ha llevado a la contaminación del medio ambiente.

3. Transporte y distribución en el medio ambiente

El arsénico se libera en la atmósfera mediante procesos de alta temperatura, como los de las centrales eléctricas alimentadas con carbón, la combustión de vegetación y los volcanes. El proceso natural de biometilación y reducción a arsinas a baja temperatura también libera arsénico en la atmósfera. La mayor parte del arsénico se libera en la atmósfera como As_2O_3, y se mantiene sobre todo adsorbido sobre la materia particulada. El viento dispersa estas partículas, que vuelven a la tierra mediante deposición húmeda o

seca. Las arsinas que se liberan de fuentes microbianas en el suelo o los sedimentos se oxidan en el aire, reconvirtiendo el arsénico en formas no volátiles que vuelven a la tierra. Entre las formas de arsénico disueltas en la columna de agua figuran el arseniato, el arsenito, el ácido metilarsónico y el ácido dimetilarsínico. En aguas y sedimentos bien oxigenados, casi todo el arsénico presente se encuentra en estado pentavalente, termodinámicamente más estable (arseniato). Algunas especies de arsenito y arseniato pueden intercambiar el estado de oxidación en función del potencial de oxidación-reducción, el pH y los procesos biológicos. Algunas especies de arsénico tienen afinidad por las superficies con minerales de la arcilla y la materia orgánica y esto puede afectar a su comportamiento en el medio ambiente. Es posible la emisión de arsénico cuando se producen fluctuaciones en el potencial de oxidación-reducción, el pH, la concentración de arsénico soluble y el contenido orgánico de los sedimentos. La erosión del viento o el agua puede transportar rocas y suelo meteorizados. Muchos compuestos de arsénico tienden a adsorberse en el suelo, y con la lixiviación suelen recorrer distancias cortas en este medio.

En el medio ambiente se han observado tres sistemas principales de biotransformación del arsénico: la transformación de oxidación-reducción entre el arsenito y el arseniato, la reducción y metilación del arsénico y la biosíntesis de compuestos orgánicos de arsénico. Hay un ciclo biogeoquímico de los compuestos formados a partir de estos procesos.

4. Niveles en el medio ambiente y exposición humana

Las concentraciones medias de arsénico total en el aire de zonas lejanas y rurales oscilan entre 0,02 y 4 ng/m^3. Las concentraciones medias de arsénico total en las zonas urbanas oscilan entre 3 y unos 200 ng/m^3; se han medido concentraciones mucho más altas (> 1000 ng/m^3) en las proximidades de fuentes industriales, aunque en algunas zonas están disminuyendo gracias a las medidas de reducción de la contaminación. Las concentraciones de arsénico en alta mar suelen ser de 1-2 µg/litro. El arsénico está ampliamente distribuido en el agua dulce superficial, siendo normalmente las concentraciones en ríos y lagos inferiores a 10 µg/litro, aunque en muestras aisladas se puedan alcanzar hasta 5 mg/litro cerca de fuentes antropogénicas. Los niveles de arsénico en el agua freática

son como promedio de alrededor de 1-2 µg/litro, excepto en las zonas con rocas volcánicas y depósitos de minerales de sulfuro, donde los niveles de arsénico pueden llegar a 3 mg/litro. Las concentraciones medias de arsénico en los sedimentos oscilan entre 5 y 3000 mg/kg, correspondiendo los niveles más altos a zonas contaminadas. La concentración de fondo en el suelo varía de 1 a 40 mg/kg, con valores medios frecuentes de alrededor de 5 mg/kg. Las concentraciones naturales elevadas de arsénico pueden estar asociadas con determinados sustratos geológicos, tales como las menas de sulfuro. Los suelos contaminados por actividades humanas pueden tener concentraciones de arsénico de hasta varios gramos por 100 ml.

Los organismos marinos suelen contener residuos de arsénico que oscilan entre < 1 y más de 100 mg/kg, predominantemente en forma de especies de arsénico orgánicas, como arsenoazúcares (macroalgas) y arsenobetaína (invertebrados y peces). En los organismos acuáticos se produce una bioacumulación de compuestos de arsénico orgánicos, tras su biogénesis a partir de las formas inorgánicas. Los factores de bioconcentración para los compuestos de arsénico son más bajos en los invertebrados y los peces de agua dulce que en los organismos marinos. No se ha observado bioamplificación en las cadenas alimentarias acuáticas. Las concentraciones de fondo de arsénico en la biota de agua dulce y terrestre son normalmente inferiores a 1 mg/kg (peso fresco). Las plantas terrestres pueden acumular arsénico por absorción radicular del suelo o mediante la adsorción del arsénico que deposita el aire en las hojas. Las concentraciones de arsénico son más elevadas en las muestras de biota recogidas en fuentes antropogénicas o en zonas con actividad geotérmica. Algunas especies acumulan niveles importantes, con concentraciones medias de hasta 3000 mg/kg en zonas con minas arsenicales.

La exposición humana no ocupacional al arsénico en el medio ambiente se produce fundamentalmente a través de la ingestión de alimentos y de agua. De éstos, suelen ser los alimentos los que más contribuyen a la ingesta diaria de arsénico total. En algunas zonas, el arsénico que contiene el agua de bebida es una fuente importante de exposición al arsénico inorgánico. En estos casos, el agua de bebida es con frecuencia lo que más contribuye a la ingesta de arsénico diaria. Los suelos contaminados, por ejemplo con desechos de minas,

son también una fuente potencial de exposición al arsénico. La ingesta diaria de arsénico total a partir de los alimentos y las bebidas oscila generalmente entre 20 y 300 µg/día. Los limitados datos disponibles indican que alrededor del 25% del arsénico presente en los alimentos es inorgánico, pero esto depende sobre todo del tipo de alimentos ingeridos. Los niveles de arsénico inorgánico en los peces y los mariscos son bajos (< 1%). Productos alimenticios como la carne, los productos lácteos y los cereales tienen niveles más elevados de arsénico inorgánico. De la exposición pulmonar pueden proceder hasta alrededor de 10 µg/día en un fumador y en torno a 1 µg/día en una persona no fumadora, siendo más elevados los valores en las zonas contaminadas. La concentración de metabolitos de arsénico inorgánico en la orina (arsénico inorgánico, ácido metilarsónico y ácido dimetilarsínico) refleja la dosis absorbida de arsénico inorgánico con carácter individual. En general, oscila entre 5 y 20 µg de arsénico/litro, pero puede incluso superar los 1000 µg/litro.

En lugares de trabajo con prácticas de higiene ocupacional actualizadas, la exposición generalmente no supera los 10 µg/m^3 (promedio ponderado por el tiempo de ocho horas). Sin embargo, en algunos lugares se han notificado concentraciones de arsénico atmosférico en los recintos de trabajo de hasta varios mg por metro cúbico.

5. Cinética y metabolismo

La absorción de arsénico en las partículas suspendidas en el aire inhaladas depende fundamentalmente de la solubilidad y del tamaño de las partículas. Los compuestos de arsénico solubles, tanto pentavalentes como trivalentes, se absorben en gran medida del tracto gastrointestinal con rapidez. En muchas especies, el metabolismo del arsénico se caracteriza por dos tipos principales de reacciones: 1) reacciones de reducción del arsénico pentavalente a trivalente, y 2) reacciones de metilación oxidativa, en las cuales las formas de arsénico trivalente experimentan una metilación secuencial que da lugar a productos mono, di y trimetilados, utilizando *S*-adenosilmetionina como donante de metilo y glutatión como un cofactor esencial. La metilación del arsénico inorgánico facilita su excreción del organismo, puesto que los productos finales, el ácido metilarsónico y el ácido dimetilarsínico, se excretan

fácilmente en la orina. Hay importantes diferencias interespecíficas cualitativas y cuantitativas en la metilación, hasta tal punto que algunas especies muestran una metilación mínima o nula (por ejemplo, el tití, el cobaya y el chimpancé). Sin embargo, el arsénico inorgánico se metila en gran parte en las personas y en los animales de laboratorio más comunes y los metabolitos se excretan fundamentalmente en la orina. Factores como la dosis, la edad, el sexo y el hábito de fumar contribuyen sólo de manera mínima a la importante variación que se observa en la metilación del arsénico en las personas. Sin embargo, sólo en un estudio de tres se ha observado una eficacia de la metilación más baja en los niños. Los estudios en las personas parecen indicar que existe una amplia diferencia en la actividad de las metiltransferasas y se ha planteado la hipótesis de la existencia de polimorfismo. De los estudios realizados en animales y personas parece deducirse que la metilación del arsénico se puede inhibir con niveles de exposición aguda elevados. El metabolismo y la eliminación del arsénico inorgánico pueden depender de su valencia, sobre todo cuando se trata de dosis elevadas. Los estudios en animales de laboratorio indican que la administración de arsénico inorgánico trivalente, como el As_2O_3 y el arsenito, produce inicialmente en la mayoría de los tejidos niveles más altos que la administración de arsénico pentavalente. Sin embargo, la forma trivalente se metila en mayor medida, dando lugar a una excreción prolongada semejante. Los compuestos organoarsenicales ingeridos, como los ácidos metilarsónico y dimetilarsínico y la arsenobetaína, se metabolizan mucho menos y se eliminan con mayor rapidez en la orina que el arsénico inorgánico, tanto en animales de laboratorio como en el ser humano.

Los niveles de arsénico o de sus metabolitos en la sangre, el pelo, las uñas y la orina se utilizan como biomarcadores de la exposición al arsénico. El arsénico en sangre es un biomarcador útil solamente en el caso de intoxicación aguda por arsénico o de exposición crónica estable elevada. El arsénico se elimina de la sangre con rapidez y la especiación de su formas químicas en la sangre es difícil. Su presencia en el pelo y las uñas puede ser un indicador de una exposición anterior al arsénico, siempre que se tenga la precaución de impedir la contaminación de las muestras con arsénico externo. El arsénico en el pelo se puede utilizar también para estimar el período de tiempo relativo desde una exposición aguda. La especiación de los metabolitos en la orina expresados

como arsénico inorgánico o bien como la suma de los metabolitos (arsénico inorgánico + ácido metilarsónico + ácido dimetilarsínico) proporciona la mejor estimación cuantitativa de la dosis de arsénico absorbida recientemente. Sin embargo, el consumo de ciertos alimentos marinos, principalmente algas y algunos bivalvos, puede confundir en la estimación de la exposición al arsénico inorgánico, debido al metabolismo de los arsenoazúcares para dar lugar a ácido dimetilarsínico en el organismo o a la presencia de ácido dimetilarsínico en los alimentos marinos. Dichos alimentos deberían evitarse durante dos o tres días antes del muestreo de la orina para vigilar la exposición al arsénico inorgánico.

6. Efectos en los animales de laboratorio y en los sistemas *in vitro*

Tanto las formas orgánicas como las inorgánicas del arsénico pueden causar efectos adversos en los animales de laboratorio. Los efectos inducidos por el arsénico van desde la letalidad aguda hasta los efectos crónicos, como el cáncer. El grado de toxicidad del arsénico depende básicamente de la forma (por ejemplo, inorgánico u orgánico) y de su estado de oxidación. En general, se considera que los compuestos arsenicales inorgánicos son más tóxicos que los orgánicos, y dentro de estas dos categorías las formas trivalentes son más tóxicas que las pentavalentes, por lo menos en dosis elevadas. El arsénico afecta a varios sistemas diferentes de órganos, como el cutáneo, el respiratorio, el cardiovascular, el genitourinario, el reproductivo, el gastrointestinal y el nervioso.

Se han realizado varios estudios de carcinogenicidad en animales, pero no se ha llegado a ninguna conclusión debido a limitaciones como las dosis elevadas, el tiempo limitado de exposición y el escaso número de animales. Sin embargo, se ha descrito recientemente un modelo en animales que podría ser un instrumento útil para futuros estudios de carcinogenicidad. En ese estudio, se relacionó la exposición de ratones C57B1/6J hembras a concentraciones de arsénico en el agua de bebida de 500 µg de As(V)/litro durante dos años con una mayor incidencia de tumores que afectaban principalmente al pulmón, el hígado, el tracto gastrointestinal y la piel. El arsénico inorgánico no induce mutaciones puntuales. Sin embargo, el arsénico puede producir aberraciones cromosómicas *in vitro*, afectar a la metilación y

reparación del ADN, inducir proliferación celular, transformar las células y favorecer la formación de tumores. En un estudio se ha indicado que en dosis elevadas el ácido dimetilarsínico puede provocar en ratas machos cáncer de la vejiga urinaria.

7. Efectos en el ser humano

El arsénico inorgánico soluble produce toxicidad aguda y la ingestión de dosis altas provoca síntomas gastrointestinales, trastornos de las funciones de los sistemas cardiovascular y nervioso y en último término la muerte. En los supervivientes se ha observado depresión de la médula ósea, hemólisis, hepatomegalia, melanosis, polineuropatía y encefalopatía.

La exposición prolongada al arsénico en el agua de bebida tiene una relación causal con un aumento de los riesgos de cáncer de piel, de pulmón, de vejiga y de riñón, así como con otros cambios cutáneos, por ejemplo hiperqueratosis y cambios de pigmentación. Estos efectos se han puesto de manifiesto en numerosos estudios utilizando diferentes diseños. Se han observado relación exposición-respuesta y riesgo alto para cada uno de estos efectos finales. Los efectos se han estudiado más detalladamente en Taiwan, pero también hay un gran número de pruebas de estudios sobre poblaciones en otros países. Se ha notificado un riesgo mayor de cáncer de pulmón y de vejiga y de lesiones cutáneas asociadas con el arsénico en relación con la ingestión de agua de bebida con concentraciones $\leq$ 50 µg de arsénico/litro.

Se ha encontrado una relación causal entre la exposición ocupacional al arsénico, principalmente por inhalación, y el cáncer de pulmón. Se han observado relaciones exposición-respuesta y riesgos altos. Se ha detectado un aumento del riesgo a niveles de exposición acumulativa $\geq$0,75 (mg/m^3) por año (por ejemplo, 15 años de exposición a una concentración en el aire del recinto de trabajo de 50 µg/m^3). El humo del tabaco se ha investigado en dos de las tres cohortes principales de fundidores y no se observó que fuera la causa del aumento del riesgo de cáncer de pulmón atribuido al arsénico; sin embargo, se comprobó que había una interacción con el arsénico que daba lugar a un aumento del riesgo de cáncer de pulmón.

A pesar de que algunos resultados han sido negativos, el valor demostrativo en conjunto indica que el arsénico puede provocar daños clastogénicos en diferentes tipos de células con distintos efectos finales en las personas expuestas y en los enfermos de cáncer. Para las mutaciones puntuales, los resultados son fundamentalmente negativos.

Se ha demostrado que la exposición crónica al arsénico en Taiwan provocó "blackfoot", una forma grave de enfermedad vascular periférica que da lugar a cambios gangrenosos. Esta enfermedad no se ha documentado en otras partes del mundo y los resultados de Taiwan pueden depender de la contribución de otros factores. Sin embargo, hay pruebas convincentes de estudios en varios países de que la exposición al arsénico produce otras formas de enfermedad vascular periférica.

Las conclusiones sobre la causalidad de la relación entre la exposición al arsénico y otros efectos en la salud son menos claras. Las pruebas más convincentes son las relativas a la hipertensión y las enfermedades cardiovasculares, son dudosas para la diabetes e insuficientes para la enfermedad cerebrovascular, los efectos neurológicos prolongados y el cáncer en lugares distintos del pulmón, la vejiga, el riñón y la piel.

8. Efectos en otros organismos en el medio ambiente

La sensibilidad de la biota acuática y terrestre para las distintas especies de arsénico es muy variable. Depende de factores biológicos y abióticos. En general, los compuestos arsenicales inorgánicos son más tóxicos que los orgánicos, y el arsenito es más tóxico que el arseniato. El mecanismo de la toxicidad y el sistema de absorción del arseniato por los organismos varían considerablemente de unos a otros. Esto puede explicar las diferencias interespecíficas en la respuesta de los organismos al arseniato y el arsenito. Se considera que el mecanismo primario de la toxicidad del arsenito se deriva de su unión a los grupos sulfhidrilo de las proteínas. Se sabe que el arseniato afecta a la fosforilación oxidativa, porque compite con el fosfato. En condiciones en las cuales la concentración de fosfato es alta, la toxicidad del arseniato para la biota es generalmente reducida. Como el arseniato es análogo al fosfato, los organismos que viven en presencia de una concentración elevada de arseniato

deben adquirir el fósforo nutritivo, pero evitando la toxicidad del arsénico.

Los compuestos de arsénico provocan efectos agudos y crónicos en las personas, las poblaciones y las comunidades a concentraciones que oscilan entre unos microgramos y miligramos por litro, dependiendo de la especie, el tiempo de exposición y los efectos finales medidos. Estos efectos incluyen la letalidad, la inhibición del crecimiento, de la fotosíntesis y de la reproducción y efectos de comportamiento. Las zonas contaminadas con arsénico se caracterizan por una abundancia y diversidad limitadas de especies. Si los niveles de arseniato son suficientemente altos, sólo es posible la presencia de especies resistentes.

2. PROPERTIES AND ANALYTICAL PROCEDURES

2.1 Identity

Elemental arsenic (As) is a member of Group 15 of the periodic table, with nitrogen, phosphorus, antimony and bismuth. It has an atomic number of 33 and an atomic mass of 74.91. The Chemical Abstract Service (CAS), National Institute for Occupational Safety and Health Registry of Toxic Effects of Chemicals (RTECS), Hazardous Substances Data Bank (HSDB), European Commission, and UN transport class numbers are 7440-38-2, HSB 509, CG 05235 000, 033-001-00-X and UN 1558, respectively.

This monograph deals with arsenic and inorganic and organic arsenic compounds, except arsine (AsH_3), for which a Concise International Chemical Assessment Document (CICAD) is being prepared.

2.2 Chemical and physical properties of arsenic compounds

Arsenic is a metalloid widely distributed in the earth's crust. It can exist in four valency states; –3, 0, +3, and +5. In strongly reducing environments, elemental arsenic and arsine (–3) can exist. Under moderately reducing conditions, arsenite (+3) may be the dominant form, but arsenate (+5) is generally the stable oxidation state in oxygenated environments.

Arsenic and its compounds occur in crystalline, powder, amorphous or vitreous forms. They usually occur in trace quantities in all rock, soil, water and air. However, concentrations may be higher in certain areas as a result of weathering and anthropogenic activities including metal mining and smelting, fossil fuel combustion and pesticide use.

Arsenical salts exhibit a range of aqueous solubilities depending on the pH and the ionic environment.

There are many arsenic compounds of environmental importance. Representative marine arsenic-containing compounds, of which some are found in terrestrial systems, are shown in Table 1; their molecular structures are shown Fig. 1. Other arsenic compounds discussed in the text are listed in Table 2.

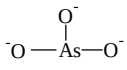
Table 1. Naturally occurring inorganic and organic As species (see Fig. 1 for structures [1]–[22])

CAS No.	Name	Synonyms	Structure
	arsenate		[1]
	arsenite		[2]
124-58-3	methylarsonic acid	monomethylarsonic acid, MMA	[3]
75-60-5	dimethylarsinic acid	cacodylic acid, DMA	[4]
4964-14-1	trimethylarsine oxide		[5]
27742-38-7	tetramethylarsonium ion		[6]
64436-13-1	arsenobetaine		[7]
39895-81-3	arsenocholine		[8]
	dimethylarsinoylribosides		[9]–[19]
	trialkylarsonioribosides		[20], [21]
	dimethylarsinoylribitol sulfate		[22]

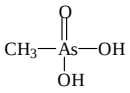
Speciation determines how arsenic compounds interact with their environment. For example, the behaviour of arsenate and arsenite in soil differs considerably. Movement in environmental matrices is a strong function of speciation and soil type. In a non-absorbing sandy loam, arsenite is 5–8 times more mobile than arsenate (Gulens et al., 1979). Soil pH also influences arsenic mobility. At a pH of 5.8 arsenate is slightly more mobile than arsenite, but when pH changes from acidic to neutral to basic, arsenite increasingly tends to become the more mobile species, though mobility of both arsenite and arsenate increases with increasing pH (Gulens et al., 1979). In strongly adsorbing soils, transport rate and speciation are influenced by organic carbon

Fig 1. Structures of naturally occurring inorganic and organic arsenic species

1



2



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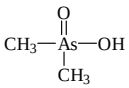


4

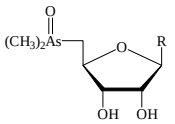
$(CH_3)_3As{=}O$ **5**

$(CH_3)_4As^+$ **6**

$(CH_3)_3As^+CH_2COO^-$ **7**

$(CH_3)As^+CH_2CH_2OH$ **8**

9 – 19



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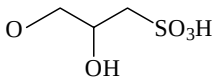
R= 

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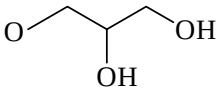
R= 

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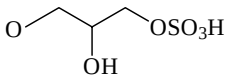
R= 

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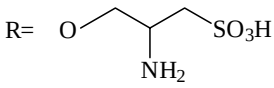
R= 

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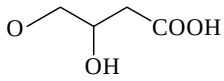
14

R= 

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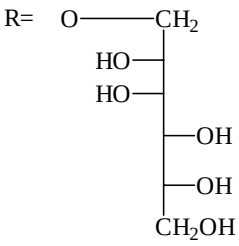
R= 

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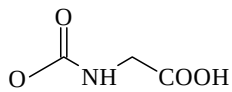
R= 

Fig. 1. (contd.)

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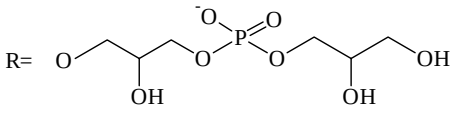

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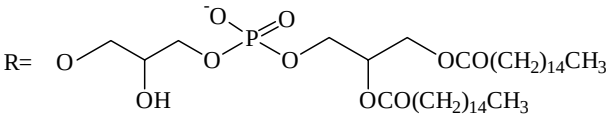

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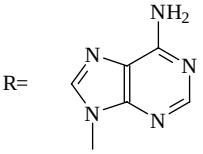

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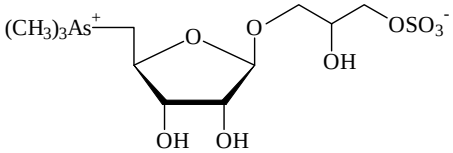

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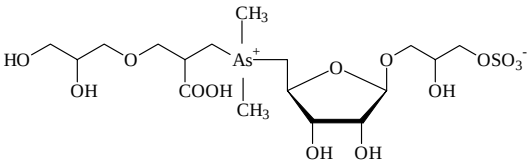

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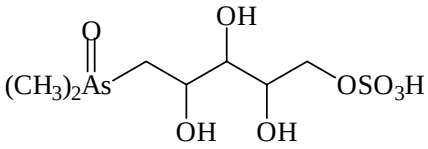
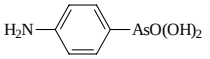
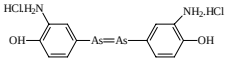
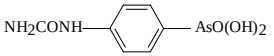
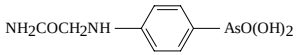
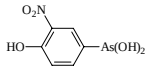
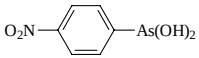


Table 2. Other As compounds of environmental significance referred to in the text

CAS No.	Name	Synonyms	Formula
	Inorganic As, trivalent		
1327-53-3	As(III) oxide	As trioxide, arsenous oxide, white As	As_2O_3 (or As_4O_6)
13768-07-5	arsenenous acid	arsenious acid	$HAsO_2$
7784-34-1	As(III) chloride	As trichloride, arsenous trichloride	$AsCl_3$
1303-33-9	As(III) sulfide	As trisulfide orpiment, auripigment	As_2S_3
	Inorganic As, pentavalent		
1303-28-2	As(V) oxide	As pentoxide	As_2O_5
7778-39-4	arsenic acid	*ortho*-arsenic acid	H_3AsO_4
10102-53-1	arsenenic acid	*meta*-arsenic acid	$HAsO_3$
	arsenates, salts of *ortho*-arsenic acid		$H_2AsO_4^-$, $HAsO_4^{2-}$, AsO_4^{3-}
	Organic As		
593-52-2	methylarsine		CH_3AsH_2
593-57-7	dimethylarsine		$(CH_3)_2AsH$
593-88-4	trimethylarsine		$(CH_3)_3As$

Table 2 (contd.)

CAS No.	Name	Synonyms	Formula
98-50-0	(4-aminophenyl)-arsonic acid	arsanilic acid, *p*-aminobenzene-arsonic acid	
139-93-5	4,4-arsenobis(2-aminophenol) dihydrochloride	arsphenamine, salvarsan	
121-59-5	[4-[aminocarbonyl-amino]phenyl] arsonic acid	carbarsone, *N*-carbamoylarsanilic acid	
554-72-3	[4-[2-amino-2-oxoethyl)amino]-phenyl] arsonic acid	tryparsamide	
121-19-7	3-nitro-4-hydroxy-phenylarsonic acid		
98-72-6	4-nitrophenylarsonic acid	*p*-nitrophenylarsonic acid	
	dialkylchloroarsine		R_2AsCl
	alkyldichloroarsine		$RasCl_2$

content and microbial population. Both arsenite and arsenate are transported at a slower rate in strongly adsorbing soils than in sandy soils.

Under oxidizing and aerated conditions, the predominant form of arsenic in water and soil is arsenate. Under reducing and waterlogged conditions (< 200 mV), arsenites should be the predominant arsenic compounds. The rate of conversion is dependent on the Eh and pH of the soil as well as on other physical, chemical and biological factors.

In brief, at moderate or high Eh, arsenic can be stabilized as a series of pentavalent (arsenate) oxyanions, H_3AsO_4, $H_2AsO_4^-$, $HAsO_4^{2-}$ and AsO_4^{3-}. However, under most reducing (acid and mildly alkaline) conditions, arsenite predominates. A pH and Eh diagram is shown in Fig. 2.

2.3 Analytical procedures

Historically, colorimetric and gravimetric methods have been used for the determination of arsenic. However, these methods are either semi-quantitative or lack sensitivity. In recent years, atomic absorption spectrometry (AAS) has become the method of choice, as it offers the possibility of selectivity and sensitivity in the detection of a wide range of metals and non-metals including arsenic. Popular methods for generating atoms for AAS are flame and electro-thermally heated graphite furnaces. However, a commonly used technique for the measurement of arsenic is the highly sensitive hydride generation atomic absorption spectrometric method (HGAAS). However, although it is suitable for total arsenic determination after appropriate digestion the technique is only routinely used to speciate a limited number of compounds – arsenite, arsenate, MMA, DMA, trimethylarsine oxide (TMAO).

Hydride generation followed by cryogenic trapping and AAS detection is a relatively inexpensive technique for the speciation of inorganic arsenic and its methylated metabolites (Ng et al., 1998a), although more expensive hyphenated techniques may also be used.

A number of other approaches have been reported for speciation of arsenic. Inductively coupled plasma-mass spectrometry (ICP-MS)

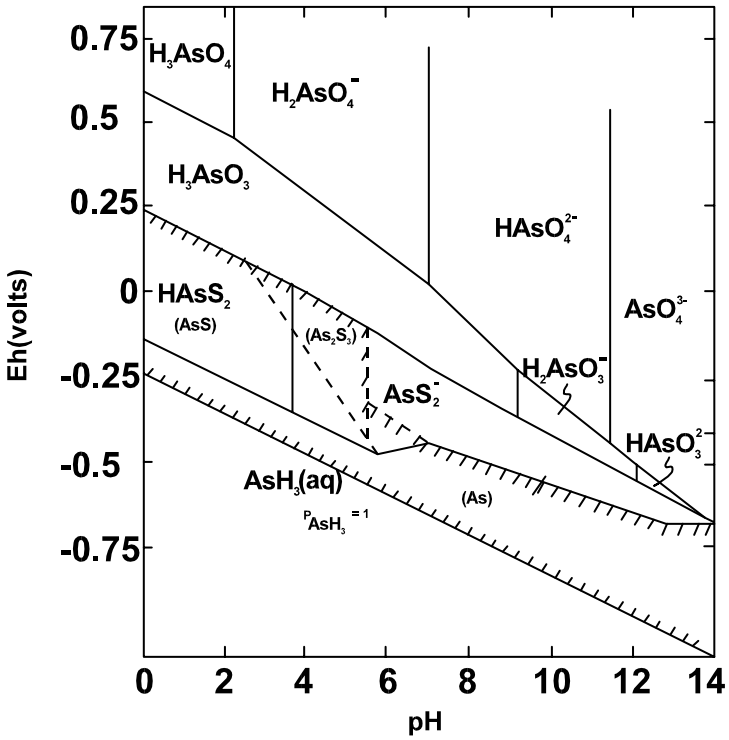


Fig. 2. The Eh-pH diagram for As at 25 °C and one atmosphere with total arsenic 10^{-5} mol/litre and total sulfur 10^{-3} mol/litre. Solid species are enclosed in parentheses in cross-hatched area, which indicates solubility less than $10^{-5.3}$ mol/litre (From: Ferguson & Gavis, 1972).

offers very high sensitivity for the determination of arsenic, and coupled with HPLC enables equally sensitive estimation of a wide variety of arsenic species.

2.4 Sample preparation and treatment

2.4.1 Sampling and collection

Care must be taken to avoid contamination and prevent speciation changes during sample collection and storage. Plastic containers should be acid washed and traces of oxidizing and reducing agents avoided to preserve the oxidation state of arsenic compounds. Freezing samples to –80 °C has also been recommended (Crecelius, 1986). Concentrated hydrochloric acid (1 ml to 100 ml urine) has been added to urine to prevent bacterial growth (Concha et al., 1998a).

For particulates in air and aerosols sampling, various types of filter have been employed including polytetrafluoroethylene (Rabano et al., 1989), cellulose ester (Yager et al., 1997), glass microfibre (Beceiro-Gonzalez et al.,1997) and filter paper (Tripathi et al., 1997).

2.4.2 Oxidative digestion

Acid digestion (George & Roscoe, 1951) and dry ashing (George et al., 1973) are the two basic methods which have been widely employed for oxidative digestion of samples before analysis. In more recent years, microwave-assisted digestion has been used (Le et al., 1994b; Thomas et al., 1997). For analysis of biological soft tissues by ICP techniques, a simple partial digestion in a closed vessel at low temperature and pressure is often sufficient for the sample preparation and pretreatment step.

2.4.3 Extraction

For speciation of arsenic, solvent extraction is often required before analysis. For example, arsenite and arsenate in soil can be speciated after a hydrochloric acid and chloroform extraction procedure (Chappell et al., 1995; Ng et al., 1998b). Water has been used for the extraction of soluble arsenic compounds from soil with the aid of ultrasonic treatment (Hansen et al., 1992). Forms of arsenic compounds can also be separated by sequential extractions based on procedures described by Tessier et al. (1979). Aqueous methanol has been widely used for the extraction of organic arsenic species (Edmonds & Francesconi, 1981a; Shiomi et al., 1988a;

Shibata et al., 1996; Kuehnelt et al., 1997). Yu & Wai (1991) and Laintz et al. (1992) described the use of sodium bis(trifluoroethyl) dithiocarbamate (NaFDDC) as a selective chelation reagent of arsenic followed by either a gas chromatograph (GC) detection or supercritical fluid chromatography (SFC) detection. The former gave a limit of detection of 10 µg As/litre in water and the latter gave similar sensitivity after 100–1000-fold preconcentration of the chelate complex in organic solvent.

2.4.4 Supercritical fluid extraction

There are very few publications on the use of supercritical fluid extraction (SFE) for the determination of arsenic. Wenclawiak & Krah (1995) reported a procedure for the measurement of arsenic species using SFE followed by GC or SFC detection. The authors described a rapid extraction of organic and inorganic arsenic species from spiked sand and soil samples by SFE with on-line derivatization using thioglycollic acid methylester (TGM) under supercritical conditions. The TGM derivatives are thermally stable, which makes them amenable to GC–SFC determination. The extracts were chromatographed without further clean-up steps. The limits of detection were 1 ng As/µl and 3 ng As/µl injection for DMA-TGM and MMA-TGM respectively.

2.5 Macro-measurement

Most procedures for the separation and determination of arsenic are based on distillation and hydrogen sulfide precipitation methods. Beard & Lyerly (1961) reported a gravimetric method for the measurement of arsenic following extraction of arsenic as $AsCl_3$ by benzene in strong hydrochloric acid. The recovery was close to 100% when 20 mg was spiked into an aqueous solution.

Vogel (1954) described the historic Marsh test, a qualitative method based on the generation of arsine (AsH_3) by the addition of Zn granules to sulfuric acid. If the gas is mixed with hydrogen, and conducted through a heated glass tube, it decomposes into hydrogen and metallic arsenic which is deposited as a brownish-black “mirror” just beyond the heated part of the tube.

2.6 Colorimetric methods

George & Roscoe (1951) reported a spectroscopic emission measurement of the blue complex formed by the reaction of ammonium molybdate and hydrazine sulfate with arsenic in various biological materials. The sensitivity was about 0.01 µg.

George et al. (1973) carried out a collaborative study for a colorimetric measurement of arsenic in poultry and swine tissues using silver diethyldithiocarbamate (AgDDTC) as the complexing agent. The sensitivity was 0.1 mg/kg in tissues. Dhar et al. (1997) reported a detection limit of 0.04 mg/litre with 95% confidence limit using AgDDTC in chloroform with hexamethylenetetramine.

Gutzeit's test (Vogel, 1954) is based on the generation of arsine from arsenic compounds by the addition of zinc granules to concentrated sulfuric acid. The arsine can be detected by means of a strip of filter paper moistened with silver nitrate or mercuric chloride. The arsine reacts with silver nitrate to give a grey spot, and with mercuric chloride to give a yellow to reddish-brown spot. The sensitivity is about 1 µg. A modification of this method, using mercuric bromide, is found in a test kit currently being used in Bangladesh for groundwater testing which has a limit of detection of 50–100 µg/litre under field conditions.

2.7 Methods for total inorganic arsenic

Methods for the analysis of inorganic arsenic based on its conversion to arsenic trichloride or arsenic tribromide by treatment with 6 mol/litre hydrochloric acid or hydrobromic acid have been described. The arsenic trihalide is separated from the remaining organic arsenic either by distillation (Maher, 1983) or by solvent extraction (Brooke & Evans, 1981). The methods have been applied routinely to the measurement of inorganic arsenic in a variety of foodstuffs, including those of marine origin where any inorganic arsenic is a small percentage of the total arsenic present (Flanjak, 1982; Shinagawa et al., 1983).

2.8 Atomic spectrometry

Common flame atomic absorption spectrometric methods are flame AAS (FAAS), electrothermal AAS (ETAAS) and hydride generation AAS (HGAAS). FAAS is relatively less sensitive for the determination of arsenic than ETAAS and HGAAS. Its detection limit is usually in the range of sub-milligram quantities per litre, and therefore it has limited application, especially for biological samples.

ETAAS, referred to also as graphite furnace-AAS (GFAAS), is generally one of the most sensitive atomic spectroscopic methods. Julshamn et al. (1996) reported factors that are known to interfere with the GFAAS determination of arsenic. The study was carried out by four participating laboratories using five marine standard reference materials. A mixture of palladium and magnesium salts has been recommended as a chemical modifier to avoid nickel contamination of the graphite furnace. The use of a pyrolytically coated graphite furnace tube with the L'vov platform improves sensitivity. Larsen (1991) achieved characteristic masses of about 16 pg of arsenic for arsenate, monomethylarsonate, DMA, arsenobetaine, arsenocholine and tetramethylarsonium ion calculated from aqueous standard solutions.

HGAAS is probably the most widely used method for the determination of arsenic in various matrices. Most of the reported errors in the determination of arsenic by HGAAS with $NaBH_4$ can be attributed to variation in the production of the hydride and its transport into the atomizer. The reaction and atomization of arsine have been reviewed and discussed by Welz et al. (1990). The addition of a solution of L-cysteine to a sample before hydride generation eliminates interference by a number of transition metals in the generation of arsine from arsenite and arsenate (Boampong et al., 1988), and improves responses of arsine generated from MMA and DMA in the presence of arsenite and arsenate (Le et al., 1994a).

Holak & Specchio (1991) described the determination of total arsenic, arsenite and arsenate in foods by HGAAS after a chloroform extraction procedure. The recovery was > 80%. Similar methods (Chappell et al., 1995; Ng et al., 1998a) have been developed for arsenic speciation in soils. Ybanez et al. (1992) described a HGAAS determination of arsenic in dry ashed mussel products and reported a detection limit of 0.017 µg As/g with a precision of 3%.

HGAAS has been used for arsenic speciation of inorganic arsenic and its urinary metabolites, MMA and DMA, since 1973, when Braman & Foreback (1973) introduced a cold-trapping step into a basic hydride generation system. Since then a number of improvements have been made to this method (Crecelius, 1978; Buchet & Lauwerys, 1981; Van Cleuvenbergen et al., 1988). Ng et al. (1998b) described an optimized procedure for the speciation of arsenic metabolites in the urine of occupationally exposed workers and experimental animals with detection limits of 1, 1.3 and 3 ng per reaction of inorganic arsenic, MMA and DMA (equivalent to 0.25 µg/litre, 0.325 µg/litre, and 0.75 µg/litre respectively), using 4 ml of urine per reaction.

HGAAS has also been widely employed for analysis of arsenic in water (Chen et al., 1994; Chatterjee et al., 1995; Mandal et al., 1996; Dhar et al., 1997; Biswas et al., 1998). Hasegawa et al. (1994) published the first report of trivalent methyl arsenicals, namely monomethylarsonous acid [MMA(III)] and dimethylarsinous acid [DMA(III)], being found and measured in natural waters. Arsenious acid, MMA(III) and DMA(III) were separated from the pentavalent species by solvent extraction using diethylammonium diethyldithio-carbamate (DDDC) and determined by HGAAS after cold trapping and chromatographic separations. The detection limits were 13B17 pmol/litre and 110–180 pmol/litre for the trivalent and pentavalent species respectively.

Atomic fluorescence spectrometry (AFS) has recently been used for the detection of arsenic hydrides in the ultraviolet spectral region because of the small background emission produced by the relatively cool hydrogen diffusion flame (Gomez-Ariza et al., 1998). The use of cold vapour or hydride generation, together with intense light sources, allows very low detection limits to be achieved. For example, arsenic species in seawater have been measured using hydride generation and cold trapping, coupled with AFS detection at 193.7 nm (Featherstone et al., 1998). They found detection limits of 2.3, 0.9, 2.4 and 3.7 ng/litre for arsenite, arsenate, MMA and DMA respectively (in a 5 ml sample), with a precision of 3.5%.

2.9 ICP methodologies

The main advantages of ICP-MS over ICP-AES are lower detection limits (sub-nanogram to sub-picogram) with wide linear range and isotope analysis capability of high precision. The detection limits of ICP-AES are typically in the range of sub-micrograms to sub-nanograms.

ICP-MS is more susceptible to isobaric interferences arising from the plasma. For example, hydrochloric acid and perchloric acid are not desirable for sample preparation, because the chloride ions generated in the plasma combine with the argon gas to form argon chloride (ArCl). This has the same mass as arsenic (75) which could lead to error if not corrected. Therefore, whenever possible, only nitric acid should be used in sample preparation. Careful sample preparation is as important as the final measurement, and special care should be taken to avoid contamination and losses by volatilization, adsorption and precipitation.

2.10 Voltammetry

Voltammetric stripping methods are mostly based on the chemical reduction of As(V) to As(III) before the deposition step, because it has been generally assumed that As(V) is electrochemically inactive. Mercury and gold (or gold-plated) electrodes are most commonly used for the determination of arsenic.

Sadana (1983) used differential pulse cathodic stripping voltammetry (DPCSV) coupled to a hanging mercury drop electrode (HMDE) to determine arsenic in drinking-water in the presence of Cu^{2+} and reported a detection limit of 1 ng/ml and a relative standard deviation of 6.4%. Zima & van den Berg (1994) reported a detection limit of 3 nmol/litre in seawater. DPCSV was employed by Higham & Tomkins (1993) to determine arsenic in canned tuna fish. They evaluated a number of digestion procedures and found the best procedure gave 93–96% recovery. No detection limit was reported.

A gold electrode affords better sensitivity than a mercury electrode. Hua et al. (1987) reported an automated determination of total arsenic in seawater by flow constant-current stripping analysis with a gold film fibre electrode, in which As(V) in the sample was

reduced to As(III) with potassium iodide; the detection limit was 0.15 µg/litre. The reduction of As(V) to As(III) can also be achieved by reaction with sulfur dioxide or hydrazinium chloride for use with a gold electrode or HMDE respectively (Esteban et al., 1994).

Huiliang et al. (1988) have shown that As(V) can be reduced to elemental arsenic provided that extremely low reduction potentials are used. They used this method to measure As(V) and total arsenic in seawater and urine. The detection limit was 0.1 µg/litre using constant-current stripping voltammetry on a gold-coated platinum-fibre electrode. Greulach & Henze (1995) developed a cathodic stripping voltammetric method for the determination of As(V) in water and stream sediment on the basis that As(V) can be reduced in perchloric acid solution containing D-mannitol, combined with the accumulation of arsenic by co-precipitation with copper on an HMDE. The detection limit was 4.4 µg/litre.

Pretty et al. (1993) developed an on-line anodic stripping voltammetry (ASV) flow cell coupled to ICP-MS for the determination of arsenic in spiked urine. The detection limit was 130 pg/ml and the recovery was 94–113%.

2.11 Radiochemical methods

Orvini et al. (1974) reported a combustion technique for sample preparation and determination of arsenic, selenium, zinc, cadmium and mercury by neutron activation analysis (NAA) in environmental matrices including a range of standard reference materials. The recoveries were 98–100%. Sharif et al. (1993) described a NAA technique for the determination of arsenic in eight species of marine fishes caught in the bay of Bengal, Bangladesh.

Haddad & Zikovsky (1985) measured several elements including arsenic in air from workroom welding fumes by NAA and reported a detection limit of 0.17 ± 0.07 µg/m^3. Landsberger & Wu (1995) reported the use of NAA to measure arsenic from environmental tobacco smoke in indoor air with a detection limit of 0.2 ng.

Chutke et al. (1994) described a radiochemical solvent extraction procedure for the determination of arsenite using an arsenic-76 tracer. The procedure is based on the complexation of arsenite with

toluene-3,4-dithiol (TDT) at pH 2 and subsequent extraction in benzene. This isotopic dilution technique was employed to measure arsenic in a range of standard and certified reference materials. The detection limit was 250 ng with an accuracy of about 4% error and 170 ng with about 12% error.

2.12 X-ray spectroscopy

Particle-induced X-ray emission spectrometry (PIXES) is an analytical technique that entails the bombardment of a sample (target) with charged particles, resulting in the emission of characteristic X-rays of the elements present. PIXES is a multi-elemental technique with a detection limit of approximately 0.1 µg As/g. It has the advantage of using small samples (1 mg or less) and being a non-destructive technique. Applications of PIXES in the environmental field have mostly focused on atmospheric particulate material (aerosol samples) (Maenhaut, 1987).

Castilla et al. (1993) described the determination of arsenite and arsenate by X-ray fluorescence (XRF) spectroscopy in water with a detection limit of 3.1 ng/g. The recovery was 97 ± 2.1% and 103 ± 1.4% for arsenite and arsenate respectively. In this method, the water sample was acidified to pH 2 and arsenite co-precipitated with sodium dibenzyldithiocarbamate (DBDTC). Arsenate in the filtrate was then reduced to arsenite with potassium iodide before the co-precipitation step for the XRF measurement.

Although there are a variety of methods to determine the concentration and oxidation states of arsenic in coal and ash, there have been few attempts to determine the mineral forms of arsenic. Huffman et al. (1994) described the use of X-ray absorption fine structure (XAFS) spectroscopy and its capability of providing speciation information at realistic concentrations of 10–100 mg/kg. They identified arsenic present as arsenopyrite in one coal sample and as aluminosilicate slag and calcium orthoarsenate in combustion ashes.

2.13 Hyphenated techniques

Hyphenated techniques is a term referring to the coupling of more than two instrumental systems to form a single technique.

The combination of chromatographic separation with element-specific spectrometric detection has been proved to be particularly useful for the speciation of arsenic compounds at trace levels in environmental samples. Woller et al. (1995) used AFS detection in combination with ultrasonically nebulized liquid chromatography (LC) for on-line speciation of arsenic, but found that the technique had limited sensitivity owing to matrix interferences. More recently, Slejkovec et al. (1998) used LC and purge-and-trap GC interfaced with AFS to separate and quantify six arsenic species with detection limits of 0.5 ng/ml As (100 µl). Gomez-Ariza et al. (1998) coupled anion-exchange HPLC, hydride generation and AFS to achieve detection limits of 0.17, 0.45, 0.30 and 0.38 µg/litre for arsenite, DMA, MMA and arsenate respectively (using a 20 µl loop). Arsenobetaine was also determined by introducing an on-line photo-oxidation step after the chromatographic separation.

Ebdon et al. (1988) described a number of coupled chromatograph–atomic spectrometry methods for arsenic speciation including GC or HPLC with detection by atomic spectrometry, namely FAAS, flame atomic fluorescence spectrometry (FAFS) and ICP-AES. The FAAS system is capable of detection at less than 1 µg/kg (0.22–0.55 ng absolute for different species) when levels permit; HPLC–hydride generation–FAAS is probably the simplest routine method and HPLC–hydride generation–ICP-AES is preferred for multi-elemental analysis. HPLC–ICP-AES has been employed for the speciation of organic arsenic of aquatic origin (Francesconi et al., 1985; Gailer & Irgolic, 1996). Gjerde et al. (1993) described the coupling of microbore columns with direct-injection nebulization to ICP-AES and reported a detection limit of 10 µg/litre (100 pg). Microbore HPLC has the advantage of analysing small sample size using low flow rates (80–100 µl/min) of mobile phases.

Numerous methods (Shum et al., 1992; Larsen et al., 1993; Magnuson et al., 1996; Thomas et al., 1997; Le & Ma, 1997) have been developed for the speciation of arsenic using the separation power of chromatography coupled to the sensitivity of ICP-MS detection. Heitkemper et al. (1989) described an anion-exchange HPLC–ICP-MS method for the speciation of arsenite, arsenate, MMA and DMA in urine with absolute detection limits ranging from 36 to 96 pg (corresponding to 0.7–1.9 µg/litre in a 50 µl injection). Beauchemin et al. (1989) reported detection limits for arsenic species

in DORM-1 (a dogfish muscle certified reference material) ranging between 50 and 300 pg using ion pairing and ion exchange HPLC-ICP-MS. Anion exchange is more tolerant because of the higher buffering capacity of the mobile phase. Cation pairing is more suitable for the determination of DMAA and arsenobetaine in biological samples containing high concentrations of salts. Pergantis et al. (1997) analysed and speciated animal feed additives using microbore HPLC–ICP-MS with detection limits ranging from 0.1 to 0.26 pg. Hakala and Pyy (1992) described an ion-pairing HPLC-HGAAS method for speciation of arsenite, arsenate, MMA and DMA in urine with detection limits of 1.0, 1.6, 1.2 and 4.7 µg/litre respectively.

Ding et al. (1995) described the coupling of micellar liquid chromatography (MLC) and ICP-MS for the speciation of arsenite, arsenate, MMA and DMA with detection limits of 90 pg for DMA and 300 pg for the other species. MLC is a type of chromatography that uses surfactants in aqueous solutions, well above their critical micelle concentration, as alternative mobile phases for reversed-phase liquid chromatography (RPLC). MLC extends the analyte candidates to almost all hydrophobic and many hydrophilic compounds providing they can partition to the micelles. Other advantages of MLC over RPLC include simultaneous separation of both ionic and non-ionic compounds, faster analysis times and improved detection sensitivity and selectivity.

Capillary electrophoresis (CE) is a versatile technique for the separation of a variety of analytes ranging from small inorganic ions to large biomolecules such as proteins and nucleic acids. CE-ICP-MS has been described for the speciation of arsenic by Liu et al. (1995) with detection limits of 100 pg arsenite/ml and 20 pg arsenate/ml and Olesik et al. (1995) with a detection limit of 8 µg/litre (1 pg injection).

Although techniques such as HPLC–ICP-MS and MLC–ICP-MS offer the advantages of high sensitivity and selectivity as well as low detection limits, species identification is based on the comparison of chromatographic retention times to those of available standards. When structure information is required, as well as quantification, electrospray HPLC–MS (Siu et al., 1991) and ionspray MS (Corr, 1997) should be considered. Corr & Larsen (1996) reported the use

of LC–MS–MS for speciation of arsenic with a detection limit of 2 pg for the tetramethylarsonium cation.

3. SOURCES AND OCCURRENCE OF ARSENIC IN THE ENVIRONMENT

3.1 Natural sources

Arsenic is the main constituent of more than 200 mineral species, of which about 60% are arsenate, 20% sulfide and sulfosalts and the remaining 20% include arsenides, arsenites, oxides and elemental arsenic (Onishi, 1969). The most common of the arsenic minerals is arsenopyrite, FeAsS, and arsenic is found associated with many types of mineral deposits, especially those including sulfide mineralization (Boyle & Jonasson, 1973). The ability of arsenic to bind to sulfur ligands means that it tends to be found associated with sulfide-bearing mineral deposits, either as separate As minerals or as a trace of a minor constituent of the other sulfide minerals. This leads to elevated levels in soils in many mineralized areas where the concentrations of associated arsenic can range from a few milligrams to > 100 mg/kg.

Concentrations of various types of igneous rocks range from < 1 to 15 mg As/kg, with a mean value of 2 mg As/kg. Similar concentrations (< 1–20 mg As/kg) are found in sandstone and limestone. Significantly higher concentrations of up to 900 mg As/kg are found in argillaceous sedimentary rocks including shales, mudstone and slates. Up to 200 mg As/kg can be present in phosphate rocks (O'Neill, 1990).

Concentrations of arsenic in open ocean water are typically 1B2 µg/litre. The concentrations of arsenic in unpolluted surface water and groundwater are typically in the range of 1–10 µg/litre. Elevated concentrations in surface water and groundwater of up to 100–5000 µg/litre can be found in areas of sulfide mineralization (Welch et al., 1988; Fordyce et al., 1995). Elevated concentrations (> 1 mg As/litre) in groundwater of geochemical origins have also been found in Taiwan (Chen et al., 1994), West Bengal, India (Chatterjee et al., 1995; Das et al., 1995, 1996; Mandal et al., 1996) and more recently in most districts of Bangladesh (Dhar et al., 1997; Biswas et al., 1998). Elevated arsenic concentrations were also found in the drinking-water in Chile (Borgono et al., 1977); North Mexico

(Cebrian et al., 1983); and several areas of Argentina (Astolfi et al., 1981; Nicolli et al., 1989; De Sastre et al., 1992). Arsenic-contaminated groundwater was also found in parts of PR China (Xinjiang and Inner Mongolia) and the USA (California, Utah, Nevada, Washington and Alaska) (Valentine, 1994). More recently, arsenic concentrations of < 0.98 mg/litre have been found in wells in south-western Finland (Kurttio et al., 1998). Levels as high as 35 mg As/litre and 25.7 mg As/litre have been reported in areas associated with hydrothermal activity (Kipling, 1977; Tanaka, 1990).

In nature, arsenic-bearing minerals undergo oxidation and release arsenic to water. This could be one explanation for the problems of arsenic in the groundwater of West Bengal and Bangladesh. In these areas the groundwater usage is very high. It has been estimated that there are about 4–10 million tube wells in Bangladesh alone. The excessive withdrawal and lowering of the water table for rice irrigation and other requirements lead to the exposure and subsequent oxidation of arsenic-containing pyrite in the sediment. As the water table recharges after rainfall, arsenic leaches out of the sediment into the aquifer.

However, recent studies seem to favour the reduction of Fe/As oxyhydroxides as the source for arsenic contamination in groundwater (Nickson et al., 1998; BGS, 2000; BGS & DPHE, 2001). Arsenic forms co-precipitates with ferric oxyhydroxide. Burial of the sediment, rich in ferric oxyhydroxide and organic matter, has led to the strongly reducing groundwater conditions. The process has been aided by the high water table and fine-grained surface layers which impede the penetration of air to the aquifer. Microbial oxidation of organic carbon has depleted the dissolved oxygen in the groundwater. The highly reducing nature of the groundwater explains the presence of arsenite (< 50%) in the water. The “pyrite oxidation” hypothesis is therefore unlikely to be a major process, and the “oxyhydroxide reduction” hypothesis (Nickson et al., 1998; Acharyya et al., 1999) is probably the main cause of arsenic contamination in groundwater. Although the oxyhydroxide reduction hypothesis requires further validation, there is no doubt that the source of arsenic in West Bengal and Bangladesh is geological, as none of the explanations for anthropogenic contamination can account for the regional extent of groundwater contamination. During the past 30 years the use of phosphate

fertilizers has increased threefold in this region. The widespread withdrawal of groundwater may have mobilized phosphate derived from fertilizers and from the decay of natural organic materials in shallow aquifers. The increase in phosphate concentration could have promoted the growth of sediment biota and the desorption of arsenic from sediments, and the combined microbiological and chemical process might have increased the mobility of arsenic (Acharyya et al., 1999).

Marine organisms naturally accumulate considerable quantities of organic arsenic compounds. In marine animals the bulk of this arsenic is present as arsenobetaine, whereas marine algae contain most of the arsenic as dimethylarsinoylribosides. Humans are therefore exposed to these arsenic compounds through any diet that includes seafoods. This subject is fully discussed in Chapter 4.

Some arsenic compounds are relatively volatile and consequently contribute significant fluxes in the atmosphere. It has been estimated that the atmospheric flux of As is about 73 540 tonnes/year of which 60% is of natural origin and the rest is derived from anthropogenic sources (Chilvers & Peterson, 1987). Volcanic action is the next most important natural source of arsenic after low-temperature volatilization, and on a local scale it will be the dominant atmospheric source.

3.2 Sources of environmental pollution

3.2.1 Industry

It has long been recognized that the smelting of non-ferrous metals and the production of energy from fossil fuel are the two major industrial processes that lead to anthropogenic arsenic contamination of air, water and soil. Other sources of contamination are the manufacture and use of arsenical pesticides and wood preservatives.

Smelting activities generate the largest single anthropogenic input into the atmosphere (Chilvers & Peterson, 1987).

Tailings from metal-mining operations are a significant source of contamination, and can lead to contamination of the surrounding

topsoils, and, because of leaching, sometimes the groundwater too. It has been estimated that several billion tons of tailings waste exist in the USA alone (Wewerka et al., 1978). As sulfur is often present in these tailings, exposure to the atmosphere in the presence of water leads to the production of an acid solution that can leach many elements including arsenic.

Elevated concentrations of arsenic in acid sulfate soils in Canada and New Zealand are associated with pyrite (Dudas, 1987). Concentrations of arsenic < 0.5% through lattice substitution of sulfur in this pyrite iron-rich bauxite have been recorded.

In the United Kingdom, the estimated arsenic releases (Hutton & Symon, 1986) were 650 tonnes/year from the non-ferrous metal industry, 9 tonnes/year emission into the atmosphere and 179 tonnes/year to landfill from iron and steel production, and 297 tonnes/year into the atmosphere and 838 tonnes/year to landfill from fossil fuel combustion. In 1996, the estimated total releases of arsenic to the air in the UK were 50 tonnes (DG Environment, 2000).

The working group of the European Union DGV (the directorate with responsibility for the environment) concluded that there were large reductions in the emissions of arsenic to air in several member countries of the European Union in the 1980s and early 1990s. In 1990, the total emissions of arsenic to the air in the member states were estimated to be 575 tonnes, of which 492 tonnes came from stationary combustion (mainly coal and oil combustion) and 77 tonnes from production processes, mainly from the iron and steel industry (35 tonnes) and the non-ferrous metal industry (31 tonnes) (DG Environment, 2000).

Arsenic is present in the rock phosphate used to manufacture fertilizers and detergents. In 1982, the United Kingdom imported 1324×10^3 tonnes of rock phosphate with an estimated arsenic burden of 10.2 tonnes (Hutton & Symon, 1986).

3.2.2 Past agricultural use

In 1983, arsenical pesticides were one of the largest classes of biocontrol agent in the USA (Woolson, 1983). From the 1960s there was a shift, in herbicide use, from inorganic compounds (including lead and calcium arsenate and copper acetoarsenite) to inorganic and

organic compounds (arsenic acid, sodium arsenate, MMA and DMA). Use of total arsenical pesticides, excluding wood preservatives, at the time of publication (1983) was estimated at $7–11 \times 10^3$ tonnes As/year. Annual historical applications of lead arsenate to orchards in the USA ranged from 32 to 700 kg As/ha. Residues in orchard soils as high as 2500 mg/kg have been reported, but they are more commonly in the range of 100–200 mg/kg. In Australia between 1900 and 1950 As_2O_3 was widely used for controlling cattle ticks (*Boophilus microplus*), resulting in widespread arsenic contamination (Seddon, 1951).

3.2.3 Sewage sludge

The levels of arsenic in sewage sludge reflect the extent of industrialization of the area served by the local sewage system. Significant quantities may be added by arsenic-contaminated wastewater runoff derived from sources including atmospherically deposited arsenic, residues from pesticide usage, phosphate detergents and industrial effluent, particularly from the metal-processing industry. Levels of 0–188 mg As/kg dry weight have been reported in the United Kingdom (Woolson, 1983). Zhu & Tabatabai (1995) reported levels of 2.4–39.6 mg As/kg with a mean of 9.8 for sewage sludges from waste treatment plants in Iowa, USA.

O'Neil (1990) estimated that in the UK as a whole about 2.5 tonnes As/year is added to the agricultural land by use of sludge, compared to 6.1 tonnes As/year when phosphate fertilizer is used.

3.3 Uses

Arsenic is produced commercially by reduction of As_2O_3 with charcoal. As_2O_3 is produced as a by-product of metal-smelting operations. It is present in flue dust from the roasting of ores, especially those produced in copper smelting. In the 1960s, the pattern of use for As_2O_3 in the USA is believed to have been 77% as pesticides, 18% as glass, 4% as industrial chemicals and 1% as medicine. However, the pattern has changed over the years as the use of arsenic compounds for timber treatment has been increasingly popular since the late 1980s. Worldwide usage in the early 1980s was estimated to be 16 000 tonnes As/year as a herbicide, 12 000 tonnes As/year as a cotton desiccant/defoliant and

16 000 tonnes As/year in wood preservative (Chilvers & Peterson, 1987). By 1990, the estimated end-use of arsenic in the USA was 70% in wood preservatives, 22% in agricultural chemicals, 4% in glass, 2% in non-ferrous alloys and 2% in other uses including semiconductors (US DOI, 1991). Arsenic pentoxide and As_2O_3 are used as additives in alloys, particularly with lead and copper; arsenic and As_2O_3 are used in the manufacturing of low-melting glasses. High-purity arsenic metal and gallium arsenide are used in semiconductor products. Fowler's solution (1% potassium arsenite solution) was used as a medication (Cuzick et al., 1992). As_2O_3 has been used for the treatment of acute promyelocytic leukaemia (Soignet et al., 1998).

Hutton & Symon (1986) reported that about 5000 tonnes/year As_2O_3 is imported to the United Kingdom for conversion to other arsenic compounds. These processes result in an estimated discharge of 87 tonnes As/year in manufacturing sludges on landfilled sites. Currently about 500 tonnes As/year is utilized in copper chrome arsenate (CCA) timber treatment, of which at most 5 tonnes/year is retained in sludges. Small amounts of arsenic are used in the production of glass, and most of the remainder is re-exported.

4. ENVIRONMENTAL TRANSPORT AND DISTRIBUTION

4.1 Transport and distribution between media

4.1.1 Air

Arsenic is primarily emitted into the atmosphere by high-temperature processes such as coal-fired power generation, smelting, burning vegetation and vulcanism. Natural low-temperature biomethylation and microbial reduction also release arsenic into the atmosphere; microorganisms can form volatile methylated derivatives of arsenic under both aerobic and anaerobic conditions, and can reduce arsenic compounds to release arsine gas (Cheng & Focht, 1979; Tamaki & Frankenberger, 1992) (see section 4.2.2). Arsenic is released into the atmosphere primarily as As_2O_3 or, less frequently, as one of several volatile organic compounds. Arsenic released to air exists mainly in the form of particulate matter (Coles et al., 1979). These particles are dispersed by the wind to a varying extent, depending on their size, and the particles are returned to the earth by wet or dry deposition. Arsines that are released from microbial sources in soils or sediments undergo oxidation in the air, reconverting the arsenic to less volatile forms that settle back to the ground (Wood, 1974; Parris & Brinckman, 1976).

Pacyna et al. (1989) studied atmospheric transport of arsenic from various sources in Europe to selected receptor sites in Norway. By modelling long-range transport they were able to calculate a dry deposition velocity for arsenic of 0.4 cm/second. Scudlark & Church (1988) measured arsenic in acid precipitation on the mid-Atlantic coast of the USA during 1985 and 1986. They calculated the total annual arsenic deposition rate to range from 38 to 266 µg/m^2, with dry deposition estimated to comprise 29–55% of the total. Davidson et al. (1985) calculated the annual dry deposition flux of arsenic to the Olympic National Park, Washington (USA) to range from 76.7 to 208 µg/m^2. The average annual wet deposition of arsenic at Chesapeake bay (Maryland, USA) was found to be 49 µg As/m^2 (Scudlark et al., 1994).

Total atmospheric arsenic emissions from both natural and anthropogenic sources have been estimated to be 31×10^9 g/year, and total atmospheric arsenic removal was estimated to be 30B50 $\times 10^9$ g/year. The global tropospheric residence time of arsenic appears to be about 9 days (Walsh et al., 1979). Nakamura et al. (1990) estimated global atmospheric emissions into the atmosphere and deposition of arsenic. Total emissions were estimated at 36×10^9 g/year, with the major source of atmospheric arsenic being anthropogenic emissions; the major natural source of arsenic was volcanic activity. Emissions from anthropogenic sources were estimated at 24×10^9 g/year, representing 64% of total arsenic influxes. Depositions from the atmosphere to the land and the oceans were estimated at 24×10^9 g/year and 9×10^9 g/year respectively. Akeredolu et al. (1994) calculated the total annual transport of arsenic into the Arctic atmosphere at 285 t (285×10^6 g) on the basis of a chemical transport modelling approach previously used for sulfur.

Arsenic in the atmosphere exists primarily adsorbed to particulate matter and mostly to particles < 2 µm in diameter (Coles et al., 1979). Waslenchuk (1978) found that atmospheric arsenate at the continental shelf of the south-eastern USA is associated exclusively with the particulate fraction. Rabano et al. (1989) collected size-fractionated aerosol samples at an urban site during 1987. A greater proportion (75%) of the arsenic was observed in the fine particles (< 2.5 µm). The As(III)/As(V) ratio for both fine and coarse (> 2.5 µm) particles was approximately 1. Similarly, Waldman et al. (1991) reported that 65% of the arsenic in aerosol samples collected at an urban site (China) was associated with fine particles (< 2.5 µm). Kelley et al. (1995) monitored arsenic in aerosol collected from the Kola Peninsula (Russia). They found 68% of arsenic associated with fine particles (< 1 µm), 26% with coarse particles (1–10 µm) and 7% with large particles (> 10 µm). The atmospheric residence time of particulate-bound arsenic depends on particle size and meteorological conditions, but a typical value is about 9 days (US EPA, 1982).

4.1.2 Freshwater and sediment

The dissolved forms of arsenic in the water column include arsenate, arsenite, monomethylarsonic acid (MMA) and

dimethylarsinic acid (DMA) (Braman & Foreback, 1973). Some As(III) and As(V) species can interchange oxidation states depending on Eh, pH and biological processes (Ferguson & Gavis, 1972). Some arsenic species have an affinity for clay mineral surfaces and organic matter, and this can affect their environmental behaviour. Methylation and demethylation reactions are also important transformations controlling the mobilization and subsequent distribution of arsenicals (Mok & Wai, 1994). Transport and partitioning of arsenic in water depends on the chemical form of the arsenic and on interactions with other materials present. Arsenic may be adsorbed from water on to clays, iron oxides, aluminium hydroxides, manganese compounds and organic material (Callahan et al., 1979; Welch et al., 1988). The distribution and transport of arsenic in sediment is a complex process that depends on water quality, native biota and sediment type. There is a potential for arsenic release when there is fluctuation in Eh, pH, soluble arsenic concentration and sediment organic content (Abdelghani et al., 1981).

Ferguson & Gavis (1972) proposed an arsenic cycle for a stratified lake. In the aerobic epilimnetic water, reduced forms of arsenic tend to be oxidized to arsenate, which co-precipitates with ferric oxyhydroxide. Turbulent dispersion and convection transports some of the arsenate across the thermocline to the oxygen-depleted hypolimnion, where reduction to $HAsO_2$ and AsS_2^- takes place, depending on the sulfur concentration and the Eh. Co-precipitation, adsorption and epitaxial crystal growth cause arsenic to be removed to the sediments, where reduction of ferric iron, arsenate and arsenite result in either solubilization or stabilization as an insoluble sulfide or arsenic metal. Microbial reduction and methylation to arsine solubilize the arsenic (see section 4.2), and diffusion through the sediments or mixing by currents or burrowing organisms (see section 4.1.4) cause arsenic to re-enter the water column.

Aurilio et al. (1994) studied the speciation and fate of arsenic in three lakes of the Aberjona watershed (Massachusetts, USA). Speciation appeared to be controlled by reduction, methylation, and oxidation processes, and by adsorption to and desorption from particles. Biologically mediated reduction, at rates of 0.2–0.5% total arsenic/day, and methylation, at rates of 0.4–0.6% total arsenic/day, occurred in the mixed layers of these lakes. These processes are slow

or even absent in the hypolimnion, however, allowing arsenate to accumulate in seasonally anoxic hypolymnetic waters. High micromolar concentrations of arsenic, predominantly arsenite, persisted in the saline, sulfidic monimolimnion of one lake.

Clement & Faust (1981) studied the release of arsenic from contaminated sediments. Anaerobic conditions led to aqueous levels of arsenic, principally as arsenite, about 10 times higher than concentrations reached with aerobic conditions. Under aerobic conditions arsenic in the overlying water comprised 70% arsenate and 20% organic arsenic. The authors found that adsorption–desorption equilibria and the amount of 'available' arsenic present in the sediment greatly influenced the soluble arsenic concentration found in the aqueous phase. In sediment under oxidized conditions arsenic solubility was low and 87% of the arsenic in solution was present as arsenate. On reduction, arsenite became the major arsenic species in solution and solubility increased (Masscheleyn et al., 1991b). Ahmann et al. (1997) identified rapid arsenic mobilization from aquatic sediments in upper Aberjona (Massachusetts, USA) sediment microcosms. The findings suggest that arsenic reduction by microorganisms may contribute to arsenic flux from anoxic sediments in this arsenic-contaminated watershed.

The predominant arsenic species in the water column of lakes is arsenate, as expected in oxidizing environments (Seyler & Martin, 1989). Arsenite is usually present and sometimes dominates in bottom water which contains high concentrations of Fe(II) and low oxygen. Peterson & Carpenter (1983) reported that the arsenate : arsenite concentration ratio was 15 : 1 in the oxic region of the water column and 1 : 12 in the anoxic zone. Seasonal trends reveal higher concentrations of arsenic in summer than in winter. The source of arsenic in the summer is most likely surface sediments that have become anoxic causing a release into the water column of arsenic adsorbed on iron and manganese oxides (Singh et al., 1988; Crecelius et al., 1994).

Pettine et al. (1992) found that arsenate was the predominant arsenic species in the river Po (Italy). The main factors affecting dissolved concentrations included flow and suspended matter concentration and biological activity. The ratio between oxidized and reduced species appears to be significantly influenced by iron and

manganese oxides. Abdel-Moati (1990) monitored arsenic in the Nile delta lakes and found arsenate to be the dominant arsenic species (85–95%). Increased arsenite (14–33%) was found near local sewage discharge points. Dimethylarsenic was the dominant organic species, reaching 22% of the total dissolved arsenic.

A temporal study of arsenic speciation in Davis Creek Reservoir, a seasonally anoxic lake in northern California (USA), demonstrated that dimethylarsinic acid increased sufficiently to become the dominant form of dissolved arsenic within the surface photic zone during late summer and early autumn. Methylated forms decreased and arsenate increased when the lake 'turned over' in early December, suggesting a degradation of dimethylarsinic acid (Anderson & Bruland, 1991).

Aggett & O'Brien (1985) report that Lake Ohakuri (New Zealand) becomes stratified during the summer. During this period arsenic released from the sediment accumulates in the hypolimnion until turnover when it is mixed with epilimnetic water. It is estimated that this turnover effect causes a temporary increase in arsenic concentrations of 10–20%. Aggett & Roberts (1986) conclude that arsenate and phosphate are incorporated into Lake Ohakuri sediments by co-precipitation at the time of formation of the hydrous oxides rather than by adsorption on existing surfaces. Aggett & Kriegman (1988) show that in sediment cores from Lake Ohakuri over 90% of arsenic in interstitial waters was present as arsenite, an indication that reduction from arsenate, the predominant form adsorbed from the lake water, was taking place. When conditions at the sediment–water interface became anoxic, arsenite diffused across the interface into the hypolimnion.

Johnson & Thornton (1987) studied the seasonal variation of arsenic in the Carnon river, south-west England (UK). Approximately 85% of the arsenic was found to originate from mine waters. Arsenic is found to a large extent (~80%) in the particulate phase; the authors suggest that sorptive or co-precipitation processes are responsible for the regulation of dissolved concentrations of arsenic in these waters. These processes are largely independent of pH. Adsorption appears to be important in the removal of arsenic from solution, with 80% being removed on entering estuarine waters.

Both adsorption of arsenic on iron-rich oxides on the surface of the sediments and incorporation of arsenic into the sediments by co-precipitation with hydrous iron oxides are factors controlling mobilization of sediment arsenic. The major arsenic species leached was arsenate; release of arsenate was found to be pH dependent and related to the total iron and free iron oxides in the sediments (Mok & Wai, 1989). Arsenate and arsenite differ in adsorption characteristics, and this influences their mobilization and subsequent distribution during water–sediment interactions. The extent of adsorption and remobilization varies with the oxidation state of arsenic, the Eh and the pH of the water. The increase in mobility of arsenate under more reducing conditions is generally attributed to the reduction of Fe^{3+} to Fe(II), with subsequent release of arsenate, and reduction of arsenate to arsenite (Mok & Wai, 1994).

Brannon & Patrick (1987) found that arsenate added to sediment became associated with relatively immobile iron and aluminium compounds. Addition of arsenate to sediments before anaerobic incubation also resulted in accumulation of arsenite and organic arsenic in the interstitial water and exchangeable phases of anaerobic sediments. Seyler & Martin (1989) report that the presence of arsenic in the anoxic zone of a permanently stratified lake was due to adsorption on to iron and manganese.

Sorption of arsenate, MMA and DMA on anaerobic bottom sediments from the Menominee river, Wisconsin (USA) is described by Langmuir isotherms (Holm et al., 1979). Singh et al. (1988) found that the adsorption of arsenite from aqueous solution followed first-order adsorption expression obeying Langmuir's model of adsorption. Similar findings were reported by Yadava et al. (1988) for adsorption of arsenite by china clay, with maximum adsorption at pH 8. Sediment adsorption of arsenate, monosodium methane-arsonate (MSMA) and DMA was positively correlated with clay content (Wauchope & McDowell, 1984). Organic sediments adsorbed arsenic more strongly than sandy sediments (Faust et al., 1987b).

The extent of uptake and the rate of adsorption of arsenate decrease with an increase in temperature from 20 °C to 40 °C. The amount of arsenate adsorbed increases as the pH of the system increases and reaches its maximum at pH 4.2 for haematite and

pH 6.2 for feldspar. The removal of arsenate from aqueous solution by adsorption on to geological materials such as haematite and feldspar follows first-order kinetics, and intraparticle diffusion seems to control the mass transfer (Prasad, 1994). The adsorption of arsenate on alumina, haematite , kaolin and quartz was influenced by the charge of the solid surface and the arsenic speciation in solution as determined by pH (Xu et al., 1988).

Adsorption of arsenate by fly ash was significantly greater at pH 4 than at pH 7 or 10 and was found to be almost irreversible. Adsorption fitted both the Freundlich and Langmuir adsorption models (Sen & De, 1987; Diamadopoulos et al., 1993). The partitioning of arsenic between acidic fly ash and leachate is controlled by sorption on iron oxyhydroxide. The leaching of arsenic is mainly controlled by sorption on hydroxylamine-extractable ("amorphous") iron oxyhydroxide; crystalline iron oxides appear to have little influence on the process (Van der Hoek & Comans, 1996). Thanabalasingam & Pickering (1986) found that arsenic sorption by humic acid varies with pH, adsorbate concentration and ash content of the substrate. At fixed pH, the amount of arsenic sorbed conformed to a Langmuir relationship, with calculated capacities in the region of maximum uptake (~pH 5.5) being of the order of 5250B6750 mg/kg for arsenite and 6750–8250 mg/kg for arsenate.

Gupta & Chen (1978) report that arsenic acid and arsenious acid species are effectively adsorbed in the pH range 4–7. Laboratory adsorption experiments indicated that arsenite is less effectively removed than arsenate. Adsorption of MMA and DMA on ferric oxyhydroxide and activated alumina decreased with increasing pH (4–11) (Cox & Ghosh, 1994).

Arsenic in porewater is controlled by the solubility of iron and manganese oxyhydroxides in the oxidized zone and metal sulfides in the reduced zone. Diagenetic sulfides are important sinks for arsenic in reduced, sulfidic sediments. During reduction, oxyhydroxides of iron and manganese dissolve, arsenic sulfides precipitate and arsenic is released to groundwater dominantly as arsenite (Moore et al., 1988). Therefore, mobilization of arsenic is more likely to occur in sediments low in iron and manganese oxyhydroxides, and calcium carbonate (Brannon & Patrick, 1987; Mok & Wai, 1990).

Bright et al. (1994) found that arsenite was the predominant arsenical in sediment porewater throughout a watershed receiving gold-mine effluent; dissolved arsenic in water column samples was mostly arsenate. Arsenic distribution in surficial sediments was controlled partially by the bulk movement of sediments, followed by burial with less-contaminated sediments in the upper reaches of the watershed. Particulate concentrations of arsenic contributed significantly (< 70%) to the total arsenic concentrations in the water column downstream of the gold-mine discharge. Azcue et al. (1994b) found that 66–83% of the arsenic in sediment porewater from a mine-polluted lake was arsenite. The concentration gradient of total dissolved arsenic indicated an upward diffusion of arsenic towards the water column, with the estimated annual fluxes being 0.8–3.8 μg/cm^2.

Mok & Wai (1990) reported that acid precipitation caused increased release of both arsenate and arsenite from contaminated sediments (pH 2.7). Arsenic release decreased with increasing pH; lowest levels of release were found at pH 8.3 for arsenite and pH 6.3 for arsenate. Release of arsenic increased at more alkaline pH values. Xu et al. (1991) concluded that environmental acidification would increase the leaching of arsenic from sediments to surface waters under reducing conditions as a result of the release of arsenite from iron oxyhydroxide phases, but could also reduce the mobility because of enhanced adsorption under oxidizing conditions. However, a large reduction in pH (to ≤4) would enhance the mobility of arsenic even under oxidizing conditions.

4.1.3 Estuarine and marine water and sediment

An arsenic cycle has also been outlined for the estuarine environment. Sanders (1980) found that the major inputs to the marine environment were river runoff and atmospheric deposition. Biological uptake caused changes in arsenic speciation resulting in measurable concentrations of reduced and methylated arsenic species. The overall cycle is similar to the phosphate cycle, but the regeneration time for arsenic is much slower. Arsenic flows into the estuary as arsenate and arsenite from river water and mine adits. There is oxidation of arsenite to arsenate, microbiological reduction of arsenate to arsenite and removal of arsenic by dilution with seawater and subsequent transport out of the estuary. Inorganic

arsenic can be adsorbed on to charged particles of iron oxyhydroxides and manganese oxides and deposited as flocculated particles to sediment. There is subsequent release of dissolved arsenite and arsenate following the reduction and dissolution of the iron and manganese carrier phases in the anoxic sediments. Arsenate can be reduced, either microbially or chemically, to arsenite within the anoxic sediment, and arsenic (as arsenate or arsenite) can enter by sediment resuspension (Sanders, 1980; Knox et al., 1984). Studies on the pH dependence of arsenate and arsenite adsorption to soils and sediments and to minerals are not consistent. For example, greater adsorption of arsenate to fly ash occurred at pH4 than at pHs 7 and 10 (Sen & De, 1987; Diamadopoulos et al., 1993), whereas Mok & Wai (1990) reported that absorption increased as pH increased for sediments.

Arsenic entering unpolluted estuaries associated with particulates remains adsorbed, and accumulates in sediment. Remobilization has only a small effect (< 7%) on the dissolved arsenic concentration in the water column. Dissolved arsenic species form complexes with low-molecular-weight dissolved organic matter, and these tend to prevent adsorption and co-precipitation interactions between arsenic and flocculating iron oxyhydroxides and humics (Waslenchuk & Windom, 1978). Langston (1983) reports that more than 80% of arsenic entering Restronguet creek in southwest England (United Kingdom) was retained by the sediment, which consequently acts as a sink for riverine inputs and limits transport of dissolved species to coastal waters. Iron oxyhydroxide scavenging seems to be a predominant factor in the removal of arsenic from the Scheldt estuary (The Netherlands) (van der Sloot et al., 1985). Millward et al. (1997) estimated an annual arsenic budget for the Thames plume (United Kingdom) and found that cycling of arsenic by phytoplankton was the dominant process. Inorganic arsenic was removed from the water column by phytoplankton and recycled during phytoplankton degradation and consumption.

An arsenic budget for Puget sound (Washington, USA) revealed that sediments accumulate less than 30% of the arsenic entering the sound (Crecelius et al., 1975). Carpenter et al. (1978) found that sedimentation processes including adsorption–desorption reactions with natural Puget sound suspended matter remove less than 15% of the dissolved arsenic input, with iron oxyhydroxides dominating

what removal does occur. Most of the arsenic entering the sound is removed by advection of surface waters out into the strait of Juan de Fuca . A similar budget for Lake Washington (USA) showed equal inputs of arsenic from the atmosphere and from rivers, and subsequent removal by outflowing water (45%) and by accumulation in the sediments (55%) (Crecelius, 1975).

Riedel (1993) studied the distribution of dissolved and solid arsenic species in contaminated estuarine sediment. Arsenite was the dominant dissolved and solid species in the deeper reduced sediment, and arsenate was dominant in the oxidized surface layer. Arsenite in the interstitial water diffused toward the surface layer, where it was mostly oxidized to arsenate.

Howard et al. (1988) found that the distribution of dissolved inorganic arsenic in an estuary appears to be determined by a combination of secondary inputs arising from old mine drainage and advective transport of arsenic-enriched sediment interstitial waters into the water column. Bioutilization of the element during the warmer months results in the release of dissolved monomethyl-arsenic and dimethylarsenic. Inorganic arsenite and methylated arsenic species can account for up to 41% and 70% of the dissolved arsenic respectively, but only when the water temperature exceeds 12 °C (Howard et al., 1984). Arsenate was the dominant form in a temperate estuary throughout the year except late winter when a dimethylarsenic species was dominant (Riedel, 1993).

Andreae (1978, 1979) monitored seawater samples from the northeast Pacific and southern Californian coast (USA). Methylation and reduction of arsenate to arsenite and methylarsenic acids occur in the photic zone. Arsenic is taken up by planktonic organisms in the surface waters and transported to deeper waters with biogenic debris. At intermediate levels regeneration of arsenate occurs. There was a good correlation between photosynthetic activity and concentration of methylated arsenicals. Andreae & Froelich (1984) and Sadiq (1990) found that arsenate is more abundant in oxic seawaters whereas arsenite is more abundant in anoxic seawaters.

Waslenchuk (1978) found that concentrations of arsenic species in continental shelf waters of the south-eastern USA are controlled mainly by simple mixing of shelf waters and Gulf Stream intrusions.

Riverine and atmospheric arsenic inputs to the shelf waters were relatively insignificant, and uptake of arsenic by biota had only a minor effect on arsenic distribution.

Byrd (1988) studied the seasonal cycle of arsenic on the continental shelf of the South Atlantic. During periods of high winds in the winter and early spring, inorganic arsenic concentrations are reduced to as little as 20% of typical open-ocean concentrations by sorption on to suspended sediments or incorporation into phytoplankton. In the late summer and early autumn arsenic is remobilized and returned to the water column, elevating arsenic concentrations to 50% more than open-ocean concentrations. Belzile (1988) analysed vertical profiles of arsenic in cores from the Laurentian trough in the gulf of St Lawrence. The surface enrichment of solid arsenic and the increase of dissolved arsenic with depth suggested that the mobile portion of arsenic is associated with iron oxyhydroxides. It follows a redox pattern of dissolution in the suboxic zone, upwards diffusion, and precipitation near the sediment–water interface under non-steady-state conditions.

Nereis succinea, a burrowing polychaete, affected distribution and flux of arsenic from sediments by its production of irrigated burrows. These burrows increased both the effective surface area of the sediment and the diffusion of arsenic by a factor of five. Although physical suspension can produce large pulses of materials from contaminated sediments, it is the continuous biological activity that is likely to be more important in the mobilization of arsenic from sediments (Riedel et al., 1987).

Riedel et al. (1989) reported that three species of burrowing invertebrates (*N. succinea*, *Macoma balthica* and *Micura leidyi*) cause a measurable flux of arsenic out of contaminated sediments which was not measurable in the absence of fauna. Arsenic release from sediment was primarily arsenate and arsenite, with trace amounts of methylated arsenic compounds.

4.1.4 Soil

Arsenic from weathered rock and soil may be transported by wind or water erosion. However, because many arsenic compounds tend to adsorb to soils, leaching usually results in transportation over

only short distances in soil (Moore et al., 1988; Welch et al., 1988). However, rainwater or snowmelt may leach soluble forms into surface water or groundwater, and soil microorganisms may reduce a small amount to volatile forms (arsines) (Woolson, 1977a; Richardson et al., 1978; Cheng & Focht, 1979; Turpeinen et al., 1999).

Under reducing conditions, arsenite dominates in soil (Deuel & Swoboda, 1972a; Haswell et al., 1985) but elemental arsenic and arsine can also be present (Walsh & Keeney, 1975). Arsenic would be present in well-drained soils as $H_2AsO_4^-$ if the soil was acidic or as $HAsO_4^{2-}$ if the soil was alkaline. Oxidation, reduction, adsorption, dissolution, precipitation and volatilization of arsenic reactions commonly occur in soil (Bhumbla & Keefer, 1994). In the porewater of aerobic soils arsenate is the dominant arsenic species, with small quantities of arsenite and MMA in mineralized areas.

The amount of arsenic sorbed from solution increases as the free iron oxide, magnesium oxide, aluminium oxide or clay content of the soil increases; removal of amorphous iron or aluminium components by treatment with oxalate eliminates or appreciably reduces the arsenic sorption capacity of the soil (Dickens & Hiltbold, 1967; Jacobs et al., 1970a; Galba, 1972; Wauchope, 1975; Livesey & Huang, 1981). Barry et al. (1995) examined the adsorption characteristics of a forest soil profile. The greatest sorption capacity for arsenic occurred at a depth of 30 cm in the profile, in the B2 horizon where there was a predominance of clay and oxyhydroxides of iron and aluminium. Adsorption of arsenic on soil colloids depends on the adsorption capacity and behaviour of these colloids (clay, oxides or hydroxides of aluminium, iron and manganese, calcium carbonates or organic matter). In general, iron oxides/hydroxides are the most commonly involved in adsorption of arsenic in both acidic and alkaline soils (Sadiq, 1997). Manning & Goldberg (1997) studied the adsorption of arsenic in three arid-zone soils. They found that the soil with the highest citrate–dithionite extractable iron and percentage of clay had the highest affinity for arsenite and arsenate and displayed adsorption behaviour similar to that of pure ferric oxide. Adsorption isotherms indicated that arsenate species adsorbed more strongly than arsenite.

The surfaces of aluminium oxides/hydroxides and clay may play a role in arsenic adsorption, but only in acidic soils. Carbonate minerals are expected to adsorb in calcareous soils (Sadiq, 1997), and Goldberg & Glaubig (1988) concluded that carbonates play a major role in arsenate adsorption at pH > 9. Phosphate substantially suppresses arsenate adsorption by soil, with the extent of the suppression varying from soil to soil (Livesey & Huang, 1981). Roy et al. (1986) found that the adsorption of arsenate was significantly reduced by competitive interactions with phosphate in three different soil types (clay, silt loam and ultisol). Darland & Inskeep (1997) found that phosphate effectively competed with arsenate for adsorption sites on sand in batch isotherms as well as in saturated transport studies. The phosphate competition was not, however, sufficient to desorb all of the applied arsenate either in simultaneously applied pulses, or in a column where arsenate was applied before a concentrated pulse of phosphate. Approximately 40% of the applied arsenate remained sorbed to the sand even after the total phosphate loading exceeded the column capacity by more than two orders of magnitude. The authors concluded that rates of arsenate desorption play an important role in transport of arsenate through porous media. Elkhatib et al. (1984) found that arsenite adsorption was not reversible, with only small amounts of sorbed arsenite released during subsequent desorption procedures. No significant correlation was found between arsenic adsorption and soil organic carbon or cation exchange capacity (CEC) (Hayakawa & Watanabe, 1982).

Jones et al. (1997) found that increased mobility of arsenic after liming appears to be consistent with the pH dependence of sorption reactions of arsenic on iron oxide minerals rather than dissolution–precipitation reactions of solid metal arsenates.

Sakata (1987) reports distribution coefficients (K_d) for arsenite for 15 subsurface soils from different sites in Japan with K_d values ranging from 75 to 1200. The distribution coefficient was significantly correlated with the extractable iron content of the soils.

Precipitation is another mechanism of arsenic removal from soil. Thermodynamic calculations showed that in acidic oxic and suboxic soils, iron arsenate may control arsenic solubility, whereas in anoxic soils, sulfides of arsenite may control the concentrations of the

dissolved arsenic in soil solutions. In alkaline, acidic, oxic and suboxic soils, precipitation of both iron arsenate and calcium arsenate may limit arsenic concentrations in soil solutions (Sadiq et al., 1983; Sadiq, 1997). Carey et al. (1996) studied the sorption of arsenic in two free-draining sandy soils in New Zealand. They concluded that arsenate sorption occurred primarily through adsorption rather than a precipitation mechanism.

Many soil organisms are capable of converting arsenate and arsenite to several reduced forms, largely methylated arsines which are volatile (see section 4.2). Woolson (1977b) proposed that about 12% of the arsenic applied and present in a soil is lost through volatilization of alkylarsines each year. Woolson & Isensee (1981) report total losses of 14–15% per year from soil treated with sodium arsenite, DMA or MMA. Most of the loss was through volatilisation, although some apparent loss was caused by movement to or mixing with subsoil. Sandberg & Allen (1975) estimated an arsenic loss of 17–35% per year through volatilization. Sanford & Klein (1988) report that arsenic volatilization showed a direct relationship with nutrient levels and microbial growth in soil.

Leaching does not appear to be a significant route of arsenic loss from soil. Arsenic as MMA was applied to three soil types over a 6-year period. Percentage recovery of applied arsenic averaged 67%, 57% and 39% in a fine sandy loam, a silt loam and a sandy loam soil respectively. All of the arsenic recovered in the soils was detected in the ploughed layer (< 30 cm) with no evidence of leaching into deeper zones (Hiltbold et al., 1974). Elfving et al. (1994) monitored the movement of arsenic following the application of lead arsenate to fruit orchards for insect control. The rate of decrease in concentration of arsenic with depth was significantly greater in a sandy soil than in clay, suggesting that downward movement occurred less readily in the former. Peryea & Creger (1994) studied the vertical distribution of arsenic in six contaminated orchard soils. Most of the arsenic was restricted to the upper 40 cm, with maximum arsenic concentrations ranging from 57.8 to 363.8 mg/kg. Absolute soil enrichment with arsenic occurred to depths between 45 and > 120 cm, with arsenic concentrations of 5.3–47.3 mg/kg at 120 cm. The authors state that the deeper movement found in this study compared with many others is due to high loading rates of lead arsenate, coarse soil texture, low organic matter content and use of

irrigation. The use of phosphate fertilizers significantly increases the amount of arsenic leached from soil contaminated with lead arsenate pesticide residues (Davenport & Peryea, 1991).

Masscheleyn et al. (1991a) found that at soil Eh levels of 200 and 500 mV arsenic solubility was low and the major part (65–98%) of the arsenic in solution was arsenate. Under moderately reduced soil conditions (at 0 and –100 mV) arsenic solubility was controlled by the dissolution of iron oxyhydroxides. Arsenic was co-precipitated as arsenate with iron oxyhydroxides and released on solubilization. On reduction to –200 mV the soluble arsenic content increased to 13 times what it was at 500 mV.

Richardson et al. (1978) monitored surface runoff of arsenic from a fine montmorillonitic clay after application of arsenic acid for desiccation of cotton (*Gossypium hirsutum*). They calculated that approximately 7% of the amount applied would be transported from the watershed by runoff and erosion, 38% in solution and 62% attached to sediment.

Tammes & de Lint (1969) calculated an average half-life of 6.5 ± 0.4 years for arsenic persistence on two Netherlands soils after application of arsenite.

4.2 Biotransformation

Most environmental transformations of arsenic appear to occur in the soil, in sediments, in plants and animals, and in zones of biological activity in the oceans. Biomethylation and bioreduction are probably the most important environmental transformations of the element, since they can produce organometallic species that are sufficiently stable to be mobile in air and water. However, the biomethylated forms of arsenic are subject to oxidation and bacterial demethylation back to inorganic forms (IPCS, 1981, section 4).

Three major modes of biotransformation of arsenic species have been found to occur in the environment: redox transformation between arsenite and arsenate, the reduction and methylation of arsenic, and the biosynthesis of organoarsenic compounds. There is biogeochemical cycling of compounds formed by these processes (Andreae, 1983).

Arsenic is released into the atmosphere primarily as As_2O_3 or, less frequently, in one of several volatile organic compounds, mainly arsines (US EPA, 1982). Trivalent arsenic and methyl arsines in the atmosphere undergo oxidation to the pentavalent state, and arsenic in the atmosphere is usually a mixture of the trivalent and pentavalent forms (Scudlark & Church, 1988). Photolysis is not considered an important breakdown process for arsenic compounds (Callahan et al., 1979).

Arsenic can undergo a complex series of transformations, including redox reactions, ligand exchange and biotransformation (Callahan et al., 1979; Welch et al., 1988). Factors affecting fate processes in water include the Eh, pH, metal sulfide and sulfide ion concentrations, iron concentrations, temperature, salinity, and distribution and composition of the biota (Callahan et al., 1979; Wakao et al., 1988).

4.2.1 Oxidation and reduction

Oscarson et al. (1980) observed oxidation of arsenite (10 mg/litre) to arsenate in sediments from lakes in Saskatchewan (Canada). The oxidation process was unaffected by flushing nitrogen or air through the system or by the addition of mercuric chloride. The authors therefore concluded that the oxidation was an abiotic process, with microorganisms playing a very minor role in the system. However, Scudlark & Johnson (1982) examined the oxidation of arsenite in seawater at low levels. They found that abiotic oxidation proceeded at a slow and constant rate with rapid oxidation occurring only in the presence of certain aquatic bacteria. The rate of abiotic oxidation, after spiking water with an initial arsenite concentration of 4 µg/litre (53 nmol/litre), was 0.2 µg/litre per day in distilled water and 0.3 µg/litre per day in artificial seawater. Baker et al. (1983a) found no methylated arsenic compounds in sterile lake sediments incubated in the presence of arsenate or arsenite.

Scudlark & Johnson (1982) studied the biological oxidation of arsenite in seawater in Narragansett bay (Rhode Island, USA). They found that oxidation was primarily due to microbial activity. Oxidation obeyed first-order kinetics with a rate constant of 0.06 h^{-1} and half-lives ranging from 8.9 to 12.8 h for initial arsenite

concentrations ranging from 7.5 µg/litre to 6.9 mg/litre (0.1B91.8 µmol/litre). Under aerobic conditions the mixed microbial cultures of lake sediments were able to reduce arsenate to arsenite and also to oxidize arsenite to arsenate. However, under anaerobic conditions only reduction was observed (Freeman et al., 1986).

In seawater containing free dissolved oxygen, arsenate is the thermodynamically stable form of the element. Arsenite is present in amounts exceeding those of arsenate only in reduced, oxygen-free porewaters of sediments and in anoxic basins such as the Baltic sea. However, significant amounts of arsenite (up to 10% of total arsenic) are found in the surface and deep waters of the oceans and, conversely, some arsenate is still present in anoxic water (Andreae, 1983). The presence of arsenite in seawater suggests that some reduction of arsenate occurs, and indeed Johnson (1972) demonstrated that bacterial arsenate reduction can take place under laboratory conditions. Matsuto et al. (1984) isolated a cyanobacterium (*Phormidium* sp.) from the coastal marine waters of Suruga bay (Japan) that was capable of readily reducing adsorbed arsenate to arsenite.

Freeman (1985) isolated an *Anabaena oscillaroides*–bacteria assemblage from the arsenic-rich Waikato river (New Zealand) capable of reducing arsenate to arsenite. In continuous culture the cyanophyte–bacteria assemblage could reduce arsenate to arsenite at a rate of 12 ng As/10^6 cells per day. Wakao et al. (1988) detected microbial arsenite oxidation occurring in acid mine waters (pH 2.0B2.4) containing 2–13 mg As/litre. Ahmann et al. (1994) isolated a microorganism from arsenic-contaminated sediment in eastern Massachusetts (USA) which used the reduction of arsenate to arsenite to gain energy for growth. Similarly, Macy et al. (1996) found that an anaerobic bacterium *Chrysiogenes arsenatis* from gold-mine wastewater grew by reducing arsenate to arsenite using acetate as the electron donor and carbon source.

On the bassis of both aqueous and solid-phase observations, McGeehan (1996) found that arsenate was reduced to arsenite in flooded soil under batch conditions. Reduction of arsenate to arsenite has also been reported for both freshwater and marine macroalgae (Blasco, 1975; Johnson & Burke, 1978; Andreae & Klumpp, 1979; Wrench & Addison, 1981). Calculations based on the measured rates

of reduction indicate that 15–20% of the total arsenic is reduced by phytoplankton during spring and autumn blooms on the continental shelf (Sanders & Windom, 1980).

4.2.2 Methylation

The biomethylation of arsenic was first recognized when arsines were produced from cultures of a fungus, *Scopulariopsis brevicaulis* (Challenger, 1945). Subsequently, the methylation of arsenic by methanogenic bacteria (McBride & Wolfe, 1971) and by reaction with methyl cobalamine (Schrauzer et al., 1972) or L-methionine-methyl-d3 (Cullen et al., 1977) has been demonstrated in laboratory work. Cox & Alexander (1973) showed that cultures of the fungus *Candida humicola* methylate arsenite, arsenate, methylarsonate and DMA to trimethylarsine. Further experiments have shown that growing cells of *C. humicola* can be induced to produce trimethylarsine from arsenate and DMA by preconditioning with DMA (Cullen et al., 1979b). Cullen et al. (1979a) incubated *C. humicola* in the presence of ^{74}As-arsenate, ^{14}C-methylarsonate or ^{14}C-DMA. They identified arsenite, methylarsonate, DMA and trimethylarsine oxide as intermediates in a biological synthesis of trimethylarsine. However, they tentatively conclude that methylarsonate does not occur as a free intermediate in the arsenate to trimethylarsine pathway.

McBride et al. (1978) reported that dimethylarsine was mainly produced by anaerobic organisms, whereas trimethylarsine resulted from aerobic methylation.

Methylated arsenic compounds were detected in aerobic sediments from various locations in Ontario (Canada) incubated with or without the addition of extraneous arsenic. Two pure bacterial cultures, *Aeromonas* sp. and *Flavobacterium* sp., isolated from lake water, were also found to methylate arsenic compounds in a synthetic medium (Wong et al., 1977).

Baker et al. (1983a) incubated lake sediment in the presence of arsenate or arsenite (7.5 mg As/litre). Methylation occurred over the pH range 3.5–7.5, with analysis revealing the presence of both methyl arsonic acid and dimethylarsinic acid. The amount of arsenic recovered in the methylated species ranged from 0 to 0.4% of the

total inorganic arsenic added. Maeda et al. (1988) exposed the cyanobacterium *Phormidium* sp. (isolated from an arsenic-polluted environment) to arsenate (128 mg/kg) and found that 3.2% of the accumulated arsenic had been methylated.

Huysmans & Frankenberger (1991) isolated a *Penicillium* sp. from evaporation pond water capable of methylating and subsequently volatilizing organic arsenic. The conditions optimum for trimethylarsine production were a minimal medium containing 100 mg/litre methylarsonic acid, pH 5–6, a temperature of 20 °C and a phosphate concentration of 0.1–50 mmol/litre.

Reimer & Thompson (1988) found a strong positive correlation between the sum of the methylarsenic compounds and the total dissolved arsenic in marine interstitial waters influenced by mine tailings discharges indicating in situ microbial methylation. Laboratory studies have shown that microorganisms present in both natural marine sediments and sediments contaminated with mine tailings are capable of methylating arsenic under aerobic and anaerobic conditions (Reimer, 1989).

Biomethylation is primarily restricted to the high-salinity regions of estuaries with the presence of methylated arsenic at lower salinities predominantly as a result of the mixing of saline water (containing bioarsenicals) with river water (Howard & Apte, 1989).

Several authors have reported arsenic methylation in macroalgae, particularly in marine organisms (Edmonds & Francesconi, 1977; Andreae & Klumpp, 1979; Wrench & Addison, 1981; Maeda et al., 1987b; Cullen et al., 1994). In fact, most diatoms, dinoflagellates and macroalgae as well as freshwater higher plants, release protein-bound arsenic as a result of sequential methylation and adenosylation (Benson et al., 1988). Baker et al. (1983b) reported that freshwater green algae were capable of methylating sodium arsenite in lake water. Analysis revealed the presence of MMA, DMA and trimethylarsine oxide; however, volatile arsine and methylarsines were not detected. Similarly, Wrench & Addison (1981) identified MMA and DMA as polar arsenic metabolites synthesized by the marine phytoplankton *Dunaliella tertiolecta*. Maeda et al. (1987b) exposed five arsenic-resistant freshwater algae from an arsenic-polluted environment to

arsenate. Small amounts of methylated arsenic compounds were detected and these were strongly bound with proteins or polysaccharides. Methylated arsenic compounds were found mainly in the lipid-soluble fractions and the major form was a dimethyl arsenic compound. No methylation occurred in algal cells (*Chlorella vulgaris*) exposed to arsenate under in vitro conditions; however, in vivo a small fraction of the arsenic accumulated was first transformed to methyl and dimethyl arsenic compounds during the early exponential phase and finally transformed to trimethylarsenic species (Maeda et al., 1992b). The marine algae *Ecklonia radiata* and *Polyphysa peniculus* methylated arsenate to produce a dimethylarsenic derivative. It was concluded that methionine or *S*-adenosylmethionine was the source of the methyl groups in this biological alkylation (Edmonds & Francesconi, 1988a; Cullen et al., 1994). *S*-adenosylmethionine is also likely to be the source of adenosyl and ribosyl groups in the arsenosugars.

The organic arsenical arsenobetaine was first identified in the late 1970s (Edmonds & Francesconi, 1981b) and has now been isolated in a variety of marine organisms (Edmonds & Francesconi, 1981b; Norin & Christakopoulos, 1982; Shiomi et al., 1984; Edmonds et al., 1992). Edmonds & Francesconi (1981a) identified arsenosugars isolated from brown kelp (*Ecklonia radiata*) as intermediates in the cycling of arsenic and stated that these compounds could be subsequently metabolized to arsenobetaine. Edmonds et al. (1982) have shown that the simpler arsenosugars in the brown alga are degraded under anaerobic conditions to dimethyloxarsylethanol. The transformation of dimethyloxarsyl-ethanol to arsenobetaine would require both a reduction-methylation step and an oxidation step; these are probably bacterially mediated (Edmonds & Francesconi, 1987a, 1988b). Edmonds & Francesconi (1988b) concluded that arsenobetaine is probably formed by the conversion of arsenate to dimethyl(ribosyl)arsine oxides by algae, and that the microbially mediated transformation to arsenobetaine or its immediate precursors occurs in sediments. Phillips & Depledge (1985, 1986) proposed that phospholipids containing arsenoethanol-amine or arsenocholine moieties may be formed as intermediates in the formation of arsenosugars and arsenobetaine. Edmonds et al. (1992) identified arsenocholine-containing lipids as natural products in the digestive gland of the rock lobster (*Panulirus cygnus*). Phillips & Depledge (1985) concluded that arsenic replaces nitrogen in

phospholipid synthesis leading to a large number of arsenic-containing intermediates, which would be either water-soluble or lipid-soluble. Arsenic-containing compounds are catabolized as they pass through the food web, yielding arsenobetaine as a stable end-product.

Inorganic arsenic administered orally to brown trout (*Salmo trutta*) was detected in tissues as organoarsenical species, whereas arsenic administered by injection was taken up as inorganic arsenic and slowly converted to the organic form. It was concluded that biosynthesis of arsenic was occurring in the gastrointestinal tract (Penrose, 1975). Oladimeji et al. (1979) reported that arsenic given as an oral dose to rainbow trout (*Oncorhynchus mykiss*) was rapidly converted to organic forms. The ratio of total organic to inorganic increased with time in all tissues, with the organic arsenic fraction accounting for about 50% after 6 h and over 80% within 24 h. The major organic arsenical appeared to be an arsenobetaine-related compound. Similarly, Penrose et al. (1977) found that sea urchins (*Strongylocentrotus droebachiensis*) were also able to convert inorganic arsenic to an organic form, but to a more limited degree than trout. However, Wrench et al. (1981) concluded that organic arsenic synthesized in the brine shrimp (*Artemia salina*) is methylated by intestinal microflora and not by the filter feeder itself.

Maeda et al. (1990c) found that 85% of arsenic accumulated by the guppy (*Poecilia* sp.) was in the di- and tri-methylated forms. The percentage of organic species was much higher than that found in phytoplankton and zooplankton in the same model ecosystem. Similarly, Maeda et al. (1990a) found that biomethylation of arsenic increased successively with trophic level in another model ecosystem: goldfish (*Carassius* sp.) > zooplankton (*Moina* sp.) > alga (*Chlorella* sp.).

4.2.3 Degradation

4.2.3.1 Abiotic degradation

The rates of photochemical decomposition of arsenite, DMA, MMA and arsenobetaine have been studied in both distilled water and seawater. All species were found to degrade rapidly in aerated distilled water. In deaerated solutions the rate of oxidation of arsenite

was almost two orders of magnitude slower. Half-lives for the degradation of DMA, MMA and arsenite were 9.2, 11.5 and 0.9 min respectively for aerated distilled water and 25, 19 and 8 min for deaerated distilled water. In seawater, the rates of photochemical decomposition were slower. For example, in seawater only 20% of DMA was converted to MMA after 300 min with no other products detected, whereas in distilled water DMA was completely degraded within 100 min (Brockbank et al., 1988). This study suggests that UV irradiation is of limited use for the pretreatment of saline samples to convert organoarsenic species to As(V) before analysis. The implications for photochemical decomposition of arsenic species in natural waters is not clear, because sunlight is deficient in the lower-wavelength bands generated by the mercury lamp used in this study. In addition, colloids and suspended particulates in the photic zone may play a significant role in arsenic decomposition in natural waters.

Von Endt et al. (1968) concluded that degradation of MSMA in soil was primarily due to soil microorganisms rather than abiotic factors. In 60-day tests in non-sterile soil 1.7–10% of the ^{14}C-MSMA was degraded, whereas under steam-sterilized conditions only 0.7% was degraded.

4.2.3.2 Biodegradation

The predominant form of arsenic in water is usually arsenate (Callahan et al., 1979; Wakao et al., 1988), but aquatic micro-organisms may reduce the arsenate to arsenite and a variety of methylated arsenicals.

Marine organisms tend to contain much higher levels of arsenic than terrestrial organisms; this is because of the high arsenate/phosphate ratio in oceans, which is a consequence of the very low phosphate concentration. Most of the arsenic accumulated in marine organisms is in a water-soluble form of arsenic, namely arsenobetaine. Hanaoka et al. (1987) incubated marine sediments in the presence of arsenobetaine and demonstrated microbial degradation, with arsenate, arsenite, MMA, DMA and arsenobetaine being identified. Further experiments revealed the formation of trimethylarsine oxide during aerobic incubation of bottom sediments with arsenobetaine as the carbon source (Kaise et al., 1987). Under

aerobic conditions, arsenobetaine is converted to its metabolites to a much greater extent than other methylarsenicals. Under anaerobic conditions little or no degradation of arsenobetaine occurred, whereas trimethylarsine oxide and DMA were converted to less methylated compounds (Hanaoka et al., 1990). Degradation of arsenobetaine has also been demonstrated in the water column in the presence of suspended substances (Hanaoka et al., 1992).

Organoarsenical pesticides (e.g. MMA and DMA) applied to soil are metabolized by soil bacteria to alkylarsines, MMA, and arsenate (ATSDR, 1993). The half-time of DMA in soil is about 20 days (ATSDR, 1993).

Cheng & Focht (1979) added arsenate, arsenite, methylarsonate and DMA to three different soil types. Arsine was produced in all three soils from all substrates but methylarsine and dimethylarsine were only produced from methylarsonate and DMA respectively. Both *Pseudomonas* sp. and *Alicaligenes* sp. produced arsine as the sole product when incubated anaerobically in the presence of arsenate or arsenite. The authors concluded that reduction to arsine, not methylation to trimethylarsine, was the primary mechanism for gaseous loss of arsenicals from soil.

Degradation of MSMA by soil microorganisms was studied by Von Endt et al. (1968). In 60-day tests they found that 1.7–10% of the ^{14}C-MSMA was degraded; four soil microorganisms isolated in pure cultures degraded 3–20% of ^{14}C-MSMA to $^{14}CO_2$ when grown in liquid culture at 10 mg MSMA/litre. Woolson & Kearney (1973) showed that sodium DMA was degraded to arsenate in soil under aerobic conditions but not under anaerobic conditions. Degradation of MSMA has been shown to be associated with soil organic matter oxidation. In a loamy soil, degradation increased with increasing organic matter content (Dickens & Hiltbold, 1967). Akkari et al. (1986) studied the degradation of MSMA in soils at concentrations up to 5 mg As/kg. It was found that degradation followed first-order kinetics. The rate constant was temperature dependent only at soil water contents less than field capacity, and the temperature effect was less under flooded conditions. The differences in degradation rate under aerobic conditions and 20% water content were related to differences in the texture of the three soils. Half-lives for the clay and silty loam soils were 144 and 88 days respectively. Under

anaerobic (flooded) soil conditions MSMA degradation occurs by reductive methylation to form arsenite and alkylarsine gases. The half-life values for the two soils indicate significantly faster degradation at 25 and 41 days respectively. The third soil, a sandy loam, produced the slowest degradation rate ($t_{½}$ = 178 days) probably because of its low organic matter content which may have supported fewer microorganisms.

The overall percentage of DMA (sodium salt) and MMA mineralized in a silty clay soil after 70 days ranged from 3% to 87% – values much higher than arsenic loss as volatile arsines (0.001B0.4%). Arsenate was the main metabolite from the degradation of both sodium DMA and MMA. The amount of sodium DMA mineralized was linearly related to the concentration of sodium DMA in the soil, indicating that the rate is first order. Mineralization of sodium DMA increased with increasing soil moisture and temperature. It was concluded that the loss of arsenic from some soils to the atmosphere may not be a major pathway and that inorganic arsenic may accumulate in soil from arsenical usage (Gao & Burau, 1997).

4.2.4 Bioaccumulation

Bioconcentration of arsenic under laboratory conditions occurs in aquatic organisms, primarily in algae and lower invertebrates. Bioconcentration factors (BCFs) measured in freshwater invertebrates for several arsenic compounds generally ranged up to 20; bioconcentration factors in fish were < 5; higher concentration factors have been observed in algae. Biomagnification in aquatic food chains does not appear to be significant (Callahan et al., 1979). Terrestrial plants may accumulate arsenic by root uptake from the soil or by adsorption of airborne arsenic deposited on the leaves, some species accumulating substantial levels.

4.2.4.1 Microorganisms

Maeda et al. (1987a) exposed cyanobacteria (*Nostoc* sp.) to arsenate concentrations of 1 and 10 mg As(V)/litre for 32 days with no effect on cell growth. *Nostoc* sp. accumulated 32 and 77 mg As/kg (dry cell) respectively at the two exposure concentrations.

Lindsay & Sanders (1990) report BCFs ranging from 1132 to 3688 for estuarine phytoplankton (*Thalassiosira pseudomonas*, *Skeletonema costatum* and *Dunaliella tertiolecta*) exposed to 25 µg As(V)/litre as arsenate for up to 48 h.

Phytoplankton take up arsenate readily and incorporate a small proportion into the cell. Most of the arsenate is reduced, methylated and released to the surrounding media. Phytoplankton batch cultures exposed to elevated levels of arsenate take up additional arsenic during the log phase of growth. Studies using ^{74}As indicate that the uptake rate varies from 0.15 ng As(V)/10^6 cells per hour in unenriched cultures to 2.3 ng As(V)/10^6 cells per hour in cultures containing 25 µg As(V)/litre. Cultured *Skeletonema costatum* increase their arsenic concentrations approximately 40% from 22 to 29 mg/kg (dry weight) in response to arsenate concentrations of 6B25 µg As(V)/litre (Sanders & Windom, 1980).

Phytoplankton readily incorporated dissolved arsenic, with average arsenic residues increasing from 5.7 to 17.7 mg/kg (dry weight) when cultured for 48–96 h at 25 µg As(V)/litre as arsenate (Sanders et al., 1989). Arsenate added to a freshwater model ecosystem was readily accumulated in plankton with arsenic residues of 37–47 mg/kg (dry weight) at 5 µg As(V)/litre and > 200 mg/kg at 50 µg As(V)/litre after 65-day exposures. Accumulation in other biota was much lower than for phytoplankton (Reuther, 1992).

Giddings & Eddlemon (1977) studied the uptake of radioactively labelled arsenic (added as sodium arsenate at 50 µg As(V)/litre) in model ecosystems (7 and 70 litres) for 5 weeks. Mean BCFs for algae ranged from 370 for sand microcosms to 4300 for lake mud microcosms. Algal arsenic concentrations were significantly greater in the 70-litre microcosms and in the sand microcosms than in the 7-litre and sediment microcosms.

Green algae (*Chlorella vulgaris*) exposed to arsenate concentrations of 7 to 9 mg As(V)/litre accumulated maximum residues of 3.75 g total As/kg (dry mass) within 10 days (Maeda et al., 1992c).

Maeda et al. (1985) found that arsenate uptake increased with an increase in the arsenic exposure concentration with *C. vulgaris*

isolated from an arsenic-polluted environment. Maximum BCFs of 200–300 were observed during the log phase. At the highest exposure concentration (10 g As(V)/litre) algae were able to accumulate 50 g As/kg (dry weight). Approximately half of the arsenic taken up was estimated to be adherent to the extraneous coat of the cell with the remainder accumulated by the cell. Arsenate accumulation was affected by the growth phase; arsenic was most actively accumulated when the cell was exposed to arsenic during the early exponential phase (Maeda et al., 1992a).

Accumulation of arsenic (1 mg As(V)/litre as arsenate) by *Dunaliella* sp. was rapid, with equilibrium established within 8 h. Arsenic accumulation was studied at temperatures ranging from 10 °C to 33 °C, pH 4–10, light intensity from 0 to 10 000 lux and sodium chloride concentrations from 1 to 100 g/litre. Maximum arsenic residues under optimum conditions (22 °C; pH 8.2; 5000B10 000 lux and 20 g NaCl/litre) were 2000 mg As/kg. Increased phosphate significantly decreased the uptake of arsenic in the culture (Yamaoka et al., 1988). Yamaoka et al. (1992) found that *D. salina* accumulated more arsenic at nitrogen concentrations of 72 mg/litre than at 4.5 mg/litre.

4.2.4.2 Macroalgae

Fucus vesiculosus accumulated approximately 120 mg As/kg during an 85-day exposure to 7.5 µg As(V)/litre as arsenate. Filamentous algae and planaria accumulated less than 40 mg As/kg (dry weight), and cyanobacteria and various zooplankton accumulated less than 20 mg As/kg (Rosemarin et al., 1985).

Klumpp (1980) studied the effect of a variety of factors on the uptake of labelled arsenic by the seaweed *Fucus spiralis*. Neither pH (pH 7–9) nor salinity (9–36 g/litre) affected the uptake of arsenic; however, uptake at 30 °C was twice that at 16 °C. Arsenate uptake was reduced with increasing phosphate concentration (40B400 µmol/litre).

Lee et al. (1991) grew the aquatic plant *Hydrilla verticillata* in both mine-waste pool water and deionized distilled water contaminated with arsenate (0.4 and 0.8 mg As(V)/litre) for up to 16 days. Accumulation of arsenic reached steady state at 2–6 days in

pool water at BCFs of 110–190. In deionized water maximum arsenic accumulation occurred after 8 days at a BCF of around 300. Phosphate (≥ 12 mg/litre) inhibited the uptake of arsenic by *H. verticillata*.

4.2.4.3 Aquatic invertebrates

Sanders et al. (1989) studied the uptake of arsenic from water and from phytoplankton by the copepod *Eurytemora affinis* and the barnacle *Balanus improvisus*. In 24-h tests, *E. affinis* exhibited no uptake of dissolved arsenic; the arsenic content of copepods fed phytoplankton increased to 11.2 mg/kg (dry weight) compared with 8.9 mg/kg in controls. In 22-day tests, *B. improvisus* exposed to dissolved arsenate (55 µg As(V)/litre) in water did not accumulate arsenic, with levels remaining around 0.88 mg/kg; however, levels in shell material increased from 0.3 mg/kg to 2 mg/kg. Barnacles fed arsenic-contaminated phytoplankton (~18 mg/kg) exhibited an increase in total arsenic concentrations from 0.3 mg/kg to 1.7 mg/kg. In further experiments with oysters (*Crassostrea virginica*) no accumulation of arsenic from water was observed in 28-day tests, but tissue concentrations increased significantly from 5.3 mg/kg to 8.2 mg/kg in oysters fed arsenic-contaminated phytoplankton. Zaroogian & Hoffman (1982) reported maximum total arsenic residues in soft tissues of oysters (*Crassostrea virginica*) of 12.6, 12.7 and 14.1 mg/kg (dry weight) at arsenite exposure concentrations of 1.2 (control), 3 and 5 mg As(III)/litre during 112-day exposures. Generally, arsenic body burdens increased with increases in phytoplankton concentration and it appears that food contributes more to arsenic uptake than do seawater arsenic concentrations. No relationship between arsenic uptake and seawater arsenic concentrations was found.

Ünlü & Fowler (1979) exposed mussels (*Mytilus galloprovincialis*) to arsenate (^{74}As) concentrations ranging from 20 to 100 µg As(V)/litre at 12 °C and 21 °C. Mean concentration factors after 20 days were low, at respectively 8.8 and 12.1 for the two temperatures; however, mussels did accumulate significantly more arsenic at 21 °C than at 12 °C. Arsenic uptake was inversely related to salinity over the range 31–19 g/litre. Arsenic loss was essentially biphasic, with biological half-times of approximately 3 and 32 days for the fast and slow compartments respectively. The

active secretion of arsenic in the byssal threads contributed to the total elimination of the element from the mussels. Similarly, Ünlü (1979) found a biphasal loss of arsenic from crabs (*Carcinus maenas*) during a 43-day depuration period. The elimination of ^{74}As by the crabs after ingestion of arsenic-contaminated mussels was dependent on the chemical form of the arsenic. After ingestion of mussel containing mostly lipid- and water-soluble arsenic species (undetermined), biological half-times were 3.4 and 19.6 days for the first and second phase of loss. After ingestion of mussel containing mostly arsenite and residual arsenic, half-times were 1.6 and 9.3 days respectively.

Naqvi et al. (1990) exposed red crayfish (*Procambarus clarkii*) to MSMA at concentrations of 0.5, 5 and 50 mg As/litre. Uptake of arsenic was dose-dependent but not time-dependent. Maximum whole-body residues were 1.36, 4.29 and 9 mg As/kg respectively for each of the exposure concentrations during the 8-week uptake period.

Gibbs et al. (1983) reported equilibrium BCFs, based on ^{74}As, for the cirratulid polychaete *Tharyx marioni* ranging from 4.5 at an exposure concentration of 10 mg As(V)/litre (as arsenate) to 111.6 at 0.01 mg/litre after 7 days. A lower BCF of only 15.9 at 0.01 mg/litre was reported for the polychaete *Caulleriella caputesocis*.

Shrimps exposed to water concentrations ranging from 0.1 to 1.5 mg As(V)/litre (as arsenate) or food (*Chlorella* sp.) containing, 1940 mg total As/kg contained arsenic residues ranging from 18.9 to 31.8 mg/kg (dry weight) (Maeda et al., 1992c).

Fowler & Ünlü (1978) reported BCFs of less than 10 for shrimps exposed to arsenate (^{74}As) concentrations of 20–100 µg As(V)/litre for 14 days. Arsenic loss was biphasic with half-lives of 3 and 26 days for the fast and slow compartments respectively. Moults shed during loss contained 2–5% of the shrimp's ^{74}As body burden.

Lindsay & Sanders (1990) found no bioaccumulation of arsenate directly from the water (25 µg As(V)/litre) or from food for the grass shrimp (*Palaemonetes pugio*). Brine shimp (*Artemia* sp.) grown in elevated arsenic concentrations exhibited small, but significant, increases in arsenic content from an average of 16.8 mg/kg (dry

weight) in controls to 17.8 mg/kg at 25 µg As(V)/litre; no accumulation was observed when brine shrimps were fed arsenic-contaminated food.

4.2.4.4 Fish

Barrows et al. (1980) exposed bluegill sunfish (*Lepomis macrochirus*) to 130 µg As(III)/litre of As_2O_3 for 28 days. The maximum BCF was found to be 4, with a half-life in tissues of 1 day. Nichols et al. (1984) found no accumulation of arsenic in a 6-month study on coho salmon (*Oncorhynchus kisutch*) exposed to As_2O_3 concentrations of < 300 µg As(III)/litre. Whole-body residues were below 0.4 mg As/kg (wet weight) and were not dose dependent.

Sorensen (1976) found that green sunfish (*Lepomis cyanellus*) exposed to higher arsenic concentrations of 100, 500 and 1000 mg As(V)/litre (as arsenate) accumulated whole-body arsenic concentrations of 33.4, 541.2 and 581.6 mg/kg (BCFs ranging from 0.3 to 1.1). Green sunfish exposed to 60 mg As(V)/litre for 6 days accumulated mean arsenic residues of 158.7, 47.7, 18.9 and 14.2 mg/kg in the gallbladder (plus bile), liver, spleen and kidney respectively (BCFs ranging from 0.2 to 2.6) (Sorensen et al., 1979).

Cockell & Hilton (1988) fed rainbow trout (*O. mykiss*) on diets containing As_2O_3 (180–1477 mg As/kg diet), disodium arsenate heptahydrate (DSA) (137–1053 mg As/kg diet), DMA (163B1497 mg As/kg diet) or arsanilic acid (193–1503 mg As/kg) for 8 weeks. For each of the arsenicals investigated, carcass arsenic concentration showed a dose–response relationship to dietary arsenic concentration and exposure rate. At lower levels of exposure (137 mg As/kg diet), dietary DSA yielded the highest mean carcass arsenic concentrations (6.9 mg As/kg), but at higher levels, dietary As_2O_3 (1477 mg As/kg diet) yielded the highest mean residues (21.6 mg As/kg). Inorganic arsenicals were accumulated from the diet to a greater degree than the organic forms. In a 16-week study, dietary DSA (8–174 mg As/kg diet) accumulated in the carcass (0.25–5.7 mg As/kg), liver (0.7–34.4 mg As/kg) and kidney (1.1B31.9 mg As/kg) in a dose-related manner (Cockell et al., 1991).

Oral administration of sodium arsenate to estuary catfish (*Cnidoglanis macrocephalus*) and school whiting (*Sillago bassensis*)

resulted in an accumulation of trimethylarsine oxide in their tissues (Edmonds & Francesconi, 1987b). Yelloweye mullet (*Aldrichetta forsteri*) fed the organic arsenicals 2-dimethylarsinylethanol, 2-dimethylarsinylacetic acid or 2-dimethyllarsinothioylethanol showed no arsenic accumulation in their tissues; fish fed arsenate-contaminated food showed a small but significant increase in arsenic concentration (muscle tissue = 1 mg As/kg wet weight). However, administering arsenobetaine or arsenocholine in the diet led to muscle concentrations of around 24 mg As/kg (wet weight) (Francesconi et al., 1989).

Oladimeji et al. (1984) fed rainbow trout (*O. mykiss*) on a diet containing 10, 20 or 30 mg As(III)/kg (as sodium arsenite) (equivalent to 0.2, 0.4 and 0.6 mg/kg fish wet weight per day) for up to 8 weeks. Arsenic accumulation was dose related, with residues ranging from 1.28 to 1.52 mg/kg (dry weight) for muscle, 1.55 to 5.21 mg/kg for liver, 0.84 to 1.88 mg/kg for gills and 1.21 to 1.98 mg/kg for skin tissue.

4.2.4.5 Terrestrial plants

Arsenic species can enter into edible tissues of food crops through absorption (i.e. not just surface contamination) (Woolson, 1973; Helgesen & Larsen, 1998). Helgesen & Larsen (1998) demonstrated that bioavailability of arsenic pentoxide to carrots in soil from a wood preservative treatment plant (soil was contaminated with CCA) was 0.47 ± 0.06% of total soil arsenic burden. This study showed that arsenite, arsenate, MMA and DMA were present in carrot tissue, where only arsenite and arsenate were present in soil. In soils dosed with arsenate (0–500 µg/g) at the concentrations which inhibited growth of vegetable crops (green bean, lima bean, spinach, cabbage, tomato and radish), high levels of accumulation when found in the edible parts of radish (76 µg/g) spinach (10 µg/g) and green bean (4.2 µg/g). Arsenic accumulation in Lima bean, cabbage and tomato ranged from 0.7–1.5 µg/g. The studies of Woolson (1973) and Helgesen & Larsen (1998) highlight the potential of movement of arsenic species from soil into agronomic crops.

Uptake of arsenate (10 mg/litre [133 µmol/litre]) by moss (*Hylocomium splendens*) from nutrient solution displayed saturation kinetics at pH 5 that could be described in terms of Michaelis–

Menten parameters with a mean K_m value of 31.4 mg/litre (418 µmol/litre). Phosphate was a competitive inhibitor of arsenate uptake with an inhibition constant (K_m phosphate) of 82 µmol/litre (Wells & Richardson, 1985).

Asher & Reay (1979) studied the uptake of arsenate (1 mg/litre [15 µmol/litre]) from nutrient solution by barley (*Hordeum vulgare*) seedlings. They found that uptake consisted of a rapid initial phase followed by a less rapid 'steady-state' phase, both of which were strongly inhibited by phosphate and positively correlated with temperature.

The marsh plant species *Spartina alterniflora* was grown in sediment treated with ~50 µg As(V)/litre (as arsenate) and accumulated significantly elevated total concentrations of arsenic after 9 days; new and old leaf blades contained mean arsenic concentrations of 6.3 and 5 mg/kg (dry weight) respectively, relative to 1 and 0.4 mg/kg in control plants (Sanders & Osman, 1985).

Meharg & Macnair (1991b) found that non-tolerant genotypes of *Holcus lanatus* accumulated significantly more arsenate than tolerant plants during a 6-h period of growth in 3.75 mg As(V)/litre (0.05 mol/m^3) arsenate. They found that tolerant plants transported a much greater proportion of arsenic to their shoots than non-tolerant plants. Phosphate (0.05 or 0.5 mol/m^3) decreased arsenate uptake in both tolerant and non-tolerant genotypes. Arsenate tolerance involves reduced accumulation of arsenate through suppression of the high-affinity phosphate–arsenate uptake system (Meharg et al., 1994).

Anastasia & Kender (1973) grew lowbush blueberry (*Vaccinium angustifolium*) plants in greenhouse soil at As_2O_3 concentrations ranging from 7.7 (controls) to 84.5 mg As(III)/kg for 17 weeks. Arsenic was accumulated in a dose-dependent manner with arsenic residues of 0.78–15 mg/kg for leaves, 0.27–13.3 mg/kg for stems and 2.4–164.2 mg/kg for roots.

Otte et al. (1990) grew *Urtica dioica* and *Phragmites australis* in soil containing up to 30 mg As/kg added as lead arsenate or sodium DMA. Concentrations of arsenic in shoots and roots of *P. australis* increased significantly only at the highest arsenic concentration in

soil with mean values of up to 1 mg/kg (dry weight) in shoots and 44.3 mg/kg in roots, whereas the arsenic content of *U. dioica* increased by a factor of 4 at 5 mg As/kg with plants accumulating mean arsenic concentrations of up to 150 mg/kg in roots at the highest exposure.

Onken & Hossner (1995) grew rice (*Oryza sativa*) in two soil types treated with up to 45 mg As(III) or As(V)/kg (as arsenite or arsenate) for 60 days. The arsenic concentration of rice plants correlated with the mean soil solution arsenate concentration in the clay soil and to the mean soil solution arsenite for the silt loam. The rate of arsenic uptake by plants increased as the rate of plant growth increased.

4.2.4.6 Terrestrial invertebrates

Meharg et al. (1998) exposed earthworms (*Lumbricus terrestris*) to arsenate (40 mg/kg dry weight) for 23 days. There was a steady-state increase in residues for depurated and undepurated worms and by 12 days earthworm residues were equivalent to those of the soil. Arsenic residues were accumulated to three times soil levels by the end of the 23-day exposure in depurated worms; however, undepurated worms did not appear to bioconcentrate arsenic beyond the level of the surrounding soil.

4.2.4.7 Birds

Daghir & Hariri (1977) administered arsanilic acid (used as a feed medication for poultry) to White Leghorn laying hens at 50 and 100 mg/kg for 15 weeks. Maximum concentrations in eggs were reached after 4–5 weeks at 0.13 and 0.24 mg As/kg (dry weight) for the two dose levels respectively. Residual arsenic was negligible 2 weeks after the withdrawal of the drug from the feed.

Proudfoot et al. (1991) found a higher concentration of arsenic in liver and muscle of broilers that were fed arsanilic acid (99 mg/kg diet) compared with controls. Mean arsenic residues of up to 1.5 mg/kg and 0.4 mg/kg were measured for the two tissues respectively. Broilers fed a diet containing 100 or 500 mg/kg arsanilic acid accumulated up to 2.3 and 8 mg As/kg in liver tissue at the two exposure concentrations respectively. Lower levels were

accumulated in muscle tissue, with arsenic concentrations of up to 0.15 and 0.67 mg/kg (VanderKop & MacNeil, 1989).

Holcman & Stibilj (1997) fed Rhode Island Red hens on diets containing 7.5, 15 or 30 mg As(III)/kg (as As_2O_3) for 19 days. Eggs were collected on days 8–19 day of the experiment, and arsenic residues were consistent throughout this period. Mean concentrations were respectively 0.2, 0.42 and 0.96 mg As/kg (dry weight) in egg yolk and in 0.06, 0.14 and 0.3 mg As/kg egg white for the three exposure concentrations.

Hoffman et al. (1992) fed mallard on a diet containing 200 mg As(V)/kg (as sodium arsenate) for 4 weeks. Arsenic accumulated in the liver at a concentration of 2.3 mg As/kg (wet weight). Birds maintained on a restricted protein and exposed to the same arsenic-contaminated diet accumulated 5.1 mg As/kg. Stanley et al. (1994) maintained mallards on diets containing 25, 100 or 400 mg As(V)/kg (as sodium arsenate) for 16–18 weeks. Arsenic was accumulated in a dose dependent manner; mean concentrations in adult livers were 0.49–6.6 mg As/kg (dry weight), in duckling livers from 0.65B33 mg/kg and in whole eggs from 0.46–3.6 mg/kg.

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