Sources and pathways of arsenic in the geochemical environment: health implications

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Abstract: Arsenic is ubiquitous in the environment, being present in small amounts in all rock, soil, dust, water and air. It is associated with many types of mineral deposits and in particular those containing sulphide minerals. The most common arsenic mineral is arsenopyrite, $FeAsS_2$. Elevated concentrations are sometimes found in fine grained argillaceous sediments and phosphorites. Some marine sediments may contain as much as 3000 mg kg^{-1}. Arsenic is co-precipitated with iron hydroxides and sulphides in some sedimentary rocks, and is precipitated as ferric arsenate in soil horizons rich in iron.
This paper reviews current knowledge on the natural geochemical sources of arsenic in several countries where high concentrations in soils, dusts, surface and groundwaters may present a hazard to human health. The chemistry and behaviour of arsenic within the weathering zone are discussed in relation to pathways leading to human exposure.

Arsenic is a metalloid element, but is often inaccurately referred to as a metal. It is a well known poison and as little as 0.1 g of arsenic trioxide may be lethal to humans (Jarup 1992). Acute arsenic poisoning is now rare, though chronic poisoning is widely recognized as a result of occupational exposure (WHO 1981). For more than a century this element at high doses has been known to be a human carcinogen and it is well established that ingestion of inorganic arsenic may cause skin cancer, whereas inhalation may produce respiratory cancer (Jarup 1992).

More recently there has been increasing concern as to the possible adverse health effects from exposure to elevated concentrations of arsenic in the natural geochemical environment. Scientific interest was initially stimulated by the results of an epidemiological study undertaken in southwest Taiwan in the 1960s which clearly showed a relationship between high concentrations of arsenic in drinking water and skin cancer, keratosis and Blackfoot Disease (a type of gangrene) (Tseng *et al.* 1968; Tseng 1977). Further investigations in Taiwan have established relationships between high arsenic exposure and cancers of the bladder and other internal organs (Chen *et al.* 1988, 1992).

Non-carcinogenic effects of inorganic arsenic have been reviewed by Abernathy & Ohanian (1992) and include hyperkeratosis and skin lesions, vascular effects including gangrene of the extremities in Taiwan (Tseng 1977), and vasoconstriction and acrocyanosis in Chile (Borgono *et al.* 1977). Neurological effects including tingling, numbness and pheripheral neuropathy have also been noted (Heyman *et al.* 1956). Hepatic effects have been recorded in India (Mazumder *et al.* 1988) and there is also evidence linking arsenic contamination of well waters with severe skin lesions (Das *et al.* 1994). Other health problems from chronic arsenic toxicity have been noted in Mexico, China and the Argentine.

Arsenic contamination of the environment has arisen as a result of mining and smelting activities in several countries. An example, where there is present-day concern, is in southwest England, where it has been suggested that the distribution of arsenic may possibly account for the high incidence of melanoma of the skin (Clough 1980), although, as yet no adverse health effects have been established (Thornton 1995). Arsenic enrichment in agricultural soils and pasture has also been recorded in association with lead/zinc mineralization elsewhere in Britain (Li & Thornton 1993). In the United States there is concern about the possible implications to human health of arsenic exposure from (a) drinking water in more arid areas, (b) exposure to arsenic used extensively as a pesticide on agricultural soils over the past 100 years and (c) exposure of communities in the vicinity of metal smelting and processing plants, power stations, etc.

The weight of evidence from the above studies has led the US Environmental Protection Agency (EPA) to consider reducing the permis-

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sible level of arsenic in drinking water from the present limit of $50\,\mu g\,l^{-1}$ to as low as $2–8\,\mu g\,l^{-1}$. In parallel the World Health Organisation (WHO) has revised their recommendation for arsenic in drinking water to a provisional guideline of $10\,\mu g\,l^{-1}$ in the place of the previously accepted $50\,\mu g\,l^{-1}$ (WHO 1994).

It is also important to realize that there is evidence to support the hypothesis that arsenic in small quantities is an essential nutrient. Studies by Neilsen *et al.* (1975) showed growth retardation in rats fed on an arsenic depleted diet, while Anke *et al.* (1976) showed reproductive effects, depressed growth and elevated mortality in arsenic-deprived goats and miniature pigs. There is, however, no evidence to support the concept of arsenic deficiency in the human, whose possible requirement has been calculated to be as little as $12\,\mu g$ per day (Uthus 1992). This author draws attention to the reported arsenic contents from diets from various parts of the world, indicating that the average human intake of arsenic is in the range $12–40\,\mu g$ per day, thus exceeding the possible human requirement. For example, the most recent Food and Drug Administration (FDA) Total Diet Study showed total mean As intakes by US adults to range from $20–35\,\mu g$ per day; fish and seafood accounted for 90% of the total food arsenic exposure, with all other foods the remaining 10% (FDA 1993).

In response to increasing concern, a Task Force was set up in 1993 by the Society for Environmental Geochemistry and Health, with funding from EPA and other sources, to examine health related arsenic issues. Subsequently a major international conference on Exposure and Health Effects of Arsenic was held in New Orleans in July 1993. This paper examines some of the issues raised and identifies some of the principal areas in which research is now urgently required.

Geochemistry and chemical behaviour of arsenic

Arsenic is ubiquitous in the environment, being present usually in small amounts in all rock, soil, dust, water and air. It is the main constituent of more than 200 mineral species, of which *c.* 60% are arsenates, 20% sulphides and sulphosalts and the remaining 20% include arsenides, arsenites, oxides, silicates and elemental As (Onishi 1969). Arsenic is found associated with many types of mineral deposits and in particular those containing sulphide minerals. The element is common in iron pyrite, galena, chalcopyrite, and more rarely in sphalerite (Goldschmidt 1954). Arsenic is in fact used as an indicator or pathfinder for gold in geochemical surveys. The most common arsenic mineral is arsenopyrite, $FeAsS_2$. The average concentration of As in igneous and sedimentary rocks is approximately $2\,mg\,kg^{-1}$, and common concentrations in most rocks range from $0.5–2.5\,mg\,kg^{-1}$ (Kabata-Pendias & Pendias 1984), though higher concentrations are found in finer grained argillaceous sediments and phosphorites. Arsenic is concentrated in some reducing marine sediments which may contain as much as $3000\,mg\,kg^{-1}$. In some sedimentary rocks arsenic is co-precipitated with iron hydroxides and sulphides. Thus iron deposits and sedimentary iron ores are rich in arsenic, as are manganese nodules. The arsenic contents of metamorphic rocks reflect those of the igneous and sedimentary rocks from which they were formed.

Weathering of rocks may mobilize arsenic as salts of arsenous acid and arsenic acid (Irgolic *et al.* 1995). The average concentration of arsenic in soil of about $5–6\,mg\,kg^{-1}$ is higher than that of rocks (Peterson *et al.* 1981) but will vary from region to region. Non-mineralized, uncontaminated soils usually contain $1–40\,mg\,kg^{-1}$ As. Lowest concentrations of arsenic are found in sandy soils and those derived from granites, with higher levels in alluvial soils and those rich in organic matter (Kabata-Pendias & Pendias 1984). Soils close to or derived from sulphide ore deposits may contain up to $8000\,mg\,kg^{-1}$ As (Levander 1977). High concentrations are also found in soils and groundwaters affected by geothermal activity (Reay 1972). The roasting of arsenic-containing (sulphide) ores and burning of arsenic-rich coal releases arsenic trioxide, which may react in air with basic oxides, such as alkaline earth oxides, to form arsenates (Irgolic *et al.* 1995). These inorganic arsenic compounds can then be deposited onto soils and may be leached into surface and ground waters.

Under oxidizing conditions, in aerobic environments, arsenates (containing pentavalent arsenic) are the thermodynamically stable species. Arsenic is precipitated as ferric arsenate in soil horizons rich in iron. Arsenic derived from the weathering of pyritic slates in Alberta, Canada, has been found to leach from surface soils and accumulate up to several hundred $mg\,kg^{-1}$ in the subsoil by adsorption onto secondary iron oxides (Dudas 1984). Elevated concentrations of arsenic ($8–40\,mg\,kg^{-1}$) in acid sulphate soils in Canada and New Zealand are associated with the presence of pyrite (Dudas 1987), which typically holds up to 0.5% As through lattice substitution for sulphur. Iron-rich bauxites have

been recorded containing in excess of 500 mg kg^{-1} As_2O_3. Under reducing conditions (< 200 mV) arsenites (containing trivalent arsenic) should be the predominant arsenic compounds. Because the reduction of arsenate to arsenite is slow, systems may not be at equilibrium. Inorganic arsenic compounds can be converted to methylated arsenic species by microorganisms, by plants, by animals, and by man. The oxidative methylation reactions act on trivalent arsenic compounds and produce methylarsonic acid, dimethylarsinic acid and trimethylarsine oxide (Irgolic *et al.* 1995). Under reducing conditions these pentavalent arsenic compounds can be reduced to volatile and easily oxidized methylarsines.

The chemical behaviour of arsenic in soil has been reviewed by O'Neill (1990) and Yan-Chu (1994). The ranges of Eh and pH in soils can lead to the presence of either As(V) or As(III), with microbial activity causing methylation, demethylation and/or change in oxidation state. The behaviour of arsenate (AsO_4^{3-}) resembles that of phosphate and vanadate. Arsenates of Fe and Al are the dominant phases in acid soils and are less soluble than calcium arsenate which dominates in many calcareous soils (Woolson *et al.* 1973; Fordyce *et al.* 1995). The presence of S species may, if the redox potential is low enough, favour the formation of arsenic sulphide minerals. A further complicating factor may be the presence of clay minerals, Fe, Mn and Al oxides and organic matter which can influence sorption, solubility and rate of oxidation of As species. Arsenic solubility is also controlled by adsorption reactions and biological activity.

The concentration of As in unpolluted fresh waters typically ranges from 1–10 $\mu g l^{-1}$, rising to 100–5000 $\mu g l^{-1}$ in areas of sulphide mineralization and mining (Fordyce *et al.* 1995). Studies in the Obuasi gold-mining area of Ghana showed arsenic concentrations in drinking water from streams, shallow wells and boreholes to range from < 2–175 $\mu g l^{-1}$ (Smedley *et al.* 1996). Both these sets of authors discuss the hydrogeochemistry of arsenic. In summary, at moderate or high redox potentials, As 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, the trivalent arsenite species (H_3AsO_3) predominates. It has been noted that the retention of As in solution is constrained by co-precipitation with elements such as Fe, Ba, Co, Ni, Pb and Zn (Fordyce *et al.* 1995). In Ghana the highest concentrations of arsenic were found in deeper more reducing waters where it was thought that the element had built up as a result of longer residence times of groundwater in a deeper part of the aquifer (Smedley *et al.* 1996).

In the marine environment, more complex organic arsenic compounds such as tetramethylarsonium salts, arsenocholine, arsenobetaine, dimethyl(ribosyl)arsine oxides, and arsenic-containing lipids have been identified (Irgolic *et al.* 1995). However, only a very minor fraction of the total arsenic in the oceans remains in solution in sea water, due to removal by suspended particulate material.

Geochemical mapping

Geochemical maps based on the systematic sampling and analysis of stream sediments, soils and/or waters have proved useful as a source of baseline data for arsenic distribution on a regional scale. At the same time, maps have delineated anomalous areas associated with natural arsenic enhancement in sulphide minerals and/or contamination with arsenic-rich mineral wastes or flue dusts. For example, a map showing the regional distribution of arsenic in stream sediments was published as part of the Wolfson Geochemical Atlas of England and Wales (Webb *et al.* 1978) and clearly shows extensive contamination of soils in southwest England, with in excess of 700 km^2 affected (Webb *et al.* 1978; Abrahams & Thornton 1987).

Geochemical surveys undertaken by the British Geological Survey have indicated anomalous levels of As in the southwest Highlands and the Grampian Highlands of Scotland, the English Lake District and North Wales, associated with specific geological units (Plant *et al.* 1989, 1991; Simpson *et al.* 1993). These identify all the known gold mineralization, and are also associated with Cu dominated multi-element mineralization and complex Pb–As mineralization, together with environments in which As has been complexed and absorbed in stream sediments by organic matter and hydrous oxides in areas of impeded drainage. It may be concluded that arsenic in stream sediments occurs mainly as As_2O_3 and As_2S_3, as heavy metal arsenates, and sorbed onto ferric hydroxides.

Collaborative geochemical and hydrogeochemical studies into mining-related arsenic contamination have been undertaken in the Ron Phibun District of Thailand by the British Geological Survey and the Government of Thailand Department of Mineral Resources (Fordyce *et al.* 1995). Here the sources of arsenic are high-grade arsenopyrite waste piles and alluvial mineral deposits. Alluvial soils contained up to 5000 mg g^{-1} As. It was found that

the waste materials contained only a small component of the primary arsenopyrite, with most of the arsenic present as secondary arsenate minerals, in particular the relatively insoluble scorodite ($FeAsO_4.2H_2O$). Concentrations of arsenic ranged up to in excess of 5000 $\mu g l^{-1}$ in shallow wells used for drinking water, while water from deeper boreholes was much less contaminated.

Geochemical mapping of Finland has clearly shown anomalies in the south of the country relating to sulphide mineralization and other geological features (Koljonen *et al.* 1992), and draws attention to possible sources of arsenic enrichment in well waters and potential increased human exposure (I. Niinisto pers. comm.).

Systematic sampling of soils and dusts in and around the ancient lead mining and smelting site at Lavrion, Greece, has indicated extensive contamination with arsenic as well as lead, and has instigated present-day studies into possible health implications to the local community (Stavrakis *et al.* 1994).

Sources of arsenic leading to human exposure

The presence of arsenic in sulphide mineral deposits has been noted above and associated mining and smelting activities have led to high concentrations ranging up to 1000 mg kg^{-1} or more of this element in both agricultural and garden soils and house dusts in old mining areas of Cornwall and Greece (Colbourne *et al.* 1975; Abrahams & Thornton 1987; Stavrakis *et al.* 1994). A further study of arsenic in surface waters in Cornwall has shown soluble arsenic in specific catchments to range from 10–50 $\mu g l^{-1}$ (Aston *et al.* 1975). However, abstraction of surface waters for processing and distribution avoids waters contaminated by past mining activities and water processing using aluminium hydroxide removes the majority of soluble arsenic. As a result arsenic in drinking water in Cornwall rarely exceeds 10 $\mu g l^{-1}$ (MAFF 1982). There are, however, some 20–30 000 private suppliers of water in south west England for which few data are available and on which further study would be advisable.

The sources of arsenic in tube wells in India and in well waters in Arizona and California are again geological, though the natures of the arsenic enriched deposits are as yet unknown. It is probable that these are sulphur- and/or iron-rich deposits of sedimentary origin or fine grained argillaceous marine deposits in which arsenic from biogenic sources has been concentrated. There is also a possibility that arsenic has been derived as a result of geothermal activity.

It has been estimated that agricultural soils in the USA contaminated with pesticide residues used over the past 100 or so years, with the use of wood preservatives, and with the current use of arsenic acid as a cotton defoliant may extend to as much as 100 000 to 1 million hectares exceeding 200 mg kg^{-1} As, with 10 million hectares with 20–30 mg kg^{-1} (Chaney pers. comm.). The extent of similar contamination in the UK and elsewhere in Europe, and indeed on a worldwide basis is difficult to estimate but is likely to be considerable.

Other anthropogenic sources of arsenic include metal processing plants, chemical works, coal combustion and geothermal power plants. In Poland sources of arsenic have been attributed to metal smelting, coal burning and use of As-rich phosphatic fertilizers (Kabata-Pendias 1994). The phytotoxicity of arsenic added to soil depends on soil type, with 90% growth reduction recorded at 1000 mg kg^{-1} on heavy soils and at 100 mg kg^{-1} on light soils (Woolson *et al.* 1973).

Exposure pathways

The main routes of environmental as opposed to industrial exposure to arsenic result from the ingestion of contaminated drinking waters, foodstuffs, soil and dust.

Taiwan

Exposure has been calculated only from the consumption of arsenic-rich drinking water. Sixty-five thousand artesian wells were sampled in the early 1970s in which concentrations of arsenic in waters ranged up to 600 $\mu g l^{-1}$ As. Unfortunately the majority of the wells were only sampled on one occasion and it is not known whether waters were filtered or not prior to analysis. The analytical method used, based on a mercury bromide stain, was probably not applicable for concentrations less than 100 $\mu g l^{-1}$ thus rendering many of the analytical results suspect (K. T. Irgolic, pers. comm.). Estimation of human exposure has been based on an assumption of 3–4.5 l consumed per day. However, it is realized that in a hot climate this could have been much greater, perhaps up to 8–10 l per day. Thus the dose may have been greatly underestimated. Exposure to arsenic in food, ingested soil/dust and by dermal contact was not taken into consideration in the overall estimate of human exposure.

Speciation of arsenic in the Taiwan well-waters showed an approximate ratio of As^{3+}: As^{5+} of 3 : 1, though this is known to change on storage and transport due to the oxidation of the former to the latter.

India: west Bengal

Drinking water from within an area of 35 000 km^2 along the River Ganga has been found to exceed 50 $\mu g l^{-1}$ in several thousand tube-wells, ranging up to 200/400 $\mu g l^{-1}$ (Das *et al.* 1994). Groundwater depletion as a result of irrigation requirements, brought about by the green revolution, has led to increasing arsenic concentrations in well waters. The reason for this has not yet been clearly established, though one possibility is that arsenic-rich pyritic beds have been exposed due to a lowering of the water table and oxidation has led to the leaching of arsenic from these beds into the well-water. There is also the possibility that the use of arsenic-rich irrigation water will have resulted in the accumulation of arsenic in surface soils used for the production of food crops. However, total arsenic exposure in local populations from both drinking water and food has not yet been determined.

Southwest England

Of 9000 km surveyed in Devon and Cornwall, 1.3% of surface soils have been described as highly contaminated with arsenic (exceeding the 90th percentile of 190 $\mu g g^{-1}$ As), and 6.6% as moderately contaminated (exceeding the 70th percentile, ranging from 110–190 $\mu g g^{-1}$ As; Abrahams & Thornton 1987). However, arsenic sometimes ranges as high as 0.1–1% or more in surface soils near old roasting ovens and smelter stacks.

Possible exposure of local populations through the consumption of dairy products and locally grown food crops has been examined. Arsenic uptake into pasture grass was found to be relatively small (it has been suggested by Kabata-Pendias & Pendias (1984) that plants take up arsenic passively with water flow), with a maximum concentration of 9.6 $\mu g g^{-1}$ As dry matter in washed grass growing on soil containing 1000 $\mu g g^{-1}$ As or more (Thoresby & Thornton 1979). However, it was subsequently shown that the majority of arsenic intake by grazing cattle resulted from the accidental ingestion of soil along with grass. Arsenic ingested in soil, determined in 11 herds of cattle, was found to range from *c.* 50–80% of the total arsenic intake (Thornton & Abrahams 1983). On moderately contaminated land, in which arsenic ranged from 160-250 $\mu g g^{-1}$, this resulted in a mean total daily intake of *c.* 50 mg arsenic.

Arsenic uptake into vegetable crops was studied in household gardens with soils ranging from around 150–900 $\mu g g^{-1}$ As (Xu & Thornton 1985). Arsenic concentrations in each of the six crops tested increased with the arsenic content of the soil, though only in lettuce did this exceed 1 $\mu g g^{-1}$ dry matter. Arsenic uptake was influenced by the iron and phosphorus content of the soil. All the vegetable samples fell below the UK statutory limit for arsenic in foods offered for sale of 1 $mg kg^{-1}$ As freshweight (Arsenic in Food Regulations 1959), and it is not thought that exposure through locally grown foods is in any way hazardous.

It is now considered that accidental ingestion of contaminated dust and soil is the main route of exposure to arsenic in this situation, and that this will be particularly important for the young child of 3–36 months of age. A recent study in several old Cornish mining villages showed a significant relationship between arsenic levels in garden soils and in house dust (Elgali 1994). One study based on 70 households in the Camborne–Hayle area of Cornwall indicated that a young child could ingest as much as 42 μg of arsenic per day by this route (Johnson 1983). This was assuming that a 2 year-old child ingests 100 μg dust per day. A further study of 23 households in old mining villages, in which arsenic in soils ranged up to 770 $\mu g g^{-1}$ As, and in housedusts up to 460 $\mu g g^{-1}$, showed amounts of arsenic on children's hands ranging up to 3.5 μg As. Total arsenic intake by hand-to-mouth activity was then estimated at between 35 and 46 μg arsenic per day, though it was accepted that this estimate was at the best semi-quantitative and that further study was required (Harding 1993). It is possible, however, that a young child in this heavily contaminated environment could have an arsenic intake exceeding the WHO Provisional Tolerable Daily Intake for inorganic arsenic of 2 $\mu g kg^{-1}$ body weight (WHO 1983). A recent pilot study showed raised levels of arsenic in dust removed from children's hands by wet-wipes and in the hair of children sampled from old mining villages compared with a control location (Elgali 1994). However, a preliminary study of inorganic arsenic and its methylated metabolites in urine from adults and children from this Cornish mining area showed only slightly elevated urinary arsenic contents (Johnson & Farmer 1989).

Assessment of exposure and risk

To date, much of the concern to human exposure to arsenic has been confined to amounts of inorganic arsenic consumed in drinking water. In a recent review, Warner-North (1992) draws attention to some of the problems inherent in the current approach to risk assessment used by USEPA which has resulted in high risk estimates for skin cancer from drinking water containing arsenic even at the present Maximum Contaminant Level (MCL) of $50\,\mu g\,l^{-1}$, which is based on systemic effects and potential carcinogenicity. This approach is based on the 'default' methodology from the Guidelines for Carcinogen Risk Assessment (USEPA 1986) and assumes a linear dose–response relationship. There is, however, clear evidence for non-linearity for the association for arsenic exposure and cancer of internal organs, and the EPA approach does not reflect the increasing evidence indicating either a threshold or sub-linear dose–response relationship for low doses of arsenic (Marcus & Rispin 1988; Petito & Beck 1990). The detoxification pathway for arsenic in the human body is one of methylation in which ingested inorganic arsenic species which are potentially toxic are converted into organic non-toxic forms. It has been proposed that an explanation for the sub-linear dose–response relationship is due to the saturation of this methylation process (USEPA 1989; Carlson-Lynch *et al.* 1994). However, the methylation threshold hypothesis for the toxicity of inorganic arsenic is not accepted by all research workers (Hopenhayn-Rich *et al.* 1993), and was not even considered by Smith *et al.* (1992) in estimating potential risks from low-level arsenic exposures typical of the US population.

It is noted that EPA is currently addressing the potential status of arsenic regulation development and is considering a potential range of MCL options from $1\,\mu g\,l^{-1}$ to $20\,\mu g\,l^{-1}$ (Shank-Givens & Auerbach 1993).

Patel (1994) has listed relevant factors to risk assessment for arsenic as (a) speciation, particularly as inorganic As is considered to be more toxic than organic; (b) exposure, including non-water sources; (c) metabolism and efficiency of detoxification by methylation; (d) genotoxicity; (e) the results of carcinogenicity studies in animals which may not be predictive of arsenic toxicity in humans; and (f) the results of epidemiological studies in Taiwan, etc. Assuming either a threshold dose or a non-threshold dose for arsenic carcinogenicity, Patel (1994) lists risk estimates derived from several studies for concentrations of arsenic in drinking water ranging from $10–20\,\mu g\,l^{-1}$, and concludes that risk assessment for arsenic in drinking water involves a lot of uncertainty.

Sage (1994) has reviewed risk assessment procedures used by WHO and within the UK for As in drinking water, contaminated land and waters. UK government departments apply quantitative risk assessments by WHO and the International Agency for Research on Cancer, who have developed practical guidelines that form the foundation for EU directives and in the interpretation of UK law. Sage (1994) draws attention to a range of areas requiring further consideration:

(i) improving the sensitivity of epidemiological studies;
(ii) creating accurate measurement techniques for individual species of arsenic at low concentrations;
(iii) the adequate quantification of all exposure routes;
(iv) obtaining bioavailability data for arsenic to plants and humans;
(v) additional research into mechanisms by which arsenic acts in the body, especially synergistic and antagonistic effects;
(vi) constructing representative models for responses at low doses;
(vii) methods to express clearly remaining uncertainties in a form which can be systematically built into risk management.

None of the risk assessment procedures for arsenic applied to date has taken into account total exposure from ingestion of diet and water, ingestion of soil and dust and inhalation of atmospheric particles.

Research requirements

The above sections review current information on the sources, chemical behaviour, exposure pathways and risk assessment to human health of arsenic in the environment. There are clearly many gaps in present day knowledge that must be filled if we are to improve our understanding of the dangers that environmental arsenic poses to human health. Priority research requirements are as follows.

Geochemistry. While it is accepted that most sources of arsenic in waters are of geological origin, there is as yet little clear understanding of the geochemical nature of As-enriched strata nor of the mineral and chemical forms of arsenic present. In the course of chemical weathering in

the surface environment, arsenic from sulphide minerals and from anthropogenic sources will, with time, interact with chemical, physical and biological components of the lithosphere. Dispersion by wind and water will in due course lead to accumulation of arsenic in river and estuarine sediments and in inshore environments. Again, little is known of the mineral and chemical forms of arsenic in the soil, sediment and marine environments. Mobility of arsenic from waste materials and contaminated soils is to be expected under oxidizing conditions and perhaps this will be accelerated as a result of acid precipitation and environmental change. Mobility will, however, be reduced by the formation of secondary minerals, such as scorodite, which have low solubilities. There is a need for research into the processes controlling solubilization and migration pathways through the soil–rock system and the implications to contamination of ground and surface waters. Drainage from disused metalliferous and coal mines is frequently a source of both soluble and particulate arsenic in surface river systems. There is a need for research into the chemical and physical factors controlling downstream dispersion in soluble, suspended particulate and sediment phases and interactions with iron, manganese and other chemical species, and processes leading to accumulation of arsenic in sediment 'sinks'.

Exposure pathways. Human exposure is thought to be mainly through the digestion route, via the diet (mainly seafood) and drinking water. In specific situations this exposure may be added to through the inhalation of atmospheric particulates derived from industrial emissions or from suspended arsenic-rich soils and waste materials. The majority of studies to date have focused on the most obvious exposure pathway, i.e. water in Taiwan and India. It is now necessary to reassess total arsenic intakes of exposed communities, taking into account both inorganic and organic forms of arsenic in the diet, soluble and particulate arsenic in drinking water (and its speciation), together with involuntary ingestion of arsenic-rich dusts and soils by hand-to-mouth activity in young children and through atmospheric contamination of food stuffs.

Risk assessment. Current risk assessment strategies have all been based on relationships established between epidemiological studies in Taiwan and arsenic concentrations in drinking water. It is now thought that some of the environmental measurements in this study are flawed due to sampling and analytical errors. There is now an urgent need to establish a new multi-disciplinary study incorporating reliable environmental measurements, assessment of total human exposure from ingestion and inhalation routes, and carefully planned epidemiological investigations. Such a study would form the basis for the development of a risk assessment study in which uncertainty was minimized.

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