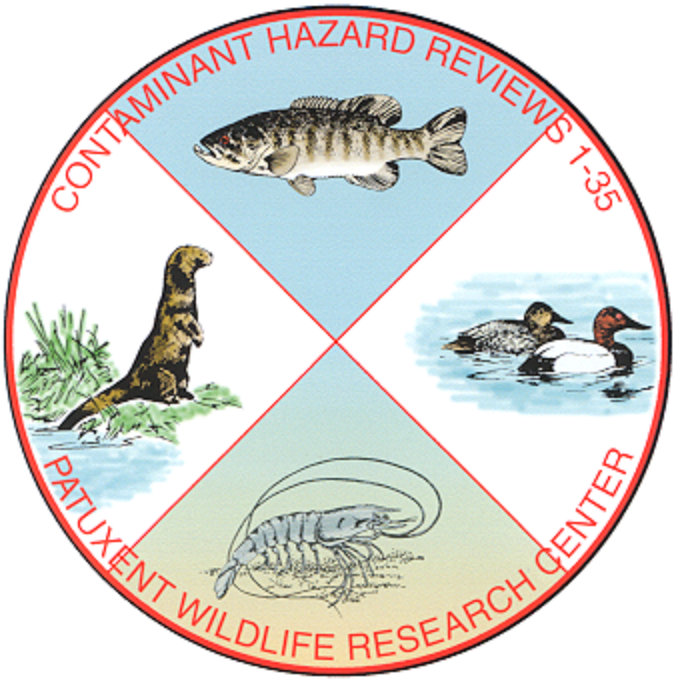
Biological Report 85(1.12)
January 1988

Contaminant Hazard Reviews
Report No. 12



ARSENIC HAZARDS TO FISH, WILDLIFE, AND INVERTEBRATES: A SYNOPTIC REVIEW

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SUMMARY

Arsenic (As) is a relatively common element that occurs in air, water, soil, and all living tissues. It ranks 20th in abundance in the earth's crust, 14th in seawater, and 12th in the human body.

Arsenic is a teratogen and carcinogen that can traverse placental barriers and produce fetal death and malformations in many species of mammals. Although it is carcinogenic in humans, evidence for arsenic-induced carcinogenicity in other mammals is scarce. Paradoxically, evidence is accumulating that arsenic is nutritionally essential or beneficial. Arsenic deficiency effects, such as poor growth, reduced survival, and inhibited reproduction, have been recorded in mammals fed diets containing <0.05 mg As/kg, but not in those fed diets with 0.35 mg As/kg. At comparatively low doses, arsenic stimulates growth and development in various species of plants and animals.

Most arsenic produced domestically is used in the manufacture of agricultural products such as insecticides, herbicides, fungicides, algicides, wood preservatives, and growth stimulants for plants and animals. Living resources are exposed to arsenic by way of atmospheric emissions from smelters, coal-fired power plants, and arsenical herbicide sprays; from water contaminated by mine tailings, smelter wastes, and natural mineralization; and from diet, especially from consumption of marine biota. Arsenic concentrations are usually low (<1.0 mg/kg fresh weight) in most living organisms but are elevated in marine biota (in which arsenic occurs as arsenobetaine and poses little risk to organisms or their consumer) and in plants and animals from areas that are naturally arseniferous or are near industrial manufacturers and agricultural users of arsenicals. Arsenic is bioconcentrated by organisms, but is not biomagnified in the food chain.

Arsenic exists in four oxidation states, as inorganic or organic forms. Its bioavailability and toxic properties are significantly modified by numerous biological and abiotic factors that include the physical and chemical forms of arsenic tested, the route of administration, the dose, and the species of animal. In general, inorganic arsenic compounds are more toxic than organic compounds, and trivalent species are more toxic than pentavalent species. Arsenic may be absorbed by ingestion, inhalation, or through permeation of skin or mucous membranes; cells take up arsenic through an active transport system normally used in phosphate transport. The mechanisms of arsenic toxicity differ greatly among chemical species, although all appear to cause similar signs of poisoning. Biomethylation is the preferred detoxification mechanism for absorbed inorganic arsenicals; methylated arsenicals usually clear from tissues within a few days.

Episodes of arsenic poisoning are ether acute or subacute; chronic cases of arsenosis are seldom encountered in any species except man. Single oral doses of arsenicals fatal to 50% of sensitive species tested ranged from 17 to 48 mg/kg body weight (BW) in birds and from 2.5 to 33 mg/kg BW in mammals. Susceptible species of mammals were adversely affected at chronic doses of 1 to 10 mg As/kg BW, or 50 mg As/kg diet. Sensitive aquatic species were damaged at water concentrations of 19 to 48 ug As/l (the U.S. Environmental Protection Agency drinking water criterion for human health protection is 50 ug/l), 120 mg As/kg diet, or (in the case of freshwater fish) tissue residues >1.3 mg/kg fresh weight. Adverse effects to crops and vegetation were recorded at 3 to 28 mg of water soluble As/l (equivalent to about 25 to 85 mg total As/kg soil) and at atmospheric concentrations >3.9 ug As/m^3.

Numerous and disparate arsenic criteria have been proposed for the protection of sensitive natural resources; however, the general consensus is that many of these criteria are inadequate and that additional information is needed in at least five categories: (1) developing standardized procedures to permit correlation of biologically observable effects with suitable chemical forms of arsenic; (2) conducting studies under controlled conditions with appropriate aquatic and terrestrial indicator organisms to determine the effects of chronic exposure to low doses of inorganic and organic arsenicals on reproduction, genetic makeup, adaptation, disease resistance, growth, and other variables (3) measuring interaction effects of arsenic with other common environmental contaminants, including carcinogens, cocarcinogens, and promoting agents; (4) monitoring the incidence of cancer and other abnormalities in natural resources from areas with relatively high arsenic levels, and correlating these with the possible carcinogenicity of arsenic compounds; and (5) developing appropriate models of arsenic cycling and budgets in natural ecosystems.

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Suggested citation for this report:

Eisler, R. 1988. Arsenic hazards to fish, wildlife, and invertebrates: a synoptic review. U.S. Fish Wildl. Serv. Biol. Rep. 85(1.12).

TABLES

ACKNOWLEDGMENTS

I thank Nancy A. Bushby, Lynda J. Garrett, and Joyce E. Haber for literature search and retrieval; Julia L. Armstrong for secretarial help; Matthew C. Perry, Charles J. Stafford, Donald H. White, and Laverne Cleveland for technical and scientific review; and Paul H. Eschmeyer and James R. Zuboy for editorial services.

INTRODUCTION

Anxiety over arsenic (As) is understandable, and frequently justifiable. Arsenic compounds were the preferred homicidal and suicidal agents during the Middle Ages, and arsenicals have been regarded largely in terms of their poisonous characteristics in the nonscientific literature (NAS 1977). Data collected on animals, including man, indicate that inorganic arsenic can cross the placenta and produce mutagenic, teratogenic, and carcinogenic effects in offspring (Nagymajtenyi et al. 1985). Correlations between elevated atmospheric arsenic levels and mortalities from cancer, bronchitis, and pneumonia were established in an epidemiological study in England and Wales, where deaths from respiratory cancer were increased at air concentrations >3 ug As/m^3 (NRCC 1978). Chronic arsenical poisoning, including skin cancer and a gangrenous condition of the hand and feet called Blackfoot's disease, has occurred in people from several communities in Europe, South America, and Taiwan that were exposed to elevated concentrations of arsenic in drinking water (EPA 1980). More recently, about 12,000 Japanese infants were poisoned (128 deaths) after consuming dry milk containing 15 to 24 mg inorganic As/kg, which originated from contaminated sodium phosphate used as a milk stabilizer. Fifteen years after exposure, the survivors sustained an elevated frequency of severe hearing loss and brain wave abnormalities Pershagen and Vahter 1979).

Many reviews on ecotoxicological aspects of arsenic in the environment are available; particularly useful are those by Woolson (1975), NAS (1977), NRCC (1978), Pershagen and Vahter (1979), EPA (1980, 1985), Hood (1985), and Andreas (1986). These authorities agree on six points. 1. Arsenic is a relatively common element, and is present in air, water, soil, plants, and in all living tissues. 2. Arsenicals have been used in medicine as chemotherapeutics since 400 BC, and organoarsenicals were used extensively for this purpose until about 1945, with no serious effects when judiciously administered. 3. Large quantities of arsenicals are released into the environment as a result of industrial and especially agricultural activities, and these may pose potent ecological dangers. 4. Exposure of humans and wildlife to arsenic may occur through air (emissions from smelters, coal-fired power plants, herbicide sprays), water (mine tailings runoff, smelter wastes, natural mineralization), and food (especially seafoods). 5. Chronic exposure to arsenicals by way of the air, diet, and other routes have been associated with liver, kidney, and heart damage hearing loss; brain wave abnormalities; and impaired resistance to viral infections. And 6. Exposure to arsenic has been associated with different types of human cancers such as respiratory cancers and epidermoid carcinomas of the skin, as well as precancerous dermal keratosis. Only recently (Deknudt et al. 1986) has the epidemiological evidence of human carcinogenicity been confirmed by carcinogenesis in experimental animals.

This report was prepared in response to requests for information from environmental contaminant specialists of the U. S. Fish and Wildlife Service. It is part of a continuing series of reviews on chemical pollutants and natural resources.

SOURCES, FATE, AND USES

Global production of arsenic is estimated to be 75,000 to 100,000 tons annually, of which the United States produces about 21,000 tons and uses about 44,000 tons; major quantities are imported from Sweden, the world's leading producer (NAS 1977; EPA 1980). Almost all (97%) of the arsenic made worldwide enters end-product manufacture in the form of arsenic trioxide (As_2O_3), and the rest is used as additives in producing special lead and copper alloys (NAS 1977). More than 80% of the As_2O_3 is used to manufacture products with agricultural application, such as insecticides, herbicides, fungicides, algicides, sheep dips, wood preservatives, dyestuffs, and the medicines for eradication of tapeworm in sheep and cattle (NAS 1977). The sole producer and refiner of As_2O_3 in the United States is a copper smelter in Tacoma, Washington (NAS 1977).

Arsenic naturally occurs as sulfides and as complex sulfides of iron, nickel, and cobalt (Woolson 1975). In one form or another, arsenic is present in rocks, soils, water, and living organisms at concentrations of parts per billion to parts per million (NAS 1977). Soil arsenic levels are normally elevated near arseniferous deposits, and in mineralized zones containing gold, silver, and sulfides of lead and zinc (Dudas 1984). Secondary iron oxides formed from the weathering of pyrite act as scavengers of arsenic (Dudas 1984). Pyrite is a known carrier of arsenic and may contain up to 5,600 mg/kg; for example, total arsenic is 10X above normal background levels in soils derived from pyritic shale (Dudas 1984). Natural weathering of rocks and soils adds about 40,000 tons of arsenic to the oceans yearly, accounting for <0.01 mg/l input to water on a global basis (NRCC 1978). Many species of marine plants and animals often contain naturally high concentrations of arsenic (NAS 1977), but it is

usually present in a harmless organic form (Woolson 1975). Anthropogenic input of arsenic to the environment is substantial, and exceeds that contributed by natural weathering processes by a factor of about 3X (NRCC 1978).

The most important concept with respect to arsenic cycling in the environment is constant change. Arsenic is ubiquitous in living tissue and is constantly being oxidized, reduced, or otherwise metabolized. In soils, insoluble or slightly soluble arsenic compounds are constantly being resolubilized, and the arsenic is being presented for plant uptake or reduction by organisms and chemical processes. Man reportedly has modified the arsenic cycle only by causing localized high concentrations (NAS 1977). The speciation of arsenic in the environment. is affected partly by indiscriminate biological uptake, which consumes about 20% of the dissolved arsenate pool and results in measurable concentrations of reduced and methylated arsenic species. The overall arsenic cycle is similar to the phosphate cycle; however, regeneration time for arsenic is much slower--on the order of several months (Sanders 1980). The ubiquity of arsenic in the environment is evidence of the redistribution processes that have been operating since early geologic time (Woolson 1975). A prehuman steady state solution to the global arsenic cycle (Austin and Millward 1984) indicates that major reservoirs of arsenic (in kilotons) are magma (50 billion), sediments (25 billion), oceanic deep waters (1.56 million), land (1.4 million), and ocean mixed layers (270,000); minor amounts occur in ocean particulates (100), and in continental (2.5) and marine tropospheres (0.069). Arsenic is significantly mobilized from the land to the troposphere by both natural and anthropogenic processes. Industrial emissions account for about 30% of the present day burden of arsenic in the troposphere (Austin and Millward 1984). Agronomic ecosystems, for example, may receive arsenic from agricultural sources such as organic herbicides, irrigation waters, and fertilizers, and from such nonagricultural sources as fossil fuels and industrial and municipal wastes (Woolson 1975). Arsenic is mobile and nonaccumulative in air, pant, and water phases of agronomic ecosystems; arsenicals sometimes accumulate in soils, but redistribution mechanisms usually preclude hazardous accumulations (Woolson 1975).

Arsenic compounds have been used in medicine since the time of Hippocrates, ca. 400 BC (Woolson 1975). Inorganic arsenicals have been used for centuries, and organoarsenicals for at least a century in the treatment of syphylis, yaws, amoebic dysentery, and trypanosomiasis (NAS 1977). During the period 1200 to 1650, however, arsenic was used extensively in homicides (NRCC 1978). In 1815, the first accidental death was reported from arsine (AsH_3) poisoning, and in 1900-1903 accidental poisonings from consumption of arsenic-contaminated beer were widely reported (NRCC 1978). In 1938, it was established that arsenic can counteract selenium toxicity (NRCC 1978). The introduction of arsphenamine, an organoarsenical, to control venereal disease earlier this century gave rise to intensive research by organic chemists, which resulted in the synthesis of at least 32,000 arsenic compounds. But the advent of penicillin and other newer drugs nearly eliminated the use of organic arsenicals as human therapeutic agents (EPA 1980). Arsenical drugs are still used in treating certain tropical diseases, such as African sleeping sickness and amoebic dysentery, and are used in veterinary medicine to treat parasitic diseases, including filariasis in dogs (*Canis familiaris*) and blackhead in turkeys (*Meleagris gallopavo*) and chickens, *Gallus* spp. (NAS 1977). Today, abnormal sources of arsenic that can enter the food chain from plants or animals include arsenical pesticides such as lead arsenate; arsenic acid, $HAsO_3$; sodium arsenite, $NaAsO_2$; sodium arsenate, Na_2AsO_4; and cacodylic acid, $(CH_3)_2$ As(OH) (NAS 1977).

The major uses of arsenic are in the production of herbicides, insecticides, desiccants, wood preservatives, and growth stimulants for plants and animals. Much smaller amounts are used in the manufacture of glass (nearly all of which contains 0.2% to 1.0% arsenic as an additive--primarily as a decolorizing agent) and textiles, and in medical and veterinary applications (NAS 1977; EPA 1980). Arsenic is also an ingredient in lewisite, a blistering poison gas developed (but not used) during World War I, and in various police riot control agents (NAS 1977). The availability of arsenic in certain local areas has been increased by various human activities: smelting and refining of gold, silver, copper, zinc, uranium, and lead ores; combustion of fossil fuels, such as coal and gasoline; burning of vegetation from cotton gins treated with arsenical pesticides; careless or extensive use of arsenical herbicides, pesticides, and defoliants; dumping of land wastes and sewage sludge (1.1 mg/l) in areas that allow leaching into groundwater; use of domestic detergents in wash water (2.5 to 1,000 mg As/1); manufacture of glass; and by the sinking of drinking water wells into naturally arseniferous rock (NRCC 1978; EPA 1980). There are several major anthropogenic sources of environmental arsenic contamination: industrial smelters--the effluent from a copper smelter in Tacoma, Washington, contained up to 70 tons of arsenic discharged yearly into nearby Puget Sound (NRCC 1978); coal-fired power plants, which collectively emit about

3,000 tons of arsenic annually in the United States (EPA 1980); and production and use of arsenical pesticides, coupled with careless disposal of used pesticide containers (NAS 1977). Elevated levels of arsenic have been reported in soils near smelters, in acid mine spoils, and in orchards receiving heavy applications of lead arsenate (NAS 1977; Dudas 1984). Air concentrations of arsenic are elevated near metal smelters, near sources of coal burning, and wherever arsenical pesticides are applied (NAS 1977). Atmospheric deposition of arsenic has steadily increased for at least 30 years, as judged by sedimentary evidence from lakes in upstate New York (Smith et al. 1987). Arsenic is introduced into the aquatic environment through atmospheric deposition of combustion products and through runoff from fly-ash storage areas near power plants and nonferrous smelters (Smith et al. 1987). Elevated arsenic concentrations in water were recorded near mining operations, and from mineral springs and other natural waters--usually alkaline and with high sodium and bicarbonate contents (NAS 1977). In the United States, the most widespread and frequent increases in dissolved arsenic concentrations in river waters were in the northern Midwest; all evidence suggests that increased atmospheric deposition of fossil fuel combustion products was the predominant cause of the trend (Smith et al. 1987).

Agricultural applications provide the largest anthropogenic source of arsenic in the environment (Woolson 1975). Inorganic arsenicals (arsenic trioxide; arsenic acid; arsenates of calcium, copper, lead, and sodium; and arsenites of sodium and potassium) have been used widely for centuries as insecticides, herbicides, algicides, and desiccants. Paris green (cuprous arsenite) was successfully used in 1867 to control the Colorado potato beetle (*Leptinotarsa decemlineata*) in the eastern United States. Arsenic trioxide has been applied widely as a soil sterilant. Sodium arsenite has been used for aquatic weed control, as a defoliant to kill potato vines before tuber harvest, as a weed killer along roadsides and railroad rights-of-way, and for control of crabgrass (*Digitaria sanguinalis*). Calcium arsenates have been applied to cotton and tobacco fields to protect against the boll weevil (*Anthonomus grandis*) and other insects, Lead arsenate has been used to control insect pests of fruit trees, and for many years was the only insecticide that controlled the codling moth (*Carpocapsa pomonella*) in apple orchards and the horn worm larva (Sphyngidae) on tobacco. Much smaller quantities of lead arsenate are now used in orchards because fruit growers rely primarily on carbamate and organophosphorus compounds to control insect pests; however, lead arsenate is still being used by some growers to protect orchards from certain chewing insects. The use of inorganic arsenicals has decreased in recent years due to the banning of sodium arsenite and some other arsenicals for most purposes, although they continue to be used on golf greens and fairways in certain areas to control annual bluegrass (*Poa annua*). In recent decades, inorganic arsenicals have been replaced by organoarsenicals for herbicidal application, and by carbamate and organophosphorus compounds for insect control (Woolson 1975). By the mid-1950's, organoarsenicals were used extensively as desiccants, defoliants, and herbicides (NRCC 1978). Organoarsenicals marketed in agriculture today, which are used primarily for herbicidal application, include cacodylic acid (also known as dimethylarsinic acid) and its salts--monosodium and disodium methanearsonate (Woolson 1975; NAS 1977). Organoarsenicals are used as selective herbicides for weedy grasses in turf, and around cotton and noncrop areas for weed control; at least 1.8 million ha (4.4 million acres) have been treated with more than 8,000 tons of organoarsenicals (NAS 1977). In 1945, it was discovered that one organoarsenical (3-nitro-4 hydroxyphenyl arsonic acid) controlled coccidiosis and promoted growth in domestic chicken (Woolson 1975). Since that time, other substituted phenylarsonic acids have been shown to have both therapeutic and growth promoting properties as feed additives for poultry and swine (*Sus* spp.), and are used for this purpose today under existing regulations (Woolson 1975; NAS 1977)--although the use of arsenicals in poultry food was banned in France in 1959 (NRCC 1978).

CHEMICAL AND BIOCHEMICAL PROPERTIES

Elemental arsenic is a gray, crystalline material characterized by atomic number 33, atomic weight of 74.92, density of 5.727, melting point of 817 °C, sublimation at 613 °C, and chemical properties similar to those of phosphorus (Woolson 1975; NAS 1977; NRCC 1978; EPA 1980; 1985). Arsenic has four valence states: -3, 0, +3, and +5. Arsines and methylarsines, which are characteristic of arsenic in the -3 oxidation state, are generally unstable in air. Elemental arsenic, As° is formed by the reduction of arsenic oxides. Arsenic trioxide (As+3) is a product of smelting operations and is the material used in synthesizing most arsenicals. It is oxidized catalytically or by bacteria to arsenic pentoxide (As+5) or orthoarsenic acid (H_3AsO_4). Arsenic in nature is rarely in its free state. Usually, it is a component of sulfidic ores, occurring as arsenide; and arsenates, along with arsenic trioxide, which is a weathering product of arsenides. Most arsenicals degrade or weather to form arsenate, although arsenite may form under anaerobic conditions. Biotransformations may occur, resulting in volatile arsenicals that normally are returned to the land where soil adsorption, plant uptake, erosion,

leaching, reduction to arsines, and other processes occur. This natural arsenic cycle reflects a constant shifting of arsenic between environmental compartments.

Arsenic species in flooded soils and water are subject to chemically and microbiologically mediated oxidation or reduction and methylation reactions. At high Eh values (i.e., high oxidation-reduction potential) typical of those encountered in oxygenated waters, pentavalent As+5 tends to exist as H_3AsO_4 H_2AsO_4, $HAsO_2$, and AsO_4^{-3}. At lower Eh, the corresponding trivalent arsenic species can be present, as well as AsS_2, (Thanabalasingam and Pickering 1986). In aerobic soils, the dominant arsenic, species was As+5, and small quantities of arsenite and monomethylarsonic acid were present in mineralized areas; in anaerobic soils, As+3 was the major soluble species (Haswell et al. 1985). Inorganic arsenic is more mobile than organic arsenic, and thus poses greater problems by leaching into surface waters and groundwater (NRCC 1978). The trivalent arsenic species are generally considered to be more toxic, more soluble, and more mobile than As+5 species (Thanabalasingam and Pickering 1986). Soil microorganisms metabolize arsenic into volatile arsine derivatives. Depending on conditions, 17% to 60% of the total arsenic present in soil may be volatilized (NRCC 1978) Estimates of the half-life of arsenic in soil varied from 6.5 years for arsenic trioxide to 16 years for lead arsenate (NRCC 1978).

In water, arsenic occurs in both inorganic and organic forms, and in dissolved and gaseous states (EPA 1980). The form of arsenic in water depends on Eh, pH, organic content, suspended solids, dissolved oxygen, and other variables (EPA 1985). Arsenic in water exists primarily as a dissolved ionic species; particulates account for less than 1% of the total measurable arsenic (Maher 1985a). Arsenic is rarely found in water in the elemental state (0), and is found in the -3 state only at extremely low Eh values (Lima et al. 1984). Common forms of arsenic encountered in water are arsenate, arsenite, methanearsonic acid, and dimethylarsinic acid (EPA 1985). The formation of inorganic pentavalent arsenic, the most common species in water, is favored under conditions of high dissolved oxygen, basic pH, high Eh, and reduced content of organic material; reverse conditions usually favor the formation of arsenites and arsenic sulfides (NRCC 1978; Pershagen and Vahter 1979; EPA 1980), although some arsenite is attributed to biological activity (Maher 1985a). Water temperature seems to affect arsenic species composition in the estuary of the River Beaulieu in the United Kingdom, where reduced and methylated species predominate during warmer months and inorganic As during colder months; the appearance of methylated arsenicals during the warmer months is attributed both to bacterial and abiotic release from decaying plankton and to grazing by zooplankton (Howard et al. 1984). Also contributing to higher water or mobile levels are the natural levels of polyvalent anions, especially phosphate species. Phosphate, for example, displaces arsenic held by humic acids and sorbs strongly on the hydrous oxides of arsenates (Thanabalasingam nd Pickering 1986).

Physical processes play a key role in governing arsenic bioavailability in aquatic environments. For example, arsenates are readily sorbed by colloidal humic material under conditions of high organic content, low pH, low phosphate, and low mineral content (EPA 1980; Thanabalasingam and Pickering 1986). Arsenates also coprecipitate with, or adsorb on, hydrous iron oxides and form insoluble precipitates with calcium, sulfur, aluminum, and barium compounds (EPA 1980). Removal of arsenic from seawater by iron hydroxide scavenging seems to be a predominant factor in certain estuaries. The process involves both As^{+3} and As^{+5} and results in a measurable increase in arsenic levels in particulate matter, especially at low salinities (Sloot et al. 1985). Arsenic sulfides are comparatively insoluble under conditions prevalent in anaerobic aqueous and sedimentary media containing hydrogen sulfide; accordingly, these compounds may accumulate as precipitates and thus remove arsenic from the aqueous environment. In the absence of hydrogen sulfide, these sulfides decompose within several days to form arsenic oxides, sulfur, and hydrogen sulfide (NAS 1977).

In reduced environments, such as sediments, arsenate is reduced to arsenite and methylated to methylarsinic acid or dimethylarsenic acids: these compounds may be further methylated to trimethylarsine or reduced to dimethylarsine, and may volatilize to the atmosphere where oxidation reactions result in the formation of dimethylarsinic acid (Woolson 1975). Arsenates are more strongly adsorbed to sediments than are other arsenic forms, the adsorption processes depending strongly on arsenic concentration, sediment characteristics, pH, and ionic concentration of other compounds (EPA 1980). An important mechanism of arsenic adsorption onto lake sediments involves the interaction of anionic arsenates and hydrous iron oxides. Current evidence suggests that arsenic is incorporated into sediments at the time of hydrous oxide formation, rather than by adsorption onto existing surfaces (Aggett and Roberts 1986). Arsenic concentrations in lake

sediments are also correlated with manganese; hydrous manganese oxides--positively charged for the adsorption of Mn^{+2} ions--play a significant role in arsenic adsorption onto the surface of lake sediments (Takamatsu et al. 1985). The mobility of arsenic in lake sediments and its release to the overlying water is related partly to seasonal changes. In areas that become stratified in summer, arsenic released from sediments accumulates in the hypolimnion until turnover, when it is mixed with epilimnetic waters; this mixing may result in a 10 to 20% increase in arsenic concentration (Aggett and O'Brien 1985). Microorganisms (including four species of fungi) in lake sediments oxidized inorganic As^{+3} to As^{+5} and reduced inorganic As^{+5} to As^{+3} under aerobic conditions; under anaerobic conditions, only reduction was observed (Freeman et al. 1986). Inorganic arsenic can be converted to organic alkyl arsenic acids and methylated arsines under anaerobic conditions by fungi, yeasts, and bacteria--although biomethylation may also occur under aerobic conditions (EPA 1980).

Most arsenic investigators now agree on the following points: (1) arsenic may be absorbed by ingestion, inhalation, or through permeation of the skin or mucous membranes; (2) cells accumulate arsenic by using an active transport system normally used in phosphate transport; (3) arsenicals are readily absorbed after ingestion, most being rapidly excreted in the urine during the first few days, or at most a week (the effects seen after long-term exposure are probably a result of continuous daily exposure, rather than of accumulation); (4) the toxicity of arsenicals conforms to the following order, from greatest to least toxicity: arsines > inorganic arsenites > organic trivalent compounds (arsenoxides) > inorganic arsenates > organic pentavalent compounds > arsonium compounds > elemental arsenic; (5) solubility in water and body fluids appears to be directly related to toxicity (the low toxicity of elemental arsenic is attributed to its virtual insolubility in water and body fluids, whereas the highly toxic arsenic trioxide, for example, is soluble in water to 12.0 g/l at 0 C, 21.0 g/l at 25 C, and 56.0 g/l at 75 C); and (6) the mechanisms of arsenical toxicity differ considerably among arsenic species, although signs of poisoning appear similar for all arsenicals (Woolson 1975; NRCC 1978; Pershagen aid Vahter 1979).

The primary toxicity mode of inorganic As+3 is through reaction with sulfhydryl groups of proteins and subsequent enzyme inhibition; inorganic pentavalent arsenate does not react as readily as As+3 with sulfhydryl groups, but may uncouple oxidative phosphorylation (Howard et al. 1984; EPA 1985). Inorganic As+3 interrupts oxidative metabolic pathways and sometimes causes morphological changes in liver mitochondria. Arsenite in vitro reacts with protein-SH groups to inactivate enzymes such as dihydrolipoyl dehydrogenase and thiolase, producing inhibited oxidation of pyruvate and beta-oxidation of fatty acids (Belton et al. 1985). Inorganic As+5 may also exert toxic effects by the reaction of arsenous acid (HAsO) with the sulfhydryl (SH) groups of enzymes. In the first reaction, arsenous acid is reduced to arsonous acid ($AsOH_2$), which then condenses to either monothiols or dithiols to yield dithioesters of arsonous acid. Arsonous acid may then condense with enzyme SH groups to form a binary complex (Knowles and Benson 1984a,b).

Methylation to methylarsonic acid ($(CH3)_2AsO_3H_2$) and dimethylarsinic acid ($(CH3)_2AsO_2H$) is usually the major detoxification mechanism for inorganic pentavalent arsenates and trivalent arsenites in mammals. Methylated arsenicals rapidly clear from all tissues except perhaps the thyroid (Marafante et al. 1985; Vahter and Marafante 1985; Yamauchi et al. 1986) Methylated arsenicals are probably common in nature. Methylation of arsenic (unlike methylation of mercury) greatly reduces toxicity and is a true detoxification process (Woolson 1975). Before methylation (which occurs largely in the liver), As+5 is reduced to As+3-- the kidney being an important site for this transformation (Belton et al. 1985). Arsenate reduction and, subsequent methylation is rapid: both. arsenite and dimethylarsinate were present in hamster (*Cricetus* sp.) plasma only 12 minutes postinjection of inorganic As+5 (Hanlon and Ferm 1986c). Demethylation of methylated arsenicals formed in vivo has not yet been reported (EPA 1980).

Toxic effects of organoarsenicals are exerted by initial metabolism to the trivalent arsonoxide form, and then by reaction with sulfhydryl groups of tissue proteins and enzymes to form an arylbis (organylthio) arsine (NAS 1977). This form, in turn, inhibits oxidative degradation of carbohydrates and decreases cellular ATP, the energy storage molecule of the cell (NRCC 1978). Among the organoarsenicals, those physiologically most injurious are methylarsonous acid ($CH3As(OH)_2$) and dimethylarsinous acid ($(CH3)_2AsOH$) (Knowles and Benson 1984b). The enzyme inhibitory forms of organoarsenicals (arsonous acid) are formed from arsenous acid and the corresponding arsonic acids by a wide variety of enzymes and subcellular particles (Knowles and Benson 1984a). Organoarsenicals used as growth promoters and drugs are converted to more easily excretable

(and sometimes more toxic) substances, although most organoarsenicals are eliminated without being converted to inorganic arsenic or to demethylarsinic acids (Pershagen and Vahter 1979).

ESSENTIALITY, SYNERGISM, AND ANTAGONISM

Limited data are available on the beneficial, protective, and essential properties of arsenic, and on its interactions with other chemicals.

Arsenic apparently behaves more like an environmental contaminant than as a nutritionally essential mineral (NAS 1977). Nevertheless, low doses (<2 ug/daily) of arsenic stimulated growth and metamorphosis in tadpoles, and increased viability and cocoon yield in silkworm caterpillars (NAS 1977). Arsenic deficiency has been observed in rats: signs include rough haircoat, low growth rate, decreased hematocrit, increased fragility of red cells, and enlarged spleen (NAS 1977). Similar results have been documented in goats and pigs fed diets containing less than 0.05 mg As/kg (NAS 1977). In these animals, reproductive performance was impaired, neonatal mortality was increased, birth weight was lower, and weight gains in second-generation animals were decreased; these effects were not evident in animals fed diets containing 0.35 mg As/kg (NAS 1977).

The use of phenylarsonic feed additives to promote growth in poultry and swine and to treat specific diseases does not seem to constitute a hazard to the animal or to its consumers. Animal deaths and elevated tissue arsenic residues occur only when the arsenicals are fed at excessive dosages for long periods (NAS 1977). Arsenic can be detected at low levels in tissues of animals fed organoarsenicals, but it is rapidly eliminated when the arsenicals are removed from the feed for the required 5-day period before marketing (Woolson 1975).

Selenium and arsenic are antagonists in several animal species. In rats, dogs, swine, cattle, and poultry, the arsenic protects against selenium poisoning if arsenic is administered in the drinking water and selenium through the diet (NAS 1977; NRCC 1978; Pershagen and Vahter 1979). Inorganic arsenic compounds decrease the toxicity of inorganic selenium compounds by increasing biliary excretion (NRCC 1978). However, in contrast to antagonism shown by inorganic arsenic-inorganic selenium mixtures, the toxic effects of naturally methylated selenium compounds (trimethylselenomium chloride and dimethyl selenide) are markedly enhanced by inorganic arsenicals (NRCC 1978).

Toxic effects of arsenic can be counteracted with saline purgatives, with various demulcents that coat irritated gastrointestinal mucous membranes, and by sodium thiosulfate (NAS 1977), and by mono- and dithiol-containing compounds and 2,3-dimercaptopropanol (Pershagen and Vahter 1979). Arsenic uptake in rabbit intestine is inhibited by phosphate, casein, and various metal chelating agents (EPA 1980). Mice and rabbits are significantly protected against sodium arsenite intoxication *by* N-(2,3-dimercaptopropyl)phthalamidic acid (Stine et al. 1984). Conversely, the toxic effects of arsenite are potentiated by excess dithiols, cadmium, and lead, as evidenced by reduced food efficiency and disrupted blood chemistry in rodents (Pershagen and Vahter 1979).

Arsenic effectively controls filariasis in cattle; new protective uses are under investigation. The control of parasitic nematodes (*Parafilaria bovicola*) in cattle was successful after 30 weekly treatments in plungement dips containing 1,600 mg As_2O_3/l; however, the muscle of treated cattle contained up to 1.3 mg As/kg, or 12X the amount in controls (Nevill 1985). Existing anionic organic arsenicals used to control tropical nematode infections in humans have sporadic and unacceptable lethal side effects. Cationic derivatives have been synthesized in an attempt to avoid the side effects and have been examined for effects on adult nematodes (*Brugia pahangi*) in gerbils (*Meriones unguiculatus*). All arsenicals were potent filaricides; the most effective-compounds tested killed 95% of adult *B. pahangi* after five daily subcutaneous injections of 3.1 mg As/kg body weight (Denham et al. 1986).

Animals previously exposed to sublethal levels of arsenic may develop tolerance to arsenic on reexposure. Although the mechanism of this process is not fully understood, it probably includes the efficiency of in vivo methylation processes (EPA 1980). For example, resistance to toxic doses of As+3 or As+5 increases in mouse fibroblast cells pretreated with a low As+3 concentration (Fischer et al. 1985). Also, growth is better in arsenic-conditioned mouse cells in the presence of arsenic than in previously unexposed cells, and inorganic arsenic is more efficiently methylated. In vivo biotransformation and excretion of inorganic arsenic as monomethylarsonic acid (MMA) and dimethylarsinic acid (DMA) has been demonstrated in a number of mammalian species,

including man. It seems that cells may adapt to arsenic by increasing the biotransformation rate of the element to methylated forms, such as MMA and DMA (Fischer et al. 1985). Pretreatment of ovary cells of Chinese hamster (*Cricetus* spp.) ovary cells with sodium arsenite provided partial protection against adverse effects of methyl methanesulfonate (MMS), and may even benefit the MMS-treated cells; however, posttreatment dramatically increases the cytotoxic, clastogenic, and mitotic effects induced by MMS (Lee et al. 1986b).

Although arsenic is not an essential plant nutrient, small yield increases have sometimes been observed at low soil arsenic levels, especially for tolerant crops such as potatoes, corn, rye, and wheat (Woolson 1975). Arsenic phytotoxicity of soils is reduced with increasing lime, organic matter, iron, zinc, and phosphates (NRCC 1978). In most soil systems, the chemistry of As becomes the chemistry of arsenate; the estimated half-time of arsenic in soils is about 6.5 years, although losses of 60% in 3 years and 67% in 7 years have been reported (Woolson 1975). Additional research is warranted on the role of arsenic in crop production, and in nutrition, with special reference to essentiality for aquatic and terrestrial wildlife.

BACKGROUND CONCENTRATIONS

GENERAL

In abundance of elements, arsenic ranks 20th in the earth's crust (1.5 to 2 mg/kg) 14th in seawater, and 12th in the human body (Woolson 1975). It occurs in various forms, including inorganic and organic compounds and trivalent and pentavalent states (Pershagen and Vahter 1979). In aquatic environments, higher arsenic concentrations are reported in hot springs, in groundwaters from areas of thermal activity or in areas containing rocks with high arsenic content, and in some waters with high dissolved salt content (NAS 1977). Most of the other elevated values reported in lakes, rivers, and sediments are probably due to anthropogenic sources, which include smelting and mining operations; combustion of fossil fuel; arsenical grasshopper baits; synthetic detergent and sewage sludge wastes; and arsenical defoliants, herbicides, and pesticides (NAS 1977). Most living organisms normally contain measurable concentrations of arsenic, but except for marine biota, these are usually less than 1 mg/kg fresh weight. Marine organisms, especially crustaceans, may contain more than 100 mg As/kg dry weight, usually as arsenobetaine, a water soluble organoarsenical that poses little risk to the organism or its consumer. Plants and animals collected from naturally arseniferous areas or near anthropogenic sources may contain significantly elevated tissue residues of arsenic. Additional and more detailed information on background concentrations of arsenic in abiotic and living resources was given by NAS (1977), Hall et al. (1978), NRCC (1978), EPA (1980), Jenkins (1980), and Eisler (1981).

NONBIOLOGICAL SAMPLES

Arsenic is a major constituent of at least 245 mineral species, of which arsenopyrite is the most common (NAS 1977). In general, background concentrations of arsenic are 0.2 to 15 mg/kg in the lithosphere, 0.005 to 0.1 ug/m in air, <10 ug/l in water, and <15 mg/kg in soil (NRCC 1978). The commercial use and production of arsenic compounds have raised local concentrations in the environment far above the natural background concentrations (Table 1).

Weathering of rocks and soils adds about 45,000 tons of arsenic to the oceans annually, accounting for less than 0.01 mg/l on a global basis (NRCC 1978). However, arsenic inputs to oceans increased during the past century both from natural sources and as a result of industrial use, agricultural and deforestation activities, emissions from coal and oil combustion, and loss during mining of metal ores. If present activities continue, arsenic concentrations in oceanic surface waters may increase overall by about 2% *by* the year 2000, with most of the increased burden in estuaries and coastal oceans--e.g., Puget Sound, Washington; the Tamar, England; and the Tejo, Portugal (Sanders 1985). Estimates of the residence times of arsenic are 60,000 years in the ocean and 45 years in a freshwater lake (NRCC 1978). In the hydrosphere, inorganic arsenic occurs predominantly as As+5 in surface waters, and significantly as As+3 in groundwaters containing high levels of total arsenic. The main organic species in freshwater are methylarsonic acid and dimethylarsinic acid, and these are usually present in lower concentrations than inorganic arsenites and arsenates (Pershagen and Vahter 1979). Total arsenic concentrations in both surface and groundwaters are usually < 10 ug/l; in certain areas, however, levels above 1 mg/l have been recorded (Pershagen and Vahter 1979).

In air, most arsenic particulates consist of inorganic arsenic compounds, often as As+3. Burning of coal and arsenic-treated wood, and smelting of metals are major sources of atmospheric arsenic contamination (i.e., >1

ug/m^3); in general, atmospheric arsenic levels are higher in winter, due to increased use of coal for heating (Pershagen and Vahter 1979).

The main carrier of arsenic in rocks and in most types of mineral deposits is iron pyrite (FeS_2) which may contain >2,000 mg/kg of arsenic (NRCC 1978). In localized areas, soils are contaminated by arsenic oxide fallout from smelting ores (especially sulfide ores) and combustion of arsenic-rich coal (Woolson 1975).

Arsenic in lacustrine sediment columns is subject to control by diagenetic processes and adsorption mechanisms, as well as anthropogenic influences (Farmer and Lovell 1986). For example, elevated levels of arsenic in surface or near surface sediments may be due to several causes (Farmer and Lovell 1986): natural processes (Loch Lomond, Scotland); or to human activities such as smelters (Lake Washington, Washington; Kelly Lake, Ontario, Canada), manufacture of arsenical herbicides (Brown's Lake, Wisconsin), and mining operations (Northwest Territories, Canada). Elevated levels of arsenic in sediments of the Wailoa River, Hawaii, are caused by As_2O_3 applied as an anti-termite agent between 1932 and 1963, and are mostly in anaerobic sediment regions where the chemical has been relatively undisturbed by biological activity; low levels of arsenic in the biota of that estuary suggest that arsenic is trapped in the anaerobic sediment layers (Hallacher et al. 1985).

Arsenic geochemistry in Chesapeake Bay, Maryland, depends on anthropogenic inputs and phytoplankton species composition (Sanders 1985). Inputs of anthropogenic arsenic into Chesapeake Bay are estimated at 100 kg daily, or 39 tons/year--probably from sources such as unreported industrial discharges, use of arsenical herbicides, and from wood preservatives (Sanders 1985). The chemical form of the arsenic in solution varies both seasonally and along the axis of the Bay. Arsenic is present only as arsenate in winter, but substantial quantities of reduced and methylated forms are present in summer in different areas. The forms and distribution patterns of arsenic during the summer suggest that separate formation processes exist. Arsenite, present in low salinity regions, may have been formed by chemical reduction in anoxic, subsurface waters and then mixed into the surface layer. Methylated arsenicals are highly correlated with ;landing crops of algae. One particular form, methylarsonate, is significantly correlated with the dominant alga *Chroomonas*. Since both arsenic reactivity and toxicity are altered by transformation of chemical form, the observed variations in arsenic speciation have considerable geochemical and ecological significance (Sanders 1985).

BIOLOGICAL SAMPLES

Background arsenic concentrations in living organisms are usually <1 mg/kg fresh weight in terrestrial flora and fauna, birds, and freshwater biota. These levels are higher, sometimes markedly so, in biota collected from mine waste sites, arsenic-treated areas, near smelters and mining areas, near areas with high geothermal activity, and near manufacturers of arsenical defoliants and pesticides (Table 2). Marine organisms, however, normally contain arsenic residues of several to more than 100 mg/kg dry weight (Lunde 1977); as discussed later, however, these concentrations present little hazard to the organism or to its consumers.

Shorebirds (seven species) wintering in the Corpus Christi, Texas, area contained an average of only 0.3 mg As/kg fresh weight in livers (maximum of 1.5 mg/kg), despite the presence of smelters and the heavy use of arsenical herbicides and defoliants; these values probably reflect normal background concentrations (White et al. 1980). Similar arsenic levels are reported in livers of brown pelicans (*Pelecanus occidentalis*) collected from South Carolina (Blus et al. 1977). The highest arsenic concentration recorded in seemingly unstressed coastal birds was 13.2 mg/kg fresh weight lipids (Table 2). This tends to corroborate the findings of others who demonstrated that arsenic concentrates in lipid fractions of marine plants, invertebrates, and higher organisms. An abnormal concentration of 16.7 mg As/kg fresh weight was recorded in liver of an osprey (*Pandion haliaetus*) from the Chesapeake Bay region (Wiemeyer et al. 1980). This bird was alive but weak, with serious histopathology including the absence of subcutaneous fat, and the presence of serous fluid in the pericardial sac and disorders of the lung and kidney. It died shortly after collection. Arsenic concentrations in liver from other ospreys collected in the same area usually were <1.5 mg As/kg fresh weight.

Arsenic concentrations in tissues of marine biota show a wide range of values, being highest in lipids, liver, and muscle tissues, and varying with the age of the organism, geographic locale, and proximity to anthropogenic activities (Table 2). In general, tissues with high lipid content contained high levels of arsenic. Crustacean tissues sold for human consumption and collected in U.S. coastal waters usually contained 3 to 10

mg As/kg fresh weight (Hall et al. 1978), or 1 to 100 mg/kg dry weight (Fowler and Unlu 1978), and were somewhat higher than those reported for finfish and molluscan tissues. Marine finfish tissues usually contained 2 to 5 mg As/kg fresh weight (Table 2). However, postmortem reduction of As+5 to As+3 occurs rapidly in fish tissues (Reinke et al. 1975), suggesting a need for additional research in this area. Maximum arsenic values recorded in elasmobranchs (mg/kg fresh weight) were 30 in muscle of a shark, *Mustelus antarcticus*, and 16.2 in muscle of a ray, *Raja* sp. (Eisler 1981). The highest arsenic concentration recorded in marine mammals, 2.8 mg As/kg fresh weight lipid, was from a cetacean captured by Norwegian whalers (Eisler 1981).

Arsenic appears to be elevated in marine biota because of their ability to accumulate arsenic from seawater or food sources, and not due to localized pollution (Maher 1985b). The great majority of the arsenic in marine organisms exists as water-soluble and lipid-soluble organoarsenicals that include arsenolipids, arsenosugars, arsenocholine, arsenobetaine ($(CH3)_3AsCH_2COOH$), monomethylarsonate ($CH3AsO(OH)_2$), and demethylarsinate ($(CH3)_2AsO(OH)$), as well as other forms. There is no convincing hypothesis to account for the existence of all the various forms of organoarsenicals found in marine organisms. One suggested hypothesis is that each form involves a single anabolic/catabolic pathway concerned with the synthesis and turnover of phosphatidylcholine (Phillips and Depledge 1986). Arsenosugars (arsenobetaine precursors) are the dominant arsenic species in brown kelp (*Ecklonia radiata*), giant clam (*Tridacna maxima*), shrimp (*Pandalus borealis*), and ivory shell (*Buccinum striatissimum*) (Shiomi et al. 1984a,b; Francesconi et al. 1985; Matsuto et al. 1986; Phillips and Depledge 1986). For most marine species, however, there is general agreement that arsenic exists primarily as arsenobetaine, a water soluble organoarsenical that has been identified in tissues of western rock lobster (*Panulirus cygnus*), American lobster (*Homarus americanus*), octopus (*Paroctopus* sp.), sea cucumber (*Stichopus japonicus*), blue shark (*Prionace glauca*), sole (*Limanda* sp.), squid (*Sepioteuthis australis*), prawn (*Penaeus latisulcatus*), scallop (*Pecten alba*), and many other species including teleosts, molluscs, tunicates, and crustaceans (Shiomi et al. 1984b; Francesconi et al. 1985; Hanaoka and Tagawa 1985a,b; Maher 1985b; Norin et al. 1985; Matsuto et al. 1986). The potential risks associated with consumption of seafoods containing arsenobetaine seem to be minor. The chemical was not mutagenic in the bacterial *Salmonella typhimurium* assay (Ames test), had no effect on metabolic inhibition of Chinese hamster ovary cells at 10,000 mg/l, and showed no synergism or antagonism on the action of other contaminants (Jongen et al. 1985). Arsenobetaine was not toxic to mice at oral doses of 10,000 mg/kg body weight during a 7-day observation period, and was rapidly absorbed from the gastrointestinal tract and rapidly excreted in urine without metabolism, owing to its high polar and hydrophilic characteristics (Kaise et al. 1985).

LETHAL AND SUBLETHAL EFFECTS

GENERAL

As discussed later, most authorities agree on 10 points: (1) inorganic arsenicals are more toxic than organic arsenicals, and trivalent forms are more toxic than pentavalent forms; (2) episodes of arsenic poisoning are either acute or subacute--cases of chronic arsenosis are rarely encountered, except in humans; (3) early developmental stages are the most sensitive to arsenic; (4) inorganic arsenic can traverse placental barriers--as little as 1.7 mg As+5/kg body weight at critical stages of hamster embryogenesis, for example, can produce fetal death and malformation; (5) biomethylation is the preferred detoxification mechanism for inorganic arsenicals; (6) arsenic is bioconcentrated by organisms, but not biomagnified in the food chain; (7) depressed crop yields were recorded at 3 to 28 mg of water soluble soil As/l, or about 25 to 85 mg total As/kg soil--adverse effects on vegetation were recorded at concentrations in air >3.9 ug As/m^3; (8) some aquatic species were adversely affected at water concentrations of 19 to 48 ug As/l, or 120 mg As/kg in the diet, or tissue residue of 1.3 to 5 mg As/kg fresh weight; (9) sensitive species of birds died following single oral doses of 17.4 to 47.6 mg As/kg body weight; and (10) adverse effects were noted in mammals at single oral doses of 2.5 to 33 mg As/kg body weight, at chronic oral doses of 1 to 10 mg As/kg body weight, and at feeding levels of 50 mg, sometimes only 5 mg, As/kg in the diet.

It is emphasized in the literature that arsenic metabolism and toxicity vary greatly between species, and that effects are significantly altered by numerous physical, chemical, and biological modifiers. Adverse health effects, for example, may involve respiratory, gastrointestinal, cardiovascular, and hematopoietic systems, and may range from reversible effects to cancer and death, depending partly on the physical and chemical forms of arsenic tested, the route of administration, and dose.

CARCINOGENESIS, MUTAGENESIS, AND TERATOGENESIS

Epidemiological studies show that increased risk of cancers in skin, lung, liver, lymph, and hematopoietic systems of humans is associated with exposure to inorganic arsenicals. These increased cancer risks are especially prevalent among smelter workers and in those engaged in the production and use of arsenical pesticides where atmospheric levels exceed 54.6 ug As/m^3 (NRCC 1978; Belton et al. 1985; Pershagen and Bjorklund 1985). Skin tumors, mainly of low malignancy, have been reported after consumption of arsenic-rich drinking waters; a total dose of several grams, probably as As+3 is usually required for the development of skin tumors (Pershagen and Vahter 1979). High incidences of skin cancer and hyperpigmentation were noted among several population groups, especially Taiwanese and Chileans, consuming water containing more than 0.6 mg As/l; the frequency of cancer was highest among people over age 60 who demonstrated symptoms of chronic arsenic poisoning (NRCC 1978).

Arsenic reportedly inhibits cancer formation in species having a high incidence of spontaneous cancers (NRCC 1978). In fact, arsenic may be the only chemical for which there is sufficient evidence for carcinogenicity in humans but not in other animals (Woolson 1975; Belton et al. 1985; Lee et al. 1985). In general, animal carcinogenicity tests with inorganic and organic arsenicals have been negative (Hood 1985), even when the chemicals were administered at or near the highest tolerated dosages for long periods (NAS 1977). Most studies of arsenic carcinogenesis in animals were presumably of insufficient duration to simulate conditions in long-lived species such as humans (NRCC 1978). However, mice developed leukemia and lymphoma after 20 subcutaneous injections of 0.5 mg As+5/kg body weight: 46% of the experimental group developed these signs vs. none in controls (NRCC 1978). Recently, pulmonary tumorogenicity has been demonstrated in hamsters administered calcium arsenate intratracheally (Pershagen and Bjorklund 1985). Cacodylic acid and other organoarsenicals are not carcinogenic, but may be mutagenic at very high doses (Hood 1985).

Several inorganic arsenic compounds are weak inducers of chromosomal aberrations, sister chromatid exchange, and in vitro transformation of mammalian cells; however, there is no conclusive evidence that arsenic causes point mutations in any cellular system (Pershagen and Vahter 1979; Belton et al. 1985; Lee et al. 1985; Deknudt et al. 1986). Studies with bacteria suggest that arsenite is a comutagen, or may inhibit DNA repair (Belton et al. 1985).

Arsenic is a known teratogen in several classes of vertebrates, and has been implicated as a cause of birth defects in humans. Specific developmental malformations have been produced experimentally in mammals using inorganic As+3 or As+5 either through a single dose or a continuous dose during embryogenesis (Hanlon and Ferm 1986b). Teratogenic effects are initiated no later than 4 hours postadministration of As; fetal abnormalities are primarily neural tube defects (Hanlon and Ferm 1985c), but may also include protruding eyes, incomplete development of the skull, abnormally small jaws and other skeletal anomalies (NRCC 1978). Inorganic As+3 and As+5, but not organoarsenicals, cross placental barriers in many species of mammals, and result in fetal deaths and malformations (NRCC 1978; EPA 1980). Recent studies with hamsters, for example, showed that sodium arsenite can induce chromatid breaks and chromatid exchanges in Chinese hamster ovary cells in a dose dependent manner (Lee et al. 1986b). In an earlier study (Lee et al. 1985), As+3 was about 10X more potent than As+5 in effecting transformations, The birth defects were most pronounced in golden hamsters exposed to As+5 during the 24-hour period of critical embryogenesis--i.e., day 8 of gestation (Ferm and Hanlon 1985)--when 1.7 mg As+5/kg body weight induced neural tube defects in about 90% of the fetuses. Hanlon and Ferm (1986a) showed that hamsters exposed to As+5 and heat stress (39 °C for 50 minutes) on day 8 of gestation produced a greater percentage of malformed offspring (18 to 39%) than did hamsters exposed to As+5 alone (4% to 8%).

TERRESTRIAL PLANTS AND INVERTEBRATES

In general, arsenic availability to plants is highest in coarse-textured soils having little colloidal material and little ion exchange capacity, and lowest in fine-textured soils high in clay, organic material, iron, calcium, and phosphate (NRCC 1978). To be absorbed by plants, arsenic compounds must be in a mobile form in the soil solution. Except for locations where arsenic content is high, e.g., around smelters, the accumulated arsenic is distributed throughout the plant body in nontoxic amounts (NAS 1977). For most plants, a significant depression in crop yields was evident at soil-As concentrations of 3 to 28 mg/l of water soluble arsenic and 25 to 85 mg/kg of total arsenic (NRCC 1978). Yields of peas (*Pisum sativum*), a sensitive species, were decreased at 1 mg/l of water soluble arsenic or 25 mg/kg of total soil As; rice (*Oryza sativum*) yields were decreased 75% at 50 mg/l of

disodium methylarsonate in silty loam; and soybeans (*Glycine max*) grew poorly when residues exceeded 1 mg As/kg (Table 3; NRCC 1978). Forage plants grown in soils contaminated with up to 80 mg total As/kg from arsenical orchard sprays contained up to 5.8 mg As/kg dry weight; however, these plants were considered nonhazardous to grazing ruminants (Merry et al. 1986).

Attention was focused on inorganic arsenical pesticides after accumulations of arsenic in soils eventually became toxic to several agricultural crops, especially on former orchards and cotton fields. Once toxicity is observed, it persists for several years even if no additional arsenic treatment is made (Woolson 1975). Poor crop growth was associated with bioavailability of arsenic in soils. For example, alfalfa (*Medicago sativa*) and barley (*Hordeum vulgare*) grew poorly in soils containing only 3.4 to 9.5 mg As/kg, provided the soils ware acidic, lightly textured, low in phosphorus and aluminum, high in iron and calcium, and contained excess moisture (Woolson 1975). Use of inorganic arsenical herbicides, such as calcium arsenate, to golf course turfs for control of fungal blight sometimes exacerbates the disease. The use of arsenicals on Kentucky bluegrass (*Poa pratensis*) is discouraged under conditions of high moisture and root stress induced by previous arsenical applications (Smiley et al. 1985).

Methylated arsenicals, whether herbicides or defoliants, are sprayed on plant surfaces. They can reach the soil during application or can be washed from the plants. Additional arsenic enters soils by exchange from the roots or when dead plant materials decay (Hood 1985). Cacodylic acid and sodium cacodylate are nonselective herbicides used in at least 82 products to eliminate weeds and grasses around trees and shrubs, and to eradicate vegetation from rights-of-ways and other noncrop areas (Hood 1985). Normal application rates of various organoarsenicals for crop and noncrop purposes rarely exceed 5 kg/ha (Woolson 1975). Under recommended treatment levels, organoarsenical soil residues were not toxic to crops, and those tested (soybean, beet, wheat) were more resistant to organoarsenicals than to comparable levels of inorganic arsenicals (Woolson 1975).

Air concentrations up to 3.9 ug As/m^3 near gold mining operations were associated with adverse effects on vegetation; higher concentrations of 19 to 69 ug As/m^3, near a coal fired power plant in Czechoslovakia, produced measurable contamination in soils and vegetation in a 6-km radius (NRCC 1978).

The phytotoxic actions of inorganic and organic arsenicals are different and each is significantly modified by physical processes. The primary mode of action of arsenite in plants is inhibition of light activation, probably through interference with the pentose phosphate pathway (Marques and Anderson 1986). Arsenites penetrate the plant cuticle to a greater degree than arsenates (NAS 1977). One of the first indications of plant injury by sodium arsenite is wilting caused by loss of turgor, whereas stress due to sodium arsenate does not involve rapid loss of turgor (NAS 1977). Organoarsenicals, such as cacodylic acid, enter plants mostly by absorption of sprays; uptake from the soil contributes only a minor fraction (Hood 1985). The phytotoxicity of organoarsenical herbicides is characterized by chlorosis, cessation of growth, gradual browning, dehydration, and death (NAS 1977). In general, plants cease to grow and develop after the roots have absorbed much arsenic (NRCC 1978). Plants can absorb arsenic through the roots and foliage, although translocation is species dependent. Concentrations of arsenic in plants correlate highly and consistently with water extractable soil arsenic, and usually poorly with total soil arsenic (NRCC 1978). For example, concentrations of arsenic in corn (*Zea mays*) grown in calcareous soils for 25 days were significantly correlated with the soil water extractable arsenic fraction, but not other fractions; extractable phosphorus was correlated positively to both arsenic in corn and to the water soluble arsenic fraction (Sadiq 1986). In the moss *Hylocomium splendens*, arsenate accumulation from solution was through living shoots, optimum uptake being between pH 3 and 5 (Wells and Richardson 1985). Some plants, such as beets (*Beta vulgaris*) accumulated arsenic more readily at elevated temperatures, but the addition of phosphate fertilizers markedly depressed uptake (Merry et al. 1986).

Soils amended with arsenic-contaminated plant tissues were not measurably affected in CO_2 evolution and nitrification, suggesting that the effects of adding arsenic to soils does not influence the decomposition rate of plant tissues by soil microorganisms (Wang et al. 1984). The half-life of cacodylic acid is about 20 days in untreated soils and 31 days in arsenic-amended soils (Hood 1985). Estimates of the half-life of inorganic arsenicals in soils are much longer, ranging from 6.5 years for arsenic trioxide to 16 years for lead arsenate (NRCC 1978).

Data on arsenic effects to soil biota and insects are limited. In general, soil microorganisms are capable of tolerating and metabolizing relatively high concentrations of arsenic (Wang et al. 1984). This adaptation seems usually to be due to decreased permeability of the microorganism to arsenic (NAS 1977). Tolerant soil microbiota can withstand concentrations up to 1,600 mg/kg; however, growth and metabolism were reduced in sensitive species at 375 mg As/kg and, at 150 to 165 mg As/kg, soils were devoid of earthworms and showed diminished quantities of bacteria and protozoans (NRCC 1978). Honeybees (*Apis mellifera*) that were killed accidentally by As+3 spray dusting contained 4 to 5 ug As per bee (NAS 1977)--equivalent to 21 to 31 mg/kg body weight (Table 3). Larvae of the western spruce budworm (*Choristoneura occidentalis*) continued to feed on As+3 -contaminated vegetation until a threshold level of about 2,300 to 3,300 mg As/kg dry weight whole larvae was reached; death then sometimes occurred (Table 3; Robertson and McLean 1985). Larvae that had accumulated sufficient energy reserves completed the first stage of metamorphosis, but developed into pupae of subnormal weight; larvae containing <2,600 mg As+3/kg ultimately developed into adults of less than normal weight, and some containing >2,600 mg/kg dry weight died as pupae (Robertson and McLean 1985).

AQUATIC BIOTA

Adverse effects of arsenicals on aquatic organisms have been reported at concentrations of 19 to 48 ug/l in water, 120 mg/kg in diets, and 1.3 to 5 mg/kg fresh weight in tissues (Table 4). The most sensitive aquatic species tested showing adverse effects were three species of marine algae, which showed reduced growth in the range of 19 to 22 ug As+3/l; developing embryos of the narrow-mouthed toad (*Gastrophryne carolinensis*), of which 50% were dead or malformed in 7 days at 40 ug As+3/l; and a freshwater alga (*Scenedesmus obliquus*), in which growth was inhibited 50% in 14 days at 48 ug As+5/l (Table 4). Chronic studies with mass cultures of natural phytoplankton communities exposed to low levels of arsenate (1.0 to 15.2 ug/l) showed that As+5 differentially inhibits certain plants, causing a marked change in species composition, succession, and predator-prey relations; the significance of these changes on carbon transfer between trophic levels is unknown (Sanders and Cibik 1985; Sanders 1986). Adverse biological effects have also been documented at water concentrations of 75 to 100 ug As/l. At 75 ug As+5/l, growth and biomass in freshwater and marine algae was reduced; at 85 to 88 ug/l of As+5 or various methylated arsenicals, mortality was 10% to 32% in amphipods (*Gammarus pseudolimnaeus*) in 28 days; at 95 ug As+3/l, marine red alga failed to reproduce sexually; and at 100 ug As+5/l, marine copepods died and goldfish behavior was impaired (Table 4). Rainbow trout (*Salmo gairdneri*) fed diets containing up to 90 mg As+5 kg were only slightly affected, but those given diets containing 120 mg As/kg (as As+3 or As+5), and higher, grew poorly, avoided food, and failed to metabolize food efficiently; no toxic effects were reported over 8 weeks of exposure to diets containing 1,600 mg/kg, as methylated arsenicals (Table 4). In bluegills (*Lepomis macrochirus*), tissue residues of 1.35 mg As/kg fresh weight in juveniles and 5 mg/kg in adults are considered elevated and potentially hazardous (NRCC 1978).

Toxic and other effects of arsenicals to aquatic life are significantly modified by numerous biological and abiotic factors (Woolson 1975; NAS 1977; NRCC 1978; EPA 1980, 1985; Howard et al. 1984; Michnowicz and Weaks 1984; Bryant et al. 1985; Sanders 1986). The LC-50 values, for example, are markedly affected by water temperature, pH, Eh, organic content, phosphate concentration, suspended solids, and presence of other substances and toxicants, as well as arsenic speciation, and duration of exposure. In general, inorganic arsenicals are more toxic than organoarsenicals to aquatic biota, and trivalent species are more toxic than pentavalent species. Early life stages are most sensitive, and large interspecies differences are recorded, even among those closely related taxonomically.

Arsenic is accumulated from the water by a variety of organisms; however, there is no evidence of magnification along the aquatic food chain (Woolson 1975; NAS 1977; NRCC 1978; Hallacher et al. 1985; Hood 1985). In a marine ecosystem based on the alga *Fucus vesiculosus*, arsenate (7.5 ug As+5/l) was accumulated by all biota. After 3 months, arsenic was concentrated most efficiently by *Fucus* (120 mg/kg dry weight in apical fronds) and filamentous algal species 30 mg/kg dry weight); little or no bioaccumulation occurred in invertebrates, although arsenic seemed to be retained by gastropods and mussels (Rosemarin et al. 1985). In a freshwater food chain composed of algae, daphnids, and fish, water concentrations of 0.1 mg cacodylic acid/l produced residues (mg As/kg dry weight), after 48 hours of 4.5 in algae and 3.9 in daphnids, but only 0.09 in fish (NAS 1977). Microcosms of a Delaware Cordgrass (*Spartina alterniflora*) salt marsh exposed to elevated levels of As showed that virtually all arsenic was incorporated into plant tissue or strongly sorbed to cell surfaces (Sanders and Osman 1985). Studies with radioarsenic and mussels (*Mytilus galloprovincialis*) showed that accumulation varied with nominal arsenic concentrations, tissues, age of the mussel, and temperature and

salinity of the medium (Unlu and Fowler 1979). Arsenate uptake increased with increasing arsenic concentration in the medium, but the response was not linear, accumulation being suppressed at higher external arsenic concentrations. Smaller mussels took up more arsenic than larger ones. In both size groups, arsenic was concentrated in the byssus and digestive gland. In general, arsenic uptake and loss increased at increasing temperatures. Uptake was significantly higher at 19 o/oo salinity than at 38 o/oo, but loss rate was about the same at both salinities. Radioarsenic loss followed a biphasic pattern; biological half-life was 3 and 32 days for the fast and slow compartments, respectively; secretion of the byssal thread played a key role in elimination (Unlu and Fowler 1979). Factors known to modify rates of arsenic accumulation and retention in a marine shrimp (*Lysmata seticaudata*) include water temperature and salinity, arsenic concentration, age, and especially frequency of molting (Fowler and Unlu 1978).

Bioconcentration factors (BCF) experimentally determined for arsenic in aquatic organisms are, except for algae, relatively low. The BCF values for inorganic As+3 in most aquatic invertebrates and fish exposed for 21 to 30 days did not exceed 17X; the maximum was 6X for As+5, and 9X for organoarsenicals (EPA 1980, 1985). Significantly higher BCF values were recorded in other aquatic organisms (NRCC 1978), but they were based on mean arsenic concentrations in natural waters that seemed artificially high. A BCF of 350X was reported for the American oyster (*Crassostrea virginica*) held in 5 ug As+3/l for 112 days (Zaroogian and Hoffman 1982). There was no relation between oyster body burdens of arsenic and exposure concentrations; however, diet seemed to contribute more to arsenic uptake than did seawater concentrations (Zaroogian and Hoffman 1982). An arsenic-tolerant strain of freshwater alga (*Chlorella vulgarus*) from an arsenic-polluted environment showed increasing growth up to 2,000 mg As+5/l, and could survive at 10,000 mg As+5/l (Maeda et al. 1985). Accumulations up to 50,000 mg As/kg dry weight were recorded (Maeda et al. 1985)--suggesting a need for additional research on the extent of this phenomenon and its implications on food web dynamics.

Some investigators have suggested that arsenic in the form of arsenite is preferentially utilized by marine algae and bacteria (Johnson 1972; Bottino et al. 1978; Johnson and Burke 1978). Arsenate reduction to arsenite in seawater depends on phosphorus in solution and available algal biomass (Johnson and Burke 1978). During algal growth, as phosphate is depleted and the P+5/As+5 ratio drops, the rate of As+5 reduction increases. The resultant As+3, after an initial peak, is rapidly oxidized to As+5, indicating the possibility of biological catalysis of oxidation as well as mediation of As+5 reduction. It is generally accepted that As+3 is more toxic than arsenates to higher organisms; however, As+5 had a more profound effect on growth and morphology of marine algae than did As+3. Possibly marine algae erect a barrier against the absorption of As+3, but not of As+5. Within the cell, As+5 can then be reduced to the possibly more toxic As+3. For example, the culture of two species of marine algae (*Tetraselmis chui, Hymenomonas carterae*) in media containing various concentrations of As+5 or As+3 showed that arsenic effects varied with oxidation state, ration, and light intensity. Arsenate was incorporated and later partly released by both species. Differences between rates of uptake and release suggest that As+5 undergoes chemical changes after incorporation into algal cells (Bottino et al. 1978). When bacterial cultures from the Sargasso Sea and from marine waters of Rhode Island were grown in As+3-enriched media, the bacteria reduced all available As+5 and utilized As+3 during the log growth phase--presumably as an essential trace nutrient. The arsenate reduction rate per cell was estimated to be 75×10^{-11} mg As/minute (Johnson 1972).

The ability of marine phytoplankton to accumulate high concentrations of inorganic arsenicals and transform them to methylated arsenicals that are later efficiently transferred in the food chain is well documented (Irgolic et al. 1977; Benson 1984; Matsuto et al. 1984; Freeman 1985; Froelich et al. 1985; Maeda et al. 1985; Norin et al. 1985; Sanders 1985; Yamaoka and Takimura 1986). Algae constitute an important source of organoarsenic compounds in marine food webs. In the food chain composed of the alga *Dunaliella marina*, the grazing shrimp *Artemia salina*, and the carnivorous shrimp *Lysmata seticaudata*, organic forms of arsenic were derived from in vivo synthesis by *Dunaliella* and efficiently transferred, without magnification, along the food chain (Wrench et al. 1979). Laboratory studies with five species of euryhaline algae grown in freshwater, or seawater, showed that all species synthesized fat soluble and water soluble arseno-organic compounds from inorganic As +3 and As+5. The BCF values in the five species examined ranged from 200X to about 3,000X--accumulations being highest in lipid phases (Lunde 1973). In Charlotte Harbor, Florida, a region that has become phosphate-enriched due to agricultural activity, virtually all of the arsenic taken up by phytoplankters was biomethylated and returned to the estuary, usually as monomethylarsonic and dimethylarsenic acids (Froelich et al. 1985). The ability of marine phytoplankton to methylate arsenic and release the products to a surrounding environment

varies between species and even within a particular species in relation to their possession of necessary methylating enzymes (Sanders 1985). The processes involved in detoxifying arsenate after its absorption by phytoplankton are not firmly established, but seem to be nearly identical in all plants, suggesting a similar evolutionary development. Like phosphates and sulphates, arsenate may be fixed with ADP, reduced to the arsonous level, and successfully methylated and adenosylated--ultimately producing the 5-dimethylarsenosoribosyl derivatives accumulating in algae (Benson 1984).

Sodium arsenite has been used extensively as an herbicide for control of mixed submerged aquatic vegetation in freshwater ponds and lakes; concentrations of 1.5 to 3.8 mg As+3/l have usually been effective and are considered safe for fish (NAS 1977). Recent data, however, have indicated that As+3 concentrations considered effective for aquatic weed control may be harmful to several species of freshwater teleosts, including bluegills, flagfish, fathead minnows, and rainbow trout (Table 4). Finfishes exposed to 1 to 2 mg total As/l for 2 to 3 days may show one or more of several signs: hemorrhagic spheres on gills; fatty infiltration of liver; and necrosis of heart, liver, and ovarian tissues (NRCC 1978). In green sunfish (*Lepomis cyanellus*), hepatocyte changes parallel arsenic accumulations in the liver (Sorensen et al. 1985). Organoarsenicals are usually eliminated rapidly by fish and other aquatic fauna. Rainbow trout, for example, fed a marine diet containing 15 mg organic arsenic/kg had only negligible tissue residues 6 to 10 days later, although some enrichment was noted in eyes, throat, gills, and pyloric caeca (Pershagen and Vahter 1979). Oral administration of sodium arsenate to estuary catfish (*Cnidoglanis macrocephalus*) and school whiting (*Sillago bassensis*) resulted in tissue accumulations of trimethylarsine oxide. Arsenobetaine levels, which occur naturally in these teleosts, were not affected by As+5 dosing. The toxicity of trimethylarsine oxide is unknown, but the ease with which it can be reduced to the highly toxic trimethylarsine is cause for concern (Edmonds and Francesconi 1987).

BIRDS

Signs of inorganic trivalent arsenite poisoning in birds (muscular incoordination, debility, slowness, jerkiness, falling hyperactivity, fluffed feathers, drooped eyelid, huddled position, unkempt appearance, loss of righting reflex, immobility, seizures) were similar to those induced by many other toxicants and did not seem to be specific for arsenosis. Signs occurred within 1 hour and deaths within 1 to 6 days postadministration; remission took up to 1 month (Hudson et al. 1984). Internal examination suggested that lethal effects of acute inorganic arsenic poisoning were due to the destruction of blood vessels lining the gut, which resulted in decreased blood pressure and subsequent shock (Nystrom 1984). Coturnix (*Coturnix coturnix*), for example, exposed to acute oral doses of As+3 showed hepatocyte damage, i.e., swelling of granular endoplasmic reticulum; these effects were attributed to osmotic imbalance, possibly induced by direct inhibition of the sodium pump by arsenic (Nystrom 1984)

Western grasshoppers (*Melanophus* spp.) poisoned by arsenic trioxide were fed, with essentially no deleterious effects, to nestling northern bobwhites (*Colinus virginianus*), mockingbirds (*Mimus polyglottos*), American robins, (*Turdus migratorius*), and other songbirds (NAS 977). Up to 134 poisoned grasshoppers, containing a total of about 40 mg As, were fed to individual nestlings without any apparent toxic effect. The species tested that were most sensitive to various arsenicals were brown-headed cowbird (*Molothrus ater*) with an LD-50 (11-day) value of 99.8 mg of copper acetoarsenite/kg diet; California quail (*Callipepla californica*) with an LD-50 single oral dose value of 47.6 mg of sodium arsenite/kg body weight; and chicken with 33 and turkey with 17.4 mg/kg body weight of 3-nitro-4-hydroxy phenylarsonic acid as a single oral dose (Table 5).

Chickens rapidly excrete arsenicals; only 2% of dietary sodium arsenite remained after 60 hours (NAS 1977), and arsanilic acid was excreted largely unchanged (Woolson 1975). Excretion of arsanilic acid by chickens was affected by uptake route: excretion was more rapid if administration was by intramuscular injection than if it was oral (NRCC 1978). Studies with inorganic As+5 and chickens indicated that arsenates rapidly penetrated mucosal and serosal surfaces of epithelial membranes; that As+5 intestinal absorption was essentially complete within 1 hour at 370 mg As+5/kg BW but only 50% complete at 3,700 mg/kg BW; that Vitamin D_3 was effective in enhancing duodenal As+5 absorption in rachitic chicks; and that As+5 and phosphate did not appear to share a common transport pathway in the avian duodenum (Fullmer and Wasserman 1986).

MAMMALS

Mammals are exposed to arsenic primarily by the ingestion of naturally contaminated vegetation and water, or through human activity. In addition, feed additives containing arsonic acid derivatives are often fed to domestic livestock to promote growth and retard disease. Some commercial pet foods contain up to 2.3 mg As/kg dry weight (NRCC 1978). Uptake may occur by ingestion (the most likely route), inhalation, and absorption through skin and mucous membranes. Soluble arsenicals are absorbed more rapidly and completely than are the sparingly soluble arsenicals, regardless of the route of administration (NRCC 1978).

Acute episodes of poisoning in warm-blooded organisms by inorganic and organic arsenicals are usually characterized by high mortality and morbidity over a period of 2 to 3 days (NAS 1977; Selby et al. 1977). General signs of arsenic toxicosis include intense abdominal pain, staggering gait, extreme weakness, trembling, salivation, vomiting, diarrhea, fast and feeble pulse, prostration, collapse, and death. Gross necropsy shows a reddening of gastric mucosa and intestinal mucosa, a soft yellow liver, and red edematous lungs. Histopathological findings show edema of gastrointestinal mucosa and submucosa; necrosis and sloughing of mucosal epithelium; renal tubular degeneration; hepatic fatty changes and necrosis; and capillary degeneration in gastrointestinal tract, vascular beds, skin, and other organs. In subacute episodes, where animals live for several days, signs of arsenosis include depression, anorexia, increased urination, dehydration, thirst, partial paralysis of rear limbs, trembling, stupor, coldness of extremities, and subnormal body temperatures (NAS 1977; Selby et al. 1977). In cases involving cutaneous exposure to arsenicals, a dry, cracked, leathery, and peeling skin may be a prominent feature (Selby et al. 1977). Nasal discharges and eye irritation were documented in rodents exposed to organoarsenicals in inhalation toxicity tests (Hood 1985). Subacute effects in humans and laboratory animals include peripheral nervous disturbances, melanosis, anemia, leukopenia, cardiac abnormalities, and liver changes. Most adverse signs rapidly disappeared after exposure ceased (Pershagen and Vahter 1979).

Arsenic poisoning in most animals is usually manifested by acute or subacute signs; chronic poisoning is infrequently seen (NAS 1977). The probability of chronic arsenic poisoning from continuous ingestion of small doses is rare, because detoxication and excretion are rapid (Woolson 1975). Chronic toxicity of inorganic arsenicals is associated with weakness, paralysis, conjunctivitis, dermatitis, decreased growth, and liver damage (NRCC 1978). Arsenosis, produced as a result of chronic exposure to organic arsenicals, was associated with demyelination of optic and sciatic nerves, depressed growth, and decreased resistance to infection (NRCC 1978).

The technical literature on arsenic (Table 6) shows general agreement on eight points: (1) Arsenic metabolism and effects are significantly influenced by the organism tested, the route of administration, the physical and chemical form of the arsenical, and the dose. (2) Inorganic arsenic compounds are more toxic than organic arsenic compounds and trivalent species more so than pentavalent. (3) Inorganic arsenicals can cross the placenta in most species of mammals. (4) Early developmental stages are the most sensitive, and man appears to be one of the most susceptible species. (5) Animal tissues usually contain low levels (<0.3 mg As/kg fresh weight) of arsenic; after the administration of arsenicals these levels are elevated, especially in liver, kidney, spleen, and lung; several weeks later, arsenic is translocated to ectodermal tissues (hair, nails) because of the high concentration of sulfur-containing proteins in these tissues. (6) Inorganic arsenicals are oxidized in vivo, biomethylated, and usually excreted rapidly in the urine, but organoarsenicals are usually not subject to similar transformations. (7) Acute or subacute arsenic exposure can lead to elevated tissue residues, appetite loss, reduced growth, loss of hearing, dermatitis, blindness, degenerative changes in liver and kidney, cancer, chromosomal damage, birth defects, and death. (8) Death or malformations have been documented at single oral doses of 2.5 to 33 mg As/kg body weight, at chronic doses of 1 to 10 mg As/kg body weight, and at dietary levels >5 and <50 mg As/kg diet.

Episodes of wildlife poisoning by arsenic are infrequent. White-tailed deer (*Odocoileus virginianus*) consumed, by licking, fatal amounts of sodium arsenite used to debark trees. The practice of debarking trees with arsenicals for commercial use has been almost completely replaced by mechanical debarking equipment (NAS 1977). Snowshoe hares (*Lepus* sp.) appear to be especially sensitive to methylated arsenicals; hares died after consuming plants heavily contaminated with monosodium methanearsonate as a result of careless silviculture practices (Hood 1985).

Unlike wildlife, reports of arsenosis in domestic animals are common in ovines and felines, less common in ovines and equines, and rare in porcines and poultry (NAS 1977). In practice, the most dangerous arsenic preparations are dips, herbicides, and defoliants in which the arsenical is in a highly soluble trivalent form, usually as trioxide or arsenite (Selby et al. 1977). Accidental poisoning of cattle with arsenicals, for example, is well documented. In one instance, more than 100 cattle died after accidental overdosing with arsenic trioxide applied topically to control lice. On necropsy, there were subcutaneous edematous swellings and petechial hemorrhages in the area of application, and histopathology of intestine, mucosa, kidney, and epidermis (Robertson et al. 1984). In Bangladesh, poisoned cattle showed depression, trembling, bloody diarrhea, restlessness, unsteady gait, stumbling, convulsions, groaning, shallow labored breathing, teeth grinding, and salivation (Samad and Chowdhury 1984). Cattle usually died 12 to 36 hours after the onset of signs; necropsy showed extensive submucosal hemorrhages of the gastrointestinal tract (Samad and Chowdhury 1984), and tissue residues >10 mg/kg fresh weight in liver and kidney (Thatcher et al. 1985). It sometimes appears that animals, especially cattle, develop an increased preference for weeds sprayed with an arsenic weed killer, not because of a change in the palatability of the plant, but probably because arsenic compounds are salty, and thus attractive to animals (Selby et al. 1977).

When extrapolating animal data from one species to another, the species tested must be considered. For example, the metabolism of arsenic in the rat (*Rattus* sp.) is unique, and very different from that in man and other animals. Rats store arsenic in blood hemoglobin, excreting it very slowly--unlike most mammals which rapidly excrete ingested inorganic arsenic in the urine as methylated derivatives (NAS 1977). Blood arsenic, whether given as As+3 or As+5, rapidly clears from humans, mice, rabbits, dogs, and primates, with a half-life of 6 hours for the fast phase and about 60 hours for the slow phase (EPA 1980). In rat, however, blood arsenic is mostly retained in erythrocytes, and clears slowly, with a Tb 1/2 of 60 to 90 days (EPA 1980). In rats, the excretion of arsenic into bile is 40X faster than in rabbits and up to 800X faster than in dogs (Pershagen and Vahter 1979). There is now general and widespread agreement that the rat is unsatisfactory for use in arsenic research (NAS 1977; NRCC 1978; Pershagen and Vahter 1979; EPA 1980; Webb et al. 1986).

Dimethylarsinic acid is the major metabolite of orally administered arsenic trioxide, and is excreted rapidly in the urine (Yamauchi and Yamamura 1985). The methylation process is true detoxification, since methanearsonates and cacodylates are about 200X less toxic than sodium arsenite (NAS 1977). The marmoset monkey (*Callithrix jacchus*), unlike all other animal species studied to date, was not able (for unknown reasons) to metabolize administered As+5 to demethylarsinic acid. Most was reduced to As+3. Only 20% the total dose was excreted in urine as unchanged As+5, another 20% as As+3, and the rest was bound to tissues giving distribution patterns similar to arsenite (Vahter and Marafante 1985). Accordingly, the marmoset--like the rat--may be unsuitable for research with arsenicals.

Arsenicals were ineffective in controlling certain bacterial and viral infections. Mice experimentally infected with bacteria (*Klebsiella pneumonias*) or viruses (pseudorabies, encephalitis, encephalomyocarditis) showed a significant increase in mortality when treated with large doses of arsenicals compared to nonarsenic-treated groups (NAS 1977; Aranyi et al. 1985).

It has been suggested, but not yet verified, that many small mammals avoid arsenic-treated feeds and consume other foods if given the choice (NAS 1977); also, that cacodylic acid, which has negligible effects on wildlife, reduces species diversity due to selective destruction of vegetation (Hood 1985). Both topics merit more research.

CURRENT RECOMMENDATIONS

Numerous criteria for arsenic have been proposed to protect natural resources and human health (Table 7). But many authorities recognize that these criteria are not sufficient for adequate or (in some cases) reasonable protection, and that many additional data are required if meaningful standards are to be promulgated (NAS 1977); NRCC 1978; Pershagen and Vahter 1979; EPA 1980, 1985). Specifically, data are needed on the following subjects: cancer incidence and other abnormalities in natural resources from areas with elevated arsenic levels, and the relation to potential carcinogenicity of arsenic compounds; interaction effects of arsenic with other carcinogens, cocarcinogens, promoting agents, inhibitors, and common environmental contaminants; controlled studies with aquatic and terrestrial indicator organisms on physiological and biochemical effects of long-term, low-dose exposures to inorganic and organic arsenicals--including effects on reproduction and genetic makeup; methodologies for establishing maximum permissible tissue concentrations for arsenic; effects

of arsenic in combination with infectious agents; mechanisms of arsenical growth-promoting agents; role of arsenic in nutrition; extent of animal adaptation to arsenicals, and the mechanisms of action, and physicochemical processes influencing arsenic cycling. In addition, techniques should be developed and procedures implemented in three fields: (1) development of more sophisticated measurements of chemical forms of arsenic in plant and animal tissues; (2) correlations of biologically observable effects with appropriate chemical forms of arsenic; and (3) management of arsenical wastes that will accommodate recycling, reuse, and long term storage.

Some proposed, arsenic criteria merit additional comment, such as those on aquatic life protection, levels in seafoods and drinking water, and use in food-producing animals as growth stimulants or for disease prevention and treatment.

For saltwater life protection, the current water quality criterion of 36 ug As+3/l (EPA 1985; Table 7) seems to offer a reasonable degree of safety; only a few species of algae show adverse effects at <36 ug/1--e.g., reduced growth at 19 to 22 ug/l. In 1980, this criterion was 508 ug/l (EPA 1980), or about 14X higher; the current downward modification seems to be indicative of the increasingly stringent arsenic criteria formulated by regulatory agencies. But the current criterion for freshwater life protection of 190 ug As+3/1 (EPA 1985; Table 7), which is down from 440 ug As+3/1 in 1980 (EPA 1980), is unsatisfactory. Many species of freshwater biota are adversely affected at <190 ug/l of As+3, As+5, or various organoarsenicals (Table 4). These adverse effects include death and malformations of toad embryos at 40 ug/l, growth inhibition of algae at 48 to 75 ug/l, mortality of amphipods and gastropods at 85 to 88 ug/l, and behavioral impairment of goldfish (*Carassius auratus*) at 100 ug/l. It seems that some downward adjustment in the current freshwater aquatic life protection criterion is warranted.

Current permissible concentrations of arsenic in seafood in Hong Kong destined for human consumption range from 6 to 10 mg/kg fresh weight (Table 7); however, these values are routinely exceeded in 22% of finfish, 20% of bivalve molluscs, 67% of gastropods, 29% of crabs, 21% of shrimp and prawns, and 100% of lobsters (Phillips et al. 1982). The highest arsenic recorded in Hong Kong seafood products was in gastropods (*Hemifusus* spp.), in which the concentrations of 152 to 176 mg/kg FW were among the highest recorded in any species to date (Phillips et al. 1982). Probably most of the arsenic in seafood products is present as arsenobetaine or in other comparatively harmless forms. In effect, arsenic criteria for seafoods are neither enforced nor enforceable. Most toxicologists from the U.S. Food and Drug Administration believe that the average daily intake of arsenic in the different food commodities does not pose a hazard to the consumer (Jelinek and Corneliussen 1977).

For maximum protection of human health from the potential carcinogenic effects due to exposure of arsenic through drinking water or contaminated aquatic organisms, the ambient water concentration should be zero, based on the nonthreshold assumption for arsenic. But zero level may not be attainable. Accordingly, the levels that may result in an incremental increase of cancer risk over a lifetime are estimated at 10^{-5}, or one additional case per 100,000 population. These values are estimated at 0.022 ug As/l for drinking water, and 0.175 ug As/l for water containing edible aquatic resources (EPA 1980; Table 7).

Various phenylarsonic acids--especially arsanilic acid, sodium arsanilate, and 3-nitro-4-hydroxy-phenylarsonic acid--have been used as feed additives for disease control and for improvement of weight gain in swine and poultry for almost 40 years (NAS 1977). The arsenic is present as As+5 and is rapidly excreted; present regulations require withdrawal of arsenical feed additives 5 days before slaughter for satisfactory depuration (NAS 1977). Under these conditions, total arsenic residues in edible tissues do not exceed the maximum permissible limit of 2 mg/kg fresh weight (Jelinek and Corneliussen 1977). It now seems that organoarsenicals will continue to be used as feed additives unless evidence indicates otherwise.

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Table 1. Total arsenic concentrations in selected nonbiological materials.

Material and units (in parentheses)	Concentration[a]	Reference[b]
Air (ug/m^3)		
Remote areas	<0.02	1
Urban areas	(0.0-0.16)	
Near smelters		
U.S.S.R.	(0.5-1.9)	2
Texas	Max. 1.4	2
Tacoma, Washington	Max. 1.5	2
Romania	Max. 1.6	2
Germany	(0.9-1.5)	2
Coal-fired power plant, Czechoslovakia	(19 to 69)	3
Orchard spraying of Pb arsenate	Max. 260,000	3
Near U.S. cotton gin burning vegetation treated with arsenic	Max. 400	3
Drinking water (g/L)		
Nationwide, U.S.A.	2.4 (0.5-214)	4
Fairbanks, Alaska	224 (1-2,450)	4
Bakersfield, California	(6-393)	4
Nevada, 3 communities	(51-123)	4
Mexico, from plant producing As_2O_3	(4,000-6,000)	2
Japan, near factory producing arsenic sulfide	3,000	2
Ghana, near gold mine	1,400	2
Minnesota, contaminated by residual arsenical grasshopper bait	(11,800-21,000)	1
Methylated arsenicals, U.S.A.	usually <0.3 (0.01-1)	5
Dust (mg/kg)		
Tacoma, Washington		
Near smelter	1,300	1
Remote from smelter	70	1
Fossil fuels (mg/kg)		
Coal		
Canada	4 (0.3-100)	3
USA	5	2

Czechoslovakia	Max. 1,500	2
Worldwide	13 (0.0-2,000)	1
Coal ash	(<20-8,000)	3
Flyash	(2.8-200)	3
Petroleum	0.2	3
Petroleum ash	Max. 100,000	3
Automobile particulates	298	3
Groundwater (ug/L)		
Near polymetallic sulfide		
deposits	Max. 400,000	3
Near gold mining activities	>50	2
USA	Usually <10	2
USA	17.9 (0.01-800)	3
Lake water (ug/L)		
Dissolved solids		
<2,000 mg/L	(0.0-100)	6
>2,000 mg/L	(0.1-2,000)	6
Lake Superior	(0.1-1.6)	6
Japan, various	(0.2-1.9)	6
Germany, Elbe River	(20-25)	6
Searles Lake, California	(198,000-243,000)	1,4
California, other lakes	(0.0-100)	1,4
Michigan	Max. 2.4	1,4
Wisconsin	(4-117)	1,4
Florida	Max. 3.6	1,4
Lake Chatauqua, New York	(3.5-35.6)	1.4
Lake Ohakuri, New Zealand	(30-60)	7
Finfeather Lake, Texas	Max. 240,000	8
Thermal waters, worldwide	(20-3,800)	
	Max. 276,000	1,2,3,9
Rain (ug/L)		
Canada	(0.01-5)	3
Rhode Island	0.8	1
Seattle, Washington	17	1
River water (ug/L)		
Polluted, USA	Max. 6,000	4
Nonpolluted, USA	Usually <5	4
Nationwide, USA, 1974-1981		
25th percentile	<1	10
50th percentile	1	10
75th percentile	3	10
Rock (mg/kg)		
Limestones	1.7 (0.1-20)	1

Sandstones	2 (0.6-120)	1
Shales and clays	14.5 (0.3-490)	1
Phosphates	22.6 (0.4-188)	1
Igneous, various	1.5-3 (0.06-113)	1
Seawater (ug/L)		
Worldwide	2 (0.15-6)	6
Pacific Ocean	(1.4-1.8)	11
Atlantic Ocean	(1.0-1.5)	11
South Australia		
Total dissolved As	1.3 (1.1-1.6)	12
As^{+5}	1.29	12
As^{+3}	0.03	12
Particulate As	<0.0006	12
U.K., Beaulieu estuary		
Water temperature <12° C		
Inorganic arsenic	(0.4-0.9)	13
Suspended arsenic	(0.02-0.24)	13
Organoarsenicals	(0.19-0.75)	13
Water temperature >12° C		
Inorganic arsenic	(0.6-1.1)	13
Suspended arsenic	(0.2-0.6)	13
Organoarsenicals	ND	13
Sediments (mg/kg dry weight)		
Near sewer outfall	35	3
From areas contaminated by smelteries, arsenical herbicides, or mine tailings		
Surface	(198-3,500)	1,7,9,14,15
Subsurface	(12-25)	1,7,9,14,15
Upper Mississippi River	2.6 (0.6-6.2)	16
Lake Michigan	(5-30)	1
Naturally elevated	>500	1,9
Oceanic	33.7 (<0.4-455)	3
Lacustrine	(5-26.9) Max. 13,000	3
Snow (mg/kg)		
Near smelter	>1,000	3
Soil pore waters (ug/L)		
Mineralized areas		
Arsenate	(79-210)	17
Arsenite	(2-11)	17
Monomethyl arsonic acid (MMAA)	(4-22)	17
Total arsenic	(93-240)	17

Unmineralized areas		
Arsenate	(18-49)	17
Arsenite	(1-7)	17
MMAA	<1	17
Total arsenic	(13-59)	17
Soils (mg/kg dry weight)		
USA, uncontaminated	7.4	18
Worldwide, uncontaminated	7.2	18
Canada		
Near Gold mine		
Air levels 3.9 mg As/m^3	21,213	3
80 km distant	(10-25)	3
Near smelter		
Japan	Max. 2,470	2
Tacoma, Washington	Max. 380	2
Treated with arsenical pesticides		
USA	165 (1-2,554)	6
Canada	121	6
Synthetic detergents (mg/kg)		

[a]Concentrations are listed as mean, minimum-maximum (in parentheses), and maximum (Max.).

[b]References: 1, NAS 1977; 2, Pershagen and Vahter 1979; 3, NRCC 1978; 4, EPA 1980; 5, Hood 1985; 6, Woolson 1975; 7, Freeman et al. 1986; 8, Sorensen et al. 1985; 9, Farmer and Lovell 1986; 10, Smith et al. 1987; 11, Sanders 1980; 12, Maher 1985a; 13, Howard et al. 1984; 14, Hallacher et al. 1985; 15, Takamatsu 1985; 16, Wiener et al. 1984; 17, Haswell et al. 1985; 18, Dudas 1984.

Table 2. Arsenic concentrations in field collections of selected species of flora and fauna. Values listed are in mg As/kg fresh weight (FW), or dry weight (DW).

Ecosystem, species, and other variables	Concentration in mg/kg[a]	Reference[b]
Terrestrial Plants		
Colonial bentgrass, *Agrostis tenuis*		
On mine waste site	1,480, Max. 3,470 DW	1
On low arsenic soil	(0.3-3) DW	1
Scotch heather, *Calluna vulgaris*		
On mine waste site	1,260 DW	1
On low arsenic soil	0.3 DW	1
Coontail, *Ceratophyllum demersum*		
From geothermal area, New Zealand	(20-1,060) DW	1
Cereal grains		
From arsenic treated areas	Usually <3 DW, Max. 252 DW	2
Nontreated areas	Usually <0.5 DW, Max. 5 DW	2
Grasses		
From arsenic treated areas	(0.5-60,000) DW	2
Nontreated areas	(0.1-0.9) DW	2
Apple, *Malus sylvestris*		
Fruit	<0.1 FW; <1.8 DW	1
Alfalfa, *Medicago sativa*		
USA	1.6 FW	1
Montana, smelter area	(0.4-5.7) FW	1
White spruce, *Picea alba*		
Arsenic-contaminated soil		
Branch	(2.8-14.3) DW	1
Leaf	(2.1-9.5)	1
Trunk	(0.3-55) DW	1
Root	(45-130) DW	1
Control site		
All samples	<2.4 DW	1
Pine, *Pinus silvestris*, needles		
Near U.S.S.R. metals smelter; soil levels 120.0 mg As/kg	22 FW	3
Trees		
Nontreated areas	Usually <1 DW	2
Lowbush blueberry, *Vaccinium angustifolium*		

Maine, leaf		
Arsenic-treated soil	(6.8-15) DW	1
Control	0.8 DW	1
Various species		
From uncontaminated soils	(<0.01-5) DW	2
From arsenic-impacted (80 mg/kg) soils	1.2 (<0.2-5.8) DW	4
Vegetables		
From arsenic-treated areas	Usually <3 DW, Max. 145 DW	2
Nontreated areas	Usually <1 DW, Max. 8 DW	2
Vegetation		
Near gold mine, Canada, Air levels up to 3.9 mg As/m^3	Max. 11,438 DW	5
80 km distant	(12-20) DW	5
Freshwater Flora		
Aquatic Plants		
Arsenic-treated areas	(20-1,450) DW	2
Untreated areas	(1.4-13) DW	2
Irish moss, *Chondrus crispus*		
Whole	(5-12) DW	1
Pondweeds, *Potamogeton* spp.		
Whole		
Near geothermal area	(11-436) DW	1
Control site	<6 DW	
Freshwater Fauna		
Alewife, *Alosa pseudoharengus*		
Whole, Michigan	0.02 FW	1
Muscle, Wisconsin	0 FW	1
White sucker, *Catostomus commersoni*		
Muscle	(0.03-0.13) FW	1
Whole	(0.05-0.16) FW	1
Common carp, *Cyprinus carpio*		
Upper Mississippi River, 1979		
Whole	0.4 (0.2-0.6) DW	6
Liver	0.4 (0.3-1) DW	6
Nationwide		
Whole	0.05 FW	1
Muscle	(0.0-0.2) FW	1
Northern pike, *Esox lucius*		
Muscle		
Canada	(0.05-0.09) FW	1

Great Lakes	<0.05 FW	1
Sweden	0.03 FW	1
New York	<0.1 FW	1
Wisconsin	<0.01 FW	1
Fish, various species		
Whole	Max. 1.9 FW	2
Whole	(0.04-0.2) FW	7
Netherlands, 1977-1984		
Muscle	(0.04-0.15) FW	8
Nationwide, USA		
Whole, 1976-1977	(0.05-2.9) FW	9
Near smelter (water arsenic 2.3-2.9 mg/L)		
Muscle, 3 species		
Total arsenic	0.05-0.24) FW	10
Inorganic arsenic	(0.01-0.02) FW	10
Liver, 2 species		
Total arsenic	0.15 FW	10
Inorganic arsenic	0.01 FW	10
Control location (water arsenic <0.5 mg/L)		
Muscle		
Total arsenic	(0.06-0.09) FW	10
Inorganic arsenic	<0.03 FW	10
Liver		
Total arsenic	0.09 FW	10
Inorganic arsenic	<0.01 FW	10
Channel catfish, *Ictalurus punctatus*		
Muscle		
Native	(0.0-0.3) FW	1
Cultured	(0.2-3.1) FW	1
Whole, nationwide	(<0.05-0.3) FW	1
Green sunfish, *Lepomis cyanellus*, liver		
Polluted waters (from manufacturer of arsenical defoliants and pesticides), Texas. Mean water concentration 13.5 mg As/L; sediment content of 4,700 mg/kg		
Age 1 to 2	(19.7-64.2) DW	11
Age 3	15 DW	11

Age 4	(6.1-11.5) DW	11
Bluegill, *Lepomis macrochirus*		
From pools treated with arsenic		
Muscle	1.3 FW	1
Skin and scales	2.4 FW	1
Gills and GI tract	17.6 FW	1
Liver	11.6 FW	1
Kidney	5.9 FW	1
Ovary	8.4 FW	1
Control locations		
All tissues	<0.2 FW	1
Whole		
Nationwide	(<0.05-0.15) FW	1
Upper Mississippi River, 1979	0.3 (0.2-0.4) DW	6
Smallmouth bass, *Micropterus dolomieui*		
Muscle		
Wisconsin	<0.13 FW	1
Lake Erie	0.22 FW	1
New York	(0.03-0.51) FW	1
Whole, nationwide	(<0.05-0.28) FW	1
Largemouth bass, *Micropterus salmoides*		
Whole, nationwide	(<0.05-0.22) FW	1
Muscle		
Wisconsin	(0.0-0.12) FW	1
New York	(0.03-0.16) FW	1
Striped bass, *Morone saxatilis*		
Muscle	(0.2-0.7) FW	1
Coho salmon, *Oncorhynchus kisutch*		
Muscle		
Wisconsin	<0.15 FW	1
Lake Erie	(0.07-0.17) FW	1
New York	<0.5 FW	1
USA	0.09 FW	1
Yellow perch, *Perca flavescens*		
All tissues	<0.16 FW	1
Rainbow trout, *Salmo gairdneri*		
All tissues	<0.4 FW	1
Atlantic salmon, *Salmo salar*		
Oil		

Liver	6.7 FW	1
Muscle	(0.8-3.1) FW	1
Lake trout, *Salvelinus namaycush*		
Whole, nationwide	(0.06-0.68) FW	1
Marine Flora		
Algae		
Green	(0.5-5) DW	2
Brown	Max. 30 DW	2
11 species	(2-58) DW	12
Various species	(10-100) DW	13
Seaweed, *Chondrus crispus*		
Alga, *Fucus* spp.		
Oil	(6-27) FW	2
Fatty acid	(5-6) FW	2
Brown alga, *Fucus vesiculosus*		
Whole	(35.2-80) DW	1
Brown alga, *Laminaria digitata*		
Whole	94 DW	2
Whole	(42-50) DW	1
Oil	(155-221) DW	2
Fatty acid	(8-36) DW	2
Alga, *Laminaria hyperborea*		
Total arsenic	142 DW	12
Organic arsenic	139 DW	12
Sargassum weed, *Sargassum fluitans*		
Total arsenic	19.5 FW	7
As^{+3}	1.8 FW	7
As^{+5}	17.7 FW	7
Organoarsenicals	0.2 FW	7
Seaweed, *Sargassum* sp.		
Total arsenic	(4.1-8.7) FW	5
As+3	(0.14-0.35) FW	5
As+5	(1.9-7.3) FW	5
Organoarsenicals	Max. 0.1 FW	5
Seaweeds		
Whole	(3.8-93.8) DW	2
Whole	(10-109) DW	12
Oil fraction	(5.7-221) FW	12
Marine Molluscs		
Ivory shell, *Buccinum striatissimum*		
Muscle		
Total arsenic	38 FW	14

Arsenobetaine	24.2 FW	14
Midgut gland		
Total arsenic	18 FW	14
Arsenobetaine	10.8 FW	14
Oysters, *Crassostrea* spp.		
Soft parts	(1.3-10) DW, (0.3-3.4) FW	12
American oyster, *Crassostrea virginica*		
Soft parts	2.9 FW	1
Soft parts	10.3 DW	15
Spindle shells, *Hemifusus* spp.		
Hong Kong 1984, Muscle		
Total arsenic	Max. 500 FW	16
Inorganic arsenic	<0.5 FW	16
Limpet, *Littorina littorea*		
Soft parts		
Near arsenic source	11.5 DW	12
Offshore	4 DW	12
Squid, *Loligo vulgaris*		
Soft parts	(0.8-7.5) FW	1
Hardshell clam, *Mercenaria mercenaria*		
Soft parts		
Age 3 years	3.8 DW	12
Age 4 years	4.7 DW	12
Age 10 years	9.3 DW	12
Age 15 years	8.4 DW	12
Molluscs, edible tissues		
Hong Kong, 1976-1978		
Bivalves	(3.2-39.6) FW	17
Gastropods	(19-176) FW	17
Cephalopods	(0.7-5.5)	17
USA		
6 species	(2-3) FW	18
8 species	(3-4) FW	18
3 species	(4-5) FW	18
4 species	(7-20) FW	18
Mussel, *Mytilus edulis*		
Soft parts	2.5 (1.4-4.6) FW	8
Soft parts	(1.6-16) DW	12
Scallop, *Placopecten magellanicus*		
Soft parts	1.6 (1.3-2.4) FW	1

Marine Crustaceans		
Blue crab, *Callinectes sapidus*		
Florida, whole	7.7 FW	1
Maryland, soft parts	(0.5-1.8) FW	1
Dungeness crab, *Cancer magister*		
Muscle	6.5 (2.2-37.8) FW	1
Muscle	4 FW	19
Alaskan snow crab, *Chinocetes bairdii*		
Muscle	7.4 FW	19
Copepods, whole	(2-8.2) DW	12
	(0.4-1.3) FW	12
Shrimp, *Crangon crangon*		
Netherlands, 1977-1984		
Muscle	3 (2-6.8) FW	8
Crustaceans, edible tissues		
Hong Kong, 1976-1978		
Crabs	(5.4-19.1) FW	17
Lobsters	(26.7-52.8) FW	17
Prawns and shrimps	(1.2-44) FW	17
USA		
6 species	(3-5) FW	18
3 species	(5-10) FW	18
4 species	(10-20) FW	18
2 species	(20-30) FW	18
1 species	(40-50) FW	18
American lobster, *Homarus americanus*		
Muscle	(3.8-7.6) DW,	
	Max. 40.5 FW	1
Hepatopancreas	22.5 FW	1
Whole	(3.8-16) DW,	12
	(1-3) FW	12
Stone crab, *Menippe mercenaria,* whole	(9-11.8)FW	1
Deep seaprawn, *Pandalus borealis*		
Head and shell	68.3 DW	1
Muscle	61.6 DW	1
Oil	42 DW, 10.1 FW	1
Egg	3.7-14 FW	1
Prawns, *Pandalus* spp.		
Whole	(7.3-11.5) FW	12
Alaskan king crab, *Paralithodes camtschatika*		
Muscle	8.6 FW	19

Brown shrimp, *Penaeus aztecus*		
Muscle	(3.1-5.2) FW	1
Whole	0.6 DW	1
White shrimp, *Penaeus setiferus*		
Muscle		
Mississippi	(1.7-4.4) FW	1
Florida	(2.8-7.7) FW	1
Shrimp, *Sergestes lucens*		
Muscle		
Total arsenic	5.5 FW	20
Arsenobetaine	4.5 FW	20
Shrimps		
Exoskeleton	15.3 FW	7
Muscle, 2 species	(18.8-41.6) FW, (3.8-128) DW	2
Marine Fishes		
Whitetip shark, *Carcharhinus longimanus*		
Muscle	3.1 FW	21
Black sea bass, *Centropristis striata*		
Muscle	6.4 DW	1
Elasmobranchs		
Muscle		
Sharks	Max. 30 FW	12
Rays	Max. 16.2 FW	12
Roundnose flounder, *Eopsetta grigorjewi*		
Muscle	20.1 FW	22
Finfishes		
Near metal smelter, water concentration 2.3-2.9 mg As/L		
Muscle, 6 species		
Total arsenic	(0.2-2.6) FW	10
Inorganic arsenic	(0.02-0.1) FW	10
Liver, 4 species		
Total arsenic	(0.4-1.8) FW	10
Inorganic arsenic	(0.02-0.07) FW	10
Control location, water concentration <2.0 mg As/L		
Muscle, 5 species		
Total arsenic	(0.1-1.2) FW	10
Inorganic arsenic	(0.02-0.15) FW	10
Liver, 4 species		
Total arsenic	(0.2-1.5) FW	10

Inorganic arsenic	(0.02-0.05) FW	10
Finfish, Hong Kong, 1976-1978		
Edible tissues	Max. 21.1 FW	17
Finfish, Netherlands, 1977-1984		
Muscle, 4 species	(2.8-10.9) FW	8
Finfish, North America		
Liver		
49 species	(0.7-5) FW	18
26 species	(5-20) FW	18
6 species	(20-50) FW	18
Muscle		
91 species	(0.6-4) FW	18
41 species	(4-8) FW	18
27 species	(8-30) FW	18
4 species		
Total arsenic	(1.4-10) FW	23
Inorganic arsenic	<0.5 FW	23
Whole		
16 species	(1-8) FW	18
Finfish, worldwide		
Various tissues		
Total arsenic	(ND-142) FW	2
Inorganic arsenic	(0.7-3.2) FW	2
Organic arsenic	(3.4-139) FW	2
Atlantic cod, *Gadus morhua*		
Muscle	2.2 FW	2
Liver	9.8 FW	2
Blue pointer, *Isurus oxyrhinchus*		
Muscle	9.5 FW	21
Striped bass, *Morone saxatilis*		
Muscle	(0.3-0.5) FW, 1.8 DW	12
Liver	0.7 FW	12
Striped mullet, *Mugil cephalus*		
Viscera	Max. 1.3 FW	24
English sole, *Parophrys vetulus*		
Muscle	1.1 (0.6-11.5) FW	1
Skate, *Raja* sp.		
Muscle	16.2 FW	1
Windowpane flounder, *Scopthalmus aquosus*		
Muscle	(1.4-2.8) FW	1

Spiny dogfish, *Squalus acanthias*		
Muscle	10 DW	25
Liver	5.7 DW	25
Spleen	9.8 DW	25
Yolk sac	9.1 DW	25
Embryo	2.6 DW	25
Amphibians and Reptiles		
Alligator, *Alligator mississippiensis*		
Egg	(0.05-0.2) FW	1
Crocodile, *Crocodylus acutus*		
Egg	0.2 FW	26
Frogs, *Rana* spp.		
All tissues	<0.4 FW	1
Toads, 2 species		
All tissues	<0.05 FW	
Birds		
American black duck, *Anas rubripes*		
Egg	0.2 FW	12
Ducks, *Anas* spp.		
All tissues	<0.4 FW	1
Scaup, *Aythya* spp.		
All tissues	<0.4 FW	1
Gulls, 3 species		
Oil	(0.6-13.2) FW	12
Osprey, *Pandion haliaetus*		
Liver	Max. 16.7 FW	27
Brown pelican, *Pelecanus occidentalis*		
Egg		
South Carolina, 1971-72	0.3 (0.08-0.8) FW	28
Florida, 1969-70	0.1 (0.07-0.2) FW	28
Liver, 1972-72,		
GA, FL, SC		
Found dead	(0.2-1) FW	28
Shot	(0.3-0.9) FW	28
Shorebirds		
Corpus Cristi, Texas, 1976-1977		
Liver, 7 species	(0.05-1.5) FW	29
New Zealand, 5 species		
Feather	<1 FW	12
Liver	Max. 2.6 FW	12
Starling, *Sturnus vulgaris*		
Whole, nationwide, USA, 1971	(<0.01-0.21) FW	2
Icelandic redshank, *Tringa totanus robusta*		

Netherlands, 1979-1982		
Feather		
Juveniles	Max. 0.8 FW	30
Adults	(0.5-3.2) FW	30
Mammals		
Fin whale,		
Balaenoptera physalis		
Blubber oil	1.8 FW	1
Cow, *Bos bovis*		
Downwind from copper smelter		
16-21 km		
Hair	8.9 FW	1
Milk	0.013 FW	1
Blood	0.026 FW	1
60 km		
Hair	0.46 FW	1
Milk	0.002 FW	1
Blood	0.009 FW	1
Controls		
Milk	<0.001 FW	31
Muscle	0.005 FW	31
Liver	(0.008-0.012) FW	31
Kidney	(0.017-0.053) FW	31
Domestic animals		
All tissues	<0.3 FW	2
Livestock		
All tissues	<0.6 FW	2
Marine mammals		
Pinnipeds		
All tissues	Max. 1.7 FW	12
Cetaceans		
Muscle	0.4 DW	12
Oil	(0.6-2.8) FW	12
White-tailed deer,		
Odocoileus virginianus		
Tennessee, killed from		
arsenic herbicide		
Liver	19 FW	1
Kidney	17.8 FW	1
Rumen contents	22.5 FW	1
Harbor seal,		
Phoca vitulina		
UK, all tissues	<0.3 FW	1

Fox, *Vulpes* sp.		
All tissues	<0.7 FW	1
Wildlife		

[a]Concentrations are listed as mean, minimum-maximum (in parentheses), and maximum (Max.).

[b]References: 1, Jenkins 1980; 2, NAS 1977; 3, Mankovska 1986; 4, Merry et al. 1986; 5, NRCC 1978; 6, Wiener et al. 1984; 7, Woolson 1975; 8, Vos and Hovens 1986; 9, Lima et al. 1984; 10, Norin et al. 1985; 11, Sorensen et al. 1985; 12, Eisler 1981; 13, Pershagen and Vahter 1979; 14, Shiomi et al. 1984a; 15, Zaroogian and Hoffman 1982; 16, Phillips and Depledge 1986; 17, Phillips et al. 1982; 18, Hall et al. 1978; 19, Francesconi et al. 1985; 20, Shiomi et al. 1984b; 21 Hanaoka and Tagawa 1985a; 22, Hanaoka and Tagawa 1985b; 23, Reinke et al. 1975; 24, Hallacher et al. 1985; 25, Windom et al. 1973; 26, Hall 1980; 27, Wiemeyer et al. 1980; 28, Blus et al. 1977; 29, White et al. 1980; 30, Goede 1985; 31, Vreman et al. 1986.

Table 3. Toxic and sublethal effects of various arsenic compounds on selected species of terrestrial plants and invertebrates.

Ecosystem, species, and other variables	Arsenic concentration and effects	Reference[a]
Terrestrial Plants		
Crops		
Total water soluble As in soils	Depressed crop yields at 3 to 28 mg/L	1
Total soil As concentrations	Depressed crop yields at 25 to 85 mg/kg	1
Common bermudagrass, *Cynodon dactylon*		
Arsenite	Plants grown on As- amended soils (up to 90 mg As^{+3}/kg) contained up to 17 mg As/kg dry weight in stems, 20 in leaves, and 304 in roots	2
Fruit orchards		
Inorganic arsenites and arsenates	Soils contain 31 to 94 mg/kg dry weight (vs. 2.4 in untreated orchards); whole rodents contain <0.002 mg As/kg fresh weight (vs. nondetectable in untreated orchards)	3
Soybean, *Glycine max*		
Total As	Toxic signs at plant residues >1 mg total As/kg	1
Grasslands		
Cacodylic acid	Kill of 75% to 90% of all species at 17 kg/ha; recovery modest	3
Rice, *Oryza sativum*		
Disodium methylarsonate	75% decrease in yield at soil (silty loam) concentrations of 50 mg/kg	1

Scots pine, *Pinus sylvestris*		
Inorganic As^{+5}	Seedlings die when soil (sandy) concentrations exceed 250 mg/kg dry weight. Maximum BCF factors low: 0.6 for roots; 0.1 for shoots. Residues >62 mg As/kg DW in shoots are toxic, and 3,300 mg/kg DW usually fatal	4
Pea, *Pisum sativum*		
Sodium arsenite	15 mg/L inhibits light activation and photosynthetic CO_2 fixation in chloroplasts	5
Sandhill plant communities		
Cacodylic acid	No lasting effect at 2.25 kg/ha. Some species defoliated st 6.8 kg/ha. Significant effect, including 75% defoliation of oaks and death of all pine trees, at 34 kg/ha	3
Cowpea, *Vigna* sp.		
Total water soluble As in soils	Decreased yields at 1 mg/L	1
Total soil As concentrations (loamy sand)	Toxic at 25 mg/kg	1
Yeast		
Arsenate	At 75 mg/L, 60% reduction in phosphate transport and glucose metabolism in 30 min; at 375 mg/L, 100% reduction	1
Terrestrial Invertebrates		
Honeybee, *Apis mellifera*		
Inorganic arsenite	Following arsenic spray dusting, dead bees contained 20.8 to 31.2 mg/kg FW (adults) or 5 to 13 mg/kg FW (larvae)	6
Beetles		
Cacodylic acid	Dietary levels of 100 to 1,000 mg/kg fatal to certain pestiferous species	3
Western spruce budworm, *Christoneura occidentalis*, sixth instar stage		
Arsenic trioxide	Dietary levels of 99.5 mg/kg FW killed 10%, 2,250 mg/kg killed 50%, and 65,300 mg/kg was fatal to 90%. Newly molted pupae and adults of As-exposed larvae had reduced weight. Regardless of dietary levels, concentrations of As ranged up to 2,640 mg/kg DW in dead pupae, and 1,708 mg/kg DW in adults	7

[a]References: 1, NRCC 1978; 2, Wang et al. 1984; 3, Hood 1985 4, Sheppard et al. 1985; 5, Marques and

Anderson 1986; 6, Jenkins 1980; 7, Robertson and McLean 1985.

Table 4. Lethal and sublethal effects of various arsenic compounds on selected species of aquatic biota.

Ecosystem, species, arsenic compound, and other variables	Arsenic concentration	Effect	Reference[b]
Freshwater Plants			
Algae, various species			
As^{+3}	1.7 mg/L	Toxic	1
As^{+3}	4 mg/L	Decomposition	1
As^{+3}	2.3 mg/L	95% to 100% kill in 2 to 4 weeks of 4 species	2,3
As^{+5}	0.075 mg/L	Decreased growth	3
Alga, *Ankistrodesmus falcatus*			
As^{+5}	0.26 mg/L	EC-50 (14 days)	3
Alga, *Scenedesmus obliquus*			
As^{+5}	0.048 mg/L	EC-50 (14 days)	3
Alga, *Selenastrum capricornutum*			
As^{+5}	0.69 mg/L	EC-50 (4 days)	3
Freshwater Invertebrates			
Cladoceran, *Bosmina longirostris*			
As^{+5}	0.85 mg/L	50% immobilization in 96 h	4
Cladoceran, *Daphnia magna*			
As^{+3}	0.63 to 1.32 mg/L	MATC[c]	3
As^{+3}	0.96 mg/L	LC-5 (28 days)	5
As^{+3}			
Starved	1.5 mg/L	50% immobilization (96 h)	6
Fed	4.3 mg/L	50% immobilization (96 h)	6
As^{+5}	0.52 mg/L	Reproductive impairment of 16% in 3 weeks	3
As^{+5}	0.93 mg/L	LC-5 (28 days); maximum bioconcentration factor (BCF) of 219X	5
As^{+5}	7.4 mg/L	LC-50 (96 h)	2

DSMA	0.83 mg/L	LC-0 (28 days)	5
SDMA	1.1 mg/L	LC-0 (28 days)	5
Total As	1 mg/L	18% decrease in body weight in 3 weeks	1
Total As	1.4 mg/L	50% reproductive impairment in 3 weeks	1
Total As	2.8 mg/L	LC-50 (21 days)	1
Total As	4.3 to 7.5 mg/L	Immobilization (21 days)	1
Cladoceran, *Daphnia pulex*			
As^{+5}	49.6 mg/L	50% immobilization (48 h)	4
As^{+3}	1.3 mg/L	LC-50 (96 h)	2,3
As^{+3}	3 mg/L	EC-50 (48 h)	7
Amphipod, *Gammarus pseudolimnaeus*			
As^{+3}	0.87 mg/L	50% immobilization (96 h)	6
As^{+3}	0.088 mg/L	LC-20 (28 days)	5
As^{+3}	0.96 mg/L	LC-100 (28 days)	5
As^{+5}	0.97 mg/L	LC-20 (28 days); no accumulations	5
DSMA	0.086 mg/L	LC-10 (28 days)	5
DSMA	0.97 mg/L	LC-40 (28 days)	5
SDMA	0.85 mg/L	LC-0 (28 days)	5
Snail, *Helisoma campanulata*			
As^{+3}	0.96 mg/L	LC-10 (28 days)	5
As^{+5}	0.97 mg/L	LC-0 (28 days); maximum BCF of 99X	5
DSMA	0.97 mg/L	LC-0 (28 days)	5
SDMA	0.085 mg/L	LC-0 (28 days)	5
SDMA	0.085 mg/L	LC-32 (28 days)	5
Stonefly, *Pteronarcys californica*			
As^{+3}	38 mg/L	LC-50 (96 h)	7
Stonefly, *Pteronarcys dorsata*			
As^{+3}	0.96 mg/L	LC-0 (28 days)	5
As^{+5}	0.97 mg/L	LC-20 (28 days); maximum BCF of 131X	5

DSMA	0.97 mg/L	LC-0 (28 days)	5
SDMA	0.85 mg/L	LC-0 (28 days)	5
Cladoceran, *Simocephalus serrulatus*			
As^{+3}	0.81 mg/L	LC-50 (96 h)	3
Zooplankton			
As^{+3}	0.4 mg/L	No effect	1
As^{+3}	1.2 mg/L	Population reduction	1
Freshwater Vertebrates			
Marbled salamander, *Ambystoma opacum*			
As^{+3}	4.5 mg/L	EC-50 (8 days) concentration producing death and malformations in developing embryos	3
Goldfish, *Carassius auratus*			
As^{+5}	0.1 mg/L	15% behavioral impairment in 24 h; 30% impairment in 48 h	1
As^{+5}	24.6 to 41.6 mg/L	LC-50 (7 days)	1
As^{+3}	0.49 mg/L	EC-50 (7 days)	3
MSMA	5 mg/L	LC-50 (96 h)	3
Narrow-mouthed toad, *Gastrophryne carolinensis*			
As^{+3}	0.04 mg/L	50% death or mal-formations noted in developing embryos in 7 days	3
Channel catfish, *Ictalurus punctatus*			
As^{+3}	25.9 mg/L	LC-50 (96 h)	8
Flagfish, *Jordanella floridae*			
As^{+3}	14.4 mg/L	LC50 (96 h)	6
As^{+3}	2.1 to 4.1 mg/L	MATC[c]	3
Bluegill, *Lepomis macrochirus*			
As^{+3}			
Juveniles	0.69 mg/L	Reduced survival 16 weeks after a single treatment	2,3
Adults	0.69 mg/L	Histopathology after 16 weekly treatments	2

As^{+3}	4 mg/L	Population reduction of 42% after several monthly applications	8
As^{+3}	30 to 35 mg/L	LC-50 (96 h)	7,8
MSMA	1.9 mg/L	LC-50 (96 h)	3
Total As	Tissue residues of 1.35 mg/kg fresh weight (juveniles) and 5 mg/kg (adults)	Threshold acute toxic value	1
Spottail shiner, *Notropis hudsonius*			
As^{+3}	45 mg/L	LC-50 (25 h)	8
As^{+3}	29 mg/L	LC-50 (48 h); survivors with fin and scale damage	8
Chum salmon, *Oncorhynchus keta*			
As^{+3}	11 mg/L	LC-50 (48 h)	8
Minnow, *Phoxinus phoxinus*			
As^{+3}	20 mg/L	Equilibrium loss in 36 h	8
As^{+5}	234 to 250 mg/L	Lethal	8
Fathead minnow, *Pimephales promelas*			
As^{+3}	14.1 mg/L	LC-50 (96 h)	6
As^{+3}	2.1 to 4.8 mg/L	MATC[c]	6
As^{+5}	25.6 mg/L	LC-50 (96 h)	3
As^{+5}	0.53 to 1.50 mg/L	MATC[c]	3
Rainbow trout, *Salmo gairdneri*			
As^{+3}	0.13 mg/L	Ec-10 (28 days)	3
As^{+3}			
Embryos	0.54 mg/L	LC-50 (28 days)	2
Adults	23 to 26.6 mg/L	LC-50 (96 h)	5
As^{+3}	0.96 mg/L	LC-50 (28 days)	7,8
As^{+3} or As^{+5}	Fed diets containing 120 to 1,600 mg As/kg for 8 weeks	Growth depression, food avoidance, and impaired feed efficiency at all levels	9
As^{+5}	Fed diets containing 10 to	No effect level at about 10 mg/kg diet.	

	90 mg As/kg for 16 weeks	Some adaptation to dietary As observed in trout fed 90 mg/kg diet, as initial negative growth gave way to slow positive growth over time	9
As^{+5}	0.97 mg/L	LC-0 (28 days); no accumulations	5
DSMA	0.97 mg/L	LC-0 (28 days)	5
SDMA	0.85 mg/L	LC-0 (28 days)	5
SC	1,000 mg/L	LC-0 (28 days)	14
DMA or ABA	Fed diet containing 120 to 1,600 mg/kg for 8 weeks	No toxic response at any level tested	9
Brook trout, *Salvelinus fontinalis*			
As^{+3}	15 mg/L	LC-50 (96 h)	3
Marine Plants			
Algae, 2 spp.			
As^{+3} or As^{+5}	1 mg/L	No effect	10
As^{+5}	1,000 mg/L	No deaths	10
Algae, 3 spp.			
As^{+3}	0.019 to 0.022 mg/L	Reduced growth	3
Red alga, *Champia parvula*			
As^{+3}	0.065 mg/L	Normal sexual reproduction	10
As^{+3}	0.095 mg/L	No sexual reproduction	10
As^{+3}	0.30 mg/L	Death	10
As^{+5}	10 mg/L	Normal growth, but no sexual reproduction	10
Phytoplankton			
As^{+5}	0.075 mg/L	Reduced biomass of populations in 4 days	3
Red alga, *Plumaria elegans*			
As^{+3}	0.58 mg/L	Arrested sporeling development 7 days posttreatment after exposure for 18 h	12

Alga, *Skeletonema costatum*			
As^{+5}	0.13 mg/L	Growth inhibition	3
Alga, *Thalassiosira aestivalis*			
As^{+5}	0.075 mg/L	Reduced chlorophyll a	3
Marine Invertebrates			
Copepod, *Acartia clausi*			
As^{+3}	0.51 mg/L	LC-50 (96 h)	3
Dungeness crab, *Cancer magister*			
As^{+3}	0.23 mg/L	LC-50 (96 h) for zoea	3
Amphipod, *Corophium volutator*			
As^{+5}			
Water temperature, °C			
5	8 mg/L	LC-50 (230 h)	11
10	8 mg/L	LC-50 (150 h)	11
15	8 mg/L	LC-50(74 h)	11
15	4 mg/L	LC-50 (140 h)	11
15	2 mg/L	LC-50 (192 h)	11
Pacific oyster, *Crassostrea gigas*			
As^{+3}	0.33 mg/L	LC-50 (96 h) for embryos	3
American oyster, *Crassostrea virginica*			
As^{+3} (eggs)	7.5 mg/L	LC-50 (48 h)	8
Copepod, *Eurytemora affinis*			
As^{+5}	0.025 mg/L	No effect	12
As^{+5}	0.1 mg/L	Reduced juvenile survival	12
As^{+5}	1 mg/L	Reduced adult survival	12
Clam, *Macoma balthica*			
As^{+5}			
Water temperature, °C			
5	220 mg/L	LC-50 (192 h)	11
10	60 mg/L	LC-50 (192 h)	11
15	15 mg/L	LC-50 (192 h)	11
Mysid, *Mysidopsis bahia*			
As^{+3}	0.63 to 1.27 mg/L	MATC[c]	3
As^{+5}	2.3 mg/L	LC-50 (96 h)	3
Blue mussel, *Mytilus edulis*			
As^{+3}	16 mg/L	Lethal in 3 to 16 days	8
Mud snail, *Nassarius obsoletus*			
As^{+3}	2 mg/L	Depressed oxygen	

		consumption in 72 h	8
Oligochaete annelid, *Tubifex costatus*			
As^{+5}			
Water temperature, °C			
5	500 mg/L	LC-50 (130 h)	11
10	500 mg/L	LC-50 (115 h)	11
15	500 mg/L	LC-50 (85 h)	11
Marine Vertebrates			
Grey mullet, *Chelon labrosus*			
As^{+3}	27.3 mg/L	LC-50 (96 h); some skin discoloration	13
Dab, *Limanda limanda*			
As^{+3}	28.5 mg/L	LC-50 (96 h); respiratory problems	13
Pink salmon, *Oncorhynchus gorbuscha*			
As^{+3}	2.5 mg/L	LC-0 (10 days)	8
As^{+3}	3.8 mg/L	LC-54 (10 days)	3
As^{+3}	7.2 mg/L	LC-100 (7 days)	3
Teleosts, 3 spp.			

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[a]As^{+3}, inorganic trivalent arsenite; As^{+5}, inorganic pentavalent arsenate; DMA, dimethylarsinic acid; ABA, p-aminobenzenearsonic acid; DMSA, disodium methylarsenate ($CH_3AsO(ONa)_2$); SDMA, sodium dimethylarsenate ($(CH_3)_2AsO(ONa)$); MSMA, monosodium methanearsonate; SC, sodium cacodylate.

[b]References: 1, NRCC 1978; 2, EPA 1980; 3, EPA 1985; 4, Passino and Novak 1984; 5, Spehar et al. 1980; 6, Lima et al. 1984; 7, Johnson and Finley 1980; 8, NAS 1977; 9, Cockell and Hilton 1985; 10, Thursby and Steele 1984; 11, Bryant et al. 1985; 12, Sanders 1986; 13, Taylor et al. 1985; 14, Hood 1985.

[c]Maximum acceptable toxicant concentration. Lower value in each pair indicates highest concentration tested producing no measurable effect on growth, survival, reproduction, or metabolism during chronic exposure; higher value indicates lowest concentration tested producing a measurable effect.

Table 5. Lethal and sublethal effects of various arsenicals on selected species of birds.

Species and arsenic compound	Effect	Reference[a]
Chukar, *Alectoris chukar*		
Silvisar-510 (mixture of cacodylic acid and tri-ethanolamine cacodylate)	Single oral LD-50 dose of ~2,000 mg/kg body weight (BW); signs of poisoning evident within 10 min and mortalities within 1 to 2 days postadminis-tration. Remission took up to 1 month	1
Mallard, *Anas platyrhynchos*		
Sodium arsenite	323 mg/kg BW is LD-50 acute oral value	1,2,3
Sodium arsenite	500 mg/kg diet is fatal to 50% in 32 days; 1,000 mg/kg diet fatal to 50% in 6 days	2
Sodium cacodylate	1,740 to 5,000 mg/kg diet fatal to 50% in 5 days	4
Silvisar 510	Single oral LD-50 >2,400 mg/kg BW; regur-gitation and excessive drinking noted	1
Lead arsenate	5,000 mg/kg diet not fatal in 11 days	2
Copper acetoarsenite	5,000 mg/kg diet fatal to 20% in 11 days	2
California quail, *Callipepla californica*		
Sodium arsenite	LD-50 single oral dose of 47.6 mg/kg BW	1
Northern bobwhite, *Colinus virginianus*		
Copper acetoarsenite	480 mg/kg in diet fatal to 50% in 11 days	2
Sodium cacodylate	1,740 mg/kg in diet for 5 days produced no effect on behavior, no signs of intoxication, and negative necropsy	4

Monosodium methanearsonate, CH_4AsNaO_3	Single oral LD-50 dose of 3,300 mg/kg BW	4
Chicken, *Gallus gallus*		
Inorganic trivalent arsenite	Up to 34% dead embryos at dose range of 0.01-1 mg As^{+3}/embryo; threshold for malformations at dose range 0.03-0.3 mg/embryo	3
Inorganic pentavalent arsenate	Up to 8% dead at dose range 0.01-1 mg As^{+5}/embryo; threshold for malformations at dose range 0.3-3 mg/embryo	3
Disodium methyl arsenate	Teratogenic to embryos when injected at 1 to 2 mg/egg	3,4
Sodium cacodylate	Developmental abnormal-ities at embryonic injected doses of 1 to 2 mg/egg	4
Dodecylamine p-chlorophenylarsonate	At dietary levels of 23.3 mg/kg, liver residues were 2.9 mg/kg FW at 9 weeks. No ill effects noted	5
3-Nitro-4-hydroxy phenylarsonic acid	At 18.7 mg/kg diet for 9 weeks, liver residues of 2.4 mg/kg FW. Those fed diets containing 187 mg/kg for 9 weeks had no ill effects; liver content of 7.5 mg/kg FW	5
3-Nitro-4-hydroxy phenylarsonic acid	LC-50 dose of 33 mg/kg BW (single oral) or 9.7 mg/kg BW (intraperitoneal injection)	2
Arsanilic acid	Fed diets containing 45 mg/kg for 9 weeks; no effect except slightly elevated liver content of 1.2 mg/kg fresh weight. At dietary levels of 455 kg/mg, liver residues were 6.4 mg/kg FW after 9 weeks; no other effects evident	5
Cacodylic acid	Dosed orally without effect at 100 mg/kg BW daily for 10 days	4

Chickens, *Gallus* spp.		
Arsanilic acid	50% excreted in 36 to 38 h	3
Arsenate	50% excreted in 60 to 63 h	3
Turkey, *Meleagris gallopavo*		
3-Nitro-4-hydroxy phenylarsonic acid	Single oral LD-50 dose of 17.4 mg/kg BW	2
Brown-headed cowbird, *Molothrus ater*		
Copper acetoarsenite	All survived 11 mg/kg diet for 6 months; maximum whole body residue of 1.7 mg As/kg dry weight	2
Copper acetoarsenite	All survived 33 mg/kg diet for 6 months (whole body content of 6.6 mg As/kg dry weight) or 7 months (8.6 DW)	2
Copper acetoarsenite	99.8 mg/kg in diet fatal to 50% in 11 days	2
Copper acetoarsenite	100 mg/kg in diet for 3 months fatal to 100%; tissue residues of 6.1 dry weight in brain, 40.6 in liver	2
Gray partridge, *Perdix perdix*		
Lead arsenate	300 mg/kg BW fatal in 52 h	2
Ring-necked pheasant, *Phasianus colchicus*		
Sodium arsenite	Single oral dose of 386 mg/kg BW is LD-50 value	1
Copper acetoarsenite	Single oral dose of 1.403 mg/kg BW is LD-50 value	3

[a]References: 1, Hudson et al. 1984; 2, NAS 1977; 3, NRCC 1978; 4, Hood 1985; 5, Woolson 1975.

Table 6. Lethal and sublethal effects of various arsenicals on selected species of mammals.

Organism and arsenical	Effect	Reference[a]
Cow, *Bos bovis*		
Arsenate	Cows fed 33 mg As^{+5} daily per animal for 3 months had slightly elevated levels in muscle (0.02 mg/kg fresh weight vs. 0.05 in controls) and liver (0.03 vs. 0.012), but normal levels in milk and kidney	1
Arsenite	Cows fed 33 mg As^{+3} daily per animal for 15 to 28 months had tissue levels, in mg/kg fresh weight, of 0.002 for milk (vs. <0.001 for controls), 0.03 for muscle (vs. 0.005), 0.1 for liver (vs. 0.012), and 0.16 for kidney (vs. 0.053)	1
Cattle, *Bos* spp.		
Arsenic pentoxide (wood ashes treated with As preservative)	Several deaths after eating wood ashes (780 mg/kg dry weight); tissue residues, in mg As/kg fresh weight, of 13.9 in liver, 23.7 in kidney, and 25.8 in rumen contents (vs. normal values of <0.5)	2
Arsenic trioxide	Single oral dose of 15 to 45 grams/animal fatal	3
Arsenic trioxide	Toxic dose is 33 to 55 mg/kg body weight (BW), or 13.2 to 22 grams for a 400-kg animal. Animals accidentally poisoned topically contained up to 15 mg As/kg fresh weight liver, 23 in kidney, and 45 in urine (vs. <1 for all normal tissues)	4
Cacodylic acid, $(CH_3)_2AsO(OH)$	Calves were anorexic in 3 to 6 days when fed diets containing 4,700 mg/kg.	
	Adult oral dosages of 10 mg/kg BW daily for 3 weeks followed by 20 mg/kg BW daily for 5 to 6 weeks was lethal. Adverse effects at 25 mg/kg BW daily for 10 days	5
Methanearsonic acid,	Calves were anorexic in 3 to 6 days	

$CH_3AsO(OH)_2$	when fed diets containing 4,000 mg/kg	5
Monosodium methanearsonate	10 mg/kg BW daily for 10 days fatal	3
Sodium arsenite	Single oral dose of 1 to 4 grams fatal	3
Dog, *Canis familiaris*		
Cacodylic acid	Single oral LD-50 value of 1,000 mg/kg BW. Fed diets containing 30 mg/kg for 90 days with no ill effects	5
Methanearsonic acid	Fed diets containing 30 mg/kg for 90 days with no ill effects	5
Sodium arsenite	50 to 150 mg fatal	3
Domestic goat, *Capra* sp.		
Arsenic acid	Single oral dose of 2.5 to 7.5 mg/kg BW (50 to 150 mg) was acutely toxic	3
Guinea pig, *Cavia* sp.		
Arsenic acid	Dietary levels of 350 mg/kg resulted in blindness and optic disc atrophy in 25 to 30 days	6
Arsenic trioxide	Fed diets containing 50 mg/kg for 21 days; elevated As residues, in mg/kg fresh weight, of 4 in blood, 15 in heart (vs. <1 for all control tissues)	7
Sodium arsanilate	Subcutaneous injection of 70 mg/kg BW caused degeneration of sensory walls of inner ear; elevated As residues in cochlea	6
Sodium arsenate	Intraperitoneal injection of 0.2 mg/kg BW at age 2 months causes deafness	6
Hamster, *Cricetus* sp.		
Arsenate	Maternal dose of 5 mg As^{+5}/kg BW caused some fetal mortality, but no malformations; higher dose of 20 mg/kg BW caused 54% fetal deaths and malformations	3
Calcium arsenate	Pulmonary tumorigenicity demonstrated 70 weeks after 15 intratracheal weekly injections of 3 mg/kg BW	8
Dimethylarsinate	50% growth reduction in Chinese hamster ovary cells (CHOC) at 90 to 112 mg/L	9
Gallium arsenide	Single oral dose of 100 mg/kg BW mostly (85%) eliminated in 5 days, usually in	

	form of organoarsenicals; all tissue levels <0.25 mg/kg	10
Sodium arsenate	Dosed intravenously on day 8 of gestation: 2 mg/kg BW had no measurable effect; 8 mg/kg produced increased incidence of malformation and resorption; 16 mg/kg BW killed all embryos	6
Sodium arsenate	50% growth reduction in CHOC at 2.25 mg/L	9
Sodium arsenite	Chinese hamster ovary cells (CHOC) show 50% growth reduction at 0.3 mg/L	9
Sodium cacodylate	Single intraperitoneal injection of 900 to 1,000 mg/kg during midgestation results in some maternal deaths, and increased incidences of fetal malformations	5
Horse, *Equus caballus*		
Sodium arsenite	Daily doses of 2 to 6 mg/kg BW (1 to 3 grams) for 14 weeks is fatal	3
Cat, *Felis domesticus*		
Inorganic arsenate or arsenite	Chronic oral toxicity at 1.5 mg/kg BW	6
Human, *Homo sapiens*		
Arsenic trioxide	Fatal at 70 to 189 mg, equivalent to about 1 to 2.6 mg As/kg BW	6
Arsenic trioxide	LD-50 dose of 7 mg/kg BW	3
Cacodylic acid	LD-50 of 1,350 mg/kg BW	3
Lead arsenate	Some deaths at 7 mg/kg BW	3
Total arsenic	Accumulations of 1 mg/kg BW daily for 3 months in children, or 80 mg/kg BW daily for 3 years produced symptoms of chronic arsenic poisoning	3
Total arsenic, daily oral dose	Prolonged dosages of 3 to 4 mg daily produced clinical symptoms of chronic arsenic intoxication	3
Total arsenic in drinking and cooking water	Prolonged use produced symptoms of chronic arsenic intoxication (0.6 mg/L) or skin cancer (0.29 mg/L)	3
Total arsenic, probably as arsenate	12,000 Japanese infants poisoned (128 deaths) from consumption of dry milk contaminated with arsenic; average exposure of 3.5 mg As daily for one month. Severe hearing loss, brain wave abnormalities, and other central nervous system disturbances noted 15 years postexposure	6

Total inorganic arsenic	Daily dose of 3 mg for 2 weeks may cause severe poisoning in infants, and symptoms of toxicity in adults	6
Cynmolgus monkey, *Macaca* sp.		
Fish arsenic meal (witch flounder, *Glyptocephalus cynoglossus*) containing 77 mg total As/kg	Given a single meal at 1 mg/kg BW; tissue residues normal after 14 days	11
As above, except arsenic trioxide substituted for total As	As above	11
Mammals, many species		
Calcium arsenate	Single oral LD-50 range of 35 to 100 mg/kg BW	3
Lead arsenate	Single oral LD-50 range of 10 to 50 mg/kg BW	3
Mammals, most species		
Arsenic trioxide	3 to 250 mg/kg BW lethal	12
Sodium arsenite	1 to 25 mg/kg BW lethal	12
Mouse, *Mus* spp.		
Arsenate	Maternal dose of 10 mg As^{+5}/kg BW results in some fetal deaths and malformations	3
Arsenic trioxide	Single oral LD-50 (96 h) value of 39.4 mg/kg BW; LD-0 (96 h) of 10.4 mg/kg BW	12
Arsenic trioxide	"Adapted" group (50 mg As/L in drinking water for 3 months) had subcutaneous LD-50 value of 14 mg/kg BW vs. 11 for nonadapted group	12
Arsenic trioxide	Air concentrations of 28.5 mg/m^3 for 4 h daily on days 9 to 12 of gestation caused fetotoxic effects and chromosomal damage to liver cells by day 18; effects included reduced survival, impaired growth, retarded limb ossification, and bone abnormalities. At 2.9 mg/m^3, a 9.9% decrease in fetal weight was recorded; at 0.26 mg/m^3, a 3.1% decrease was measured	13
Cacodylic acid	Oral dosages of 400 to 600 mg/kg BW on days 7 to 16 of gestation produces fetal malformations (cleft palate), delayed	

	skeletal ossification, and fetal weight reduction	5
Sodium arsenate	Maximum tolerated doses in terms of abortion or maternal death over 24 h in 18-day pregnant mice were 20 mg As^{+5}/kg BW, intraperitoneal route, and 50 mg/kg BW when administered orally. Residue half-life was about 10 h regardless of route of administration	14
Sodium arsenite	Fed 5 mg/kg diet for three generations: reduced litter size, but outwardly normal	6
Sodium arsenite	LD-50 of 9.6 mg/kg BW, subcutaneous route; LD-90 (7 days postadministration) of 11.3 mg/kg BW, subcutaneous route	15
Sodium arsenite	LD-50 of 12 mg/kg BW intraperitoneal route. At 10 mg/kg BW, damage to bone marrow and sperm	16
Sodium cacodylate	Single intraperitoneal injection of 1,200 mg/kg BW during midgestation results in increased rates of fetal skeletal malformations	5
Mule deer, *Odocoileus hemionus hemionus*		
Silvisar-510 (mixture of cacodylic acid and tri-ethanolamine cacodylate)	Single oral LD-50 dose >320 mg/kg BW; appetite loss	17
White-tailed deer, *Odocoileus virginianus*		
Sodium arsenite (used to debark trees)	Lethal dose of 923 to 2,770 mg equivalent to about 34 mg/kg BW; liver residues of 40 mg/kg fresh weight	12
Arsenic acid (herbicide to control Johnson grass)	23 deer killed from apparent misuse. Arsenic levels, in mg/kg fresh weight, in deer found dead were 19 in liver, 18 in kidney, and 22.5 in rumen contents. Soils from area contained ~2.4 mg As/kg, and water 0.42 mg As/L	12
Domestic sheep, *Ovis aries*		
Arsanilic acid	One-year-old castrates fed diets with 273 mg As/kg for 28 days had 0.54 mg As/L in blood, 29 mg/kg dry weight in liver, 24 in kidney,	

	and 1.2 in muscle (vs. <0.01 in all control tissues). After 6 days on an As-free diet, liver residues were <5 mg/kg DW. Maximum tissue levels in sheep fed diets containing 27 mg As/kg for 28 days were 3.2 mg/kg DW kidney; for a 144 mg/kg DW diet, the maximum tissue level was 27 mg/kg DW liver	7
Sodium arsenite	Single oral dose of 5 to 12 mg/kg BW (0.2 to 0.5 grams) was acutely toxic	3
Soluble arsenic	Lambs fed supplemental arsenic for 3 months at 2 mg As/kg dry weight diet contained maximum concentrations of 2 mg/kg fresh weight brain (vs. 1 in controls), 14 in muscle (2), 24 in liver (4), and 57 in kidney (10)	18
Total arsenic	Sheep fed on diets containing lakeweed, *Lagarosiphon major* (288 mg As/kg DW) at 58 mg total As/kg diet for 3 weeks without ill effect. Tissue residues increased during feeding, but rapidly declined when lakeweed was removed from diet	7
Rat, *Rattus* spp.		
Arsanilic acid	No teratogenesis observed in 7 generations at dietary level of 17.5 mg/kg; positive effect on litter size and survival	6
Arsenate	Fed diets containing 50 mg/kg for 10 weeks with no effect on serum uric acid levels	19
Arsenic trioxide	Single oral LD-50 (96 h) value of 15.1 mg/kg BW	12
Arsenic trioxide	Single dose of 17 mg/kg BW administered intratracheally is maximally tolerated nonlethal dose; 2 weeks later, blood As elevated (36 mg/L) and lung histopathology evident	20
Arsenic trioxide	After 21 days on diet containing 50 mg/kg, tissue arsenic levels were elevated in blood (125 mg/L vs. 15 in controls), heart (43 mg/kg FW vs. 3.3), spleen (60 vs. <0.7) and kidney (25 vs. 1.5)	7

Arsenite	Oral administration of 1.2 mg/kg BW daily for 6 weeks reduced uric acid levels in plasma by 67%	19
Arsenite	Oral administration of 1.2 mg/kg BW daily for 6 weeks reduced uric acid levels in plasma by 67%	19
Cacodylic acid	Fetal and maternal deaths noted when pregnant rats dosed by gavage at 50 to 60 mg/kg BW daily during gestation days 6 to 13. Fetal abnormalities observed when dams given oral dosages of 40 to 60 mg/kg BW on days 7 to 16 of gestation	5
3-Nitro-4-hydroxy-phenylarsonic acid	Single oral LD-50 value of 44 mg/kg BW	12
Sodium arsenate	LD-75 (48 h) value of 14 to 18 mg/kg BW (intraperitoneal route)	12
Sodium arsenate	Single intraperitoneal injection of 5 to 12 mg/kg on days 7 to 12 of gestation produced eye defects, exencephaly, and faulty development of kidney and gonads	6
Sodium arsenite	LD-75 (48 h) value of 4.5 mg/kg BW (intraperitoneal injection	12
Rodents, various species		
Cacodylic acid	LD-50 (various routes) values range from 470 to 830 mg/kg BW	5
Sodium cacodylate	LD-50 (various routes) values range from 600 to 2,600 mg/kg BW	5
Pig, *Sus* sp.		
Sodium arsenite	Drinking water containing 500 mg/L lethal at 100 to 200 mg/kg BW	12
3-Nitro-4-hydroxy-phenylarsonic acid	Arsenosis documented after 2 months on diets containing 100 mg/kg, or after 3 to 10 days on diets containing 250 mg/kg	12
Rabbit, *Sylvilagus* sp.		
Cacodylic acid	Adverse effects at dermal dosages equivalent to 4 to 6 grams/kg BW	5
Calcium arsenate	Single oral dose of 23 mg/kg BW fatal in 3 days	12
Copper acetoarsenite	Single oral dose of 10.5 mg/kg BW fatal in 50 h	12
Inorganic arsenate	Single oral LD-50 value of 8 mg/kg BW	3

[a]References: 1, Vreman et al. 1986; 2, Thatcher et al. 1985; 3, NRCC 1978; 4, Robertson et al. 1884; 5, Hood 1985; 6, Pershagen and Vahter 1979; 7, Woolson 1975; 8, Pershagen and Bjorklund 1985; 9, Belton et al. 1985; 10, Yamauchi et al. 1986; 11, Charbonneau et al. 1978; 12, NAS 1977; 13, Nagymajtenyi et al. 1985; 14, Hood et al. 1987; 15, Stine et al. 1984; 16, Deknudt et al. 1986; 17, Hudson et al. 1984; 18, Veen and Vreman 1986; 19, Jauge and Del-Razo 1985; 20, Webb et al. 1986.

Table 7. Proposed arsenic criteria for protection of selected natural resources and human health.

Resource, criterion, and other variables	Criterion or effective arsenic concentration (reference)
Aquatic Life	
Freshwater biota: medium concentrations	Four-day mean water concentration not to exceed 190 ug total recoverable inorganic As^{+3}/L more than once every 3 years; 1-h mean not to exceed 360 ug inorganic As^{+3}/L more than once every 3 years. Insufficient data for criteria formulation for inorganic As^{+5}, or for any organoarsenical (EPA 1985)
Freshwater biota: tissue residues	Diminished growth and survival reported in immature bluegills (*Lepomis macrochirus*) when total arsenic residues in muscle >1.3 mg/kg fresh weight (FW) or >5 mg/kg in adults (NRCC 1978)
Saltwater biota: medium concentrations	Four-day average water concentration not to exceed 36 ug As^{+3}/L more than once every 3 years; 1-h mean not to exceed 69 ug As^{+3}/L more than once every 3 years. Insufficient data for criteria formulation for inorganic As^{+5}, or for any organoarsenical (EPA 1985)
Saltwater biota: tissue residues	Depending on chemical form of arsenic, certain marine teleosts may be unaffected at muscle total arsenic residues of 40 mg/kg FW (NRCC 1978)
Birds	
Tissue residues	Residues, in mg total As/kg FW, liver or kidney in 2 to 10 range are considered elevated; residues >10 mg/kg are indicative of arsenic poisoning (Goede 1985)
Turkey, *Meleagris gallopavo*	
Arsanilic acid in diet	Maximum dietary concentration for turkeys less than 28 days old is 300 to 400 mg/kg feed (NAS 1977)
Phenylarsonic feed additives for disease control and improvement of weight gain in domestic poultry; safe dietary levels	Maximum levels in diets, in mg/kg feed, are 50 to 100 for arsanilic acid, 25 to 188 for 3-nitro-4-hydroxy-phenylarsonic acid (for chickens and turkeys, not recommended for ducks and geese), and 180 to 370 for others (NAS 1977)
Domestic Livestock	
Prescribed limits for arsenic in feedstuffs	
Straight feedstuffs, except	<2 mg total As/kg FW (Vreman et al. 1986)

those listed below	
Meals from grass, dried	<4 mg total As/kg FW (Vreman et al. 1986)
lucerne, or dried clover	
Phosphate mealstuffs	<10 mg total As/kg FW (Vreman et al. 1986)
Fish meals	<10 mg total As/kg FW (Vreman et al. 1986)
Tissue residues	
Poisoned	
Liver, kidney	5 to >10 total As/kg FW (Thatcher et al. 1985; Vreman et al. 1986)
Normal, muscle	<0.3 mg total As/kg FW (Veen and Vreman 1986)
Vegetation	
No observable effects	<1 mg total water soluble soil As/L; <25 mg total As/kg soil; <3.9 ug As/m^3 air (NRCC 1978)
Human Health	
Diet	
Permissible levels	
Total diet	<0.5 mg As/kg dry weight diet (Sorensen et al. 1985)
Fruits, vegetables	The tolerance for arsenic residues as As_2O_3, resulting from pesticidal use of copper, magnesium, and sodium arsenates is 3.5 mg/kg (Jelinek and Corneliussen 1977)
Muscle of poultry and swine, eggs, swine edible byproducts	< 0.5 mg total As/kg FW (Jelinek and Corneliussen 1977)
Edible byproducts of chickens and turkey, liver and kidney of swine	<2 mg total As/kg FW (Jelinek and Corneliussen 1977)
Seafood products	In Hong Kong, limited to 6 mg total As/kg FW for edible tissues of finfish, and 10 mg/kg for molluscs and crustaceans (Phillips et al. 1982)
Adverse effects	
Consumption of aquatic organisms living in As-contaminated waters	
Cancer risk of	
10^{-5}	0.175 ug As/L (EPA 1980)
10^{-6}	0.0175 ug As/L (EPA 1980)
10^{-7}	0.00175 ug As/L (EPA 1980)
Drinking water	
Allowable concentrations	
Total arsenic	<10 ug/L (NAS 1977)
Total arsenic	<50 ug/L (Pershagen and Vahter 1979; EPA 1980; Norin et al. 1985)
Adverse effects	
Cancer risk of	

10^{-5}	0.022 ug As/L (EPA 1980)
10^{-6}	0.0022 ug As/L (EPA 1980)
10^{-7}	0.00022 ug As/L (EPA 1980)
Symptoms of arsenic toxicity observed	9% incidence at 50 ug As/L, 16% at 50 to 100 ug/L, and 44% at >100 ug As/L (NRCC 1978)
Harmful after prolonged consumption	
"Cancer"	In Chile, cancer rate estimated at 0.01% at 82 ug As/L, 0.17% at 600 ug As/L (NRCC 1978)
Skin cancer	0.26% frequency at 290 ug/L and 2.14% at 600 ug/L (EPA 1980)
Total intake	
No observable effect	
North America	0.007 to 0.06 mg As daily (Pershagen and Vahter 1979)
Japan	0.07 to 0.17 mg As daily (Pershagen and Vahter 1979)
USA	
1960's	0.05 to 0.1 mg As daily (Pershagen and Vahter 1979)
1974	0.015 mg As daily (Pershagen and Vahter 1979)
Canada	0.03 mg As daily (NRCC 1978)
Netherlands	
Acceptable	2 ug total inorganic As/kg body weight (BW) (about 0.14 mg daily for 70 kg adult); 0.094 mg daily through fishery products (Vos and Hovens 1986)
Adverse effects (Prolonged exposure)	
Subclinical symptoms	0.15 to 0.6 mg As daily (NRCC 1978)
Intoxication	3 to 4 mg As daily (NRCC 1978)
Blackfoot disease	Total dose of 20 grams over several years increases prevalence of disease by 3% (Pershagen and Vahter 1979)
Mild chronic poisoning	0.15 mg As daily or about 2 ug/kg BW daily (NRCC 1978)
Chronic arsenicism	Lifetime cumulative absorption of 1 gram As, or intake of 0.7 to 2.6 grams/year for several years (in medications) can produce symptoms after latent period of 4 to 24 years (NRCC 1978)
Tissue residues	
No observed effect levels	
Urine	<0.05 mg As/L (NRCC 1978)
Liver, kidney	<0.5 mg As/L (NRCC 1978)
Blood	<0.7 As/kg (NRCC 1978)
Hair	<2 mg As/kg (NRCC 1978)
Fingernail	<5 mg As/kg (NRCC 1978)

Arsenic-poisoned	
Liver, kidney	2 to 100 mg As/kg FW; confirmatory tests >10 mg As/kg FW; residues in survivors several days later 2 to 4 mg/kg FW (NAS 1977)
Whole body	In child, symptoms of chronic arsenicism evident at 1 mg As/kg BW, equivalent to intake of about 10 mg/month for 3 months; for adults, these values were 80 mg/kg BW, equivalent to about 2 grams/year for 3 years (NRCC 1978)
Air	
Allowable concentrations	
Arsine	<200 ug/m^3 for USA industrial workers; proposed mean arsine limit of <4 ug/m^3 in 8 h period and <10 ug/m^3 maximum in 15 min (NAS 1977)
Arsine	<4 ug/m^3 (NRCC 1978)
Total As	<3 ug/m^3 in USSR and Czechoslovakia, <500 µg/m^3 for USA industrial workers (NAS 1977)
Total As (threshold limit value-time weighted average: 8 h/day, 40-h work week)	Proposed limit of <50 ug/m^3, maximum of 2 µg/m^3 in 15 min, <10 ug airborne inorganic As/m^3 (EPA 1980)
Arsenic trioxide	<0.3 ug/m^3 in USSR, <0.1 ug/m^3 in USA (Nagymajtenyi et al. 1985)
Adverse effects	
Increased mortality	Associated with daily time-weighted average As exposure of >3 ug/m^3 for 1 year (NRCC 1978)
Respiratory cancer (increased risk)	Associated with chronic exposure >3 ug As/m^3, or occupational exposure (lifetime) of >54.6 ug As/m^3 (NRCC 1978)
Respiratory cancer (increased risk)	Exposure to 50 ug As/m^3 for more than 25 years associated with 3X increase (Pershagen and Vahter 1979)
Skin diseases	Associated with ambient air concentrations of 60 to 13,000 ug As/m^3 (NRCC 1978)