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Guidelines for Interpretation of the Biological Effects of Selected Constituents in Biota, Water, and Sediment

Arsenic

Participating Agencies:

Bureau of Reclamation
U.S. Fish and Wildlife Service
U.S. Geological Survey
Bureau of Indian Affairs

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Arsenic

Description

Arsenic (As) is a metalloid, with properties intermediate between those of a metal and a nonmetal. In its pure state, it generally takes the form of a dense, gray metal, although a much lighter, yellowish powder may be formed through sublimation of the vapor. In nature, arsenic exists in four oxidation states (As^{-3}, As^{0}, As^{+3} [referred to as "As (III)"], and As^{+5} ["As (V)"]), and it may be in either organic or inorganic forms. Its common ores include the minerals arsenopyrite (FeAsS) and realgar (As_2S_2). Arsenopyrite is a white to gray orthorhombic mineral resembling pyrite, commonly found in lead and silver veins. Realgar forms red to reddish-orange nodules in ore veins and similarly colored coatings around some hot springs.

Occurrence

Arsenic is ubiquitous—present in air, water, soil, plants, and other living organisms. In water, common forms of arsenic are As (III), As (V), methanearsonic acid, and dimethyl-arsinic acid (EPA 1985). Inorganic As (V) is the most common species in water. As (III) in water converts readily to As (V) under aerobic conditions (Clement and Faust 1973), but some As (III) may persist depending on microorganisms, temperature, and other factors.

Background Concentrations.—The arsenic concentration in soil normally ranges from 1 to 50 mg/kg, though it does not generally exceed 10 mg/kg (Brown et al. 1983), and in water it is normally <10 μg/L (Eisler 1988). Terrestrial flora and fauna, birds, and freshwater biota usually contain <1 mg As/kg by wet weight (ww). Arsenic at 0.27 mg/kg ww (≈1 mg/kg dry weight [dw]) is reported to be the 85th percentile concentration for freshwater fish (Schmitt and Brumbaugh 1990), and background concentrations in terrestrial plants range from 0.01 to 1.7 mg/kg dw (Bodek et al. 1988). Concentrations of arsenic in livers of adult amphibians collected in an apparently uncontaminated area averaged 0.164 mg/kg ww (Byrne et al. 1975). These levels are sometimes much higher in biota collected near areas with high geo-thermal activity and near manufacturers of arsenical defoliants and pesticides (Eisler 1988).

Each year, as a result of agricultural and industrial activities, large quantities of arsenicals that may be hazardous to fish and wildlife are released into the environment (Eisler 1988). Agricultural applications provide the largest artificial source of arsenic in the environment (Eisler 1988). It is contained in wastes from the production of certain herbicides, fungicides, insecticides, algicides, and wood preservatives (Brown et al. 1983); in particular, sodium arsenite was commonly used as an aquatic herbicide between 1940 and the 1970's, especially in the United States (Tanner and Clayton 1990). Arsenic is also present in large amounts in water contaminated by mine tailings, smelter wastes, and natural mineralization (Eisler 1988). EPA also states that sources of arsenic in drinking water include glass, electronic wastes, and orchards (EPA 1994).

Summary of Effects

Arsenic is not normally considered an essential element to most species, and it has been shown to be both teratogenic and carcinogenic in many

mammal species (Eisler 1988, 1994). However, beneficial effects have been reported in tadpoles, silkworm, rats, goats, and pigs at low dietary concentrations (Eisler 1988). Mammals with arsenic deficiencies display poor growth, reduced survival, and inhibited reproduction, whereas low doses of arsenic actually stimulate growth in plants and animals (Eisler 1994).

Arsenic's toxicity and bioavailability may vary significantly, depending on the chemical forms and routes of exposure. In general, inorganic arsenic compounds are more toxic than organic compounds, and As (III) is more toxic than As (V) (Eisler 1988, 1994). Hence, the natural conversion of As (III) to As (V), which is favored in most aquatic environ-ments (Manahan 1989), somewhat reduces the overall hazard of this element. It should be noted, though, that most dietary studies rely on only a single species of arsenic—generally inorganic—and that such studies thus do not reflect the diversity of arsenic species present in the environment. The varying effects of different arsenic compounds should be considered before using experimental data to assess the toxicity of arsenic in the environment.

In the aquatic environment, adverse effects of arsenic have been reported at a wide range of concentrations in water, sediment, and diets. Suter and Mabrey (1994) evaluated a series of toxicological benchmarks for screening various contaminants for their potential effects on aquatic biota. In addition to the national ambient water quality (NAWQ) criteria, they provided secondary acute and chronic values, lowest chronic values (including those for fish, daphnids, nondaphnid invertebrates, aquatic plants, and all organisms), test EC20s (concentrations that cause observable ill effects in 20 percent of specimens), sensitive species test EC20s, and population EC20s. These data were used to establish the general biotic effect levels presented in table 1. As listed there, "No effect" is the lowest chronic value for all organisms; "Toxicity threshold" is the NAWQ chronic criterion (if established) or the secondary chronic value; and "Level of concern" is the range between the two other values.

Field Cases

Though arsenic is ubiquitous in the environment, the incidence of wildlife poisoning by arsenic is relatively rare (Eisler 1988). Sandhu (1977) reported an intensive fish kill in a reservoir at Orangeburg, South Carolina, after aerial spraying of arsenic defoliants in a nearby cotton field. The arsenic concentration in the water was elevated to 2,500 μg/L, and catfish in the reservoir were reported to contain 5 and 12 mg As/kg in skeletal muscle after 5-hour and 7-week exposures (weight basis not specified).

Arsenic is also relatively persistent in the aquatic environment. Tanner and Clayton (1990) reported elevated concentrations of arsenic in macrophytes (193–1,200 mg/kg dw) and surficial sediments (540–780 mg/kg dw) in Lake Rotoroa, New Zealand, 24 years after an application of sodium arsenite herbicide; arsenic levels in a nearby reference lake (Lake Rotokauri) were <20 mg/kg dw in macro-phytes and 16.5–40 mg/kg dw in sediments. (Note, however, that the "reference lake" had arsenic concentrations in the sediments that are in the middle of the levels of concern in table 1, and the detection limit for the macro-phyte datum was four times the toxicity threshold for plants in table 1. Alternatively, the "living" macrophytes had arsenic con-centrations of between 39 and 240 times the toxicity threshold and are obviously tolerant species.)

Natural sources, such as hot springs and volcanic activity, also contribute to elevated levels of arsenic in the environment. Lacayo et al. (1992) determined arsenic levels in water, fish, and sediments from Xolotlán, Managua, Nicaragua, a lake which contained high levels of arsenic from such sources.

Table 1.—Summary of comprehensive biotic effects of arsenic
[See Appendix II for explanation of abbreviations and technical terms]

Medium	No effect	Level of concern	Toxicity threshold	Comments/Explanation
Water (µg/L)	48	48–190	190	48 µg/L is lowest chronic value for As (V) in aquatic plants; 190 µg/L is NAWQ chronic criterion for As (III). See Suter and Mabrey (1994).
Sediment (mg/kg dw)	8.2	8.2–70	70	"ERL" and "ERM" values of Long et al. 1995.
Plants (mg/kg dw)	1–1.7	2–5	5	Levels in plants (Kabata-Pendias and Pendias 1992) and invertebrates (see table 2) not well established, but at least some show no effects below these tissue concentrations.
Invertebrates (mg/kg dw)	30	30–50	50	
Fish (mg/kg dw)	1.0	1–12	12	No-effect level is 85th percentile concentration from Schmitt and Brumbaugh (1990). Toxicity threshold from Sandhu (1977).
Bird eggs (mg/kg dw)	1.3	1.3–2.8	<2.8	J.P. Skorupa, unpub. data, 1996.
Amphibians/reptiles	—	—	—	Diagnostic effect levels not available. Mammals, in particular are poor biomonitors for As (Talmage and Walton 1991).
Mammals	—	—	—	

Note: Although diagnostic levels for biota concentrations are generally not well defined, arsenic concentrations in biota are usually <1 mg/kg fresh weight except near sources of arsenic pollution (Eisler 1988, 1994). (Dry-weight concentrations, such as those shown above, are generally several times higher than fresh-weight concentrations, although no reliable conversion factor can be defined.)

In Texas, Clark et al. (in press) reported what they believed to be the highest concentrations of arsenic found in tadpoles (6.87 mg As/kg ww). Their report provides a good review of information concerning arsenic (as well as chromium and zinc) in amphibians and reptiles. Tadpoles were collected in 1994 from areas immediately downstream from Finfeather Lake, which had been directly contaminated during 53 years of industrial production of arsenic-based cotton defoliants. No tadpoles were found in Finfeather Lake, probably because arsenic, chromium, or zinc concentrations there were still toxic, even though contaminated sediments had been removed about 10 years earlier.

Dead and blind turtles (red-eared slider, *Trachemys scripta*, and common snapper, *Chelydra serpentina*) were found at Finfeather Lake in 1973, when waterborne arsenic concentrations in the lake averaged 7.9 milligrams per liter (mg/L) (Cearley 1973). The turtles showed symptoms similar to those of arsenic-poisoned domestic mammals. These included keratinization (leathery appearance) of the eyelids, nasal areas, and roof of the mouth. The nasal passages of one turtle were completely occluded with the keratinized tissue, forcing the turtle to breathe through its mouth. Clark et al. (in press) observed no turtles or snakes in Finfeather Lake in 1994 or 1995, leading them to speculate that few or none were present.

Fish populations in Finfeather Lake also were affected (Cearley 1973, Sorensen et al. 1985). Green sunfish (*Lepomis cyanellus*) in the system exhibited liver pathology related to arsenic. In 1991, Cantu et al. (1991) found that

large-mouth bass (*Micropterus salmoides*) from Finfeather Lake had deformed fins, jaws, heads, and eyes; waterborne arsenic concen-trations at the time were 0.54 mg/L.

Abiotic Factors Affecting Bioavailability

Water

Many factors influence arsenic toxicity in water, including water temperature, pH, organic content, phosphate concentration, suspended soils, the presence of other substances and oxidants, and arsenic speciation. A study by McGeachy and Dixon (1990) confirmed that more arsenic is taken up as the water temperature increases.

Sediment

Higher levels of arsenic in sediment were correlated with levels in macrophytes in a study done by Tanner and Clayton (1990), but other studies (Cain et al. 1992, Smith et al. 1992) reported low bioavailability and little partitioning of arsenic from contaminated sediments. Long and Morgan (1990) and Long et al. (1995) made a comprehensive evaluation of chemical concentrations in sediments that were associated with adverse biological effects. They concluded that arsenic concentrations of 8.2 mg/kg dw or less do not usually produce adverse effects, but concen-trations of 70 mg/kg or higher usually do. Although many of the data evaluated were for estuarine and marine sediments, Hull and Suter (1994) concluded that those screening levels also were appropriate for freshwater sediments until more specific guidelines become available. However, it is also recommended that these concentrations be compared to local background levels when possible.

Biotic Effects

Tables 2, 3, and 4 at the end of this chapter list the reported biotic effects of arsenic in water, sediment, and diet, respectively.

Plants

Arsenic is not an essential element in plants (Kabata-Pendias and Pendias 1992), although small increases in yield have been observed for several species at low levels of soil arsenic (Woolson 1975). Some forms of arsenic, such as sodium arsenate and arsenic trioxide, are extremely toxic to plants. Arsenic uptake seems to be passive (Bodek et al. 1988) from terrestrial soil to plants. The major symptoms of arsenic toxicity in plants are red-brown necrotic spots on old leaves, yellowing or browning of the roots, wilting of new leaves, and depressed tillering (Kabata-Pendias and Pendias 1992). Sensitive species such as spinach (*Spinacia oleracea*) showed 40-percent reduction in growth when exposed to As (V) at 10 mg/kg in soil (table 3). Low concen-trations of As (V) in water (1–15.2 µg/L) have been reported to inhibit certain aquatic plants, resulting in noticeable changes throughout the ecosystem. Sanders and Cibik (1985) have reported consequent changes in the composi-tion and succession of species and in predator-prey relations in chronic studies.

Amphibians/Reptiles

Very few studies have investigated the effects of arsenic on amphibians and reptiles. Khangarot et al. (1985) determined the acute toxicity of As (III) to tadpoles (*Rana hexadactyla*). Under the conditions of pH 6.1, temperature 15 °C, and total hardness 20 mg/L (calcium carbonate), they found that a concentration of 249 µg As/L

would kill 50 percent of specimens in 4 days (96-h LC50). Average arsenic concentrations in the livers of adult frogs and toads were 0.164 mg/kg ww at an uncontaminated area (Hall and Mulhern 1984). This value was considerably lower than the levels of arsenic in many other freshwater animals (Wagemann et al. 1978).

Birds

There are great differences in tolerance to arsenic among bird species. As shown in table 4, female mallard (*Anas platyrhynchos*) ducklings showed a reduced growth rate when they were fed 30 mg As (V)/kg dw over 10 weeks (Camardese et al. 1990). In adult mallards, arsenic toxicity from sodium arsenate in the diet was significant at 400 mg/kg dw (Stanley et al. 1994). Other sensitive species, such as the brown-headed cowbird (*Molothrus ater*), showed 50-percent mortality in 11 days when fed copper acetoarsenite at 99.8 mg/kg dw (table 4). Opresko et al. (1994) estimated the no-observed-adverse-effect levels (NOAEL) for dietary concentrations of arsenic in several species of aquatic and terrestrial birds. The belted kingfisher (*Ceryle alcyon*) and great blue heron (*Ardea herodias*) are the most relevant species for aquatic habitats. For those two species, the dietary NOAELs were 19 to 22 mg/kg ww when based on sodium arsenite in the diet and 3.4 to 3.9 mg/kg ww when based on copper acetoarsenite (Paris green).

Stanley et al. (1994) found that adult mallards fed arsenic as sodium arsenate showed reduced weight gain, reduced liver weight, delayed egg laying, reduced egg weight, and eggshell thinning. Adult mallards exposed to dietary concentrations of 300 mg As/kg (dw) as sodium arsenate rapidly accumulated the compound but also rapidly eliminated it; the compound had a half-life of 1 to 3 days after removal from the diet and reached equili-brium levels in 10 to 30 days (Pendleton et al. 1995). The greatest accumulation of arsenic was in the liver, and lower levels were found in the blood and brain. Arsenic also reduced the growth and the body and liver weights in mallard ducklings (Stanley et al. 1994).

Some studies indicate that arsenic is extremely toxic to avian eggs when injected (Birge and Roberts 1976, Gilani and Alibhai 1990). However, elevated levels of arsenic rarely occur naturally in eggs, even in those collected at agricultural drainwater evaporation ponds where arsenic was present at high concentra-tions. Among 81 eggs collected during 1987–89 in the San Joaquin Valley of California, only one contained arsenic above the detection limit of 0.4 mg/kg dw (Ohlendorf et al. 1993). Libby et al. (1953) found that domestic poultry fed a diet containing high levels of arsenic (arsanilic acid at 180 mg/kg dw) nevertheless produced eggs that contained an average of only 1.3 mg As/kg and showed normal embryo viability. Many studies have shown that arsenic actually stimulates growth and egg pro-ductivity in poultry. Stute and Vogt (1968) fed 3-nitro-4-hydroxyphenylarsonic acid to hens at 50 mg/kg dw and observed a 4-per-cent increase in egg production.

Mammals

Although arsenic is officially classified as a human carcinogen (EPA 1995), there is little evidence that it is carcinogenic to other mammals (Eisler 1988). It does, however, cause teratogenic effects in many species. Mammals are exposed to arsenic mainly by the ingestion of contaminated vegetation and water. Adverse effects were noted in rats at dietary levels of 20 mg/kg dw (table 4). Acute or subacute arsenic poisoning is much more common than chronic poisoning in mammals (National Academy of Sciences 1977). The probability of chronic arsenic exposure is rare because detoxification and excretion are rapid (Woolson 1975). As various studies have noted (see review by Talmage and Walton 1991), mammals normally are not good biomonitors for arsenic in the

environment. Sharma and Shupe (1977), for instance, observed no relationship between arsenic concentrations in soil and vegetation and those in the liver of ground squirrels.

Bioaccumulation

Waterborne arsenic is known to accumulate to high concentrations in some species (table 2). The accumulated arsenic concentrations in stoneflies, snails, and Daphnia were as much as 131, 99, and 219 times, respectively, the water concentration according to a study by Spehar et al. (1980), whereas rainbow trout and amphipods showed no sign of bioaccumulation. Though the bioaccumulation of arsenic from the water has been well documented, there is no evidence of magnification along the aquatic food chain (Eisler 1988).

Arsenic has been found to accumulate in the lipid fractions of marine plants, invertebrates, and higher organisms (Eisler 1994). Marine biota, in particular, contain unusually high levels of arsenic in their lipids because of their ability to accumulate the element from both seawater and food sources. For mallards, Stanley et al. (1994) found that arsenic accumulated in both adult and duckling livers and in whole eggs (table 4). Pendleton et al. (1995) found that arsenic (as sodium arsenate) accumulated in all tissues but was also rapidly eliminated when birds were switched to an uncontaminated diet.

In order to evaluate the cumulative toxicity of arsenic and various metals (Cd, Cu, Hg, Pb) along the food chain, Yannai et al. (1979) raised a large quantity of algae (*Micractinium* and *Chlorella*) on metal-rich waste water, fed the algae to chickens and carp, and then fed the meat of these chickens and carp to rats. They found that bioaccumulation did not increase the levels of any of these metals in chickens or carp except for chickens' livers (which contained higher arsenic than the livers of control chickens), and they observed no change in the general appearance, behavior, and survival of the rats that ate the chicken and carp meat. They concluded that such meat would pose no hazard to consumers.

Interactions

An antagonistic interaction between arsenic and selenium is found in several animal species, including rats, dogs, swine, cattle, and poultry, and it is best documented for non-domestic birds in a study done by Stanley et al. (1994). According to the study, "As reduced Se accumulation in liver and egg, and alleviated the effects of Se on hatching success and embryo deformities" in mallards. However, exposure to As and Se at contaminated sites may not be in the chemical forms administered in that study, and exposure levels, especially for As, may be lower than those administered. Thus, the interactions observed may not occur under natural conditions and, therefore, may not be an important consideration in the management of contaminated sites.

Regulatory Standards

Standards and criteria established by the U.S. Environmental Protection agency are listed in table 5. For standards and criteria set by State agencies, contact those agencies directly. See Appendix I for a listing of water quality officials in the 17 Western States.

Table 2.—Biological effects of various waterborne arsenicals on selected species in aquatic environments

[dw, dry weight; ww, wet weight. See Appendix II for explanation of other abbreviations and technical terms]

Species	As compound	Concentration in water (µg/L)	Effects	Reference
Aquatic plants				
Algae, various species	As (V)	75	Decreased growth	Eisler 1988
Alga (*Ankistrodesmus falcatus*)	As (V)	260	14-day EC50; inhibited growth	
Alga (*Scenedesmus obliquus*)	As (V)	48	14-day EC50; inhibited growth	
Alga (*Selenastrum capricornutum*)	As (V)	690	4-day EC50; inhibited growth	
Stoneworts (*Chara corallina*)	Total As	<10	As in biomass 340–400 mg/kg (dw)	Tanner and Clayton 1990
Aquatic invertebrates				
Amphipod (*Gammarus pseudolimnaeus*)	As (III)	88	28-day LC20	Eisler 1988
		1,000	Significant reduction in survival after 7 days	Spehar et al. 1980
	Disodium methyl-arsenate	85	28-day LC10	Eisler 1988
	Sodium dimethyl-arsenate	850	28-day LC0	
Cladoceran (*Daphnia magna*)	As (III)	600–1,320	MATC[1]	Eisler 1988
		960	28-day LC5	
	As (V)	520	Reproductive impairment of 16% in 3 weeks (at pH 7.74)	Eisler 1988, SJVDP 1990
		930	28-day LC5. Maximum BCF of 219	Eisler 1988
	Total As	1,000	18% decrease in body weight in 3 weeks	Eisler 1988
		1,400	50% reproductive impairment in 3 weeks	
		2,800	21-day LC50	
Cladoceran (*Daphnia pulex*)	As (III)	1,300	96-h LC50	Eisler 1988
Midge larvae (*Chironomus tentans*)	As (III)	680	48-h LC50	Khangarot and Ray 1989
		1,310	24-h LC50	

Table 2.—Biological effects of various waterborne arsenicals on selected species in aquatic environments—Continued

Species	As compound	Concentration in water (µg/L)	Effects	Reference
Aquatic invertebrates—Continued				
Snail (*Helisoma campanulata*)	As (III)	960	28-day LC10. As in biomass 80 mg/kg dw. Maximum BCF 83	Eisler 1988, Spehar et al. 1980
	As (V)	970	28-day LC0. Maximum BCF 99	
Stonefly (*Pteronarcys california*)	As (III)	960	28-day LC0	Eisler 1988
Stonefly (*Pteronarcys dorsata*)	Total As	1,000	No effect. As in biomass 29–44 mg/kg (dw); BCF 33–45 in 28 d	Spehar et al. 1980
	As (V)	89	No effect. As in biomass 12 mg/kg (dw); BCF 131 in 28 d	
Zooplankton	As (III)	400	No effect	Eisler 1988
Fish				
Arctic grayling (*Thymallus arcticus*)	As (III)	13,700	96-h LC50 for juvenile	Buhl and Hamilton 1991
		27,700	96-h LC50 for alevin	
Black crappie (*Pomoxis nigromaculatus*)	Total As	22,400–114,800 (mean=49,000)	As 0.14–2.04 mg/kg (ww) 2.9–41.6 bioaccumulation ratio	Foley et al. 1978
Brown bullhead (*Ameiurus nebulosus*)	Total As	<10	As = 0.9 mg/kg (ww) in flesh	Tanner and Clayton 1990
Chinook salmon fry (*Oncorhynchus tshawytscha*)	As (III)	21,400	96-h LC50. Mean weight 1.99 g	Hamilton and Buhl 1990
		25,100	96-h LC50. Mean weight 0.5 g	
		56,500	24-h LC50. Mean weight 1.99 g	
		59,600	24-h LC50. Mean weight 0.5 g	
	As (V)	66,500	96-h LC50. Mean weight 1.99 g	
		78,000	24-h LC50. Mean weight 1.99 g	
		90,000	96-h LC50. Mean weight 1.99 g	
		167,000	24-h LC50. Mean weight 0.5 g	
Midas cichlid (*Cichlasoma citrinellum*)	Total As	10–30	No effect. As in fish muscle <0.01–0.37 mg/kg (ww)	Lacayo et al. 1992
Jaguar guapote (*Cichlasoma managuense*)			No effect. As in fish muscle <0.01–0.24 mg/kg (ww)	
Coho salmon (*Oncorhynchus kisutch*)	As (III)	18,500	96-h LC50 for juveniles	Buhl and Hamilton 1991
		49,400	96-h LC50 for alevins	
Eel (*Angulia australis*)	Total As	<10	As = 0.4 mg/kg (ww) in flesh	Tanner and Clayton 1990

Table 2.—Biological effects of various waterborne arsenicals on selected species in aquatic environments—Continued

Species	As compound	Concentration in water (µg/L)	Effects		Reference
Fish—Continued					
Pallas (*Notopterus notopterus*)	As (III)	30,930	96-h LC50		Gosh and Chakrabarti 1990
		40,000	50% mortality in 43 h		
Perch (*Perca fluviatilis*)	Total As	<10	As = 0.3–0.5 in flesh; 0.2 in scales (mg/kg, ww)		Tanner and Clayton 1990
Rainbow trout (*Oncorhynchus mykiss*)	As (III)	960	28-day LC0; no bioaccumulation		Buhl and Hamilton 1991, Spehar et al. 1980
		16,000	96-h LC50 for juveniles		
		91,000	96-h LC50 for alevins		
	Sodium arsenate	18,000	8% mortality, whole-body As 2–3 mg/kg ww after 11 weeks at 15°C		McGeachy and Dixon 1990
		36,000	34% mortality, whole-body As 2–3 mg/kg ww after 11 weeks at 5°C		
Rainbow trout larvae	As (III)	42	1% mortality (in moderately hard water of pH 6.9–7.8)		SJVDP 1990
Rudd (*Scardinius erythrophthalmus*)	Total As	<10	As (mg/kg, ww) <0.2 in flesh; 0.3 in scales; 5.5 in gut contents		Tanner and Clayton 1990
Birds					
Shag (*Phalacrocorax* sp.)	Total As	<10	As <0.2 mg/kg (ww) in flesh, liver, and brain		Tanner and Clayton 1990
Amphibians					
Frog (*Rana hexadactyla*) tadpoles	As (III)	249	96-h LC50	Conditions: 15°C, pH 6.1, hardness 20 mg/kg (as $CaCO_3$)	SJVDP 1990
		270	48-h LC50		
		368	24-h LC50		

[1] Maximum acceptable toxicant concentration. Lower value in the range shown 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 3.—Biological effects of concentrations of various arsenicals in sediment

[Concentrations in milligrams per kilogram; dw, dry weight; ww, wet weight]

Species	As compound	Concentration in sediment	Concentration in biomass and other effects	Reference
Plants				
Mixed submerged macrophytes	Total As	3.6–5.0 (ww)	2.3–26 (ww)	Tanner and Clayton 1990
		19–38 (ww)	5.7–7.9 (ww)	
		20–105 (ww)	66–80 (ww)	
Stoneworts (*Nitella hookeri*)	Total As	100–780 (dw)	2,400–1,128 (dw)	
Stoneworts (*Chara corallina*)		<0.01 (dw)	200–240 (dw)	
		100–780 (dw)	235–300 (dw)	
Mixed submerged macrophytes (contaminated by mine and industrial effluent)	Total As	40–3,500 (ww)	250–920 (ww)	
		6.3–3,300 (ww)	150–3,700 (ww)	
Spinach plants (*Spinacia oleracea*)	As (V)	10 (in soil)	40% growth reduction	Woolson 1973
Fish				
Midas cichlid (*Cichlasoma citrinellum*)	Total As	5.37–8.65 (dw)	<0.01–0.37 (ww) in muscle. No effect	Lacayo et al. 1992
Jaguar guapote (*Cichlasoma managuense*)			<0.01–0.12 (ww) in muscle. No effect	
Rudd (*Scardinius erythrophthalmus*)	Total As	100–780 (dw)	<0.2 in flesh; 0.3 in scales; 5.5 in gut contents (ww)	Tanner and Clayton 1990
Perch (*Perca fluviatilis*)			0.3–0.5 in flesh; 0.2 in scales (ww)	
Catfish (*Ameiurus nebulosus*)			0.9 in flesh (ww)	
Eel (*Angulia australis*)			0.4 in flesh (ww)	
Birds				
Shag (*Phalacrocorax* sp.)	Total As	100–780 (dw)	<0.2 in flesh; <0.2 in liver; <0.2 in brain (ww)	Tanner and Clayton 1990

Table 4.—Biological effects of arsenicals in the diet on selected species

[LC50, median lethal concentration—50% mortality after a stated time interval. Similarly, LC100 denotes 100% mortality; dw, dry weight]

Species	As compound	Concentration in diet (mg/kg dw)	As concentration in biomass and other effects	Reference
Fish				
Rainbow trout (*Oncorhynchus mykiss*)	As (V)	10	No effect	Eisler 1988
		90	Some adaptation to dietary As observed, as initial negative growth gave way to slow positive growth over time	
	Sodium arsenite	30	Reduced weight gains after 8 weeks	SJVDP 1990
Birds				
Brown-headed cowbird (*Molothrus ater*)	Copper aceto-arsenite	11	1.7 mg/kg dw (maximum whole-body concentration). All survived after 6 months	Eisler 1988
		33	6.6 mg/kg dw (whole body). All survived after 6 months	
		99.8	11-day LC50	
		100	3-month LC100. Brain 6.1 mg/kg dw; liver 40.6 mg/kg dw	
Mallards (*Anas platyrhynchos*)	Sodium arsenate	25	Adult liver 0.49, duckling liver 0.65 mg/kg dw. No significant differences in body weight or growth rate in ducklings, compared to controls fed 0.26 mg/kg dw	Stanley et al. 1994
		100	Adult liver 2.4, duckling liver 4.5 mg/kg dw. Reduced body weight and lower growth rate at 14 days in ducklings from parents fed As. Antagonistic interactions observed between As and Se	Stanley et al. 1994
		200	Duckling liver 5.1 mg/kg ww. Increased mortality, decreased growth, and liver histopathology in ducklings fed a low-protein (7%) diet. As reduced effects of Se when fed together in a diet with adequate protein (22%)	Hoffman et al. 1992
		400	Adult liver 6.6, duckling liver 33 mg/kg dw. Arsenic accumulated in adult liver and egg, reduced adult weight gain and liver weight, delayed onset of egg laying, decreased whole egg weight, and caused eggshell thinning. Reduced body weight, growth, and liver weight in ducklings. Antagonistic interactions observed between As and Se	Stanley et al. 1994
One-day-old mallard ducklings (female)	As (V)	300	Brain 0.8 mg/kg dw; liver 1.3 mg/kg dw. Reduced growth rate, and standing and bathing time; increased resting time over 10 weeks	Camardese et al. 1990
		30	Reduced growth rate over 10 weeks; no significant bioaccumulation in brain or liver when compared to controls	

Table 4.—Biological effects of arsenicals in the diet on selected species—Continued

Species	As compound	Concentration in diet (mg/kg)	Concentration in biomass and other effects	Reference
Birds—Continued				
Chicken	Arsanilic acid	180	1.3 mg/kg in eggs; embryo viability normal	Libby et al. 1953
		>1,000	Depressed growth	Abbott et al. 1959
		2,000	Mortality increased 33%	
		2,250	Mortality increased 40%	
Mammals				
Domestic sheep	Total As	58	No outwardly visible effect. Tissue As increased after 3-week exposure, then declined rapidly after return to low-As diet	Eisler 1988
Mice	As (III)	0.46	No significant difference in growth and survival	Schroeder and Balassa 1967
Rats	As (III)+(V)	5	No significant differences in growth and survival	Sharpless and Metzger 1940
		20	Growth decreased by 50%	

Table 5.—U.S. Environmental Protection Agency standards and criteria for arsenic

[See Appendix II for explanation of terms. Sources: EPA, 1985, 1995]

Status	Known carcinogen; EPA priority pollutant
Drinking water MCL[1]	50 µg/L
Freshwater criteria (AS-III)	360 µg/L for acute exposure 190 µg/L for chronic exposure
Freshwater LOAEL[2] (As-V)	850 µg/L for acute exposure
1/10,000 cancer risk	2 µg/L
1/1,000,000 cancer risk	0.018 µg/L (water and organisms) 0.14 µg/L (organisms only)

[1] Maximum contaminant level
[2] Lowest-observed-adverse effect level.

References

Abbott, O.J., H.R. Bird, and C.A. Baumann. 1959. Arsenic deposition in tissues of chickens fed arsanilic acid. *Poult. Sci.* 34: 1175.

Birge, W.J., and O.W. Roberts. 1976. Toxicity of metals to chick embryos. *Bull. Environ. Contam. Toxicol.* 16:319–324.

Bodek, I., W.J. Lyman, W.F. Reehl, and D.H. Rosenblatt. 1988. *Environmental inorganic chemistry: properties, processes, and estimation methods*. Pergamon Press, New York.

Brown, K.W., G.B. Evans, Jr., and B.D. Frentrup. 1983. *Hazardous waste land treatment.* Butterworth Publishers, Woburn, Massachusetts. p. 240–243.

Buhl, K.J., and S.J. Hamilton. 1991. Relative sensitivity of early life stages of arctic grayling, coho salmon, and rainbow trout to nine inorganics. *Ecotoxicol. Environ. Safety* 22:184–197.

Byrne, A.R., L. Kosta, and P. Stegner. 1975. The occurrence of mercury in amphibia. *Environ. Lett.* 8:147-155.

Cain, D.J., S.N. Luoma, J.L. Carter, and S.V. Fend. 1992. Aquatic insects as bioindicators of trace element contamination in cobble-bottom rivers and streams. *Can. J. Fish. Aquat. Sci.* 10:2141–2154.

Camardese, M.B., D.J. Hoffman, L.J. LeCaptain, and G.W. Pendleton. 1990. Effects of arsenate on growth and physiology in mallard ducklings. *Environ. Toxicol. Chem.* 9:785-795.

Cantu, R., M. Stacell, and M. Webb. 1991. *Observations of abnormalities in largemouth bass fry exposed to arsenic contaminated water*. Texas Parks and Wildlife Department, Austin, Texas.

Cearley, J.E. 1973. *Arsenic contamination in Finfeather and Municipal Lakes on the City of Bryan*. Texas Water Quality Board, Austin, Texas.

Clark, D.R., Jr., R. Cantu, D.F. Cowman, and D.J. Maxon. In press. Uptake of arsenic and metals by tadpoles at an historically contaminated Texas site. *Ecotoxicology*.

Clement, W.H., and S.D. Faust. 1973. A new convenient method for determining arsenic (+3) in natural waters. *Environ. Lett.* 5:155–164.

Eisler, R. 1988. *Arsenic hazards to fish, wildlife, and invertebrates: A synoptic review.* U.S. Fish and Wildlife Service Biological Report 85(1.12). 92 p.

Eisler, R. 1994. A review of arsenic hazards to plants and animals with emphasis on fishery and wildlife resources. In: *Arsenic in the environment, part II: Human health and ecosystem effects.* J.O. Nriagu, ed. John Wiley & Sons, New York. p. 185–259.

EPA (Environmental Protection Agency). 1985. Ambient water quality criteria for arsenic—1984. U.S. Environmental Protection Agency Report 440/5–84–033. 66 p.

EPA (Environmental Protection Agency). 1994. National primary drinking water standards. U.S. Environmental Protection Agency Report 810–F–94–001.

EPA (Environmental Protection Agency). 1995. Drinking water regulations and health advisories. U.S. Environmental Protection Agency, Office of Water, Washington. 11 p.

Foley, R.E., J.R. Spotila, J.P. Giesey, and C.H. Wall. 1978. Arsenic concentrations in water and fish from Chautauqua Lake, New York. *Environ. Biol. Fishes* 3(4):361–367.

Gilani, S.H., and Y. Alibhai. 1990. Teratogenicity of metals to chick embryos. *J. Toxicol. Environ. Health* 30:23–31.

Gosh, A.R., and P. Chakrabarti. 1990. Toxicity of arsenic and cadmium to a freshwater fish *Notopterus notopterus*. *Environ. Ecol.* 8:576–579.

Hall, R.J., and B.M. Mulhern. 1984. Are anuran amphibians heavy metal accumulators? In: *Vertebrate ecology and systematics—A tribute to Henry S. Fitch.* R.A. Seigel et al., eds. University of Kansas Museum of Natural History, Lawrence, Kansas. p. 123–133.

Hamilton, S.J., and K.J. Buhl. 1990. Safety assessment of selected inorganic elements to fry of Chinook salmon (*Oncorhynchus tshawytscha*). *Ecotoxicol. Environ. Safety* 20:307–324.

Hoffman, D.J., C.J. Sanderson, L.J. LeCaptain, E. Cromartie, and G.W. Pendleton. 1992. Interactive effects of arsenate, selenium, and dietary protein on survival, growth, and physiology in mallard ducklings. *Arch. Environ. Contam. Toxicol.* 22:55–62.

Hull, R.N., and G.W. Suter, II. 1994. *Toxicological benchmarks for screening contaminants of potential concern for effects on sediment-associated biota: 1994 revision.* Oak Ridge National Laboratory, Environmental Sciences Division, Oak Ridge, Tennessee. Rept. ES/ER/TM-95/ R1. Prepared for U.S. Department of Energy, Office of Environmental Restoration and Waste Management.

Kabata-Pendias, A., and H. Pendias. 1992. *Trace elements in plants and soils.* CRC Press, Inc., Boca Raton, Florida. 365 p.

Khangarot, B.S., and P.K. Ray. 1989. Sensitivity of midge larvae of *Chironomus tentans* Fabricius (*Diptera Chironomidae*) to heavy metals. *Bull. Environ. Contam. Toxicol.* 42:325–330.

Khangarot, B.S., A. Sehgal, and M.K. Bhasin. 1985. Man and biosphere—Studies on the Sikkim Himalayas. Part 5: Acute toxicity of selected heavy metals on the tadpoles of *Rana hexadactyla*. *Acta Hydrochim. et Hydrobiol.* 13:259–263.

Lacayo, M.L., A. Cruz, S. Calero, J. Lacayo, and I. Fomsgaard. 1992. Total arsenic in water, fish and sediments from Lake Xolotlán, Managua, Nicaragua. *Bull. Environ. Contam. Toxicol.* 49:463–470.

Libby, D.A., A.C. Groschke, R.J. Evans, and S.L. Bandemer. 1953. Feeding of arsenic acid to pullets. *Michigan Agr. Expt. Sta. Quart. Bull.* 34:419–427.

Long, E.R., D.D. MacDonald, S.L. Smith, and F.D. Calder. 1995. Incidence of adverse biological effects within ranges of chemical concentrations in marine and estuarine sediments. *Environ. Manage.* 19:81–97.

Long, E.R., and L.G. Morgan. 1990. The potential for biological effects of sediment-sorbed contaminants tested in the National Status and Trends Program. U.S. National Oceanic and Atmospheric Administration, Seattle, Washington. Technical Memorandum NOS OMA 52. 175 p.

Manahan, S.E. 1989. *Toxicological chemistry.* Lewis Publishers, Chelsea, Michigan.

McGeachy, S. M., and D.G. Dixon. 1990. Effect of temperature on the chronic toxicity of arsenate to rainbow trout (*Oncorhynchus mykiss*). *Can. J. Fish. Aquat. Sci.* 47:2228–2234.

National Academy of Sciences. 1977. *Arsenic.* National Academy Press, Washington. 332 p.

Ohlendorf, H.M., J.P. Skorupa, M.K. Saiki, and D.A. Barnum. 1993. Food-chain transfer of trace elements to wildlife. In: *Management of irrigation and drainage systems: Integrated perspectives.* R.G. Allen and C.M.U. Neale, eds. Proceedings of the 1993 National Conference on Irrigation and Drainage Engineering. Park City, Utah; July 21–23, 1993. American Society of Civil Engineers, New York. p. 596–603.

Opresko, D.M., B.E. Sample, and G.W. Suter. 1994. *Toxicological benchmarks for wildlife: 1994 revision.* Oak Ridge National Laboratory, Health Sciences Research Division and Environmental Sciences Division, Oak Ridge, Tennessee. Rept. ES/ER/TM–86/R1. Prepared for United States Department of Energy, Office of Environmental Restoration and Waste Management.

Pendleton, G.W., M.R. Whitworth, and G.H. Olsen. 1995. Accumulation and loss of arsenic and boron, alone and in combi-nation, in mallard ducks. *Environ. Toxicol. Chem.* 14(8):1357–1364.

Sanders, J.G., and S.J. Cibik. 1985. Adaptive behavior of euryhaline phytoplankton communities to arsenic stress. *Marine Ecol. Prog. Ser.* 22:199–205.

Sandhu, S.S. 1977. Study on the post-mortem identification of pollutants in the fish killed by water pollution: Detection of arsenic. *Bull. Environ. Contam. Toxicol.* 17:373–470.

Schmitt, C.J., and W.G. Brumbaugh. 1990. National contaminant biomonitoring program: Concentrations of arsenic, cadmium, copper, lead, mercury, selenium, and zinc in U.S. freshwater fish, 1976–1984. *Arch. Environ. Contam. Toxicol.* 19:731–747.

Schroeder, H.A., and J.J Balassa. 1967. Arsenic, germanium, tin, and vanadium in mice: Effects on growth, survival and tissue levels. *Nutrition* 92:245–252.

Sharma, R.P., and J.L. Shupe. 1977. Lead, cadmium, and arsenic residues in animal tissues in relation to those in their surrounding habitat. *Sci. Total Environ.* 7:53–62.

Sharpless, G.R., and M. Metzger. 1940. Arsenic and goiter. *J. Nutr.* 21:341–346.

SJVDP (San Joaquin Valley Drainage Program). 1990. *Fish and wildlife resources and agricultural drainage in the San Joaquin Valley, California.* San Joaquin Valley Drainage Program, Sacramento, California. Vol. 1, sec. 3.

Smith, K.S., W.H. Ficklin, G.S. Lumlee, and A.L. Maier. 1992. Metal and arsenic partitioning between water and suspended sediment at mine-drainage sites in diverse geologic settings. In: *Water-Rock Interaction: Proceedings of the 7th International Symposium on Water-Rock Interaction, Park City, Utah, 13-18 July 1992.* A.S. Maest and Y.K. Kharaka, eds. A.A. Balkema, Rotterdam and Brookfield, Vermont. p. 443–447.

Sorensen, E.M., R.R. Mitchell, A. Pradzynski, T.L. Bayer, and L.L. Wenz. 1985. Stereological analyses of hepatocyte changes parallel arsenic accumulation in the livers of green sunfish. *J. Environ. Pathol. Toxicol.* 6:195–210.

Spehar, R.L., J.T. Fiandt, R.L. Anderson, and D.L. De Foe. 1980. Comparative toxicity of arsenic compounds and their accumulation in invertebrates and fish. *Arch. Environ. Contam. Toxicol.* 9:53–63.

Stanley, T.R., Jr., J.W. Spann, G.J. Smith, and R. Rosscoe. 1994. Main and interactive effects of arsenic and selenium on mallard reproduction and duckling growth and survival. *Arch. Environ. Contam. Toxicol.* 26:444–451.

Stute, K., and H. Vogt. 1968. The effect of 3-nitro-4-hydroxyphenylarsonic acid (3-Nitro-HOECHST) on egg production and food conversion of laying hens and residue analyses of muscle, organs and eggs. *Br. Poult. Sci.* 9:121–128.

Suter, G.W., II, and J.B. Mabrey. 1994. *Toxicological benchmarks for screening potential contaminants of concern for effects on aquatic biota: 1994 revision*. Oak Ridge National Laboratory, Environmental Sciences Division, Oak Ridge, Tennessee. Rept. ES/ER/TM–96/R1. Prepared for U.S. Department of Energy, Office of Environmental Restoration and Waste Management.

Talmage, S.S., and B.T. Walton. 1991. Small mammals as monitors of environmental contaminants. *Rev. Environ. Contam. Toxicol.* 119:47–145.

Tanner, C.C., and J.S. Clayton. 1990. Persistence of arsenic 24 years after sodium arsenite herbicide application to Lake Rotora, Hamilton, New Zealand. *N.Z. J. Marine Freshwater Res.* 24:173–179.

Wagemann, R., N.B. Snow, D.M. Rosenberg, and A. Lutz. 1978. Arsenic in sediments, water and aquatic biota in lakes from the vicinity of Yellowknife, Northwest Territories, Canada. *Arch. Environ. Contam. Toxicol.* 7:169–191.

Woolson, E.A. 1973. Arsenic phytotoxicity and uptake in six vegetable crops. *Weed Sci.* 21:524–527.

Woolson, E.A. (ed.). 1975. Arsenical pesticides. *Am. Chem. Soc. Symp. Ser.* 7. 176 p.

Yannai, S.M., K. Sachs, B. Kantorowitz, and Z. Berk. 1979. Secondary toxicology and contaminants of algae grown on waste water. *Nutr. Rep. Int.* 19:391–400.