ENVIRONMENTAL CONTAMINANTS ENCYCLOPEDIA

ARSENIC ENTRY

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Like a library or most large databases (such as EPA's national STORET water quality database), this document contains information of variable quality from very diverse sources. In compiling this document, mistakes were found in peer reviewed journal articles, as well as in databases with relatively elaborate quality control mechanisms [366,649,940]. A few of these were caught and marked with a "[sic]" notation, but undoubtedly others slipped through. The [sic] notation was inserted by the editors to indicate information or spelling that seemed wrong or misleading, but which was nevertheless cited verbatim rather than arbitrarily changing what the author said.

Most likely additional transcription errors and typos have been added in some of our efforts. Furthermore, with such complex subject matter, it is not always easy to determine what is correct and what is incorrect, especially with the "experts" often disagreeing. It is not uncommon in scientific research for two different researchers to come up with different results which lead them to different conclusions. In compiling the Encyclopedia, the editors did not try to resolve such conflicts, but rather simply reported it all.

It should be kept in mind that data comparability is a major problem in environmental toxicology since laboratory and field methods are constantly changing and since there are so many different "standard methods" published by EPA, other federal agencies, state agencies, and various private groups. What some laboratory and field investigators actually do for standard operating practice is often a unique combination of various standard protocols and impromptu "improvements." In fact, the interagency task force on water methods concluded that [1014]:

> It is the exception rather than the rule that water-quality monitoring data from different programs or time periods can be compared on a scientifically sound basis, and that...
>
> No nationally accepted standard definitions exist for water quality parameters. The different organizations may collect data using identical or standard methods, but identify them by different names, or use the same names for data collected by different methods [1014].

Differences in field and laboratory methods are also major issues related to (the lack of) data comparability from media other than water: soil, sediments, tissues, and air.

In spite of numerous problems and complexities, knowledge is often power in decisions related to chemical contamination. It is therefore often helpful to be aware of a broad universe of conflicting results or conflicting expert opinions rather than having a portion of this information arbitrarily censored by someone else. Frequently one wants to know of the existence of information, even if one later decides not to use it for a particular application. Many would like to see a high percentage of the information available and decide for themselves what to throw out, partly because they don't want to seem uniformed or be caught by surprise by potentially important information. They are in a better position if they can say: "I knew about that data, assessed it based on the following quality assurance criteria, and decided not to use it for this application." This is especially true for users near the end of long decision processes, such as hazardous site cleanups, lengthy ecological risk assessments, or complex natural resource damage assessments.

For some categories, the editors found no information and inserted the phrase "no information found." This does not necessarily mean that no information exists; it

simply means that during our efforts, the editors found none. For many topics, there is probably information "out there" that is not in the Encyclopedia. The more time that passes without encyclopedia updates (none are planned at the moment), the more true this statement will become. Still, the Encyclopedia is unique in that it contains broad ecotoxicology information from more sources than many other reference documents. No updates of this document are currently planned. However, it is hoped that most of the information in the encyclopedia will be useful for some time to come even with out updates, just as one can still find information in the 1972 EPA Blue Book [12] that does not seem well summarized anywhere else.

Although the editors of this document have done their best in the limited time available to insure accuracy of quotes as being "what the original author said," the proposed interagency funding of a bigger project with more elaborate peer review and quality control steps never materialized.

The bottom line: The editors hope users find this document useful, but don't expect or depend on perfection herein. Neither the U.S. Government nor the National Park Service make any claims that this document is free of mistakes.

The following is one chemical topic entry (one file among 118). Before utilizing this entry, the reader is strongly encouraged to read the README file (in this subdirectory) for an introduction, an explanation of how to search for power key section headings, an explanation of how to use this document in general, an explanation of the organization of each entry, an information quality discussion, a discussion of copyright issues, and a listing of other entries (other topics) covered.

See the separate file entitled REFERENC for the identity of numbered references in brackets.

HOW TO CITE THIS DOCUMENT: As mentioned above, for critical applications it is better to obtain and cite the original publication after first verifying various data quality assurance concerns. For more routine applications, this document may be cited as:

> **Irwin, R.J., M. VanMouwerik, L. Stevens, M.D. Seese**, and **W. Basham.** 1997. Environmental Contaminants Encyclopedia. National Park Service, Water Resources Division, Fort Collins, Colorado. Distributed within the Federal Government as an Electronic Document (Projected public availability

on the internet or NTIS: 1998).

Arsenic (As, CAS number 7440-38-2)

NOTE: This entry contains information on both elemental arsenic and various arsenic compounds.

Brief Introduction:

Br.Class: General Introduction and Classification Information:

Arsenic is a steel-gray brittle metalloid (exhibiting both metallic and non-metallic properties) with chemical properties similar to phosphorous [375,445,488].

Arsenic is a naturally-occurring element. Pure arsenic is a gray metal-like material which is usually found in the environment combined with other elements such as oxygen, chlorine, and sulfur. Arsenic combined with these elements is called inorganic arsenic. Arsenic combined with carbon and hydrogen is called organic arsenic [716].

The two primary forms of arsenic in water are trivalent arsenic (As+3, arsenite (III), CAS number 22569-72-8) and pentavalent arsenic (As+5, arsenate (V), CAS number 17428-41-0). Arsenic as a free element (0-oxidation state) is rarely encountered in natural waters [366]. Soluble inorganic arsenate (+5-oxidation state) predominates under normal conditions since it is thermodynamically more stable in water than arsenite (+3 oxidation state) [366]. Arsenic +3 tends to be more toxic than arsenic +5 [483]. Trivalent arsenicals (like mercury but unlike pentavalent arsenic) react with sulfhydryl protein groups [488] (NOTE: An "arsenical" is a compound which contains arsenic [492].)

Arsenic is listed by the Environmental Protection Agency as one of 129 priority pollutants [58]. Arsenic is also listed among the 25 hazardous substances thought to pose the most significant potential threat to human health at priority superfund sites [93]. Arsenic is included as one of the 19 most regulated chemicals in the 1990 publication "List of lists of worldwide hazardous chemicals and pollutants" [621].

Arsenic is a toxic pollutant designated pursuant to section 307(a)(1) of the Clean Water Act and is subject to effluent limitations [40 CFR 401.15 (7/1/88)] [366].

Br.Haz: General Hazard/Toxicity Summary:

Potential Hazards to Fish, Wildlife, and Other Non-Human

Biota:

Organic forms are usually less harmful than inorganic forms [716,1024]. A typical statement in the literature is that "Trivalent inorganic arsenicals are more toxic to mammals and aquatic species than the pentavalent forms" [375]. EPA has separate water quality criteria for the two forms (see W.General section below).

Generally speaking, inorganic trivalent arsenic is more hazardous to animals than the pentavalent forms [366,716,1024]. Inorganic trivalent arsenic is systematically more poisonous than the pentavalent form [445].

Plants can take up arsenic in a variety of ways: including from fly ash, sludge and by manure dumped on the land [666]. However, it has been found that the edible portions of plants grown on contaminated sources seldom accumulate dangerous levels of As. This is due to a variety of reasons: 1) before the plant can assimilate dangerous levels of arsenic in it's edible portion it may be killed due to the toxic levels of arsenic to the plant; and 2) phosphorus competes with arsenic to gain entry into plants and because it is more readily available it can hinder the entrance of arsenic into the plant [666].

Animals are generally less sensitive to arsenic than plants [196].

Arsenic is one of the most toxic elements to fish [488]. Acute exposures can result in immediate death because of As-induced increases in mucus production, causing suffocation, or direct detrimental effects on the gill epithelium [488]. Chronic exposures can result in the accumulation of the metalloid to toxic levels; the detoxification role of the liver places the liver at considerable risk [488]. In fish, bizarre morphological alterations, as well as early neoplastic alterations are produced in the liver [488].

Changes in water hardness did not significantly alter the acute toxicities of arsenic to larval striped bass [445]. Arsenic water criteria are not governed by hardness [893].

The signs of inorganic trivalent arsenite poisoning in birds (mallard, quail, pheasant) include ataxia, goose-stepping ataxia, asthenia, slowness, jerkiness, falling, hyporeactivity, fluffed

feathers, ptosis, huddled position, unkempt appearance, loss of righting reflex, immobility, and tetanic seizures. Signs appeared as soon as 1 hour and mortalities usually after exposure; remission took up to 1 month [445].

Unlike mercury, inorganic arsenic has generally been thought to be more toxic and otherwise hazardous to animals than organic arsenic [366]. Methylation and excretion is one way arsenic has sometimes been considered a detoxification mechanism in mammals [366]. However, much of the arsenic ingested orally tends to remain in the body, and almost no information is available on the effects of organic arsenic forms in humans [716]. It was only recently recognized that a potentially significant amount of the arsenic in food is inorganic, and that even organic arsenic may pose some risk (Willard R. Chappell, University of Colorado at Denver, personal communication, 1995). In animals, very high doses of organic arsenic can produce some of the same effects as inorganic arsenic [716]. This fact, coupled with the fact that arsenic in certain forms and low quantities seems to have beneficial effects, means that the full ramifications of significant quantities of organic arsenic in the diets of animals (as well as humans, where seafood and chicken are among the many sources) is not completely understood. For a long time, inorganic arsenic was thought of the main culprit, so relatively little work has been done on organic arsenic. However, in a 1997 review, Neff once again concluded that organic arsenic compounds are usually not very hazardous [1024].

Examples of the "beneficial uses" (beneficial to the farmer perhaps) of arsenic include the use of organic arsenic compounds as animal feed additives in swine and chickens to increase the rate of weight gain and to control swine dysentery [483]. Carbarsone and nitarsone (4-nitrophenylarsanilic acid) act as anti-histomonads in chickens [483]. Arsanilic acid, sodium ansenilate, and Roxarsone have a growth promoting effect similar to antibiotics [740].

Certain (organic) arsenicals have been approved for use in chickens, turkeys, and swine; claimed benefits in fowl include increasing egg production in layers. However, the redeeming qualities of certain arsenic compounds are not as well publicized as the hazards of arsenic. Even the use of arsenic in animal feed has prompted some

concern. For example, in East Texas, reports of cattle being poisoned after drinking from chicken-manure impacted creeks were reported (Suzanne Dodson, Fish and Wildlife Service, personal communication, 1993).

The use of organic arsenic growth-promoter feed additives in chicken and swine can result in arsenic poisoning (when the dose is too high) and increased arsenic levels in liver, skin, and muscle tissues [772].

In 1985, Hem updated a summary of many basic water quality issues related to this element, including its sources and species, solubility controls, and its occurrence in natural water [190].

Although arsenic has been shown to be a required nutrient in several animal species and, as such, may also be required for humans, there is no evidence that arsenic is essential for plant growth [445].

Ron Eisler summarized effects of arsenic on non-humans in 1988 (Arsenic Hazards to Fish, Wildlife, and Invertebrates: A Synoptic Review, Report No 12, 1988)[21] and in 1994 in the form of a chapter in Nriagu's book [654] on arsenic effects on both humans and non-humans.

Potential Hazards to Humans:

Relatively high doses of arsenic have been reported to cause bone marrow suppression in humans; other marrow effects associated with arsenic and arsine (arsenic hydride) are seen in rats and other species [893].

Smokers and those regularly consuming large amounts of seafood may be exposed to higher than average levels of arsenic [716].

Inhalation of arsenic from ambient air is usually a minor exposure route for the general population [716]. For example, the dose to a person who breathes 20 m3/day of air containing 20-30 ng/m3 would be about 0.4-0.6 mg/d [716]. However, smokers may be exposed to arsenic by inhalation of mainstream smoke [716]. Assuming that 20% of the arsenic in cigarettes is present in smoke, an individual smoking two packs of cigarettes per day would inhale about 12 mg of arsenic [716].

Occupational exposure to arsenic may be significant

in several industries, mainly nonferrous smelting, arsenic production, wood preservation, glass manufacturing, and arsenical pesticide production and application [716]. The National Institute for Occupational Safety and Health (NIOSH) estimated that about 55,000 workers were exposed to arsenic in the early 1980s [716]. The principal exposure pathway is probably inhalation of arsenic adsorbed to particulates, but ingestion and possibly dermal exposure may also be common [716]. However, no information was located on typical exposure levels in the workplace [716]. Since arsenic is no longer produced in the United States, and many arsenical pesticide uses have recently been banned, it is likely that the number of workers occupationally exposed to arsenic has decreased in recent years [716].

Inorganic arsenic in high amounts has been known for centuries as a fast acting human poison [716]. In humans recently in contact with arsenic contaminated soil, a more subtle hazard would be that some arsenic could be ingested from arsenic contaminated hands while eating (this would be particularly dangerous if a child licked fingers while eating).

The organic compound arsenobetaine is the principle arsenic compound in marine animals and seafood [604,1024]. This compound is not toxic to mammals [0124]. Marine arsenic represents a low risk to human consumers of seafood [1024].

Arsenic is thought to account for about 1/2 the risk from coal fly ash and about 2/3 of the risk from oil fly ash; it has been implicated in black-foot gangrene and suspected related to ischemic heart disease and diabetes (Willard R. Chappell, University of Colorado at Denver, personal communication, 1995).

Some immunological and neural effects have been documented for arsenic, but study information is incomplete, especially for organic arsenic [716].

A comprehensive toxicological profile for arsenic and its compounds, especially as it relates to human health, is available from ATSDR [716]. Due to lack of time, important highlights from this ATSDR document have not yet been completely incorporated into this entry.

Potential Benefits of Certain Forms of Arsenic:

Although most references to arsenic in the environmental literature refer only to its potentially harmful effects, there are some references which indicate that arsenic in certain forms, and doses can have some redeeming qualities in living things. One author stated that there is growing evidence that arsenic is an essential micro co-nutrient in animals and humans [1024].

The need for arsenic in nutrition was shown by three laboratories in four mammalian species [366]. Reported data indicate that we have yet to learn the optimal intake levels for arsenic and how its decreased bioavailability affects human health [366].

The Canadian government quoted a 1988 EPA document which postulates that although arsenic may be essential to some animals, there is no indication of its need in humans [604]. Arsenic has had a long use in medicine and certain generally less toxic forms continue to be used to treat parasites in 1994 [654]. Arsenic has also been detected in several homeopathic medicines [716] (see details in Uses/Sources section far below).

Although (in the past) arsenic has more often been thought of as a contaminant than as an nutritionally essential mineral, some alleged benefits of low doses (possibly to counteract deficiencies) have been reported for tadpoles, caterpillars, rats, goats, poultry, pigs, potatoes, corn, rye and wheat [654].

Potential beneficial effects of low doses of certain arsenic compounds and many other details of arsenic toxicity are not yet completely understood (Willard R. Chappell, University of Colorado at Denver, personal communication, 1995).

Br.Car: Brief Summary of Carcinogenicity/Cancer Information:

Arsenobetaine, the principle arsenic compound in seafood [604,1024] is not carcinogenic to mammals [0124]. The EPA 10-6 cancer risk water criterion of 0.0175 ug/L is much lower than the ambient concentration of inorganic arsenic in clean ocean water and may be overly restrictive based on a lack of understanding that most organic seafood arsenic is not easily converted to the inorganic arsenite form of concern related to cancer risk

[1024].

EPA 1996 IRIS database information [893]:

> Evidence for classification as to human carcinogenicity; weight-of-evidence classification:
>
> Classification: A; human carcinogen [893].
>
> BASIS: Based on sufficient evidence from human data. [893].
>
> HUMAN CARCINOGENICITY DATA: Sufficient. [893].
>
> ANIMAL CARCINOGENICITY DATA: Inadequate. There has not been consistent demonstration of carcinogenicity in test animals for various chemical forms of arsenic administered by different routes to several species. [893].

Arsenic has long been a concern to man because small amounts can be toxic to humans [190]. Indeed, arsenic is often thought of as a carcinogenic priority pollutant [446]. Ingestion of arsenic by the oral route has caused an increased incidence of tumors of the liver, blood, and lungs [480]. However, intraspecies comparisons show remarkable differences in the processing of arsenic by humans and rodents; rats excrete less arsenic than humans but rabbits excrete more [488]. Some workers argue that no established mammalian model simulates carcinogenicity in humans [488].

Inorganic arsenic ingested by humans is associated with increased risk of skin cancer [716]. Inhaled inorganic arsenic seems to increase the risk of lung cancer in humans [716]. Arsenic does not seem to directly impact DNA but may inhibit some DNA repair mechanisms [716]. Arsenic is somewhat unusual among contaminants, in that the (epidemiological) evidence suggesting carcinogenic effects on humans seems more convincing or complete than the data relating to lab animals. Inorganic arsenic has not been proven to be a carcinogen in animals and there is no animal model for arsenic carcinogenicity (the data derives from entirely from humans) [Smith, A.H. et al, 1992. Cancer risks from arsenic in drinking water, Environ Health Persp., 97, 259-267].

Recent reviews indicate arsenic has been associated with carcinogenic impacts [21,129,168]. It has been implicated in numerous types of cancer (including skin, bladder, kidney, liver, prostrate, and nasal cavity) (Willard R. Chappell, University of Colorado at Denver, personal communication, 1995).

Although most references to arsenic suggest it is a carcinogen [21,129,168,277,446,480,654], at least one reference counters that arsenic, like selenium, can act as an essential nutrient with anticancer value [Frost DV; Sci Total Environ 28: 455-66 (1983)] [366]. The potential for carcinogenic and noncarcinogenic thresholds are not yet completely understood (Willard R. Chappell, University of Colorado at Denver, personal communication, 1995).

As of September 1995, EPA was considering changing the drinking water MCL from 50 ug/L to 2 ug/L (based on a 1E-04 cancer risk). This proposal was subject to considerable controversy due to the following (Willard R. Chappell, University of Colorado at Denver, personal communication, 1995):

1) There is a fair amount (up to 100 ug/L) of arsenic in well water in parts of California, Utah, New Mexico, Alaska, and Oregon. Many commenting felt 2 ug/L would cost too much to achieve.

NOTE: Even higher (up to 4 mg/L) concentrations are found in aerated-pyrite contaminated well water in India, where thousands of people are showing arsenic poisoning signs such as skin cancer and wart-like (keratosis) skin growths.

2) Two ug/L is below the practical quantitation limit for arsenic in water.

3) Regulating arsenic in soil at the same (1E-04 cancer risk) level would require limiting soil concentration to 37 ppm at CERCLA sites; this would be hard to achieve (the median concentration at the Leadville, CO, site is about 37 ppm).

This compound has been treated as a carcinogen for model calculation purposes in some EPA risk-based (RBC and PRG) models; sometimes non-cancer endpoints are also calculated for comparison [868,903].

Br.Dev: Brief Summary of Developmental, Reproductive, Endocrine, and Genotoxicity Information:

Study results are somewhat mixed and incomplete (especially for organic arsenic), but recent reviews indicate arsenic has been associated with genotoxic, fetotoxic, mutagenic and teratogenic impacts [21,129,168,716]. Arsenic does not seem to directly impact DNA but may inhibit some DNA repair mechanisms [716].

Injection of sublethal concentration of arsenic into chicken eggs produced ectopic conditions, but no teratogenic effect (National Research Council. Drinking Water & Health Volume 1. Washington, DC: National Academy Press, 1977. 339)[366].

In humans, inorganic arsenic crosses the placenta and was reported to cause death without early chelation therapy [363].

There is no evidence that adverse effects on human reproduction will occur at permissible exposure limits (Council on Scientific Affairs, 1985)[363].

Arsenic (inorganic) is teratogenic in rodents at doses of 20 mg/kg or greater [363].

Among smelter workers exposed to a mixture of metals including arsenic, the frequency of congenital malformations did not differ from non-exposed populations. However, mean birth wt were reported to be decr in offspring of female employees of the smelter (Friberg, L., Nordberg, G.F., Kessler, E. and Vouk, V.B. (eds). Handbook of the Toxicology of Metals. 2nd ed. Vols I, II.: Amsterdam: Elsevier Science Publishers B.V., 1986.,p. V1 414)[366].

Testicular and ovarian degeneration was observed in freshwater perch exposed to arsenite (arsenic+3 oxide) [445].

Arsenic may inhibit some DNA repair mechanisms [716].

The genotoxic potential of 48 inorganic derivatives was studied using the bacterial colorimetric assay: the SOS Chromotest. Some of these compounds are known carcinogens (arsenic, chromium(VI), cadmium, nickel) or suspected carcinogens for human beings (mercury, lead), others are non-carcinogens. Among these 48 derivatives, only the two chromium(VI) and the tin(II) compounds gave positive results (Olivier P, Marzin D; Mutat Res 189, 3: 263-70, 1987)[366].

Br.Fate: Brief Summary of Key Bioconcentration, Fate, Transport, Persistence, Pathway, and Chemical/Physical Information:

The ultimate sink for most environmental arsenic is ocean sediment. Because of its reactivity and mobility, arsenic can cycle extensively through both biotic and abiotic components of local aquatic and terrestrial systems. Here it can undergo a variety of chemical and biochemical transformations, such as oxidation,

reduction, methylation, and demethylation [604].

Most arsenic in the environment exists in soil or rock [716]. This material may be transported by wind or water erosion of small particles, or may be transported by leaching into rainfall or snowmelt [716]. However, because many arsenic compounds tend to adsorb to soils or sediments, leaching usually results in transportation over only short distances in soil [716].

Ground water normally contains higher concentrations of arsenic than are found in associated surface water [604].

Transport and partitioning of arsenic in water depends upon the chemical form (oxidation state and counter ion) of the arsenic and on interactions with other materials present [716]. Soluble forms move with the water, and may be carried long distances through rivers [716]. However, arsenic may be adsorbed from water onto sediments or soils, especially clays, iron oxides, aluminum hydroxides, manganese compounds, and organic material [716]. Sediment-bound arsenic may be released back into the water by chemical or biological inter-conversions of arsenic species [716].

Arsenate (As+5) is the main species found in oxidizing environments [445,1024]. Reducing conditions (such as reducing sediments) favor the much more hazardous chemical form of arsenite (As+3) [445,1024].

Although arsenic is bioconcentrated by aquatic organisms (especially invertebrates), it does not appear to biomagnify up the food chain [445]. Preliminary data suggests the potential for bioaccumulation or bioconcentration of arsenic is moderate for the following biota: mammals, birds, fish, mosses, lichens, and algae [83]. The potential is considered high to very high for mollusks, crustacea, lower animals, and higher plants [83]. The best potential mediums for biological monitoring appear to include animal hair, clams, algae, and higher plants [83]. Uptake of arsenic by phytoplankton can be significant [196].

One reference stated that "Arsenic is one of the few metals which tends to concentrate in axial muscles of fish" [29]. However, Sorensen's summary of 1991 stated that when compared to food web organisms, fish generally concentrate strikingly low concentrations of arsenic in skeletal muscles [488]. One study showed that planktivorous fish accumulated more arsenic than omnivores [488]. Bottom feeders tend to accumulate more arsenic than pelagic fish [488].

In animals, arsenic tends to slowly build up in the

liver, from which it is slowly redistributed to other tissues [772]. Arsenic compounds which are not very soluble, such as arsenic trioxide, are poorly absorbed [772]. Relatively soluble arsenic compounds such as sodium arsenite are rapidly absorbed through intact skin (and even more rapidly absorbed through wounds) [772].

Although fish and shellfish can build up arsenic in their tissues, most of this is in an (organic) form thought to less toxic (than inorganic arsenic) [716].

Arsenic can enter the environment in several ways [716]. Even though it does not evaporate, arsenic can get into air as dust [716]. This can happen when smelters heat ores containing arsenic, when people burn any material containing arsenic, or when wind blows soil that contains arsenic into the air [716]. Once in the air, the arsenic particles will travel with the wind for a while, but will then settle back to the ground [716]. Most arsenic compounds can also dissolve in water [716]. Thus, arsenic can get into lakes, rivers, or underground water by dissolving in rain or snow, or through the discharge of industrial wastes [716]. Some of the arsenic will stick to the sediment on the bottom of the lake or river, and some will be carried along by the water [716].

Arsenic is not broken down or destroyed in the environment [716]. However, it can change from one form to another by natural chemical reactions, and also by the action of bacteria that live in soil or water [716]. Although some fish and shellfish build up arsenic in their tissues, most of this is in a form (often called "fish arsenic") that is not toxic [716].

Biogeochemical Cycling Summary [445]:

> Arsenic is very mobile in the environment. Arsenic transport is governed by complex conditions in sediment, soil, air, water, and organisms in which chemical and/or biochemical processes take place. Rocks are a natural source of arsenic, but rivers seem to cleanse themselves of soluble arsenic [445].
>
> An important factor in the natural circulation of arsenic is the volatility of the element and some of its compounds [190,445].

Breakdown of arsenic compounds stops with elemental arsenic (arsenic itself is not broken down or destroyed in the environment) [716], so many remediation efforts are aimed at immobilizing (often by combination into less soluble compounds) or removing arsenic to hazardous waste sites. Surface and groundwater as well as wind-blown

dust are important media for arsenic transport pathways [716].

Arsenic as a free element (0-oxidation state) is rarely encountered in natural waters [366]. Soluble inorganic arsenate (+5-oxidation state) predominates under normal conditions since it is thermodynamically more stable in water than arsenite (+3 oxidation state, USEPA; Ambient Water Quality Criteria Doc: Arsenic p.A-1, 1980, EPA 440/5-80-021)[366].

Arsenic +5 can form relatively insoluble metallic salts with a number of cations (e.g., arsenates of aluminum, calcium, copper, iron, nickel, lead, magnesium, and zinc) [445]. Arsenic trioxide is the primary product of arsenic smelters and is only slightly soluble in water; once in the general environment, arsenic trioxide undergoes oxidation, reduction, methylation, and demethylation [445]. Oxidation of elemental arsenic or arsenic trioxide yields arsenic pentoxide, which is very soluble in water [445].

Arsenic is volatilized from the soil as arsine, which is produced through chemical reduction by soil microorganisms. Arsenic is also lost from surface soils through leaching. The amount removed by leaching is related to the solubility of arsenic, which is greater in sandy or low-clay soils; the solubility of arsenic is reduced by the adsorption of arsenic onto organic matter and charged surfaces of clays, and the binding of arsenic to metallic compounds. In general, organisms can have a significant influence on the distribution of arsenic in the environment by accumulating, transporting, and transforming it. Some of the transformations, such as oxidation and reduction, are probably catalyzed by the presence of organisms, but occur in their absence; other processes, such as methylation, occur only in the presence of organisms [445].

Synonyms/Substance Identification:

ARSENIC BLACK [366]
ARSENIC-75 [366]
COLLOIDAL ARSENIC [366]
Gray arsenic [366]
Metallic arsenic [366]

Associated Chemicals or Topics (Includes Transformation Products):

NOTE: The chemistry of arsenic is complex [445]. Arsenic is classified as a metalloid, but it exhibits both metallic and non-metallic properties. For example, the four oxidation states in which arsenic forms

inorganic compounds are +5, +3, 0, and -3 [445]. Arsenic combined with carbon and hydrogen is called organic arsenic [716]. See information from reference [445] in the Forms/Preparations/Formulations section for various inorganic and organic arsenic compounds.

In the open literature, information may be searched for under the following topics:

Arsenic III, Arsenic V, Arsenicals, various topics starting with letters "arsen", Calcium arsenate, Sodium Arsenite, Cupric acetoarsenite, Arsenic Acid (Desiccant L-10), various other individual arsenic compounds, and Arsenic Trioxide.

Arsenic and gold are often found together and often elevated in the same plants, soils, or rocks, so that arsenic concentrations are used in prospecting for gold [951].
Metabolites of Arsenic [366]:

The major metabolite in urine of experimental animals exposed to inorganic arsenic is dimethylarsinic acid. The marmoset monkey is the only species to date which has been found to be unable to methylate inorganic arsenic. In man the urinary excretion at low levels consists of about 20% inorganic arsenic, 20% methylarsonic acid and 60% dimethylarsinic acid. [Friberg, L., Nordberg, G.F., Kessler, E. and Vouk, V.B. (eds). Handbook of the Toxicology of Metals. 2nd ed. Vols I, II.: Amsterdam: Elsevier Science Publishers B.V., 1986.,p. V2 53].

The arsenic compound in which flounder appears to be stable and intact during metabolism since it does not adhere to the erythrocytes of rats as all arsenic cmpd were previously expected to do. It was also indicated that when crab meat containing arsenic was ingested, the cmpd appeared to be excreted intact. Therefore, it appears that dietary arsenic, as that occurring in marine products, is metabolically stable. [Siewicki TC; J Nutr 111: 602-9 (1981)].

Factors which were discussed influence the methylation of inorganic arsenic by rat liver. S-adenosylmethionine alone administered or in association with reduced or oxidized glutathione or acetylcysteine and the increase of hepatic reduced or oxidized glutathione level by butylated hydroxytoluene pretreatment do not stimulate the urinary excretion of the methylated arsenic metabolites following a challenge dose of inorganic arsenic. Conversely a reduction of the hepatic reduced or oxidized glutathione level by phorone pretreatment greatly modifies the metabolism of inorganic arsenic in vivo. A reduction exceeding 90% of the control value leads to a decreased urinary excretion of

monomethylarsonic and dimethylarsinic acid and an increased urinary excretion of inorganic arsenic. This is also associated with an increased accumulation of inorganic arsenic in the liver. A drastic reduction of reduced or oxidized glutathione level in liver not only impairs the methylation of inorganic arsenic but also impairs its biliary excretion. When reduced or oxidized glutathione depletion is less severe, the total amount of arsenic excreted in urine after a challenge dose of sodium arsenate is not significantly different from that found in un-pretreated animals but the proportion of the three metabolic forms is different: MMA is reduced whereas ASI and DMA tend to increase. These changes resemble those found in patients with liver insufficiency. Long-term pretreatment of rats with carbon tetrachloride slightly reduces the amount of monomethyl arsenic and dimethylarsinic acids excreted in urine following a challenge dose of inorganic arsenic. This effect may result from a reduction of reduced or oxidized glutathione transferase activity by CCl4. [Buchet JP, Lauwreys R; Toxicol Appl Pharmacol 91 (1): 65-74 (1987)].

Water Data Interpretation, Concentrations and Toxicity (All Water Data Subsections Start with "W."):

W.Low (Water Concentrations Considered Low):

No information found.

W.High (Water Concentrations Considered High):

Elevated arsenic in groundwater, in one case 30 ppb, has been found around several pre-1910 cemetaries impacted by arenic embalming fluid (see also: uses/sources section below) [976].

In Texas, a total of 2,552 samples were tested for arsenic from 100 countries for the period 1968-1990 of which 119 (4.66 %) samples exceeded the drinking water standard of 50 ug/L [424].

1971: Concentrations above 50 ug/L accounted for 2% of the samples taken in stream waters and were presumed to be the result of waste disposal [190].

In parts of the U.S., 50 to 4,000 ppm arsenic in water would be considered "elevated" (Willard R. Chappell, University of Colorado at Denver, personal communication, 1995).

There is a fair amount (up to 100 ug/L) of arsenic in well water in parts of California, Utah, New Mexico, Alaska, and Oregon. Even higher (up to 4 mg/L)

concentrations are found in aerated-pyrite contaminated well water in India, where thousands of people are showing arsenic poisoning signs such as skin cancer and wart-like (keratosis) skin growths (Willard R. Chappell, University of Colorado at Denver, personal communication, 1995).

Arsenic concentrations are greater in waters of the Tulare Basin than in the San Joaquin Basin. For example, median (minimum-maximum) dissolved and total waterborne concentrations of arsenic in subsurface agricultural drainage water inflow to the Tulare Lake Drainage District South Evaporation Ponds were 79.5 (11-110) and 97 (64-190) ug/L (ppb), respectively (Fujii, 1988) [445].

Elevated concentrations of arsenic have been reported in surface waters in the vicinity of gold-mining or ore-roasting operations. Mean levels of about 45 ug/L/(maxima about 140 ug/L) were found near abandoned gold mines in Nova Scotia and Ontario during the 1980s. Highest arsenic levels were found in samples of water collected from several lakes near the gold mines and roasters at Yellowknife in the mid-1970s. For example, Keg and Kam Lakes contained from 700 to 1500 ug As/L and 1500 to 5500 ug As/L, respectively. Limited data from the early 1990s indicated that concentrations in these lakes had declined appreciably, to about 545 and 645 ug/L, in Keg Lake and Kam Lake, respectively [604].

Gold mining activities in Nova Scotia have been reported to contribute to high arsenic levels in local ground waters. High arsenic concentrations (up to 11,000 ug/L) were also detected in ground water in the vicinity of an abandoned arsenical wood preservative facility near Vancouver, British Columbia [604].

Concentrations of arsenic have been reported to range up to 90 ug/L in ground water in areas with a high content of arsenic in the bedrock, such as in regions of Ontario, Quebec, New Brunswick, and Nova Scotia [604].

Five percent of samples of well water from contaminated supplies in Nova Scotia contained more than 500 ug As/L [604].

W.Typical (Water Concentrations Considered Typical):

EPA 1981: 0.0004 mg/l [83].

1971: Concentrations below 10 ug/L accounted for 79% of the samples taken in stream waters [190].

USGS 1974-1981: 50th percentile of 293 (not especially

clean) NASQWAN and NWQSS river sites in the U.S. was 1 ug/l; 25th percentile was <1 ug/l, and 75th percentile was 3 ug/l, with concentrations trending upwards more often than downward, possibly due to atmospheric deposition [219].

In parts of the U.S., less than 5 ug/L (ppb) arsenic in water would be considered "typical" (Willard R. Chappell, University of Colorado at Denver, personal communication, 1995).

Typical Ocean Concentrations of Arsenic: EPA 1981: 0.003 mg/l [83].

Average total arsenic in oceans is about 1.7 ug/L, much higher than the EPA 10-6 cancer risk water criterion of 0.0175 ug/L [1024].

Arsenic has been found in many natural waters including seawater, hot springs, ground water, rivers, and lakes (Lemmo et al., 1983). The concentrations of arsenic generally average less than 10 ppb [445,716]. The concentration of arsenic in freshwaters shows considerable variation with the geologic composition of the drainage area and the extent of anthropogenic input [445]. Arsenic concentrations in waters of the San Luis Drain and Kesterson Reservoir were <1-2 ug/l (ppb) and <1-2 ug/l, respectively (USBR, Oct 1986) [445].

Arsenic levels in Canadian surface waters away from point sources of contaminants are typically less than 2 ug/L [604].

Surveys of arsenic concentrations in rivers and lakes indicate that most values are below 10 ppb, although individual samples may range up to 1,000 ppb [716]. The median arsenic concentration for surface water samples recorded in the STORET database was 3 ppb [716]. Arsenic has also been detected in rainwater at average concentrations of 0.2-0.5 ppb and in seawater at an average level of 2 ppb [716].

Arsenic levels in groundwater average about 1-2 ppb, except in some western states with volcanic rock and sulfide mineral deposits high in arsenic, where arsenic levels up to 3,400 ppb have been observed [716]. In western mining areas, groundwater arsenic concentrations up to 48,000 ppb have been reported [716].

Surveys of drinking water in the United States have found that more than 99% of public water supplies have arsenic concentrations below the EPA Maximum Contaminant Level (MCL) of 50 ppb [716]. In an EPA study of tap water from 3,834 U.S. residences, the average value was 2.4 ppb

[716]. However, drinking water in polluted areas may have much higher levels; mean arsenic concentration in tapwater from homes near a smelter was 90 ppb [716].

Available data indicates that most Canadian ground waters contain less than 50 ug As/L [604].

In three River Nile coastal lakes in Egypt (areas away from pollution sources), As levels lie within the range 1.2 to 18.2 mu g L super(-1) (dissolved) and between 1.2 and 8.7 mu gg super(-1) (particulate). As(V) is the predominant dissolved As species constituting between 85 and 95% of total dissolved As (TDAs).(Abdel-moati, A.R. 1990. Speciation and behavior of arsenic in the Nile Delta lakes. Water Air Soil Pollut. 51:107-132).

Most arsenic in natural water is a mixture of arsenate and arsenite, with arsenate usually predominating [716]. Methylated forms have also been detected in both surface and groundwater, at levels ranging from 0.01 to 7.4 ppb with most values below 0.3 ppb [716].

W.Concern Levels, Water Quality Criteria, LC50 Values, Water Quality Standards, Screening Levels, Dose/Response Data, and Other Water Benchmarks:

W.General (General Water Quality Standards, Criteria, and Benchmarks Related to Protection of Aquatic Biota in General; Includes Water Concentrations Versus Mixed or General Aquatic Biota):

Notes on total vs. acid soluble vs. dissolved metals:

Although most of the lab tests done to develop water quality criteria and other benchmarks were originally based on "total" values rather than "dissolved" values, some regulatory authorities nevertheless recommend comparing criteria with dissolved or acid soluble metals concentrations. EPA gave 11 reasons why water quality criteria should be compared to acid soluble values [129]. For detailed discussion, see the Laboratory and/or Field Analyses section (far below).

NOTE: This section includes information on several forms of arsenic: Arsenic (+0), Arsenic (III), and Arsenic (V).

Water Quality Criteria for CAS 7440-38-2: Arsenic (+0, also known as inorganic arsenic) in ug/L [446,689,893]:

Freshwater Acute Criteria: None Published.

Freshwater Chronic Criteria: None Published

Marine Acute Criteria: None Published

Marine Chronic Criteria: None Published

Human Health: See Water.Human section (below)

Criteria Federal Register Notice Number: 45 FR 79325

See also: [USEPA; Ambient Water Quality Criteria Doc: Arsenic p.A-1 (1980) EPA 440/5-80-021].

Note: Before citing a concentration as EPA's water quality criteria, it is prudent to make sure you have the latest one. Work on the replacement for the Gold Book [302] was underway in March of 1995.

Water Quality Criteria for Arsenic (III) in ug/L:

EPA 1996: Acute Freshwater Criteria: 3.6E+2 ug/L [893]. Older Reference for Freshwater Acute Criteria: 360 ug/L [446,928].

EPA 1996: Chronic Freshwater Criteria: 1.9E+2 ug/L [893]. Older Reference: Freshwater Chronic Criteria: 190 ug/L [446,928].

Marine Acute Criteria 1996: 6.9E+1 ug/L [893]. Older reference 69 ug/L [446].

Marine Chronic Criteria 1996: 3.6E+1 ug/L [893]. Older reference: 36 ug/L [446].

Criteria Federal Register Notice Number: 50 FR 30786

NOTE: Before citing a concentration as EPA's water quality criteria, it is prudent to make sure you have the latest one. Work on the replacement for the Gold Book [302] was underway in March of 1996, and IRIS is updated monthly [893].

Water Quality Criteria for Arsenic (V) in ug/L [446]:

Freshwater Acute Criteria: Insufficient data

to develop criteria. Lowest Observed Effect Level: 850 [446]. Lowest Observed Effect Level for arensic (pent) oxide: 850 [928].

Freshwater Chronic Criteria: None Published

Marine Acute Criteria: Insufficient data to develop criteria. Lowest Observed Effect Level: 2319 ug/L [446]. Lowest Observed Effect Level for arsenic (pent) oxide): 2319 ug/L [928].

Marine Chronic Criteria: None Published

Human Health (10-6 Risk Level for Carcinogens)

IRIS Recalculated (9/90) Criteria for Water and Organisms: None Published

IRIS Recalculated (9/90) Criteria for Organisms Only: None Published

Drinking Water MCL: None Published

Criteria Federal Register Notice Number: 50 FR 30789

NOTE: Before citing a concentration as EPA's water quality criteria, it is prudent to make sure you have the latest one. Work on the replacement for the Gold Book [302] was underway in March of 1992.

Oak Ridge National Lab, 1994: Ecological Risk Assessment Freshwater Screening Benchmarks for concentrations of contaminants in water [649]. To be considered unlikely to represent an ecological risk, field concentrations should be below all of the following benchmarks (in mg/L) [649]:

Benchmarks for CAS 22541-54-4 (ARSENIC III: applies to Arsenic III only!):

NATIONAL AMBIENT WATER QUALITY CRITERION - ACUTE: 360

NATIONAL AMBIENT WATER QUALITY CRITERION - CHRONIC: 190

SECONDARY ACUTE VALUE: no information found.

SECONDARY CHRONIC VALUE: no information

found.

LOWEST CHRONIC VALUE - FISH: 2962

LOWEST CHRONIC VALUE - DAPHNIDS: 914.1

LOWEST CHRONIC VALUE - NON-DAPHNID INVERTEBRATES: no information found.

LOWEST CHRONIC VALUE - AQUATIC PLANTS: 2320

LOWEST TEST EC20 - FISH: 2130

LOWEST TEST EC20 - DAPHNIDS: 633

SENSITIVE SPECIES TEST EC20: 55

POPULATION EC2O: 1995

Benchmarks for CAS 17428-41-0 (ARSENIC V: applies to Arsenic V only!):

NATIONAL AMBIENT WATER QUALITY CRITERION - ACUTE: no information found.

NATIONAL AMBIENT WATER QUALITY CRITERION - CHRONIC: no information found

SECONDARY ACUTE VALUE: 170

SECONDARY CHRONIC VALUE: 8.11

LOWEST CHRONIC VALUE - FISH: 891.6

ESTIMATED LOWEST CHRONIC VALUE - DAPHNIDS: 450

LOWEST CHRONIC VALUE - NON-DAPHNID INVERTEBRATES: no information found

LOWEST CHRONIC VALUE - AQUATIC PLANTS: 48

LOWEST TEST EC20 - FISH: 1500

LOWEST TEST EC20 - DAPHNIDS: >932

SENSITIVE SPECIES TEST EC20: no information found

POPULATION EC2O: 185

Other Concern Levels/Benchmarks for Water

Concentrations of Arsenic:

The USEPA has established both acute and chronic water quality criteria for the protection of freshwater aquatic life for arsenic+3 (360 ug/l [ppb] and 190 ug/l [ppb], respectively); however, it does not believe that adequate toxicity data exist with which to establish such criteria for arsenic +5 or any organic arsenic compounds (50 FR 30786, Jul 29, 1985). The State of California has established no water quality objectives for arsenic, for the protection of fish and wildlife in the San Joaquin Valley [445].

To protect livestock/cattle use, arsenic levels should be less than 0.05 mg/L (ppm) and general irrigation water should not exceed 1 ppm in coarse soils or 10 ppm in firm soils [671].

For the protection of freshwater aquatic life the average concentration of dissolved trivalent inorganic arsenic (operationally defined as the trivalent inorganic arsenic that passes through a 0.45 micron membrane filter) should not exceed 0.072 mg/l in any 30 consecutive days, nor should the concentration exceed 0.14 mg/l for more than 96 consecutive hours [375]. Pentavalent inorganic arsenic is actively toxic to freshwater aquatic animals at concentrations as low as 0.850 mg/l [375,446,928].

W.Plants (Water Concentrations vs. Plants):

Irrigation Water Recommendations for Arsenic: A maximum arsenic concentration of 0.100 mg/l is recommended as a limit for irrigation water [375].

It is suggested that a maximum concentration of 0.100 mg/l be observed for the protection of aquatic vegetation [302].

Pentavalent arsenic at concentrations as low as 0.048 mg/l may be toxic to freshwater aquatic plants [375].

Shallow Groundwater Ecological Risk Assessment Screening Benchmark for Terrestrial Plants Listed by Oak Ridge National Lab, 1994 [651]:

To be considered unlikely to represent an

ecological risk, field concentrations in shallow groundwater or porewater should be below the following benchmark for any aqueous solution in contact with terrestrial plants. Toxicity of groundwater to plants may be affected by many variables (pH, Eh, cation exchange capacity, moisture content, organic content of soil, clay content of soil, differing sensitivities of various plants, and various other factors). Thus, the following solution benchmark is a rough screening benchmark only, and site specific tests would be necessary to develop a more rigorous benchmark for various combinations of specific soils and plant species [651]:

For CAS 7440-38-2 (Arsenic), the benchmark is 0.001 mg/L (groundwater or porewater).

W.Invertebrates (Water Concentrations vs. Invertebrates):

EC50 (96 hr) Daphnia magna 4.3 mg/l (with food), 1.5 mg/l (without food). LC50 (48 hr) Aplexa hypnorum 24.5 mg/l [692]. LC50 Daphnia magna 5.26 mg/l; Daphnia pulex LC50 1.34 mg/l [970].

Information on arsenic concentrations vs. invertebrates from 1990 report entitled: "Fish and Wildlife Resources and Agricultural Drainage in the San Joaquin Valley, California," quoted word for word with the Permission of Senior Author Stephen Moore, Fish and Wildlife Service, Portland Oregon, Regional Office (see original publication for embedded references) [445]:

Biesinger and Christensen (1972) performed acute and chronic studies of sodium arsenate with daphnids (Daphnia magna). Reproductive impairment and growth were evaluated in the chronic (3-week) toxicity test. Water pH was 7.74 and total hardness was 45.3 ppm. Over the 3-week period, daphnids exposed to 0.52 or 1.4 mg/l (ppm) arsenate exhibited a 16 or 50% reduction in reproduction, respectively. An arsenic concentration of 0.996 mg/l caused daphnid weight to decrease by 18% and total protein content to decrease by 15%; glutamic oxalacetic transaminase (GOT) activity was also reduced by 18%. The 3-week LC50 was 2.85 mg/l and the 48-hour LC50 was 7.4 mg/l [445].

Arsenic toxicity changes in Daphnia magna in the presence of sediment were investigated by Burton et al. (1987). Consecutive 48-hour

exposure periods were conducted in beakers containing only reconstituted hard water (pH = 7.9, temperature 20 degrees C) or water and lake sediment, which had been allowed to settle. Arsenic acid was dissolved in water before daphnid exposure. Survival of daphnids during exposure to several arsenite concentrations in sediment/water beakers generally increased over time. For example, a nominal arsenite concentration in water of 67 mg/l (ppm) caused 100% mortality in repetitive 48-hour exposures through day 12. For the periods ending at days 20 or 28, the percentage of daphnids surviving the 48-hour exposure periods increased to 37% and 100%, respectively. A similar trend was observed for arsenite concentrations of 3.4, 6.8, 13.5, 27.0, 33.5, and 100.0 mg/l. The highest concentration of 133 mg arsenite/l caused 100% mortality throughout the test period, whereas the lowest concentration of 1.3 mg arsenite/l caused no mortality [445].

Percent survival in beakers containing only water and arsenite did not change over time, except at the low arsenite concentration of 3.4 mg/l. At this arsenite water concentration, 48-hour percent survival increased from 0 to 17% at day 6 and by day 10 was 39%. The lowest concentration of 1.3 mg arsenite/l caused no mortality throughout the test period, whereas the concentrations ranging from 6.8 to 133.0 mg arsenite/l caused 100% mortality. Thus, daphnid survival was significantly reduced during exposure to arsenite in water only compared to survival during arsenite exposure in water and sediment. The authors proposed that arsenite in the water column became adsorbed to sediment through time, thereby becoming less available to the organisms [445].

Passino and Novak (1984) investigated arsenate toxicity in daphnids (Daphnia pulex) and in the cladoceran Bosmina longirostris, a widespread and ecologically important crustacean for larval fish of the Great Lakes. For the static bioassays, sodium arsenate was added to softened well water (pH = 6.8, 120 mg/L hardness [ppm as calcium carbonate], temperature 17 degrees C) prior to adding the test organisms. The 96-hour EC50 (the estimated concentration of toxicant that caused 50% of test organisms to stop moving) for the cladoceran was 0.85 +/-0.12 mg

arsenate/l (+/-SE) and the 48-hour EC50 for D. pulex was 49.6 +/- 9.0 mg arsenate/l [445].

Schaefer and Pipes (1973) studied temperature and toxicity of sodium arsenate to the rotifer (Philodina roseala). Rotifers were exposed to arsenate ion concentrations of 4, 8, 10, 32, or 64 mg arsenic/l (ppm). The temperature range of the toxicity bioassays was 5-35 degrees C. The results clearly show that there was greater toxicity at higher temperatures for 24-,48-, 72-, and 96-hour exposure times. The 24-hr TLm (median tolerance limit) was 150 mg/l at 5 degrees C, 84 mg/l at 20 degrees C, and 56 mg/l at 30 degrees C. The 72-hour TLm was 29 mg/l at 5 degrees C, 21 mg/l at 20 degrees C, and 11 mg/l at 30 degrees C. Rotifer susceptibility to the temperature effect decreased with increasing exposure time (that is, the differences between the TLm values at different temperatures decreased with increasing exposure time). Thus, the difference between the TLm values for arsenate at 5 degrees or 30 degrees C at 24 hours was 94 mg/l, but at 72 hours it was 18 mg/l. Increasing temperature was correlated to an exponential decrease in the median life span of rotifers but temperature did not appear to influence the life span TLm concentrations: the life span (10.2 days) TLm at 20 degrees C was 9.0 mg/l and the life span (3.0 days) TLm at 35 degrees C was 11 mg/l [445].

In their bioaccumulation study, Spehar et al. (1980) observed toxicity of the four arsenic compounds, arsenic+3, arsenic+5, SDMA, and DSMA, in stoneflies, snails, amphipods, daphnids, and trout. (Refer to the [445] "Bioaccumulation" information in the Bio.Detail section below for a description of experimental design and results of arsenic bioaccumulation.) Toxicity was characterized by survival. The survival of stoneflies, snails, and trout was not significantly affected by any of the four arsenic compounds (at 100 or 1,000 ug/L [ppm]) after 28 days of exposure. Amphipod survival, however, was significantly decreased to 20% after 7 days exposure to arsenic+3 (1,000 ug/L); after 14 days, none of the animals had survived. Due to large variability in amphipod response to the other arsenic compounds at high and low concentrations, significant statistical differences were not determined. None of the

four arsenic compounds (at 100 or 1,000 ug/L) significantly reduced survival or reproduction of Daphnia magna after 14 days of exposure [445].

In the study by Gilderhus (1966) in which outdoor pools were used to assess the effects of sodium arsenite on aquatic organisms (refer to the [445] "Bioaccumulation" information in the Bio.Detail section below for a description of experimental design), the total numbers of bottom macro- and microinvertebrates were reduced in numbers when the treatment over the year totalled 4.0 ppm sodium arsenite. Species diversity was also reduced in the higher concentrations. For example, mayfly nymphs were absent from the four highest treatment pools but present in all others. The pools which received 1.2 ppm of sodium arsenite weekly or monthly had reduced populations of rotifers; however, rotifers in other treated pools did not appear to be affected and exceeded numbers in control pools. Cladocera were abundant only in the control and the pool which received a single treatment of 0.4 ppm [445].

Naqvi and Flagge (1990) assessed the chronic effects of sublethal exposure to MSMA herbicide in the American red crayfish. Fecundity, hatchability, and juvenile growth-rate were observed. Adult male and female crayfish were exposed to 100 ppm waterborne MSMA for a period of 12 weeks. The males were removed after mating, but exposure to the females continued for an additional 12 weeks at the same concentration. The exposure concentration (100 ppm) was based on the 96-hour LC50 (1,019 ppm) reported for MSMA by Naqvi et al. (1987). Aged tap-water, which was used throughout the study, was characterized by the following parameters: temperature 17.7-19.6 degrees C, total water hardness 25.6-29.3 ppm, and pH 7.8-8.3. Hatchlings produced by the treated females (100 ppm) were exposed to 15 ppm MSMA for 36 weeks during which time their growth-rate was monitored. This sublethal concentration (15 ppm) was selected on the basis of the 96-hour LC50 value for juveniles (101 ppm) determined by Naqvi et al. (1987). The total number of eggs produced by MSMA-exposed crayfish (1,149 eggs) was not significantly different from the total number produced by the controls (1,419 eggs). However, hatching success of treated

crayfish (16.97%) was significantly reduced compared to that of controls (78.08%). The weight gain and final length of MSMA-exposed hatchlings over the 36-week exposure period were not different from those of the controls. In addition, there was very little difference in molting frequencies of MSMA-exposed and control hatchlings, the frequencies of which were 53 and 56, respectively [445].

W.Fish (Water Concentrations vs. Fish):

LC50s for various fish 1.57 mg/L (fathead minnow) to 41 mg/L (bluegill) [970].

EC50 (96 hr) fathead minnow 141-144 mg/l. LC50 (96 hr) knifefish 31 mg/l. Oral (24 wk) rainbow trout 0.52 mg/kg/day caused chronic inflammatory changes in subepithelial tissues of the gall bladder wall in 71% of group. LC50 (96 hr) striped bass 30 mg/l [692].

Additional information on arsenic vs. fish from 1990 report entitled: "Fish and Wildlife Resources and Agricultural Drainage in the San Joaquin Valley, California," quoted word for word with the Permission of Senior Author Stephen Moore, Fish and Wildlife Service, Portland Oregon, Regional Office (see original publication for embedded references) [445]:

A review by Shukla and Pandey (1985) indicated that fish exposed to arsenic have difficulty breathing due to the clogging of gills by coagulated mucous film and to the direct damage of arsenic ions on blood vessels, resulting in vascular collapse in the gills and anoxia. Arsenic has been reported to cause sloughing of external epidermal layers, including the gill, leading to the coughing reflex which has been observed during exposures (Sorensen et al., 1979) [445].

A behavioral study was carried out by Weir and Hine (1970) on conditioned goldfish exposed to low concentrations of waterborne arsenic (as sodium arsenate). Comet goldfish (Carassius auratus) were trained for conditioned avoidance response to light and electric shock stimuli. Water hardness was 50 ppm (as calcium carbonate), pH was 6.0-6.9, and temperature was 19-25 degrees C. Exposure tanks contained arsenic in concentrations of decreasing percentages of the LC50, which was determined to be 32.0 ppm (based on 7-day

survival time after 48-hour exposure). The LC1 was determined to be 1.5 ppm. After the training period, the fish were transferred to the exposure tank for 24 hours, tested for their response, then returned to the exposure tank for a second 24 hours, retested for response and returned to holding tanks. The fish were exposed to arsenic for a total of 48 hours. The lowest concentration of arsenic which gave a significant impairment of the conditioned avoidance response was 0.1 ppm, equivalent to 1/320 of the LC50 or 1/15 of the LC1 [445].

In Japanese medaka (Oryzias latipes), Biddinger (1983) observed that sodium arsenite caused delayed hatching by 1.0-1.36 days at concentrations of 5.0, 7.5, and 10.0 mg arsenic/l (ppm). Although the time to hatch was delayed, length at hatch and survival at these arsenic concentrations did not differ significantly from controls. However, the total length of larvae reared for the first two weeks post-hatch at 5.0, 7.5, and 10.0 mg arsenic/l was significantly less than that of controls by ratios of 0.80, 0.77, and reduced 20%, 23%, and 30%), respectively [445].

Acute toxicity of arsenic was investigated by Passino and Kramer (1980). Mature ciscoes (Coregonus sp.) were captured from Lake Superior in 1976 in order to hatch eggs and to measure arsenic content. Whole body arsenic concentrations ranged from 0.75 to 0.84 ug/g (ppm, wet weight). The difference in arsenic concentrations between male and female ciscoes was not significant and arsenic was not preferentially stored in the eggs (26 ug/g wet weight). Fry were exposed to arsenic (as arsenic trioxide) in reconstituted soft water (total hardness 40-48 mg/l [ppm, as calcium carbonate], temperature 7 degrees C). The 96-hour LC50 for fry 15-19 days old was 26 mg/l. For fry 22-26 days old, the 96-hour LC50 value was 17 mg/l. When the toxicity test was extended to 5 or 14 days after renewal of solutions at 96 hours, the LC50 values for 22-day-old fry were 14 and 6.7 mg/l, respectively [445].

In the same study, Passio and Kramer (1980) compared 96-hour and 5-day LC50 values of 22-day-old fry for arsenic and PCBs, singly and in combination. As stated above, the 96-our LC50 for arsenic alone was 17 mg/l; however,

the 96-hour LC50 for arsenic in the presence of PCBs was 11 mg/l. (The 96-hour LC50 values for PCBs alone and in combination with arsenic were >10 and 3.5 mg/l, respectively.) Similarly, the 5-day LC50 for arsenic alone was 14 mg/l (as stated above), whereas the 5-day LC50 for arsenic in the presence of PCBs was 6.3 mg/l. (The 5-day LC50 values for PCBs alone and in with arsenic were 3.2 and 2.0 mg/l, respectively.)[445].

The effect of arsenic on protein and amino acid content and membrane permeability in the freshwater fish (Clarias batrachus L.) was examined. The fish were exposed to sodium arsenate (1.0 mg arsenic/l [ppm]) in soft water for 14 days (water pH = 8.5, temperature = 22 degrees C). Protein content decreased in the muscle whereas it increased in liver, kidney, stomach, intestine, testis, and ovary. Amino acid content increased in all organs analyzed. Tissue dry weight decreased and tissue membrane permeability increased. For the above biochemical parameters, the effect was most pronounced in the liver and kidneys, followed by intestine, stomach, muscle, testis, and ovary [445].

Sorensen (1976a) investigated effects of short-term exposures to high concentrations of waterborne sodium arsenate in green sunfish. Acute effects were assessed on the basis of mortality: lethal time for 50% mortality (LT50) and lethal dose for 50% mortality (LD50). Water temperature was 20 degrees C. Cumulative percent mortality increased with increasing exposure concentrations. LT50 values for 100, 500, or 1,000 ppm were 46, 17, and 12 hours, respectively. No excessive variation in cumulative percent mortality was apparent for three class sizes of fish in the 500 or 1,000 ppm exposure concentrations, but variation was observed in the 100 ppm exposure....(sic).. for small, intermediate, and large fish exposed to 100 ppm arsenic were 39, 55, and 73 hours, respectively. The LD50 values were calom arsenic exposure versus cumulative percent mortality. The LD50 values for 12, 18, 24, or 48 hours were 1,000, 350, 175, and 150 ppm, respectively. Arsenic accumulation increased with increasing arsenic concentration; for exposure concentrations of 100, 500, or 1,000 ppm, mean whole body arsenic concentrations (based on dry weight) were 33.4, 541.2, and 581.6 ppm, respectively.

No correlation was observed between arsenic accumulation and fish total length, wet weight, dry weight, or condition [445].

Sorensen (1976b) studied thermal effects of arsenic vs. survival. Arsenic tissue concentration was measured as a function of temperature, exposure time, and waterborne arsenic concentration. Green sunfish were exposed to or 60 ppm arsenic as sodium arsenate in water temperatures of 10 degrees, 20 degrees, or 30 degrees C for an initial uptake period of 5 weeks. m hardness concentration was 92 ppm and pH was 8.37-8.46. Liver, gut, and muscle showed increasing arsenic concentrations with the three measured parameters (i.e., temperature, exposure time, and waterborne arsenic concentration); however, no patterns between fish weight and arsenic uptake in liver and gut were observed. The temperature quotient values (plots of temperature versus log of uptake rate) for arsenic uptake in liver ranged from 1.41 to 11.42 (mean 4.47). These values are elevated compared to typical temperature quotient values for the genus Lepomis which range from 1.6 to 3.0, suggesting that elevated heat and high arsenic concentrations act synergistically in arsenic uptake (Sorensen, 1976b). For the 10 degrees C treatments only, a 3-week retention period followed the 5-week uptake period in which fish were moved from 30 or 60 ppm of arsenic to no arsenic. The biological half-life of arsenic in specimens exposed to 10 degrees C Survivorship, as measured by the lethal time for 50% mortality (LT50), was affected by temperature and/or arsenic concentration. For the 60 ppm treatment, increasing the temperature from 10 degrees to 20 degrees to 30 degrees C decreased respective LT50 values from 678 to 210 to 124 hours. Similarly, for the 30 ppm treatment, increasing the temperature from 20 degrees to 30 degrees C decreased respective values from 527 to 209 hours [445].

In another study, green sunfish were exposed to arsenic (as sodium arsenate) under controlled, experimental conditions in order to correlate arsenic accumulation, tissue distribution, and cytotoxicity (Sorensen et al., 1979). Exposure times were 2, 4, or 6 days to 60 ppm waterborne arsenic at 20 degrees C. No histological changes were observed in proximal convoluted tubules of the

kidneys after mean arsenic accumulation of 8.1, 7.2, or 14.2 ppm (fresh weight) for 2, 4, or 6 days, respectively. However, hepatic intranuclear 48.3, 51.5, and 55.0% of all nuclei examined for the 2, 4, and 6 day exposure times, respectively. Corresponding liver arsenic residues were 23.8, 42.3, and 47.4 ppm (fresh weight) for the same exposure periods [445]. Note: For the results of arsenic accumulation in other tissues for which a histopathological analysis was not done, refer to the Moore [445] "Bioaccumulation" information in the Bio.Detail section below.)

Sorenson et. al (1985) compared cellular changes in hepatocytes of green exposed to arsenic-contaminated lake water with the concentration of arsenic in the liver. Fish were collected from Municipal Lake, Texas, where the arsenic concentration in the water was 13.6 ppm and from a farm pond which had no detectable arsenic (control). Arsenic was not detected in the livers of control fish; however, arsenic was detected in livers from fish from Municipal Lake at concentrations ranging from 6.1 to 64.2 ppm (dry weight). The severity of several cytotoxic reactions to arsenic exposure was directly correlated with the concentration of arsenic accumulated in the liver. Both volume and numbers of parenchymal hepatocyte nuclei increased slightly with increasing arsenic concentrations in the liver. The volume occupied by necrotic bodies and fibrous bodies increased significantly with increasing liver arsenic concentrations. The volume areas, and autophagic vacuoles also showed increases with increasing liver arsenic concentrations. Additionally, the surface density of rough endoplasmic reticulum increased with increasing liver arsenic concentrations [445].

In the study by Gilderhus (1966), fewer bluegills survived in outdoor pools treated with varying concentrations of sodium arsenite compared to controls. In the control pool, 90% of the immature fish survived whereas only 18% survived in the pool which received 1.2 ppm sodium arsenite weekly for 16 weeks. The adult fish were more tolerant to arsenic than the immature fish. In the same treatment pool, 31% of adult fish survived compared to 60% survival in the control pool. In all pools having the same treatment, the growth

rate of immature fish was slower at each succeedingly higher concentration. Adults in the four highest concentrations lost 25-37% of their weight. Histopathology of adult fish from pools which received weekly treatments revealed a greater number of hemorrhagic globes on the gills compared to the controls. Higher concentrations of sodium arsenite also prevented development of the ova. The author remarked that highest concentrations exhibited poor growth and survival of fish and reduced bottom fauna numbers, no correlation between the decreased rates of survival/growth and the shortage of food was obvious for each treatment. Competition for food was probably an influencing factor, but it was likely that the physiology of the fish was also affected by arsenic and its accumulation in the fish body [445].

Pandley and Shukla (1982) assessed the effects of arsenite on survival and of fingerlings of a freshwater perch (Colisa fasciatus). The fingerlings were introduced to water (pH 7.1) into which arsenite (as arsenic trioxide) had been mixed. LC50 values in 24, 48, 72, and 96 hours were determined to be 16.06, 14.02, 10.08, and 8.04 mg/l (ppm), respectively. After 6 hours of exposure to arsenic at 5.0 mg/l (as arsenic trioxide), the fingerlings exhibited fast swimming, gulping of air at the water surface, and mucous secretion. Growth (estimated by weight) began to fall significantly after 8 days of exposure and had decreased 12% after 32 12 days of exposure, fingerling length also showed a significant decline, dec after 32 days [445].

Testicular degeneration was observed in freshwater perch exposed to arsenite. Shukla and Pandey (1984a; 1984b) exposed male perch to 2.0 or 14.0 mg/l (ppm) of arsenic+3 oxide for 15 or 30 days. No marked alterations in the architecture of the testis were observed in fish exposed to 2.0 mg/l for 15 or 30 days or in fish exposed to 14.0 mg/l for 15 days. However, 30 at 14.0 mg/l resulted in noticeable structural and cellular changes of the testicular lobules. The lobules were distorted in shape and edematous. The interstitial Leydig cells underwent significant reduction and showed varying degrees of necrosis, pyknosis (shrinkage of nucleus into a dark staining body), and reduced secretory function. The above changes

were observed in the preparatory and mature phases of the testicular cycle but did not occur during the spawning and post-spawning phases. The DNA and RNA content of the testis showed a significant decline in all testicular phases [445].

Ovarian degeneration was observed in freshwater perch exposed to arsenite. Shukla and Pandey (1984c) exposed female perch to 2.0 or 14.0 mg/l (ppm) of arsenic+3 oxide for 15 or 30 days. Histological observations were made during the mature phase of the ovarian cycle. No marked changes of the ovary were produced by exposure to 2.0 mg/l for 15 or 30 days or to 14.0 mg/l for 15 days. However, 14.0 mg/l exposure for 30 days resulted in marked degenerative changes of the ovary, including prominent follicular spaces, reduction in the development of second and third stage of oocyte, reduced number and diameter of nucleoli, and increased atretic follicles [445].

Spotila and Paladino (1979) determined lethal concentrations of arsenic for newly hatched muskellunge fry (Esox masquinongy) and for fry at 5 an weeks of age. In the first experiment, arsenic (as sodium arsenite) was added to tank water to yield concentrations of 0.05, 1.0, or 5.0 mg arsenic/l (ppm). Fry were exposed to arsenic represented two different developmental periods, the first, a sessile non-active stage of yolk absorption and the second, a swimup stage of rapid developmental change and increasing activity. In the second and third experiments, 5- and 12-week-old fry, respectively, were exposed to varying concentrations of arsenic (as sodium arsenite). Water temperature was 15 degrees C in the first experiment and 17 degrees C in the second and third experiments. Water pH ranged from 7.2 to 7.9. Prior to swim up, the fry did not appear to be affected by arsenic and no LC50 would be determined; however, a rapid rise in mortality was observed in fry age. All fry died by day 15 post-hatch, 7 days after swim up began. The 96-hour LC50 determined for swimup fry was 1.1 mg/l. The 96-hour LC50 values for 5- and 12-week-old fry were 2.6 mg/l and 16.0 mg/l, respectively. Thus, as development progressed from the swimup stage to 12 weeks of age, the fry became more tolerant to arsenic; however, arsenic concentrations toxic to fry during

swim up were not toxic to these fry during their pre swimup stage [445].

Cardwell et al. (1976) investigated the acute toxicity of sodium arsenite on six species of fish: bluegill, channel catfish (Ictalurus punctatus), fathead minnow (Pimephales promelas), brook trout (Salvelinus fontinalis), flagfish (Jordanella floridae), and Ozark-strain goldfish (Carassius auratus). The fish were juveniles, except for the brook trout which were adults. Water temperature was maintained at 15 degrees C for tests using brook trout and at 25 degrees C for ties. Total water hardness was in the range of 140-152 mg/l (ppm, as calcium carbonate) and pH ranged from 7.61 to 7.98. The 96-ho/l for bluegill, 48.5 mg/l for flagfish, 44.9 mg/l for goldfish, 31.2 mg/l for channel catfish, 27.0 mg/l for fathead minnow, and 25.8 mg/l for brook trout. With prolonged exposures (i.e., >96 hours) to the lower concentrations, mortality in fathead minnows, bluegill, and channel catfish increased; for brook trout and goldfish, however, t LC50 became constant with time. The minimum concentration of sodium arsenite was found to be acutely lethal in the tests of adult brook trout, where the LC50 for 262 hours of exposure was 18.0 mg/l [445].

Palawski et al. (1985) measured acute toxicities of arsenic to young striped bass (Morone saxatilis) in water of two hardnesses. Striped bass larvae were exposed to arsenic (as arsenic pentoxide) in 20 degrees C water. The 96-hour LC50 for 63-day-old striped bass was 40.5 mg/l (ppm) in soft water (40 mg/l calcium carbonate, pH 8.1), and 30.5 mg/l in hard water (285 mg/l calcium carbonate, pH 7.9). Changes in water hardness did not significantly alter the acute toxicities of arsenic to larval striped bass [445].

Long-term effects of arsenic accumulation in rainbow trout were investigated by Oladimeji et al. (1984). Trout diets were supplemented with sodium arsenite to yield diets containing 10, 20, or 30 mg arsenic/kg (ppm, dry weight), the concentrations of which were equivalent to a daily dose of 0.2, 0.4, or 0.6 mg arsenic/kg body weight, respectively (based on a feeding rate of 2% body weight per day). Exposure times were 2, 4, 6, or 8 weeks. Weight gains of fish exposed to 10 or 20 mg arsenic/kg diet were not different from those of control fish;

however, the weight gains of fish exposed to 30 mg arsenic/kg diet were significantly less than those for the control fish throughout the duration of exposure. After 8 weeks exposure, blood hemoglobin levels showed a significant drop of 12%, 20%, and 29% for treatment groups, respectively. The mean corpuscular hemoglobin concentrations (MCHC) also dropped significantly by 25, 25, and 22% for the 10, 20, and 30 mg treatment groups, respectively, after 8 weeks. Because of the observed reduction in MCHC, the authors suggested that the primary effect of arsenic on blood was a decrease in red blood cell hemoglobin and not hemolysis of the cells. The hematocrit levels also dropped significantly after 6 weeks in all treatment groups. In general, arsenic accumulation correlated to the concentrations of arsenic to which the fish were exposed (for discussion of tissue arsenic residues, see the [445] "Bioaccumulation" information in the Bio.Detail section below) [445].

In another study, arsenic trioxide affected the weight and fat content of rainbow trout. When exposed to 1.0 mg arsenic/l (ppm), fish lost dry weight but the wet weight remained unchanged. This change in weight may be explained by a disruption in osmoregulation due to resultant kidney damage, causing water to be retained in the body. However, when exposed to 6.0 mg arsenic/l, the wet weight also decreased. Arsenic affected the synthesis of lipids necessary as energy reserves and the development of eggs. Statistical signiion (sic) in rainbow trout were investigated by Oladimeji et al. (1984). Trout diets were supplemented with sodium arsenite to yield diets containing 10, 20, or 30 mg arsenic/kg (ppm, dry weight), the concentrations of which were equivalent to a daily dose of 0.2, 0.4, or 0.6 mg arsenic/kg body weight, respectively (based on a feeding rate of 2% body weight per day). Exposure times were 2, 4, 6, or 8 weeks. Weight gains of fish exposed to 10 or 20 mg arsenic/kg diet were not different from those of control fish; however, the weight gains of fish exposed to 30 mg arsenic/kg diet were significantly less than those for the control fish throughout the duration of exposure. After 8 weeks exposure, blood hemoglobin levels showed a significant drop of 12%, 20%, and 29% for treatment groups, respectively. The mean corpuscular hemoglobin concentrations (MCHC) also dropped

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McGeachy and Dixon (1989) studied the impact of temperature on the acute toxicity of waterborne arsenate and arsenite to rainbow trout. Total water hardness was 362 mg/l (ppm as calcium carbonate)as 8.0. At a water temperature of 5 degrees or 15 degrees C, the trout were exposed to arsenite (as arsenic trioxide) or to arsenate (as sodium arsenate). Temperature had no effect on the acute toxicity of ar rainbow trout: the 144-hour LC50 of 17.7 mg/l (ppm) at 5 degrees C was not significantly different from the 144-hour LC50 of 20.7 mg/l at 15 degrees C. However, temperature did affect arsenic toxicity, body burden, uptake and depuration in trout exposed to arsenate. Trout acclimated to 5 degrees C showed a greater tolerance toward arsenate than those acclimated to 15 degrees C: the 144 hour LC50 of 114.1 mg/l at 5 degrees C was twice the 144-hour LC50 of 58.0 mg/l at 15 degrees C. When fish were exposed to 70 mg/l arsenate for 72 hours, those held at 15

degrees C had mean whole-body arsenic concentrations that were five times higher than those in fish held at 5 degrees C. Uptake of arsenic was enhanced at 15 degrees C compared to uptake at 5 degrees C and fish that were held in arsenate depurated significantly more arsenic at 15 degrees C (49%) than at 5 degrees C (31%). When the exposure concentrations are expressed as proportions) of the temperature-dependent 144-hour LC50, any temperature effects were eliminated. Thus, whole body arsenic concentrations demonstrated essentially the same uptake patterns with time at both 5 degrees and 15 degrees C [445].

Creek water from a mining region of South Dakota was used to determine the toxicity of mining wastes (Hale, 1977). Two-month old rainbow trout were placed in creek water (pH 6.4-8.3) with added gradients of sodium arsenate. The 96-hour TL50 was estimated to be 10.8 ppm for waterborne pentavalent arsenic [445].

Using the rainbow trout, embryo larval toxicity tests were performed with , 1979). Treatment was maintained continuously from fertilization through 4 days post-hatching, giving an exposure period of 28 days. Water hardness ranged from 92 to 119 mg/l (ppm as calcium carbonate), pH varied from 6.9 to 7.8, and temperature was 12-13 degrees C. The LC50 value for arsenic (as sodium arsenite) was 0.55 mg/l (ppm); the LC10 and LC1 values e 134 and 42.1 ug/l (ppb), respectively [445].

The USFWS-NFCRC (Dec 1987) conducted twelve experiments to determine the 24- and 96-hour LC50's for fall-run chinook salmon (Onchorhynchus tshawytsha) exposed to waterborne arsenic+3 and arsenic+5. Two life stages of salmon were tested in water standardized to simulate the cation and anion concentrations of San Luis Drain water (without the trace elements) which was diluted 10-fold with either freshwater (for 0.5 g swimup fish) or brackish water (salinity approximately 1.2 ppth, for 2 g advanced fry). Tests were conducted using arsenic+3 (as arsenic trioxide), arsenic+5 (as arsenic pentoxide) in a non-pH-buffered solution, or arsenic+5 (as arsenic pentoxide) in a pH-buffered solution. Water hardnesses were 211 ppm (as calcium carbonate) in the freshwater-

diluted test solutions and 347 ppm (as calcium carbonate) in the brackish-water-diluted solutions. pH of test solutions were as follows: 7.9 and 7.8 for arsenic+3 (for the freshwater-diluted and brackish-water respectively), 7.0 for buffered arsenic+5, and 3.0-6.9 for unbuffered arsenic+5 [445].

The acute toxicity of waterborne arsenic+3 was greater than arsenic+5 (unbuffered), which was greater than arsenic+5 (buffered). The 24- and ppm, 167 and 90.4 ppm, and >470 and 167 ppm, for arsenic+3, arsenic+5 (unbuffered), and arsenic+5 (buffered), respectively. The same values for the larger salmon were 56.5 and 21.4 ppm, 78.8 (unbuffered), and arsenic+5 (buffered), respectively [445].

W.Wildlife (Water Concentrations vs. Wildlife):

Information from 1990 report entitled: "Fish and Wildlife Resources and Agricultural Drainage in the San Joaquin Valley, California," quoted word for word with the Permission of Senior Author Stephen Moore, Fish and Wildlife Service, Portland Oregon, Regional Office (see original publication for embedded references) [445]:

Khangarot et al. (1985) determined the acute toxicity of arsenite on tadpoles of the frog (Rana hexadactyla). Toxicity tests were conducted under the following water conditions: pH 6.1, temperature 15 degrees C, and total hardness 20 ppm (as calcium carbonate). The LC50 values at 24, 48, 72, or 96-hour exposure to arsenite (as arsenic trioxide) were 0.368, 0.270, 0.270, and 0.249 ppm, respectively. In the higher test concentrations, tadpoles exhibited abnormal behavior such as surfacing and loss of equilibrium before death [445].

Mammals and birds are exposed to arsenic through vegetation and water contaminated naturally or through human activity; though not as likely, arsenic exposure may also occur via inhalation and ion (NRC of Canada, 1978) [445].

Oak Ridge National Lab, 1994: Risk Assessment Screening Benchmarks for Wildlife derived from No-Observed-Adverse-Effect (NOAEL) levels (see Tis.Wildlife, B) section below for these). To be considered unlikely to represent an ecological risk, water concentrations should be below the

following benchmarks for each species present at the site [650]:

For CAS 7440-38-2 (ARSENIC AS ARSENITE), the bencmarks are:

SPECIES	WATER CONCENTRATION (ppm)
Mouse (test species)	0.00000
Short-tailed Shrew	0.72000
Little Brown Bat	1.24400
White-footed Mouse	0.46500
Meadow Vole	0.81400
Cottontail Rabbit	0.38600
Mink	0.40000
Red Fox	0.28600
Whitetail Deer	0.16000

Comment: Actually, the number of significant figures for a benchmark value should never be more than one; even if these values have been taken directly from another report, they should be rounded; otherwise the impression is given of a level of accuracy that is simply unwarranted. The uncertainties are too large to justify such a fine distinction (Owen Hoffman, SENES Oak Ridge, Personal Communication, 1997).

W.Human (Drinking Water and Other Human Concern Levels):

EPA 1996 IRIS database information on Arsenic (CAS 7440-38-2) [893]:

Maximum Contaminant Level Goal [893]:

Value: 0.05 mg/L Status/Year: Proposed 1985 Econ/Tech?: No, does not consider economic or technical feasibility Reference: 50 FR 46936 (11/13/85) [893].

Contact: Health and Ecological Criteria Division / (202)260-7571 Safe Drinking Water Hotline / (800)426-4791 [893].

Discussion: An MCLG of 0.05 mg/L for arsenic is proposed based on the current MCL of 0.05 mg/L. Even though arsenic is potentially carcinogenic in humans by inhalation and ingestion, its potential essential nutrient value was considered in determination of an MCLG. The basis

for this evaluation is nutritional requirements by NAS (NAS, 1983, Vol. 5, Drinking Water and Health, National Academy of Sciences Press, Washington, DC.)[893].

Maximum Contaminant Level (MCL) [893]:

Value: 0.05 mg/L [893,952].

Status/Year: Interim 1980 Econ/Tech?: Yes, does consider economic or technical feasibility Reference: 45 FR 57332 (08/27/80); 50 FR 46936 (11/13/85) [893].

Contact: Drinking Water Standards Division / OGWDW / (202)260-7575 Safe Drinking Water Hotline / (800)426-4791 [893].

Discussion: As an interim measure the U.S. EPA is using the value previously derived by the Public Health Service. [893].

Ambient Water Quality Criteria for Human Health [893]:

Water & Fish: 2.2E-3 ug/liter [893].

Older Citations for Human Health Water Quality Criteria for Carcinogens (risk of one additional case in 1 million, 1E-06): Published Criteria for Water and Organisms: 0.0022 ug/L [446,689,928]. Note: Four states, SD, UT, NH, and OR had adopted either 0.0022 or 0.002 ug/L as official surface water quality standards as of July 1996 (L. Loehr. SETAC News, November, 1996).

Older citation: IRIS Recalculated (7/93) Criteria for Water and Organisms: 0.018 ug/L [689,928]. Note: Three states, CT, VT, and MA had adopted either 0.0018 ug/L as official surface water quality standards as of July 1996 (L. Loehr, SETAC News, November, 1996).

Fish Only: 1.75E-2 ug/liter [893].

Note: Arsenobetaine, the principle arsenic compound in seafood [604,1024] is not carcinogenic to mammals [0124]. The EPA 10-6 cancer risk water criterion of 0.0175 ug/L is much lower than the ambient concentration of inorganic arsenic in clean ocean water and may be overly restrictive based on a lack of understanding that most organic seafood arsenic is not easily converted to the inorganic arsenite form of concern related to cancer risk [1024].

Older Citation for Human Health Water Quality Criteria for Carcinogens (risk of one additional case in 1 million, 1E-06): Published Criteria for Organisms Only: 0.0175 ug/L [446].

References: 45 FR 79318 (11/28/80). See also: [USEPA; Ambient Water Quality Criteria Doc: Arsenic p.A-1 (1980) EPA 440/5-80-021, Criteria Federal Register Notice Number: 45 FR 79325] [893].

Contact: Criteria and Standards Division / OWRS / (202)260-1315 [893].

Discussion: For the maximum protection from the potential carcinogenic properties of this chemical, the ambient water concentration should be zero. However, zero may not be attainable at this time, so the recommended criteria represents a E-6 estimated incremental increase of cancer risk over a lifetime. [893].

Quantitative estimate of carcinogenic risk from oral exposure ————Cancer Slope Factor: 1.5E+0 per mg/(kg/day) [893,952].

Unit Risk: 5E-5 per ug/liter Extrapolation Method: Time- and dose-related formulation of the multistage model (U.S. EPA, 1988) [893].

Drinking Water Concentrations at Specified Risk Levels:

Risk Level Concentration E-4 (1 in 10,000) 2E+0 ug/liter E-5 (1 in 100,000) 2E-1 ug/liter E-6 (1 in 1,000,000) 2E-2

ug/liter [893].

EPA Region 9 Preliminary remediation goals (PRGs) for tap water [868]: 4.5E-02 (0.045) ug/L.

Up to 4 mg/L concentrations are found in aerated-pyrite contaminated well water in India, where thousands of people are showing arsenic poisoning signs such as skin cancer and wart-like (keratosis) skin growths (Willard R. Chappell, University of Colorado at Denver, personal communication, 1995).

Drinking Water Standards for Arsenic: An arsenic concentration not greater than 0.05 mg/l is recommended for the domestic drinking water supply [375]. A concentration not greater than 0.05 mg/l is also recommended by the World Health Organization as the international standard for drinking water [375]. Drinking water concentrations even lower than a concentration commonly used as a detection limit (10 ug/L) for this compound may result in an unacceptable human cancer risk [209].

FDA Requirements: Bottled water shall, when a composite of analytical units of equal volume from a sample is examined by the methods described in paragraph (d)(1)(ii) of this section, meet the standards of chemical quality and shall not contain arsenic in excess of 0.05 mg/l (Arsenic as As ion. 21 CFR 103.35, 4/1/88) [366].

Safe drinking water act (SDWA): MAXIMUM CONTAMINANT LEVEL GOAL (MCLG) for Drinking Water Value (status) -- 0.05 mg/L (Proposed, 1985) [337].

The maximum contaminant level (MCL) of arsenic is 0.05 mg/l (40 CFR 141.11, 7/1/88) [366].

Secondary maximum contaminant level (SMCL) for Drinking Water: No data available [337].

> NOTE: As of September 1995, EPA was considering changing the drinking water MCL from 50 ug/L to 2 ug/L; this proposal was subject to considerable controversy due to the following (Willard R. Chappell, University of Colorado at Denver, personal communication, 1995).:
>
> > 1) There is a fair amount (up to 100 ug/L) of arsenic in well water in parts of California, Utah, New Mexico, Alaska, and Oregon. Many commentators felt 2 ug/L would cost too much to achieve.

2) Two ug/L is below the practical quantitation limit for arsenic in water.

3) Regulating arsenic in soil at the same (1E-04 cancer risk) level would require limiting soil concentration to 37 ppm at CERCLA sites; this would be hard to achieve (the median concentration at the Leadville, CO, site is about 37 ppm).

Bureau of Land Management RMC Benchmarks, 1995: Risk Management Criteria (RMC) developed from the mostly dry BLM lands in the western U.S. These risk management criteria should be used by the land manager as a cautionary signal that potential health hazards are present and that natural resource management or remedial actions are needed [715]. Exceedances of the criteria should be interpreted as follows 715]:

Less than criteria: low risk
1-10 times the criteria: moderate risk
10-100 times the criteria: high risk
>100 times the criteria: extremely high risk

Human RMC criteria for arsenic in surface waters. These categories of humans not exposed to surface waters with concentrations of arsenic exceeding the below RMCs are not expected to experience adverse toxic effects [715]:

Camp host: 93 ug/L
Child Camper: 85 ug/L
Boater: 81 ug/L
Swimmer: 144 ug/L

Human RMC criteria for arsenic in ground water. These categories of humans not exposed to ground waters with concentrations of arsenic exceeding the below RMCs are not expected to experience adverse toxic effects [715]:

Child resident (living on properties adjacent to BLM lands): 0.1 ug/L
Camp host: 1 ug/L
Child Camper: 3 ug/L
Worker: 0.7 ug/L
Surveyor: 7 ug/L

The current recommended maximum contaminant level of drinking water for arsenic is 50 mcg/L (ppb), set by the EPA [363]. Drinking water containing

arsenic above that level results in increases in hair and urine arsenic levels (Valentine et al, 1978) [363].

Wells in Minnesota containing up to 21000 ppb have caused severe arsenic poisoning (Feinglass, 1973) [363].

A well drilled into old mine tailings in upstate New York yielded water with arsenic concentrations of 9000 to 10,900 ug/L; two patients were seriously poisoned (Franzblan & Lilis, 1989) [363].

Water Quality Criteria for Arsenic (III) in ug/L [446] (information for Arsenic III only!):

Human Health (10-6 Risk Level for Carcinogens)

IRIS Recalculated (9/90) Criteria for Water and Organisms: None Published [446].

IRIS Recalculated (9/90) Criteria for Organisms Only: None Published [446].

State Surface Water Quality Standards for Arsenic:

While four states had adopted either 0.0022 or 0.002 ug/L as official surface water quality standards as of July 1996, and 31 states had standards of 50 ug/L or lower, the Arizona standard was 1450 ug/L (L. Loehr, SETAC News, November, 1996)

W.Misc. (Other Non-concentration Water Information):

According to one author, the overly restrictive EPA 10-6 cancer risk water criterion of 0.0175 ug/L is much lower than the ambient concentration of inorganic arsenic in clean ocean water [1024]. The very low fish consumption criteria can lead to unwarranted conclusions of unacceptable risk to humans in risk assessments done in marine and estuarine ecosystems [1024]. Some of the lowest criteria seem to ignore that most organic seafood arsenic is not easily converted to the inorganic arsenite form of concern related to cancer risk [1024].

Ground water normally contains higher concentrations of arsenic than are found in associated surface water [604].

Liming soil, a remediation sometimes used to reduce soil acidity and reduce the mobility of metals such as copper, can result in the (unintended) consequence of increasing

the mobility of arsenic and its transport to groundwater (see more detailed note in Soil.Human section.

In IRIS, The criteria given are for Arsenic III [893]. Much less data are available on the effects of Arsenic V to aquatic organisms, but the toxicity seems to be less [893].

Relatively high levels of As have been reported in agricultural drainage water and in evaporation pond sediments in Kern County, California. As(V), MAA, and DMA added to water samples at concentrations from 0.1 to 1000 mg/l showed no effect on the colony forming units (CFUs) compared with no As supplementation, while arsenite (III) (> 1.0 mg/L) inhibited the population. The As-resistant bacteria showed a relatively high tolerance to metals and antibiotics (Huysmans KD; Frankenberger WT Jr., 1990. Arsenic resistant microorganisms isolated from agricultural drainage water and evaporation pond sediments. Water air soil pollut 53 (1-2): 159-168).

For members of the general population, above-average exposure to arsenic from drinking water is possible in areas of high natural arsenic levels in groundwater or elevated arsenic levels in drinking water due to industrial discharges, pesticide applications, or leaching from hazardous waste facilities [716].

Total vs. Dissolved Arsenic in Water:

> A potential complication in comparing contaminants data is that different investigators have sometimes meant different things when they put the words "dissolved" or "total" in front of a reported measurement. In the case of nutrients, the "dissolved" portion is usually simply that portion which has passed through a 0.45-micrometer membrane filter and the "total" measurements implies that it was not filtered and includes both dissolved and other forms of the nutrient [141]. However, usage of the words dissolved and total has not been uniform in the past and there is still considerable debate about which methods should truly be considered "dissolved" or "total" (Merle Schlockey, USGS, personal communication).
>
> Water bodies are often marked by heterogeneity of the distribution of undissolved materials [691]. The size of any effects depends on the difference in density of the undissolved materials and the water, the size of the particles or bubbles of the materials, and various hydrodynamic factors such as the degree of turbulence in the water. Thus, undissolved inorganic materials in rivers and other natural water-bodies tend to increase in

concentration with increasing depth because the particles tend to settle [691]. On the other hand, certain biological detritus may tend to rise towards the surface of the water because its density is less than that of water; oils also commonly demonstrate this effect markedly [691]. The surface microlayer is usually higher in concentration of many metallic and organic contaminants than the water column further down.

If the only change one makes is to use the prefix "dissolved" rather than the prefix "total" in an otherwise identical water quality standard, the effect can be a weakening of the standard related to total loading of a system. Many contaminants which are not currently dissolved can become dissolved at a later time, when encountering different conditions (perhaps downstream), such as changes in pH, additions of surfactants or humic substances, bioturbation, methylating organisms, and various other physical, chemical, or biological changes.

One problem with relying too heavily on dissolved fractions of metals is that the dissolved fraction misses the metals carried by colloids. Colloids were found to carry toxic metals 140 miles downstream of mining sources in Leadville, Colorado, to be repeatedly washed from flood deposited lowlands back into the river year after year in spring runoff (Briant Kimball, USGS Salt Lake City, as quoted in U.S. Water News, April 5th, 1995).

See Laboratory section below for EPA generic (guesstimate) conversion factors to convert total to dissolved concentrations.

Some environmental toxicologists make the argument that dissolved metals in surface water and porewaters represent most of what is bioavailable and thus "total" metals parameters are not good as a measure of potential biological effects. This is mostly true in many situations, but it should be kept in mind that fish and other aquatic organisms do not typically live in filtered water and that many fish and other aquatic organisms live in the sediments and in other situations in which they come in contact with toxic or otherwise harmful compounds (as certain colloids, precipitates, oxides, adsorbed metals), etc. Sometimes the effect of total metals is partially related to physical or chemical aspects, such as when ferric oxide coats or covers benthic organisms. Another factor to consider: contaminants carried downstream

by erosion of bottom sediments or colloids can be mobilized when they come in contact with different physical/chemical environments downstream (for example, a tributary bringing low pH into the system).

Misc. Notes on colloids (Briant Kimball, USGS, Salt Lake City Office, Personal Communication, 1995):

There is no question that dissolved metals are critical to fish and invertebrates, but less well recognized is the potential impact and movement of metals in colloids. The possibility of having colloidal material present means there is a readily available supply of metals in a state in which the metals can quickly be reduced and mobilized. In river banks, reducing environments form just under the surface quickly.

Note: in reducing environments: toxic metals of concern would include the relatively hazardous arsenite forms [1024].

Colloids do move in surface water (for example, transport of metal in colloids 140 miles downstream of Leadville, CO), but also in groundwater, especially related to radionuclides.

Colloidal metals may effect biota more than is widely recognized. Brown trout are effected by colloids which travel kind of like dissolved fractions, don't settle out. There may be little understood colloidal pathways of metals to fish, for example. Colloidal metals become part of the caddis cast which are ingested, once part of acid gut, metals can be released. On the Arkansas River of Colorado below Leadville, the dissolved metals have gone down with treatment, but Will Clements of CSU has discovered the toxicity has not been reduced to the same extent as have the dissolved metals. Treatment has not eliminated colloidal fractions loaded with cadmium and copper, and this is possibly impacting the fish.

In rivers, there is annual flushing of the colloids, loads are much greater

during runoff.

The following article related to arsenic in water was summarized by Susan Dodson, Arlington Field Office, US Fish and Wildlife Service, Arlington, Texas in January 1992:

Richardson, C.W., J.D. Price and Earl Burnett. 1978. Arsenic Concentrations in Surface Runoff from Small Watersheds in Texas. J. Environ. Qual. 7:198-192. Summary:

A study was conducted to determine the movement of arsenic in surface water runoff after arsenic acid application for desiccation of cotton. Six small watersheds were studied from 1971 -1974. The cropping sequence on the watersheds was a 3-year rotation of grain sorghum, cotton, and oats. Each of the three crops was planted on two of the watersheds each year. Arsenic acid was applied each year in the fall on the two watersheds planted in cotton. Application rate was 4.6 l/ha of a 75% commercial concentrate of arsenic acid, resulting in 6.6 kg/ha arsenic acid (or 3.5 kg/ha elemental arsenic) to each watershed once every three years. Runoff from these watersheds was then monitored. The concentration of dissolved arsenic in the runoff was highest (250-18 ppb) during the first runoff event after application, then decreased and levelled off to 10-20 ppb, remaining fairly constant until the next arsenic application 3 years later. Tillage incorporating plant material into the soil reduced arsenic concentration in the runoff. Soil arsenic levels were not measured in this study. Arsenic of suspended sediment was measured and averaged 20 ppb. Authors concluded that arsenic concentration in sediment was related more to the arsenic content of the soil rather than length of time or tillage between arsenic acid application and the first runoff. About 7% of the arsenic applied would be transported by runoff and erosion. Of the total arsenic removed, 38% would be in solution, 62% would be attached to sediment.

Sediment Data Interpretation, Concentrations and Toxicity (All Sediment Data Subsections Start with "Sed."):

Sed.Low (Sediment Concentrations Considered Low):

Great Lakes Harbors, EPA 1977: Sediments having sediment

concentrations lower than 3.0 mg/kg were classified as "non-polluted [145]."

Sed.High (Sediment Concentrations Considered High):

Texas: The statewide 90th percentile value for this compound was 15.7 mg/kg dry weight [7].

NOAA National Status and Trends Program (1984-1990) [698]: High concentration for arsenic in fine-grained sediment (n=233) = 24 ug/g dry weight at 4.6% TOC dry weight. The above concentration was adjusted for sediment grain-size in the following way: the raw concentrations were divided by the fraction of particles less than or equal to 64 um. "High" NOAA concentrations are equal to the geometric mean plus one standard deviation on the log normal distribution [696].

> Note: Fine-grained sediment would typically contain more arsenic than course-grained sediment, and sediments higher in total organic carbon (TOC) would typically have more arsenic than sediments which are similar except for being lower in TOC, which is why NOAA and many others are now normalizing sediment values for grain size, and reporting TOC.

Great Lakes Harbors, EPA 1977: Sediments having sediment concentrations higher than 8.0 mg/kg dry weight were classified as "heavily polluted" [145].

Three to 8 ppm dry weight is considered moderately polluted and >8 ppm is considered heavily polluted [347,386,761].

Illinois EPA, 1984: Sediments having sediment concentrations higher than 11.0 mg/kg dry weight were classified as "elevated" [145].

Eisler, 1988: Sediments from areas contaminated by arsenical herbicides had arsenic concentrations ranging from 198 to 3500 mg/kg [21].

Concentrations of arsenic are highest in sediments near base- and precious-metal mining and ore-processing operations. Average levels of 100 to 200 mg/kg (maximum 650 mg/kg) were reported near base-metal mines and smelters in several provinces. Near gold mines and an abandoned precious-metal refinery, mean concentrations in sediments ranged from about 700 to 5000 mg As/kg [604].

Arsenic accumulation (up to 65 mg/kg) has been reported in contaminated Halifax harbor sediments. 262 mg As/kg was found in a sediment downstream from an arsenical wood preservation facility near Elmsdale, Nova Scotia [604].

Sed.Typical (Sediment Concentrations Considered Typical):

The International Joint Commission considered 1.1 mg/kg as a background sediment level [145].

Sediments in aquatic systems often have higher arsenic concentrations than those of the water [716]. Most sediment arsenic concentrations reported for U.S. rivers, lakes, and streams range from 0.1 to 4,000 ppm, but much higher levels may occur in areas of contamination [716].

Riverine sediment arsenic concentrations of 300 ppm dry weight are considered high even for mountain mineralized areas; there are a few high values in Yellowstone National Park; 12-15 ppm arsenic dry weight in parts of Soda Butte Creek (NE Yellowstone Park) are typical (not higher or lower than elsewhere) for the general area (Maurice Chaffee, USGS, personal communication, 1995).

Uncontaminated ocean sediments contain from 5 to 40 ug/g dry weight total arsenic [1024].

Guidelines for the pollution classification of Great Lakes harbor sediments (in ppm; dry weight) (1977): non-polluted <3, moderately polluted 3-8, heavily polluted >8 ppm of arsenic [347,761].

Background concentrations in relatively uncontaminated surface sediments are generally less than 20 mg As/kg dry weight; arsenic levels in deeper sediment are normally only a few mg/kg [604].

Analyses of sewage sludges from 50 publicly owned treatment works by the U.S. Environmental Protection Agency (1985): The mean concentration of arsenic was 5.9 ppm (dry weight) [347].

Analyses of 74 Missouri sewage sludges (in ppm; dry weight) (1985): Median 6.1, range 2-39 [347].

Sed.Concern Levels, Sediment Quality Criteria, LC50 Values, Sediment Quality Standards, Screening Levels, Dose/Response Data and Other Sediment Benchmarks:

Sed.General (General Sediment Quality Standards, Criteria, and Benchmarks Related to Protection of Aquatic Biota in General; Includes Sediment Concentrations Versus Mixed or General Aquatic Biota):

Wisconsin interim criteria for sediments from Great Lakes harbors for disposal in water (1985): Arsenic should not exceed 10 ppm (dry weight) [347].

EPA Region 6, 1973: The arsenic concentration

proposed by EPA Region 6 as a guideline for determining acceptability of dredged sediment disposal was 5.0 mg/kg [143].

Ontario, 1978, 1986: The arsenic concentration proposed by the Ontario Ministry of the Environment as a threshold for evaluations of dredging projects was 8.0 mg/kg [145]. Ontario Ministry of the Environment guidelines for open lake disposal of sediments (1986): 8 ppm of Arsenic [347].

International Joint Commission, 1988: The IJC suggested sediment concentrations of arsenic not exceed background levels of 1.1 mg/kg [145].

AET 1988: The apparent effects threshold concentrations for arsenic in sediments proposed for Puget Sound ranged from 57 mg/kg (Benthic Species) to 700 mg/kg dry weight (microtox) [416]. Although the authors of the Puget Sound AETs have cautioned that Puget Sound AETs may not be appropriate for comparison with data from other geographic areas, so few concern levels for this chemical have been published that the proposed Puget Sound concern level is included in this text as a reference item.

NOAA 1995 Concern Levels for Coastal and Estuarine Environments: After studying its own data from the National Status and Trends Program as well as many literature references concerning different approaches to determining sediment criteria, NOAA suggested that the potential for biological effects of this contaminant sorbed to sediments was highest in sediments where its concentration exceeded the 70 ppm dry weight Effects Range-Median (ERM) concentration and was lowest in sediments where its concentration was less than the 8.2 ppm dry weight Effects Range-Low (ERL) concentration [664]. To improve the original 1990 guidelines [233], the 1995 report included percent (ratios) incidence of effects for ranges below, above, and between the ERL and ERM values. These numbers represent the number of data entries within each concentration range in which biological effects were observed divided by the total number of entries within each range [664]:

<ERL	5.0
ERL-ERM	11.1
>ERM	63.0

Oak Ridge National Lab, 1994: Risk Assessment Screening Benchmarks for Sediment Concentrations: To be considered unlikely to represent an

ecological risk, field concentrations should be below all of the following benchmarks in mg/kg (ppm) dry weight [652]:

LOWEST EFFECT LEVEL (ONTARIO MOE): 6
EFFECTS RANGE - MEDIAN (NOAA): 70
EFFECTS RANGE - LOW (NOAA): 8.2

Ontario Ministry of Environment Freshwater Sediment Guidelines, 1993 (mg/kg dry weight) [761]:

Lowest effect level: 6

Severe effect level: 33

St. Lawrence River Interim Freshwater Sediment Criteria, 1992 (mg/kg dry weight) [761]:

No effect level: 3

Minimal effect level: 7

Toxic effect level: 17

Environment Canada interim sediment quality assessment values, 1994 (mg/kg dry weight) [761]:

Threshold effect level: 5.9

Probable effect level: 17

Sed.Plants (Sediment Concentrations vs. Plants):

No information found.

Sed.Invertebrates (Sediment Concentrations vs. Invertebrates):

No information found.

Sed.Fish (Sediment Concentrations vs. Fish):

No information found.

Sed.Wildlife (Sediment Concentrations vs. Wildlife or Domestic Animals):

Bureau of Land Management RMC Benchmarks, 1995: Risk Management Criteria (RMC) were developed for the mostly dry BLM lands in the western U.S. These risk management criteria should be used by the land manager as a cautionary signal that potential health hazards are present and that natural resource management or remedial actions are needed [715]. Exceedances of the criteria should be

interpreted as follows [715]:

Less than criteria: low risk
1-10 times the criteria: moderate risk
10-100 times the criteria: high risk
>100 times the criteria: extremely high risk

Wildlife criteria for arsenic in soils and sediments. Wildlife not exposed to soils/sediments with concentrations of arsenic exceeding the below RMCs are not expected to experience adverse toxic effects [715]:

Deer/Mouse: 14 mg/kg
Rabbit: 70 mg/kg
Bighorn Sheep: 123 mg/kg
Whitetailed Deer: 216 mg/kg
Mule Deer: 110 mg/kg
Elk: 63 mg/kg
Mallard: 205 mg/kg
Canada Goose: 90 mg/kg
Trumpeter Swan: 96 mg/kg

Sed.Human (Sediment Concentrations vs. Human):

Bureau of Land Management RMC Benchmarks, 1995: Risk Management Criteria (RMC) were developed for the mostly dry BLM lands in the western U.S. These risk management criteria should be used by the land manager as a cautionary signal that potential health hazards are present and that natural resource management or remedial actions are needed [715]. Exceedances of the criteria should be interpreted as follows 715]:

Less than criteria: low risk
1-10 times the criteria: moderate risk
10-100 times the criteria: high risk
>100 times the criteria: extremely high risk

Human criteria for arsenic in sediments. These categories of humans not exposed to sediments with concentrations of arsenic exceeding the below RMCs are not expected to experience adverse toxic effects [715]:

Camp host: 46 mg/kg
Child Camper: 21 mg/kg
Boater: 166 mg/kg
Swimmer: 72 mg/kg

Sed.Misc. (Other Non-concentration Sediment Information):

The ultimate sink for most environmental arsenic is ocean sediment [604].

Limited data on the composition of pore waters from two areas in Canada suggest that most (> and equal to 85% of) bioavailable arsenic in sediment is present as inorganic As(III) and As(V) [604].

Soil Data Interpretation, Concentrations and Toxicity (All Soil Data Subsections Start with "Soil."):

Soil.Low (Soil Concentrations Considered Low):

Texas: Arsenic occurs naturally in the earth's crust in concentrations of approximately 1.8 ppm and in virgin soils ranges from 0.2 to 40 ppm with an average content of about 5 ppm. A total of 28 Texas counties had in their soils averages less than 1 ppm arsenic. This level is considered very low. Fifty-five (55) counties had average arsenic concentrates between 1 ppm and 2 ppm. This concentration would be considered low, but source of arsenic (native or man introduced) is not established [424].

Soil.High (Soil Concentrations Considered High).

NOTE: "High" depends partly on whether or not the natural concentrations in soils are high in the area.

In Canada, near smelters the mean concentrations ranged from 50-110 mg/kg, with one sample as high as 2000 mg/kg [604].

Arsenic in German Gulch (Upper Clark Fork Superfund Site Area, Montana) samples ranged from 47.2 to 136.4 ppm and averaged 89.5 ppm. Overall mean values for arsenic in U.S. soils are reported to be between 5.8 and 10 ppm (range <0.1-93 ppm) [699].

Information From Arsenic Survey of Texas Soils [424]: Texas volcanic ash deposits frequently have elevated concentrations of naturally occurring arsenic. These ash deposits are a result of ancient volcanic eruptions in New Mexico and other regions. From the total data set of 4100 samples, 10 samples had arsenic levels above 15 ppm. Approximately 25% of the soils having arsenic levels greater than 5 ppm were from urban areas. This situation is due to the use of herbicides, insecticides, paints, and composting.

In parts of the U.S., 120 ppm dry weight arsenic in soil would be considered "elevated" (Willard R. Chappell, University of Colorado at Denver, personal communication, 1995).

Arsenic concentrations up to 27,000 ppm were reported in soils contaminated with mine or smelter wastes [716]. Soil on agricultural lands treated with arsenical pesticides may retain substantial amounts of arsenic [716]. One study reported an arsenic concentration of 22 ppm in treated soil compared to 2 ppm for nearby untreated soil [716].

In the Netherlands, moderate soil contamination of Arsenic is 30 ppm [347].

Highest arsenic concentrations (up to 75,000 mg/kg; typically 3000 to 4000 mg/kg) were found in tailings at base-and-precious-metal mine sites in Ontario and Nova Scotia [604].

Elevated arsenic levels have also been reported in soils where arsenical pesticides (including wood-preservation compounds) have been used; for example, concentrations of arsenic of up to 290 mg/kg (mean values of up to 54 mg/kg) have been detected in soil from orchards in Ontario and up to 10,860 mg/kg (mean values of up to 6000) at active wood-preservation facilities in Atlantic Canada [604].

Elevated arsenic in soils, has been found around several pre-1910 cemeteries impacted by arsenic embalming fluid; a cemetery of 2,000 people could contain 330 pounds of arsenic (see also: uses/sources section below) [976].

Soil.Typical (Sediment Concentrations Considered Typical):

Arsenic is found in the earth's crust at an average level of 2 ppm [716]. Most natural soils contain low levels of arsenic, but industrial wastes and pesticide applications may increase concentrations [716]. Background arsenic concentrations in soil range from about 1 to 40 ppm, with a mean value of about 5 ppm [716]. Soils overlying arsenic-rich geologic deposits such as sulfide ores may have soil concentrations two orders of magnitude higher [716].

EPA 1981: 6.0 mg/kg dry weight not considered elevated [83].

In Canada, unpolluted areas tend to have dry weight soil concentrations from 4.8 to 13.6 mg/kg [604].

In parts of the U.S., 5 to 10 ppm dry weight arsenic in soil would be considered "background" (Willard R. Chappell, University of Colorado at Denver, personal communication, 1995).

What is typical depends on the area being examined. The range of arsenic in U.S. soils is broad, <0.1 - 93 ppm,

and most soils are in the less than 10 ppm range (Kabata-Pendias and Pendias, 1992) [699]. Baseline values for arsenic in soils overlying granitic rock typically range from 0.7 to 15 ppm (Kabata-Pendias and Pendias, 1984, as cited in Morrison Knudsen, 1991) [699]. The lowest concentrations of arsenic are found in sandy soils, particularly sandy soils derived from granites (means in the range 2-6 ppm). Higher arsenic concentrations are found in alluvial soils and soils rich in organic matter (means in the range of 5-25 ppm). Acid sulfate soils are reported to accumulate a high proportion of native arsenic, up to 30 to 50 ppm. An overall mean value for arsenic in U.S. soils is reported to be between 5.8 and 10 ppm [699].

Western U.S. Soil Median Concentrations: 7 mg/kg [715].

In Texas, eight (8) counties' soil had average arsenic concentrations greater than 2 ppm. These levels would be considered only slight but the presence of man's activities is beginning to show in some of these samples. No As were above 3 ppm in this data set [424].

Based on a review of available data, mean concentrations of arsenic in several uncontaminated soil types in Canada were reported to range from 4.8 to 13.6 mg/kg dry weight (dw) [604].

In the Netherlands, background concentrations in soil or detection limits of Arsenic are 20 ppm [347].

Igneous Rocks (Earth's Crust) Concentrations not Considered Elevated:

EPA 1981: 1.8 mg/kg dry weight [83].

Arsenic accounts for 5e-04 % of the earth's crust. Minerals containing arsenic include: ... Orpiment ... Realgar ... Claudetite ... Cobaltite ... Enargite [366].

The occurrence of arsenic in the continental crust of the earth is generally given as 1.5-2 ppm [445].

Analyses of sewage sludges from 50 publicly owned treatment works by the U.S. Environmental Protection Agency (1985): The mean concentration of arsenic was 5.9 ppm (dry weight) [347].

Analyses of 74 Missouri sewage sludges (in ppm; dry weight) (1985): Median 6.1, range 2-39 [347].

Soil.Concern Levels, Soil Quality Criteria, LC50 Values, Soil Quality Standards, Screening Levels, Dose/Response Data and Other Soil Benchmarks:

Soil.General (General Soil Quality Standards, Criteria, and Benchmarks Related to Protection of Soil-dwelling Biota in General; Includes Soil Concentrations Versus Mixed or General Soil-dwelling Biota):

Regulating arsenic in soil at the same (1E-04 cancer risk) level that EPA is proposing for water would require limiting soil concentration to a soil screening level of 37 ppm; this would be hard to achieve at some sites since the median concentration at the Leadville, CO, site is about 37 ppm (Willard R. Chappell, University of Colorado at Denver, personal communication, 1995).

Soil Criteria for evaluating the severity of contamination under the Dutch Soil Cleanup (Interim) Act (1982): Background concentrations in soil or detection limits of Arsenic are 20 ppm. Moderate soil contamination of Arsenic is 30 ppm. Soil concentrations of Arsenic which require immediate cleanup are 50 ppm [347].

Soil cleanup criteria for Arsenic for decommissioning industrial sites in Ontario (1987) [347]:

Benchmark for Agricultural land = 14 ppm

Benchmark for Residential/parkland = 25 ppm

Commercial/industrial = 50 ppm

Suggested safe applications (kg/ha) of arsenic to Missouri soils without further investigations (1988): 112 (maximum cumulative addition) [347].

Soviet Union Maximum Allowable Concentration in Soils, 1984: 2.0 mg/kg [347].

The 1987 soil (clean up) criteria given by the New Jersey Department of Environmental Protection for arsenic is 20 mg/kg dry weight [347, 386].

Proposals for Maximum Acceptable Concentrations (MAC) of Arsenic in Agricultural Soils as given by various authors [719]:

Proposal of European Economic Commission for MAC in soils treated with sewage sludge: 20 ppm dry weight (London)

Proposal of Ontario Ministry of Agriculture and Food for MAC in soils treated with sewage sludge: 14 ppm dry weight (published in Tokyo; work done for Ontario)

Other MAC levels: 20 ppm dry weight (Stuttgart).

Soil.Plants (Soil Concentrations vs. Plants):

Arsenic levels above 7 ppm will begin to affect some sensitive plants (such as rice). Arsenic levels above 17 ppm will eventually kill newly established vegetation [424].

Minimum soil concentration causing phytotoxicity: 15-50 [699].

Levels (ppm dry weight) considered phytotoxic: 50 (Vienna), 30 (Warsaw), 15 (Tokyo), 20 (Warsaw), 25 (Ontario) [719].

Oak Ridge National Lab, 1994: Risk Assessment Screening Benchmarks for Terrestrial Plants: To be considered unlikely to represent an ecological risk to terrestrial plants, field concentrations in soil should be below the following dry weight benchmark for soil in mg/kg (ppm) [651]:

For CAS 7440-38-2 (Arsenic, the soil concentration benchmark is 10 mg/kg dry weight.

Soil.Invertebrates (Soil Concentrations vs. Invertebrates):

No information found.

Soil.Wildlife (Soil Concentrations vs. Wildlife or Domestic Animals):

See Sed.Wildlife section above.

Soil.Human (Soil Concentrations vs. Human):

EPA 1996 National Generic Soil Screening Level (SSL) designed to be conservative and protective at the majority of sites in the U.S. but not necessarily protective of all known human exposure pathways, land uses, or ecological threats [952]:

SSL = 0.4 mg/kg for ingestion pathway [952].

SSL = 750 mg/kg for inhalation pathway [952].

SSL = 1 to 29 mg/kg for protection from migration to groundwater at 1 to 20 Dilution-Attenuation Factor (DAF) [952].

Editor's note: According to an article in

the Bozeman (Montana) Daily Chronicle Newspaper of December 30, 1996, increasing soil pH by adding lime, a remediation sometimes used to reduce soil acidity and reduce the mobility of metals such as copper, can result in the (unintended) consequence of increasing the mobility of arsenic and its transport to groundwater. The article stated that Bill Inskeep, soil scientist at Montana State University had seen an increase of arsenic percolation of 10 to 100 times after lime was added to arsenic contaminated soils (News Media Report, not yet independently confirmed, but included since lime is such a common treatment for acidic metals contaminated soils).

EPA 1995 Region 3 Risk based concentration (RBC) to protect from transfers to groundwater:

15 mg/Kg dry weight [903].

EPA 1995 Region IX Preliminary remediation goals (PRGs) for cancer risk [868]:

Residential Soil: 3.8E-01 mg/kg wet weight
Industrial Soil: 2.4 mg/kg wet weight

NOTE:

1) Values are based on a one-in-one million cancer risk.
2) PRGs focus on the human exposure pathways of ingestion, inhalation of particulates and volatiles, and dermal absorption. Values do not consider impact to groundwater or ecological receptors.
3) PRGs are slightly lower concentrations than EPA Region III RBCs, which consider fewer aspects [903].

Acceptable level for production of healthy food: 2 ppm dry weight (Moscow) [719].

Maximum Acceptable Concentration (MAC) trigger (of concern) concentration for domestic gardens and playing fields: 10 ppm dry weight (London) [719].

Bureau of Land Management RMC Benchmarks, 1995: Risk Management Criteria (RMC) were developed for the mostly dry BLM lands in the western U.S. These

risk management criteria should be used by the land manager as a cautionary signal that potential health hazards are present and that natural resource management or remedial actions are needed [715]. Exceedances of the criteria should be interpreted as follows [715]:

Less than criteria: low risk
1-10 times the criteria: moderate risk
10-100 times the criteria: high risk
>100 times the criteria: extremely high risk

BLM Human criteria for arsenic in soil. These categories of humans not exposed to soil with concentrations of arsenic exceeding the below RMCs are not expected to experience adverse toxic effects [715]:

Child resident (living on properties adjacent to BLM lands): 0.6 mg/kg
Camp host: 15 mg/kg
Child Camper: 11 mg/kg
ATV Driver: 218 mg/kg
Worker: 13 mg/kg
Surveyor: 134 mg/kg

Soil.Misc. (Other Non-concentration Soil Information):

Liming soil, a remediation sometimes used to reduce soil acidity and reduce the mobility of metals such as copper, can result in the (unintended) consequence of increasing the mobility of arsenic and its transport to groundwater (see more detailed note in Soil.Human section.

Some soils have naturally high arsenic, and other soils have been impacted by arsenic from pesticides.

The plant to soil ratio for arsenic varies from 0.2 to 39.7 [951].

Analysis of whole-soil and soil pore water from a limited number of areas indicates that most (>90%) of the arsenic in soils is inorganic [604].

Individuals living in the vicinity of large smelters and other industrial emitters of arsenic may be exposed to above average arsenic levels both in the air and, as a result of atmospheric deposition, in water and soil [716].

Soils can be contaminated with inorganic arsenic or methylated arsenical contained in herbicides. Methylated arsenical are eventually degraded (half-lives = 0.5 to 2.9 years) to carbon dioxide and arsenate by soil

microorganisms [604].

Tissue and Food Concentrations (All Tissue Data Interpretation Subsections Start with "Tis."):

Tis.Plants:

A) As Food: Concentrations or Doses of Concern to Living Things Which Eat Plants:

Based on very limited data, concentrations of arsenic in produce grown in the vicinity of industrial sources may be somewhat higher than those reported in the duplicate diet study [604].

B) Body Burden Residues in Plants: Typical, Elevated, or of Concern Related to the Well-being of the Organism Itself:

Arsenic concentrations in plant tissue collected across Smelter Hill (Upper Clark Fork Superfund Site Area, Montana) averaged 24.6 ppm. Arsenic content in a wide variety of plants from uncontaminated regions typically averages between 0.01 and 5.0 ppm (Kabata-Pendias and Pendias, 1984, as cited in PTI, 1991a) [699].

Levels of arsenic in the tissue of freshwater macrophytes in areas remote from point sources of contamination generally range from <10 mg As/kg dry weight (dw). Concentrations of arsenic up to 538 and 206 mg As/kg dw have been detected in aquatic macrophytes growing near a gold mine in Nova Scotia in 1981 and a base-metal smelter in Manitoba from 1975 to 1976 respectively [604].

High levels of arsenic were also found in aquatic macrophytes from Kam Lake: maximum 3700 mg As/kg dry weight (dw); mean 1010 mg/kg dw [604].

Results of a more recent study (1990-91) indicate that levels of arsenic in aquatic macrophytes near Yellowknife remain high (up to 4900 mg/kg dry weight) [604].

In freshwater aquatic plants, arsenic is present mainly as lipid and water-soluble, "lipid-related" compounds; lesser amounts of arsenite and methylated As(V) species are also present. Although little is known about the behavior of arsenicals in terrestrial plants, methylation has been reported in some plants grown in nitrate- or phosphate-deficient conditions. Arsenosugars and arsenic containing lipid compounds, as well as methylated arsenicals, have been found in marine

plants [604].

Arsenic is frequently found in plants, often as a result of pesticide treatment [716]. Concentrations typically vary from 0.01 to 5 ppm [716]. Tobacco levels of arsenic average 1.5 ppm, or about 1.5 mg per cigarette [716].

Tis.Invertebrates:

A) As Food: Concentrations or Doses of Concern To Living Things Which Eat Invertebrates:

No information found.

B) Concentrations or Doses of Concern in Food Items Eaten by Invertebrates:

No information found.

C) Body Burden Residues in Invertebrates: Typical, Elevated, or of Concern Related to the Well-being of the Organism Itself:

See also information on clams in the Trinity River Report information in Tis.Fish, C) section below.

Zooplankton from Kam Lake had high levels of arsenic: maximum 2400 mg As/kg dry weight (dw); mean 1875 mg/kg dw [604].

The following information summarizes data gathered from the NOAA National Status and Trends (NS&T) Program for the year 1990 [697]:

> For arsenic in mussels and oysters combined (n=214), the Geometric Mean was 10 ug/g dry and the "high" concentration was 17 ug/g dry weight [697]. NOAA "high" concentrations are equal to the geometric mean plus one standard deviation on the log normal distribution [696].

Tis.Fish:

A) As Food: Concentrations or Doses of Concern to Living Things Which Eat Fish (Includes FDA Action Levels for Fish and Similar Benchmark Levels From Other Countries):

Arsenic has been detected in most foodstuffs consumed by humans; however the proportion of inorganic arsenic varies considerably. Much of the arsenic in fish is present in highly complex forms that are not bioavailable, or as organic compounds that are rapidly excreted from the body [604].

Legal Limits for Concentrations in Fish and Fishery Products:

The lowest legal limit was 0.1 mg/kg (Venezuela) [216,418]. Seven countries have limits less than or equal to 1.0 mg/kg, but the U.S. apparently has no limit [216,418].

NOTE: One reference stated that "Arsenic is one of the few metals which tends to concentrate in axial muscles of fish" [29]. However, Sorenson's summary of 1991 stated that when compared to food web organisms, fish generally concentrate strikingly low concentrations of arsenic in skeletal muscles [488].

Bureau of Land Management RMC Benchmarks, 1995: Risk Management Criteria (RMC) were developed for the mostly dry BLM lands in the western U.S. These risk management criteria should be used by the land manager as a cautionary signal that potential health hazards are present and that natural resource management or remedial actions are needed [715]. Exceedances of the criteria should be interpreted as follows 715]:

Less than criteria: low risk
1-10 times the criteria: moderate risk
10-100 times the criteria: high risk
>100 times the criteria: extremely high risk

Human criteria for arsenic in fish consumed by humans. These categories of humans not exposed to fish with concentrations of arsenic exceeding the below RMCs are not expected to experience adverse toxic effects [715]:

Child resident (living on properties adjacent to BLM lands): 24 ug/kg
Camp host: 48 ug/kg
Child Camper: 133 ug/kg

For risk to human adults eating fish, separate carcinogenic and non-carcinogenic risk-based fish tissue concentrations were calculated [903]. The following EPA Region III fish tissue risk-based concentration (RBC) benchmark utilizes the lower of the two concentrations (carcinogenic), rounded to two

significant figures [903]:

RBC = 0.00041 mg/Kg for cancer risk for arsenic CAS 7440382; for risks other than cancer the RBC is 0.41 mg/kg. These concentrations are presumably wet weight, although EPA does not say whether wet or dry [903].

B) Concentrations or Doses of Concern in Food Items Eaten by Fish:

No information found.

C) Body Burden Residues in Fish: Typical, Elevated, or of Concern Related to the Well-being of the Organism Itself:

See also [445] information in W.Fish above.

NOTE: One reference stated that "Arsenic is one of the few metals which tends to concentrate in axial muscles of fish" [29]. However, Sorenson's summary of 1991 stated that when compared to food web organisms, fish generally concentrate strikingly low concentrations of arsenic in skeletal muscles [488].

Fish/Seafood Concentrations of Arsenic:

Mean NCBP Levels (Tissue Concentrations): The geometric mean of whole-body concentrations of fish in a 1980-1981 and 1976-1984 national surveys was 0.14 mg/kg (wet weight) of arsenic [23,384].

Livers of fish from the great lakes contain 5.6 to 80 ppb mainly in the fat fraction. Fish generally contain lower arsenic levels than other aquatic organisms (Lundi J; Science Food Agriculture 21: 242, 1970)[366].

Arsenic levels in fish muscle from Abu Quir Bay ranged from 0.97 to 10.5 ppm. Levels in Tilapia muscle from Idku and Margut Lakes ranged from 0.11 to 0.18 ppm. Arsenic levels in fish livers were not consistently higher than in fish muscle (El Nabawi A et al; Bull Environ Contam Toxicol 39,5: 889-97, 1987)][366].

The arsenic content of edible muscle of 2 tuna species (Thunnus thynnus and Thunnus toggel) caught in Arabian Sea waters was 2.88 and 2.51 ug/g dry wt, respectively, for the 2 species.

A marked increase in arsenic content was found with increasing wt of the 2 fish species (Ashraf M, Jaffar M; Bull Environ Contam Toxicol 40, 2 : 219-25, 1988)[366].

Other Edible Tissue (Mostly Fillet) Concentrations for Arsenic in Freshwater Fish:

The highest concentrations of arsenic in 4 studies of edible fish tissues in several states ranged from 0.1 to 2.9 mg/kg wet wt [57].

Tissue Concentrations in Texas [201]:

The following text is quoted from the Trinity River Report [201] for reference comparison with values from other areas):

Due to cost, we analyzed for arsenic in only 50 Trinity River samples. Arsenic was found above the detection limit (0.05 mg/kg) in all but 7 of these samples.

Predator Protection Level: Arsenic whole-body levels above 0.5 mg/kg are considered to be harmful to fish and predators [20]. All four Trinity River samples above the 0.5 mg/kg level were clam flesh samples rather than fish, including one sample (0.93 mg/kg) of unionid clams from site 14 and Asian clams from sites 14, 26, and 5 (0.72 to 0.89 mg/kg). Clams were not found at the most polluted sites below Dallas. These high levels for clams tend to confirm previous observations that clams, unlike fish, are efficient arsenic accumulators [57,83,95]. A nationwide study of arsenic in bivalves showed less variation in levels from various stations than was found for most other contaminants, with greater variation between different bivalve species from the same location [62].

Mean NCBP Levels: The geometric mean of whole-body concentrations of fish in a 1980-1981 national survey was 0.14 mg/kg arsenic [23], a level exceeded in 24 of 50 Trinity River samples. Included were numerous species of fish and turtles from both upstream and downstream sites. However, since this group of samples included a variety of turtle samples, it

is not directly comparable to the NCBP means for fish only.

Gradient Monitoring Levels: Elevated concentrations of arsenic (above recommended criteria) in water and sediments have previously been reported for an area downstream of Dallas [42,71]. However, another summary seemed to suggest that arsenic may not be as highly elevated in sediments of the upper Trinity as are several heavy metals [7].

In our study, plots and statistical analyses of arsenic levels in mosquitofish versus river miles, location groups, and runoff types revealed no clear trends or correlations. Certain clam species may be better than mosquitofish as indicator species for gradient monitoring of arsenic.

Although most Trinity River tissue samples do not show highly elevated levels, arsenic is a compound for which we need more data to assess risks to fish and wildlife. A zero level of arsenic would be most effective at protecting from carcinogenic risk [21]. However, a zero level is probably not currently attainable due to the many potential sources of arsenic in the river.

Tis.Wildlife: Terrestrial and Aquatic Wildlife, Domestic Animals and all Birds Whether Aquatic or not:

A) As Food: Concentrations or Doses of Concern to Living Things Which Eat Wildlife, Domestic Animals, or Birds:

Predator Protection Level (Tissue Concentrations):

Arsenic whole-body levels above 0.5 mg/kg are considered to be harmful to fish and predators [20].

No regulatory standards currently exist for the protection of fish and wildlife from dietary exposure to arsenic [445].

B) Concentrations or Doses of Concern in Food Items Eaten by Wildlife, Birds, or Domestic Animals (Includes LD50 Values Which do not Fit Well into Other Categories, Includes Oral Doses Administered in Laboratory Experiments):

In arsenic poisoned cattle, the average arsenic concentration in the ingesta (food they had been eating) was 35.7 ppm wet weight [772].

Information from 1990 report entitled: "Fish and Wildlife Resources and Agricultural Drainage in the San Joaquin Valley, California," quoted word for word with the permission of Senior Author Stephen Moore, Fish and Wildlife Service, Portland Oregon, Regional Office (see original publication for embedded references) [445]:

> Camardese et al. (1990) and USFWS-PWRC (Jan 1989; Jan 1988) conducted feeding trials to assess the toxicity of arsenic+5 to mallard ducklings. One day-old mallards were placed on commercial duck mash (12.1-14.2% moisture) diets containing 0, 30, 100, or 300 ppm arsenic+5 (dry weight, as sodium arsenate) for 10 weeks. Survival was not affected by any of the tested dietary arsenic+5 concentrations. Liver, brain, spleen, and body weights were not significantly different between the groups and the organ to body weight ratios did not differ for any of these. Arsenate treatment did in significant histopathological lesions, nor did it result in significant effects on hematocrit percentages or hemoglobin concentrations. Although duckling body weights were not significantly different after 10 weeks, growth rates among females were reduced in all of the arsenic+5 treatment groups and males experienced delayed growth in the 300 ppm group. The authors suggested that reduced consumption of feed may have contributed to the growth delays [445].
>
> Arsenate treatment resulted in the following effects on blood, liver, and brain biochemistry: decrease in plasma creatine kinase (CK) activity in at 30 ppm, but increase in plasma CK activity at 300 ppm; increase in plasma sorbitol dehydgenase activity (indicative of hepatic alteration) at 300 ppm; increase in plasma glucose at 300 ppm; increase in plasma triglyceride concentrations at all treatment concentrations; increase in liver adenosine triphosphate (ATP) concentration treatment concentrations; decrease in liver DNA to RNA ratcrease in liver protein-bound sulfhydryls (PBSH) and protein concentrations in females at 300 ppm; increase in liver nonprotein sulfhydione) at all treatment concentrations; decrease in glutathione peroxidase

concentrations at 300 ppm; decrease in liver malondialdehyde concentrations at all treatment concentrations (indicating decreased hepatic lipid peroxidation); increase in total liver thiols in females at 100 ppm; decrease in brain ATP (primary energy source of the brain) at 300 ppm; increase in brain sodium/potassium ATPase activity at 30 and 100 ppm; increase in total brain ATPase activity at 30 and 300 ppm; and increase in brain acetylcholinesterase (AChE) activity in females at 30 ppm [445].

The USFWS-PWRC (Jan 1989; Jan 1988) studied the interactive effects of three trace elements and nutrition on mallard ducklings. Various concentrations of arsenic+5 (as sodium boron (as boric acid), and/or selenium-2 (as seleno-DL-methionine) were added to ducklings' diets containing low (7%) or normal (21%) amounts of protein. Two separate sets of tests were conducted over 4 weeks. In the first, six groups of ducklings were fed diets containing both levels of protein and arsenic+5 and selenium-2, singly and in combination. In the second experiment, six groups of ducklings were fed diets containing both levels of protein and boron and selenium-2, singly in combination. Preliminary results regarding the effects of arsenic in combination with selenium and both dietary levels of protein are available and are discussed here [445].

Arsenate in the normal protein diet appeared to protect ducklings from exposure to high concentrations (60 ppm) of selenium-2, reducing mortality from 40% to 0%. This protective effect was less pronounced when the dietary protein level was low [445].

Bell (1972) determined that the oral LD50 for arsenic trioxide in the opossum (Trichosurus vulpecula) was 8.22 mg/kg (ppm) (dose administered via enterogastric catheter). In the same study, Bell observed the dose mortality rate for arsenic trioxide given as a single oral dose. At 6 and 10 mg arsenic trioxide/kg body weight, 3 of 9 animals died in 17-120 hours and 7 of 9 animals died in 7-120 hours, respectively [445].

Inns et al. (198) compared the acute systemic toxicity of sodium arsenite and dichloro(2-chlorovinyl)arsine (lewisite) in rabbits. The

LD50 of sodium arsenite was 7.6 mg/kg (ppm); that for lewisite was 1.8 mg/kg. After rabbits were treated with an LD10 dose of the two arsenicals, higher concentrations of arsenic were found in all tissues, except for lung, in arsenite-treated animals compared to the lewisite-dosed animals. Intravenously injected lewisite was preferentially distributed to the lungs, the tissues of which showed a variety of histological changes [445].

The effects of low dietary intake of methionine, choline, and proteins on arsenite accumulation and excretion in the rabbit were investigated by Vahter and Marafante (1987). Groups of rabbits on a standard diet, a choline-deprived diet, a methionine-deprived diet, and a low-protein diet were given a single intravenous injection of arsenite (as arsenic trioxide). The low dietary intake of methionine, choline, or proteins was found to decrease urinary arsenic excretion by 20% (mainly due to a lower excretion of dimethylarsinic acid in the urine) and to increase tissue retention of arsenic, especially in the liver and the lungs. Based on the results of this study, the authors indicated that rabbits with a poor nutritional status have a lower capacity of methylating and thereby detoxifying inorganic arsenic [445].

The LD50 of a single oral dose of sodium arsenite in the California quail (Callipepla cals 47.6 mg/kg body weight; in the ring-necked pheasant (Phasianus colchicus) the LD50 was 386 mg/kg body weight; and in the mallard duck (Anas platyrhynchos) the LD50 was 323 mg/kg body weight [445].

Chaineau et al. (1990) studied the direct embryo-lethal and teratogenic effects of sodium arsenite and sodium arsenate on mouse embryos in culture. Post-implantation mouse embryos were cultured in a serum medium containing 1-40 uM sodium arsenite or 10-400 uM sodium arsenate for 48 hours. A comparison to control embryos showed that: 1) sodium arsenite was teratogenic above 4 uM and embryo-lethal above 15 uM, and 2) sodium arsenate was teratogenic above 40 uM and embryo lethal above 150 uM. None of the dead embryos displayed appearance of development. Both compounds produced growth retardation,

indicated by reduced crown-rump length, head length, and yolk sac diameter, and a similar pattern of defects, characterized by prosencephalon hypoplasia with open neural tube, somite alterations, hydropericardium, and failure of development of limb buds and sensory placodes [445].

Indirect fetal toxicity of sodium arsenite in mice was studied by Hood (1972). Albino Swiss-Webster female mice were mated then received a single intraperitoneal injection of sodium arsenite on one of days 7-12 of gestation. Dose levels were 10 or 12 mg arsenite/kg body weight. Females were sacrificed on day 18 at which time fetal observations were made. Arsenite treatment resulted in a significant increase in fetal deaths for all days (7-12) and both dose levels (10 or 12 mg/kg). A decrease in fetal weight was correlated to both day of treatment and dose. The period of greatest susceptibility to teratogenic effects was found to be from gestation days 8 through 10. The most common malformations associated with arsenite treatment were exencephalpy, micrognathia, open eye, and skeletal anomalies of the ribs and vertebrae; bent, shortened, or missing tails were also noted in several fetuses [445].

No regulatory standards currently exist for the protection of fish and wildlife from dietary exposure to arsenic [445].

Oak Ridge National Lab, 1994: Risk Assessment Screening Benchmarks for Wildlife derived from No-Observed-Adverse-Effect (NOAEL) levels (mg contaminant per kg body weight per day). To be considered unlikely to represent an ecological risk, wet-weight field concentrations should be below the following (right column) benchmarks for each species present at the site [650]:

For CAS 7440-38-2 (ARSENIC AS ARSENITE), the bencmarks are:

SPECIES	NOAEL (mg/kg/day)	FOOD CONCEN-TRATION (ppm)
Mouse (test species)	0.12600	0.00000
Short-tailed Shrew	0.15800	0.26400
Little Brown Bat	0.19900	0.59700
White-footed Mouse	0.14000	0.90300
Meadow Vole	0.11100	0.97700

Cottontail Rabbit	0.03700	0.18900
Mink	0.04000	0.28900
Red Fox	0.02400	0.24100
Whitetail Deer	0.01000	0.34000

Comment: Actually, the number of significant figures for a benchmark value should never be more than one; even if these values have been taken directly from another report, they should be rounded; otherwise the impression is given of a level of accuracy that is simply unwarranted. The uncertainties are too large to justify such a fine distinction (Owen Hoffman, SENES Oak Ridge, Personal Communication, 1997).

Other Information on Dietary Concentrations of Arsenic of Concern to Waterfowl:

Aquatic vegetation concentrations of arsenic as low as 30 mg/kg wet weight could alter the growth, physiology, and development of ducklings [429]. Sodium arsenite in the diet of ducklings at 250 ppm caused 12% mortality [424] Sodium arsenite had a LD50 of 323 ppm in mallard hens [429].

Predator Protection Level (Tissue Concentrations):

Arsenic whole-body levels above 0.5 mg/kg are considered to be harmful to fish and predators [20].

LD50/LC50 Values for rats (Done, 1971) [363]:

Sodium arsenite: 42 mg/kg
Arsenic trioxide: 385 mg/kg
Ortho crabgrass killer (8% methane arsonate, 8% dodecyl NH4 methane-arsonate): 595 mg/kg

The following article was summarized by Susan Dodson, Arlington Field Office, US Fish and Wildlife Service, Arlington, Texas in January 1992:

Casteel, S.W., E.M. Bailey, Jr., M.J. Murphy, A.C. Ray and J.C. Reagor. 1986. Arsenic poisoning in Texas cattle: The implications for your practice. Vet. Med. 81:1045-1049.

Case reports of confirmed arsenic poisoning of Texas cattle in 1985 by the Texas Veterinary Medical Diagnostic Laboratory were reviewed in this article. The toxicity of arsenic varies with

form, trivalent arsenic compounds being the most toxic, followed by pentavalent aliphatics. Phenylarsonic compounds are least toxic and are approved for use in feed additives. For most species of animals, the lethal oral dose of sodium arsenite is 1-25 mg/kg body weight. The average lethal oral dose of sodium arsenite in cattle is about 1-4 grams. Another study of cattle found the lethal oral dose was five daily doses of 10 mg/kg body weight of monosodium methanearsonate (MSMA), and six daily doses of 25 mg/kg body weight disodium methanearsonate (DMSA). Clinical signs and lesions are similar for these aliphatic organic arsenicals and with inorganic arsenicals.

The mechanism of arsenic intoxication is related to its reaction with sulfhydryl groups vital to enzyme function and arsenic ability to uncouple oxidative-phosphorylation. Arsenic toxicity can result in a range of effects from rapid cardiovascular collapse and sudden death, to watery diarrhea, salvation, weakness, trembling and other symptoms for days to weeks prior to death. Application of MSMA and DSMA, even at recommended rates, can pose risk to cattle allowed to graze treated areas, and by cotton desiccant spray drift from cotton field to adjacent pasture. Case studies included a liver arsenic level of 3.3 ug/g with a duration of illness for two weeks, and 5.18 ug/g with illness of 24 hr.

C) Body Burden Residues in Wildlife, Birds, or Domestic Animals: Typical, Elevated, or of Concern Related to the Well-being of the Organism Itself:

Values as low as 1.5-5 ppm and as high as 30-38 ppm were found in the liver and kidneys of some cattle [772]. In arsenic poisoned cattle, the average arsenic concentration in the ingesta (food they had been eating) was 35.7 ppm wet weight, while the average concentration in livers was 14 ppm and the average concentration in kidneys was 13.3 ppm [772]. Liver concentrations of more than 10-15 ppm arsenic wet weight are usually found in animals which have been exposed to lethal doses of arsenic [772].

Tis.Human:

A) Typical Concentrations in Human Food Survey Items:

See also Tis.Fish, C) above.

Arsenic is found in many types of food [716]. The highest levels are detected in seafood, meats, and grains [716]. Typical U.S. dietary levels of arsenic in these foods range from 0.02 ppm in grains and cereals to 0.14 ppm in meat, fish, and poultry, but there is a wide range of values [716]. Shellfish and other marine foods contain the greatest arsenic concentrations [716]. Mean levels in fish and seafood are usually about 4-5 ppm, but may be as high as 170 ppm [716].

It is important to bear in mind that much of the arsenic present in fish and shellfish exists in an organic form that is essentially nontoxic [716]. However, some of the arsenic in these foods is in inorganic form [716]. For example, a recent study in the Netherlands reported that inorganic arsenic comprised 0.1-41% of the total arsenic in seafood [716].

Patients consuming opium for long periods of time have developed arsenic neuropathy [363]. Arsenic content of the opium has been measured to be as high as 74.1 mcg/100 g (Datta, 1977) [363].

The percentage of total arsenic that is inorganic in various foods has been determined to range from 0% in saltwater fish to 75% in milk, dairy products, beef, and pork. Mean concentrations of total arsenic in 10 food groups surveyed in a duplicate diet study in five cities in Canada ranged from 0.46 ng/g or ug/L (drinking water) to 60.1 ng/g (0.0601 mg/kg) [meat, fish, and poultry] [604].

Moonshine ethanol: Contaminated moonshine has been found to contain up to 415 mcg/L of arsenic (Gerhardt et al, 1980) [363].

Seafood, especially shellfish, have significant arsenic concentrations [363]. Ingestion may result in urinary arsenic levels of 200 to 1700 mcg/L within 4 hours (Baselt & Cravey, 1989) [363].

Arsenic has also been detected in several homeopathic medicines at concentrations up to 650 ppm [716].

B) Concentrations or Doses of Concern in Food Items Eaten by Humans (Includes Allowable Tolerances in Human Food, FDA, State and Standards of Other Countries):

Note: One author stated, that with the exception of localized area of heavy pollution from inorganic arsenic, which can accumulate in the gills and certain digestive organs such as the glands and livers, the consumption of seafood with relatively high concentrations of total arsenic does not pose much of a threat to humans [1024].

EPA 1996 IRIS database information [893]:

Crit. Dose: 0.0008 mg/kg-day [Study 1 NOAEL(adj)] UF: 3 MF: 1

RfD: 3E-4 mg/kg-day [893]. Reference dose: 3.0E-04 mg/kg-d [952]. Confidence: Medium [893].

NOTE: There was not a clear consensus among Agency scientists on the oral RfD. Applying the Agency's RfD methodology, strong scientific arguments can be made for various values within a factor of 2 or 3 of the currently recommended RfD value, i.e., 0.1 to 0.8 ug/kg/day. It should be noted, however, that the RfD methodology, by definition, yields a number with inherent uncertainty spanning perhaps an order of magnitude. New data that possibly impact on the recommended RfD for arsenic will be evaluated by the Work Group as it becomes available. Risk managers should recognize the considerable flexibility afforded them in formulating regulatory decisions when uncertainty and lack of clear consensus are taken into account [893].

Quantitative estimate of carcinogenic risk from oral exposure ———

Cancer Slope Factor: 1.5E+0 per mg/(kg/day) [868,893,952]

Unit Risk: 5E-5 per ug/liter Extrapolation Method: Time- and dose-related formulation of the multistage model (U.S. EPA, 1988)[893].

For risk to human adults eating fish, separate carcinogenic and non-carcinogenic risk-based fish tissue concentrations were calculated [903]. The following EPA Region III fish tissue risk-based concentration (RBC) benchmark utilizes the lower of the two concentrations (carcinogenic), rounded to

two significant figures [903]:

RBC = 0.00041 mg/Kg (presumably wet weight) for cancer risk for arsenic CAS 7440382; for risks other than cancer the RBC is 0.41 mg/kg.

See also Tis.Fish, A) above.

Allowable Tolerances of Arsenic for Other Human Foods and Drinks [366]:

Tolerances for total residues of combined arsenic (calculated as As) in food are established as follows: In edible tissues and in eggs of chickens and turkeys: 0.5 ppm in uncooked muscle tissue; 2 ppm in uncooked edible by-products; 0.5 ppm in eggs. In edible tissues of swine: 2 ppm in uncooked liver and kidney; 0.5 ppm in uncooked muscle tissue and by-products other than liver and kidney. [21 CFR 556.60 (4/1/88)].

FDA Requirements: Bottled water shall, when a composite of analytical units of equal volume from a sample is examined by the methods described in paragraph (d)(1)(ii) of this section, meet the standards of chemical quality and shall not contain arsenic in excess of 0.05 mg/l. /Arsenic as As ion/ [21 CFR 103.35 (4/1/88)].

Legal Limits for Concentrations in Fish and Fishery Products:

The lowest legal limit was 0.1 mg/kg (Venezuela) [216,418]. Seven countries have limits less than or equal to 1.0 mg/kg, but the U.S. apparently has no limit [216,418].

For the general population, food is generally the greatest source of arsenic exposure [716]. In the United States, food intake of arsenic has recently been estimated to be about 46 mg/day, with the largest contribution from meat, fish, poultry, grain, and cereal products [716]. Some of this is probably in the form of organic arsenicals [716]. Drinking water may also be a significant source of arsenic exposure [716]. Estimates of arsenic intake for adults drinking 2 liters of water per day average about 5 mg/d, but could be higher (10-100 mg/d) where levels in water are above average [716]. It is assumed that nearly all arsenic in drinking water is inorganic [716].

C) Body Burden Residues in Humans: Typical, Elevated, or

of Concern Related to the Well-being of Humans:

Elevated arsenic in human remains from pre 1910 cemeteries, in one case 28,000 parts per million = 2.8 percent, has been found in the tissue remains of civil war soldiers impacted by arsenic embalming fluid (see also: uses/sources section below) [976].

Asian folk remedies have been reported to contain levels of arsenic that have resulted in arsenic poisoning with elevated arsenic levels of up to 3,334 mcg/24hr in Hmong Southeast Asian refugees (Hall et al, 1989) [363].

Milk Concentrations [366]:

Colostrum and transitional milk were obtained from 15 healthy mothers living in the Athens area /Greece/ with mean age of 26 yr. Mature milk was obtained from 5 of the 15 mothers. The concn of arsenic and some other trace metals in human milk were determined using neutron activation analysis. There were no differences between levels in human colostrum, transitional, and mature milk, all of which were about 3 ug/l (range 0.6-6.3 ug/l). /Inorganic arsenic/ [WHO; Environ Health Criteria: Arsenic p.64 (1981).

Body Burdens [366]:

Normal values of arsenic in urine ... vary from 0.013 to 0.046 mg/l, to 0.13, to 0.25. The urinary excretion in mg/l, of elements that are freely eliminated by this route, such as fluorine, mercury, and arsenic, is at most 2.5 to 5 times the occupational exposure in mg/cu m of air. [American Conference of Governmental Industrial Hygienists. Documentation of the Threshold Limit Values and Biological Exposure Indices. 5th ed. Cincinnati, OH:American Conference of Governmental Industrial Hygienists, 1986. 37].

Colostrum and transitional milk were obtained from 15 healthy mothers living in the Athens area /Greece/ with mean age of 26 yr. Mature milk was obtained from 5 of the 15 mothers. The concn of arsenic and some other trace metals in human milk were determined using neutron activation analysis. There were no differences between levels in human colostrum, transitional, and mature milk, all of which were about 3 ug/l (range 0.6-6.3 ug/l). /Inorganic arsenic/ [WHO; Environ Health

Criteria: Arsenic p.64 (1981)].

Human body burden less than 100 mg/70 kg /from table/. /Inorganic arsenic/ [Doull, J., C.D. Klaassen, and M. D. Amdur (eds.). Casarett and Doull's Toxicology. 2nd ed. New York: Macmillan Publishing Co., 1980. 410].

Tis.Misc. (Other Tissue Information):

The toxicity of arsenic to plants is moderate; arsenic and gold are often found together and often elevated in the same plants, soils, or rocks, so arsenic concentrations are used in prospecting for gold [951]. The plant to soil ratio for arsenic varies from 0.2 to 39.7 [951].

Wood preservative: Chromium-copper-arsenate (CCA) used as a wood preservative has been claimed to cause arsenic poisoning in a family using CCA-impregnated wood as firewood (Peters et al, 1984) [363]. Arsenic levels in blood, urine, hair, and nails have been shown to be higher in opium eaters in India when compared to a control population (Narang et al, 1987) [363].

Limited data on arsenic speciation in plant and aquatic-animal tissue suggest that most arsenic is in the form of organo-arsenic compounds; however, a small amount (<1-30%) may be present as inorganic As(III) [604].

Since the EPA ban on the sale of sodium arsenate-containing ant poisons, the number of arsenate related poisoning calls has decreased in Michigan (Kuslikis et al, 1991) [363].

Bio.Detail: Detailed Information on Bioconcentration, Biomagnification, or Bioavailability:

Various aquatic organisms, including algae, crustaceans and fish, bioaccumulate arsenic. Bioconcentration factors (BCFs) of up to several thousand have been reported for these aquatic organisms. Arsenic does not biomagnify through the aquatic or terrestrial food chains [604]. A study from the mid-1970's found the BCFs to range from 53 to 80 in Keg Lake near Yellowknife [604].

The best potential mediums for biological monitoring appear to include animal hair, clams, algae, and higher plants [83].

Information from HSDB [940]:

Hair samples collected from common hare (Lepus europaeus), common vole (Microtus arvallis), and wood mouse (Apodemus sylvaticus) were subjected to instrumental neutron activation analysis (INAA). Up to 18 elements (arsenic, gold, bromine, cesium, cobalt, chromium, copper, iron, mercury, potassium, lanthanum,

sodium, antimony, scandium, selenium, samarium, thorium and zinc) were detected in each hair sample. Animal hair samples from areas polluted by thermal power plants burning coal were taken and compared with hair samples from the animals living in relatively nonpolluted control areas. Animal hair samples from areas with higher levels of pollution contain usually higher concn of toxic and essential elements as As, Co, Cr, Fe, and Se. Muride rodents can be used for more detailed monitoring of environmental exposure than the hare. Moreover, the hair of the common vole shows usually highest levels of contamination as compared with the wood mouse, which could be explained by different components of feed. Animal hair was a rather sensitive indicator of environmental exposure and INAA proved to be a suitable analytical tool for this purpose. [Obrusnik I, Paukert J; J Radioanal Nucl Chem 83 (2): 397-406 (1984)]

Additional information on arsenic bioaccumulation from 1990 report entitled: "Fish and Wildlife Resources and Agricultural Drainage in the San Joaquin Valley, California," quoted word for word with the permission of Senior Author Stephen Moore, Fish and Wildlife Service, Portland Oregon, Regional Office (see original publication for embedded references) [445]:

Bioaccumulation: The sorption of arsenate ions in the soil by iron, zinc, and aluminum greatly restricts the availability of arsenic to plants (Walsh, 1977); bioavailability can also be affected by soil pH, texture, phosphorous and calcium content, organic matter content, and moisture (Woolson, 1975). The arsenic content of plants grown on soils containing natural concentrations of arsenic (1-20 ppm) (Wauchope, 1983) varies from 0.01 to approximately 5 mg/kg (ppm, dry weight) (NRC-Committee on Medical and Biological Effects of Environmental Pollutants, 1977). Once arsenic enters a plant it is, like phosphorous, freely transported both actively and passively into all active tissues and tissue concentrations are essentially proportional to arsenic availability (Wauchope, 1983). Plants grown on soils contaminated with arsenic through such anthropogenic activities as smelting, mining, or arsenical pesticide application may contain considerably higher concentrations of arsenic, most of which is accumulated in the roots (Cullen and Reimer, 1989). Vascular plants are able to concentrate the widely used herbicide monosodium methanearsonate (aka MSMA [CH4AsNaO3]) from water and thus serve as a sink for concentrating the arsenical from the aquatic environment (Anderson et al., 1981). For the water hyacinth (Eichhornia crassipes), the calculated bioaccumulation ratio for root tissue (which showed the greatest absorption) was 24, after 3 weeks of exposure to 10 ug/ml (ppm) MSMA [445].

Many aquatic organisms bioconcentrate arsenic. A review

by Woolson (1975) and a study by Isensee et al. (1973) reported that lower food-chain organisms such as algae and daphnids typically have higher bioconcentration factors than higher food-chain organisms such as fish. Spehar et al. (1980) investigated arsenic accumulation and toxicity in several freshwater invertebrates and one species of fish. In this study, an intermittent-flow exposure system delivered arsenic+3 (as arsenic trioxide), arsenic+5 (as arsenic pentoxide), sodium dimethylarsonate (aka sodium cacodylate or SDMA [C2H6AsNaO2]) and disodium monomethanearsonate (aka DSMA [CH3AsNa2O3]) at two concentrations (100 or 1,000 ug/L [ppb]) to stoneflies (Pteronarcys dorsata), snails (Helisoma campanulata and Stagnicola emarginata), amphipods (Gammarus pseudolimnaeus), and rainbow trout (Salmo gairdneri). The length of exposure was 28 days. A static system utilizing test water from the flow-through system was also employed for a 3-week life-cycle test with daphnids (Daphnia magna). Water hardness was 42-45 ppm as calcium carbonate (CaCO3) and pH was 6.9-7.3 [445].

Stoneflies exposed to the four arsenic compounds at 1,000 ug/L accumulated similar residue concentrations, resulting in bioconcentration factors ranging from 33 to 45. Stoneflies exposed to arsenic at 100 ug/L accumulated highest residues when exposed to arsenic+5, which resulted in a bioconcentration factor of 131. The resulting bioconcentration factors of the snail (H. campanulata) exposed to arsenic+3 (at 1,000 ug/L) and arsenic+5 (at 100 ug/L) were 83 and 99, respectively. The snail (S. emarginata) accumulated similar arsenic residues when exposed to the high concentration of arsenic+3, arsenic+5, and DSMA, resulting in bioconcentration factors ranging from 16 to 17. Residue concentrations were significantly less in animals exposed to SDMA. Residue accumulation was also highest in S. emarginata exposed to the low concentration of arsenic+5, resulting in a bioconcentration factor of 92. Amphipods and trout did not accumulate arsenic when exposed to any of the compounds after 28 days [445].

Arsenic accumulation in daphnids increased with increased exposure concentration and residues were highest in daphnids exposed to arsenic+3. After the 21-day exposure, bioconcentration factors calculated for daphnids exposed to arsenic+3 (at 1,000 and 100 ug/L) were 50 and 219, respectively [445].

Although arsenic is bioconcentrated by aquatic organisms, it does not appear to biomagnify up the food chain (Woolson, 1975). In a recent study, Maeda et al. (1990) investigated the accumulation of arsenic in a three-step freshwater food chain consisting of an autotroph (algae, Chlorella vulgaris), a grazer (zooplankton, Moina

macrocopa), and a carnivore (goldfish, Carassius auratus) [445].

The algae cells were cultured for 14 days in Modified-Detmer medium under aeration containing 30 or 100 ppm atomic arsenic (as sodium arsenate [Na2HAsO4]). Zooplankton in one-tenth diluted Modified-Detmer medium under aeration were fed arsenic-accumulated algae (from the 100 ppm culture) for 7 days; to measure direct arsenic accumulation from water, zooplankton in medium containing 0.1, 1, or 2 ppm were fed arsenic-free bread yeast for 7 days. Juvenile goldfish in one-tenth diluted Modified-Detmer medium under aeration were fed arsenic-accumulated zooplankton for 7 days; to measure direct arsenic accumulation from water, goldfish in medium containing 0.5 or 1 ppm arsenic were fed arsenic-free artificial food for 7 days [445].

Direct accumulation of arsenic from water by algae, zooplankton, and goldfish was correlated to the arsenic concentration in the medium. Tissue concentrations of total arsenic are on a dry-weight basis. The concentrations of arsenic in algae exposed to 30 or 100 ppm arsenic were 745 and 2,850 ppm, respectively. The concentrations of arsenic in zooplankton exposed to 0.1, 1, or 2 ppm arsenic were 9.5, 10.3, and 17.9 ppm, respectively. The concentrations of arsenic in goldfish exposed to 0.5 or 1 ppm were 33.2 and 51.3 ppm, respectively. The arsenic accumulation from food by zooplankton was increased about one order of magnitude over that from water (225 ppm); however, the arsenic accumulated in goldfish via the food chain was relatively low (37.0 ppm). Thus, in this freshwater food chain, the total arsenic accumulated decreased one order of magnitude [445].

Naqvi et al. (1990) conducted a study to evaluate accumulation of arsenic by the American red crayfish (Procambarus clarkii). Crayfish were exposed to 3 different concentrations (0.5, 5, and 50 ppm) of monosodium methanearsonate (MSMA) herbicide for a period of 8 weeks in water of pH 7.1-7.8 and total hardness of 32 ppm. Arsenic uptake by crayfish (whole-body) during the 8 weeks of exposure was dose-dependent but not time-dependent. The respective ranges of arsenic uptake at 0.5, 5, or 50 ppm exposure concentrations were 0.23-1.36, 1.28-4.29, and 2.81-9.02 ppm. Most of the arsenic accumulated during the uptake period was rapidly lost within the first two weeks of depuration and continued to be depurated thereafter [445].

Sorensen et al. (1979) exposed green sunfish (Lepomis cyanellus) to arsenic (as sodium arsenate) in order to correlate arsenic accumulation, tissue distribution and cytotoxicity. Exposure times were 2, 4, or 6 days to 60

ppm arsenic at 20 degrees C. (Refer to "Toxicity" for kidney and liver cytotoxicity evaluation.) The gall bladder and bile (combined) accumulated the highest concentrations of arsenic. The arsenic concentrations increased from 35 to 78 to 159 ppm (fresh weight) during the 2-, 4-, and 6-day exposures, respectively. The liver, spleen, and kidney also accumulated more arsenic as exposure time increased, reaching 47.7, 18.9, and 14.2 ppm (fresh weight) arsenic by 6 days, respectively. Ovaries and testes were not observed to accumulate arsenic to significantly different concentrations during the 6-day exposure period; their respective arsenic residues were 8.5 and 2.3 ppm at day 6. A noticeable decrease in arsenic residues of gill tissue was observed; at day 2, the gill arsenic concentration was 6.8 ppm and by day 6, it had dropped to 3.8 ppm [445].

Bluegills (Lepomis macrochirus) were exposed to varying sublethal concentrations of sodium arsenite in outdoor pools (Gilderhus, 1966). The pools were filled with well water (total hardness 310 ppm); temperature 60-83 degrees F, depending on outdoor temperature) and soil (silt loam type) was spread on the bottom of each pool to support aquatic plants and bottom fauna. Nine pools were treated as follows: once at the start of the experiment (4.0, 1.2, or 0.4 ppm sodium arsenite); monthly for 4 months (1.2 or 0.4 ppm); weekly for 16 weeks (1.2, 0.4, or 0.04 ppm); and no treatment (control). Residues of arsenic increased in fish during the season in all treated pools, with the highest concentrations detected in the 16-week samples. In pools that were treated only once, the arsenic concentrations in the fish increased as the concentration in the water decreased. In all but one pool, the fish arsenic concentration was higher than the water arsenic concentration at the end of 16 weeks. Immature and adult bluegills sampled at the same time contained approximately equal concentrations of arsenic. Residues (ppm, dry weight) in fish from the pool treated only once at 4.0 ppm were as follows: flesh, 1.3; skin and scales, 2.4; gills and digestive tract, 17.6; liver, 11.6; kidney, 5.9; and ovaries, 8.4 [445]. (For discussion of survival and other effects on fish and bottom organisms, refer to "Toxicity").

Arsenic accumulation in rainbow trout resulted from long-term dietary exposure to sodium arsenite ($NaAsO_2$) (Oladimeji et al., 1984). Supplemented diets contained 0, 10, 20, or 30 mg arsenic/kg (ppm, dry weight) and exposure time for each treatment was 2, 4, 6, or 8 weeks. (Refer to "Toxicity" for discussion of long-term toxic effects.) The pattern of arsenic accumulation in liver, skin, gill, and muscle did not always correlate with exposure concentration for each exposure period. In order to compare arsenic residues in each tissue, the final 8-week residue concentration (ug/g [ppm], dry

weight) is given here unless stated otherwise. Arsenic residues for the 10, 20, and 30 mg/kg diet groups in liver were 1.55, 3.41, and 5.21, respectively; in skin, arsenic residues were 1.21, 1.45, and 1.98 respectively; and in muscle, arsenic residues were 1.28, 1.28, and 1.52, respectively. After 2 weeks at 10 or 30 mg/kg dietary exposure, residues in gill reached maximum levels of 2.80 and 3.37, respectively, then declined thereafter at 8 weeks to 0.84 and 1.88, respectively. However, at 20 mg/kg dietary exposure, arsenic residues in gill varied over time and reached 1.71 after 8 weeks [445].

Arsenic was found to accumulate in the livers of adult chickens fed a diet of algae harvested from waste-water ponds (Yannai, 1979). In this study, chickens were raised for 7 weeks on a ration containing 15% dried sewage- grown algae. Arsenic concentrations in 3 different species of algae ranged from 1.1 to 3.6 ppm (dry weight). The arsenic concentration in control chicken livers was in the range of 0.5 ppm; comparatively, arsenic in the livers of chickens grown on two of the three algae diets ranged from 1.07 to 1.46 ppm [445].

Dietary exposure of mallard ducklings to arsenate resulted in significant arsenic accumulation in the liver and brain (Camardese et al., 1990). Arsenic concentrations in livers of ducklings exposed to 100 or 300 ppm dietary arsenic (as sodium arsenate) were 0.3 and 1.3 ppm (or 3 and 13 times the concentration found in unsupplemented controls), respectively. Arsenic concentrations in brains of ducklings exposed to 100 or 300 ppm dietary arsenic were 0.4 and 0.8 ppm (or 4 and 8 times the concentration found in controls), respectively. (Refer to "Toxicity" for experimental design and discussion of toxic effects) [445].

Interactions:

Eisler (in Niragu et al.) discussed essentiality, synergism, and antagonistic interaction of arsenic and other chemicals in 1994 [654]. Selenium and arsenic appear to be antagonistic in many animals [654].

Arsenate in the normal protein diet appeared to protect ducklings from exposure to high concentrations (60 ppm) of selenium-2, reducing mortality from 40% to 0%. This protective effect was less pronounced when the dietary protein level was low [445].

Information from HSDB [366]:

When selenium is injected almost simultaneously with arsenic into test animals biliary excretion of both elements is enhanced seven to tenfold. [Nat'l Research Council Canada; Effect of Arsenic in the Canadian Environment p.215 (1978)

NRCC No.15391].

The toxicity of 3 doses of a mixture of 10 heavy metals arsenic, cadmium, chromium, copper, iron, lead, mercury, nickel, selenium, and zinc at 0.5, 1, or 2 fold the maximum recommended concn to size fractionated natural phytoplankton from the North American Great Lakes was determined. [Munawar M et al; Ergeb Limnol 25: 123-39 (1987)].

The effects of selenium and arsenic on tumor size and tumor number were examined in mice using the urethane pulmonary adenoma model. Female Swiss cross mice were administered 3 ug/ml selenium and 80 ug/ml arsenic in drinking water on alternate days for 15 weeks. Urethane was administered 3 weeks post metal treatment; the incidence and size of pulmonary adenomas were determined after 12 weeks. Weight gain was diminished in mice exposed to arsenic but not selenium; no other clinical signs were seen. [Blakley BR; Drug Nutrient Interact 5 (2): 97-102 (1987)].

Specific hazards associated with painting, printmaking, photography, ceramics, and sculpturing are discussed. The major dangers associated with painting are from toxic pigments that contain lead, arsenic, chromium, mercury, and solvents. Hazards associated with printmaking include exposure to solvent vapors in silk screening, corrosive chemicals in plate etching, and dust inhalation in lithography. Photographic developers irritate the skin, eyes, and respiratory tract. Many are sensitizers, but others produce systemic effects if inhaled or ingested. Ceramic artists are exposed to hazards related to dry clays, glazes, kiln use; ingestion and inhalation are the important routes of exposures. Sculptors are exposed to fumes in welding of steel structures, sensitizing epoxy resins in plastic sculptors, and dust, solvent and preservatives in wood sculpturing. Recommended control measures for preventing exposure to health and safety hazards in the arts are listed. [Hart C; J Environ Health 49 (5): 282-7 (1987)].

The interactions of tobacco smoking with exposure to occupational chemicals were reviewed. ... A multiplicative interaction was found in one, and an intermediate interaction between additive and multiplicative in two studies of arsenic exposure in copper smelters. ... [Saracci R; Epidemiologic Reviews 9: 175-93 (1987)].

The effects of selenium and arsenic on tumor size and tumor number were examined in mice using the urethane pulmonary adenoma model. Female Swiss cross mice were administered the metals in drinking water at levels of 3 ug/ml selenium and 80 ug/ml arsenic on alternate days for 15 weeks. The urethane was administered after 3 weeks of the metal treatment, and the incidence and size of pulmonary adenomas were determined 12 weeks later. Weight gain was diminished in mice exposed to arsenic but not selenium. No other clinical signs were present

due to metal exposure. Urethane induced sleeping times were significantly reduced in animals given both metals relative to those administered either arsenic or selenium. Both arsenic and selenium administered alone reduced tumor size; the effect of arsenic was greater than that of selenium and arsenic treatment also resulted in a decreased number of tumors per animal. No interactive effects between the metals were determined with regard to tumor production. Both arsenic and selenium alter urethane induced adenoma formation. [Blakely BR; Drug-Nutrient Interactions 5 (2): 97-102 (1987)].

Uses/Sources:

See also additional information regarding sources in Forms/Preparations/Formulations section below.

Of special interest in the National Park Service, due to fact that there are so many civil war battlefield sites, is the fact that arsenic was heavily used as a primary active ingredient in embalming fluids from the time of the civil war until about 1910 [976]. A popular formula contained about four ounces of arsenious acid (an arsenic trioxide) per gallon of water, and up to 12 pounds of non-degradable arsenic was sometimes used per body [976].

Arsenic compounds have applications as animal feed additives, veterinary medicines, pharmaceuticals, fungicides, herbicides, corrosion inhibitors, tanning agents, and wood preservative [483].

Arsenic compounds are used or found in the following industries: agriculture, forestry, mining or smelting, glass manufacture, semiconductors, among others [363].

Information on uses from HSDB [366]:

> Alloying constituent, mfr of certain types of glass; in metallurgy for hardening copper, lead alloys, to make gallium arsenide for dipoles & other electronic devices; doping agent in germanium & silicon solid state products; special solders; medicine, component of alloys; component of electrical devices, medication: to mfr arsenical org cmpd for therapeutic use, As radioactive tracer in toxicology, Used as a catalyst in the manufacture of ethylene oxide, and in semiconductor devices. Arsenic is one of the metals used by ceramic artists; the others are lead, antimony, arsenic, barium, beryllium, boron, chromium, cobalt, cadmium, copper, and vanadium.

Arsenic is ubiquitous in the biosphere. It is present in one form or another in air, water, soil, and living organisms [445].

Wine produced from grapes grown in vineyards treated with an arsenical pesticide has been postulated to cause arsenic poisoning in at least one case [363]. Criminal activity should always be suspected when arsenic poisoning is diagnosed [363].

Plants take up arsenic from soil, groundwater, sewage sludge, biocides, fertilizers and air pollution [83]. Worldwide, perhaps 30% comes from weathering of soils [196]. Animals take up arsenic from drugs, biocides, industrial sources, contaminated water, and

contaminated food [83]. Arsenic enters rivers from air pollution (fossil fuel combustion) and soil erosion as well as from pesticides and industrial sources.

Arsenic Pesticides include:

> Arsenic trioxide, sodium arsenite, calcium arsenite, copper acetoarsenite, copper arsenite, arsenic acid, lead arsenate, cacodylic acid, arsine (can be produced during manufacture of arsenical pesticides), sodium arsenate, calcium arsenate, zinc arsenate, methane arsenic acid (MAA), monosodium methane arsonate (MSMA), disodium methane arsonate (DSMA), monoammonium methane arsonate (MAMA), Calcium acid methane arsonate (CAMA) (Morgan, 1989) [363].
>
> With the exception of Grant's Ant Control Ant Stakes(R) containing 84.1 mg arsenic trioxide per sealed container, other arsenical ant control products are no longer registered by the EPA and are not sold [363].

Significant amounts of arsenic are known to leach from municipal landfills [46]. Pesticides are an additional source of arsenic in water [57]. Arsenic is produced as a by-product of zinc, copper, and lead smelters--and possibly also produced through the large-scale burning of coal--poisons both livestock and humans [335].

In Iran, an arsenic sulfide (AS2S3) compound with calcium oxide and starch is mixed with water to form calcium hydroxide, and then used as a depilatory [363].

Arsenic is normally found in surface waters as an industrial pollutant or a product of agricultural runoff [424]. However, arsenic is found widely in nature and most abundantly in sulfide ores [366]. Arsenopyrite (FeAsS) is the most abundant one [366]. Arsenic compounds in nature may be organic or inorganic but occur mostly as arsenides and arsenopyrites [375]. Arsenicals are used widely in industry and are a constituent of herbicides used in forest management and agriculture [302,129].

Information from Environment Canada [604]:

> Arsenic is present naturally in the aquatic and terrestrial environments from weathering and erosion of rock and soil. In areas of arsenic-enriched bedrock, background concentrations can be significantly elevated. In Canada, for example, large amounts of arsenic have been reported in soil, sediment and water in the vicinity of arsenic-bearing precious metal deposits near Cobalt, Ontario, and Halifax, Nova Scotia [604].
>
> Arsenic is released naturally into the atmosphere by volcanic eruptions and the escape of volatile methylarsines from soil. Atmospheric releases from one of the two rosters (arsenic is produced mainly as arsenic trioxide through the roasting of arsenious gold mines)

currently operating in Canada, located in Yellowknife, NWT (Northwest Territories), are about 8.8 tons of arsenic per year. Some arsenic (both organic and inorganic forms) can also be lost to the atmosphere as a result of production of volatile methylarsines [604].

Significant amounts of arsenic are released in liquid effluent from Canadian gold-milling operations using cyanide, as well as in stack gases from roasting of gold ores. In 1972, about 1750 tons of arsenic were emitted into the Canadian atmosphere by four gold-ore roasters. Gold mining activities in Nova Scotia have been reported to contribute to high arsenic levels in local ground waters [604].

Weathering of acidic mill tailings, as well as waste rock and mine workings, can also result in the release of arsenic, especially at abandoned base and precious metal mine sites where leachates are not treated [604].

Other anthropogenic sources of arsenic include the use of arsenical pesticides in fruit and vegetable production prior to 1975 and in wood preservation, coal-fired power generation, and disposal of domestic and industrial wastes. High arsenic concentrations (up to 11,000 ug/L) were also detected in ground water in the vicinity of an abandoned arsenical wood preservative facility near Vancouver, British Columbia [604].

Information on sources of arsenic in fish [366]:

Present, background, and anthropogenic loading rates of copper, nickel, zinc, lead, cadmium, chromium, mercury, arsenic, and selenium to lake sediments were calculated, and compared to concentrations in several fishes. ... The majority of lakes had anthropogenic loadings of zinc, cadmium, mercury, and arsenic, which were presently 1.8-2.6 times background loadings. ... Enrichment by zinc, cadmium, arsenic, and especially lead was greater closer to industrialized regions. Anthropogenic and precipitation loadings for zinc, lead, cadmium, and arsenic were similar, suggesting that anthropogenic inputs are atmospheric and that current atmospheric loadings are mostly anthropogenic. ... [Johnson MG; Can J Fish Aquat SCI 44 (1): 3-13 (1987)].

HOMEOPATHIC MEDICINES have been found to contain arsenic in the following concentrations [363]:

ARSENIC IN HOMEOPATHIC PREPARATIONS:

Preparation	Calculated Arsenic*	Measured Arsenic
Alpha Homeopathic	757 mcg/g	323 mcg/g

Remedy 38 3x		286 mcg/g
Hyland's Homeopathic 555 6x	0.757 mcg/g	0.680 mcg/g
Hyland's Homeopathic Arsenicum Album 3x	757 mcg/g	623 mcg/g 650 mcg/g
Luyties Arsenicum Homeopathic 12x	0.000757 mcg/g	0.005 mcg/g
Natra-Bio 519 Hay Fever 6x	0.757 mcg/mL	0.024 mcg/mL
Pain Eased 21 Natural Homeopathic Formula 12x	0.000757 mcg/mL	0.006 mcg/mL

NOTE: * = Expected arsenic is calculated on the assumption that the homeopathic preparations contain the stated amount as arsenic trioxide (As2O3) (elemental arsenic content of arsenic trioxide is 75.7%) [363].

Additional Information from 1990 report entitled: "Fish and Wildlife Resources and Agricultural Drainage in the San Joaquin Valley, California," quoted word for word with the permission of Senior Author Stephen Moore, Fish and Wildlife Service, Portland Oregon, Regional Office (see original publication for embedded references) [445]:

> Arsenic is found in high concentrations in sulfide deposits, where it is present as the native element, in combination with minerals (as an alloy, arsenide, sulfide, or sulfosalt), or as the sulfide of arsenic with metals such as copper, lead, silver, and thallium; oxidation products of the foregoing compounds are also found (NRC-Committee on Medical and Biological effects of Environmental Pollutants, 1977). Major arsenic-containing minerals are arsenopyrite (FeAsS), realgar (As4S4), orpiment (As2S3), and enargite (Cu3AsS4) (Tamaki and Frankenberger, Mar 1989; UNEP et al., 1981). High concentrations of arsenic may also occur in some coals (UNEP et al., 1981; Adriano, 1983) [445].
>
> Arsenic is present in all soils and originates primarily from rocks and minerals weathered to form that soil (NRC of Canada, 1978). Thus, the geologic history of a particular soil determines its native arsenic content (NRC-Committee on Medical and Biological Effects of Environmental Pollutants, 1977; Adriano, 1983). Arsenic in soils on the west side of the San Joaquin Valley probably originated in the Coast Ranges (Tidball et al., 1989) which were derived from marine sedimentary parent material. The natural arsenic content in soils seldom exceeds 10 ppm (Adriano, 1983). However, soils overlaying sulfide ore deposits usually contain arsenic at several hundred ppm, the reported average being 126 ppm and the range being 2-8,000 ppm (NRC-Committee on Medical and Biological Effects of Environmental Pollutants, 1977; NRC of Canada, 1978; Adriano, 1983).

Unnaturally high levels of arsenic can also occur in soils where arsenic pesticides, herbicides, or defoliants were repeatedly used for agricultural purposes and in soils which receive fallout from ore smelting and fossil fuel combustion (NRC of Canada, 1978) [445].

Arsenic has been found in many natural waters including seawater, hot springs, ground water, rivers, and lakes (Lemmo et al., 1983). The concentrations of arsenic generally average less than 10 ppb (NRC of Canada, 1978). The concentration of arsenic in freshwaters shows considerable variation with the geologic composition of the drainage area and the extent of anthropogenic input (Cullen and Reimer, 1989). Arsenic concentrations in waters of the San Luis Drain and Kesterson Reservoir were <1-2 ug/l (ppb) and <1-2 ug/l, respectively (USBR, Oct 1986). Arsenic concentrations are greater in waters of the Tulare Basin than in the San Joaquin Basin. For example, median (minimum-maximum) dissolved and total waterborne concentrations of arsenic in subsurface agricultural drainage water inflow to the Tulare Lake Drainage District South Evaporation Ponds were 79.5 (11-110) and 97 (64-190) ug/L (ppb), respectively (Fujii, 1988).

Forms/Preparations/Formulations:

Four arsenic species common in natural waters are arsenate (+5), arsenite (+3), methanearsonic acid and dimethylarsinic acid [375]. Biomethylation can form organic arsenicals [488]. The chemical and toxicological properties of the forms appear to be quite different, so some recommend that the toxicities of these forms should be treated separately [375]. Arsenic III [arsenite (III)] tends to be more toxic than arsenic V [arsenate (V)] [483]

Recent studies suggest that arsenic in ground water is present mainly as inorganic As(III) and As(V) species [604]. Most, > or = to 80%, of the arsenic in contaminated waters as well as in surface waters remote from point sources, is expected to be present as inorganic As(V) and to a lesser extent As (III) species [604].

Arsenobetaine is the principle arsenic compound in marine animals [604]. In freshwater aquatic plants, arsenic is present mainly as lipid and water-soluble, "lipid-related" compounds; lesser amounts of arsenite and methylated As(V) species are also present. Although little is known about the behavior of arsenicals in terrestrial plants, methylation has been reported in some plants grown in nitrate- or phosphate deficient conditions. Arsenosugars and arsenic containing lipid compounds, as well as methylated arsenicals, have been found in marine plants [604].

Limited data on arsenic speciation in plant and aquatic-animal tissue suggest that most arsenic is in the form of organo-arsenic compounds; however, a small amount (<1-30%) may be present as inorganic As(III) [604].

Additional Information on arsenic forms from 1990 report entitled: "Fish and Wildlife Resources and Agricultural

Drainage in the San Joaquin Valley, California," quoted word for word with the permission of Senior Author Stephen Moore, Fish and Wildlife Service, Portland Oregon, Regional Office (see original publication for embedded references) [445]:

> Chemical Forms: The chemistry of arsenic is complex. Arsenic is classified as a metalloid, but it exhibits both metallic and non-metallic properties (Phillips, 1990). The four oxidation states in which arsenic forms inorganic compounds are +5, +3, 0, and -3. When found in the natural environment, the elemental form of arsenic (As0) occurs in three colors: gray, yellow, or black (Dickerson, 1980). Arsenate (As+5) is the main species found in oxidizing environments, such as in unflooded, aerobic soils (Wauchope, 1983); mildly reducing conditions favor the chemical form of arsenite (As+3) (Ferguson and Gavis, 1972). The highly reduced state (As-3) is found mainly as arsine (AsH3) (Lemmo et al., 1983). In general, though, interchanges in valence state may occur in water solutions depending on the pH and the presence of other substances which can be reduced or oxidized (Ishinishi et al., 1986) [445].
>
> Arsenic covalently bonds carbon, hydrogen, oxygen, and sulfur (Ferguson and Gavis, 1972). In anaerobic sediments and waters containing hydrogen sulfide, arsenic sulfides precipitate and thus remove arsenic from the water column (NRC-Committee on Medical and Biological Effects of Environmental Pollutants, 1977). A large variety of organic arsenic compounds are made possible by the ability of the arsenic atom to bond from one to five organic groups, aromatic or aliphatic (NRC-Committee on Medical and Biological Effects of Environmental Pollutants, 1977). Valences not used in bonding organic groups can be linked to other atoms, for example halogens [445].
>
> Arsenic+5 can form relatively insoluble metallic salts with a number of cations (e.g., arsenates of aluminum, calcium, copper, iron, nickel, lead, magnesium, and zinc) (Lemmo et al., 1983; Dickerson, 1980). Also known are metallic arsenites of the formulas MH2AsO3, M2HAsO3, and M3AsO3, where M represents a univalent metal cation or one equivalent of a multivalent cation (NRC-Committee on Medical and Biological Effects of Environmental Pollutants, 1977) [445].
>
> There are many arsenic compounds of environmental importance, including: arsenic trioxide (aka arsenous acid [As2O3]) and arsenic pentoxide (As2O5), arsenic acid (AsO(OH)3), salts of arsenous acid (e.g., arsenites [HAsO3-2]), salts of arsenic acid (e.g., arsenates [HAsO4-2]), methylarsonic acid (CH3AsO(OH)2), dimethylarsinic acid (aka cacodylic acid [(CH3)2AsO(OH)]), and arsanilic acid (C6H8AsNO3) (NRC-

Committee on Medical and Biological Effects of Environmental Pollutants, 1977). Arsenic trioxide is the primary product of arsenic smelters (NRC-Committee on Medical and Biological Effects of Environmental Pollutants, 1977; Dickerson, 1980). It is only slightly soluble in water and once in the general environment, arsenic trioxide undergoes oxidation, reduction, methylation, and demethylation (Dickerson, 1980). Oxidation of elemental arsenic or arsenic trioxide yields arsenic pentoxide, which is very soluble in water. Methylated arsenic compounds are derived from arsenic acid by replacing one or more of the hydroxyl groups with a methyl group (Lemmo et al., 1983) [445].

Chem.Detail: Detailed Information on Chemical/Physical Properties:

Physical and Chemical Properties: The chemical and physical properties of the arsenic species of chief toxicological and environmental concern are sufficiently well characterized to allow estimation of the environmental fates of these compounds [716]. However, more information regarding the K ow and K oc values of the organic arsenicals would help predict the fate of these compounds in the environment [716].

Solubilities [366]:

Sol in nitric acid; insol (sic) in water [Weast, R.C. (ed.) Handbook of Chemistry and Physics, 68th ed. Boca Raton, Florida: CRC Press Inc., 1987-1988.,p. B-73].

Note: "INSOL" really means "relatively insoluble," and the exact solubility usually depends on chemical speciation and other details.

Insol in caustic and nonoxidizing acids [Sax, N.I. and R.J. Lewis, Sr. (eds.). Hawley's Condensed Chemical Dictionary. 11th ed. New York: Van Nostrand Reinhold Co., 1987. 98].

Vapor Pressure [366]:

1 mm Hg at 372 deg C; 10 mm Hg at 437 deg C; 40 mm Hg at 483 deg C; 100 mm Hg at 518 deg C; 400 mm Hg at 579 deg C [Weast, R.C. (ed.) Handbook of Chemistry and Physics, 68th ed. Boca Raton, Florida: CRC Press Inc., 1987-1988.,p. D-192].

Density/Specific Gravity [366]:

5.727 @ 14 DEG C [Weast, R.C. (ed.) Handbook of Chemistry and Physics, 68th ed. Boca Raton, Florida: CRC Press Inc., 1987-1988.,p. B-74].

Melting Point [366]:

817 DEG C @ 28 ATM [Weast, R.C. (ed.) Handbook of Chemistry and Physics, 68th ed. Boca Raton, Florida: CRC Press Inc.,

1987-1988.,p. B-73].

Molecular Weight [366]:

74.92 [Weast, R.C. (ed.) Handbook of Chemistry and Physics, 68th ed. Boca Raton, Florida: CRC Press Inc., 1987-1988.,p. B-73].

Critical Temperature and Pressure [366]:

Critical temp: 1673 deg K; Critical pressure: 22.3 MPa [Weast, R.C. (ed.) Handbook of Chemistry and Physics, 68th ed. Boca Raton, Florida: CRC Press Inc., 1987-1988.,p. F-64].

Heat of Vaporization [366]:

11.2 KCAL/G-ATOM [The Merck Index. 10th ed. Rahway, New Jersey: Merck Co., Inc., 1983. 116].

Color/Form [366]:

A silver-grey brittle, crystalline, metallic-looking substance [International Labour Office. Encyclopedia of Occupational Health and Safety. Vols. I&II. Geneva, Switzerland: International Labour Office, 1983. 179].

It exists in three allotropic forms, the yellow (alpha), black (beta) and grey (gamma) forms [International Labour Office. Encyclopedia of Occupational Health and Safety. Volumes I and II. New York: McGraw-Hill Book Co., 1971. 115].

Hexagonal, rhombic, gray, metallic [Weast, R.C. (ed.) Handbook of Chemistry and Physics, 68th ed. Boca Raton, Florida: CRC Press Inc., 1987-1988.,p. B-73].

Odor [366]:

Odorless [Gosselin, R.E., R.P. Smith, H.C. Hodge. Clinical Toxicology of Commercial Products. 5th ed. Baltimore: Williams and Wilkins, 1984.,p. III-42].

Taste [366]:

Nearly tasteless [Gosselin, R.E., R.P. Smith, H.C. Hodge. Clinical Toxicology of Commercial Products. 5th ed. Baltimore: Williams and Wilkins, 1984.,p. III-42].

Other Chemical/Physical Properties [366]:

A yellow modification which has no metallic properties is obtained by sudden cooling of arsenic-vapor. This yellow arsenic is converted back to the gray modification upon very short exposure to ultraviolet light. [The Merck Index. 10th ed. Rahway, New Jersey: Merck Co., Inc., 1983. 116].

Vaporization becomes apparent at 100 deg c and is already rapid at 450 deg c; brinell hardness: 147; mohs' scale: 3.5; Heat of sublimation: 30.5 Kcal/g-atom; 7.63 Kcal/g-atom; specific heat: 0.0822 For 0 deg c to 100 deg c; heat of fusion: 22.4 Kcal/g-atom; 6.620 Kcal/g-atom; not attacked by cold sulfuric acid or hydrochloric acid; converted by hno3 or hot h2so4 into arsenous or arsenic acid; dielectric constant: 10.23 @ 20 Deg c & 60 cycles [The Merck Index. 10th ed. Rahway, New Jersey: Merck Co., Inc., 1983. 116].

613 deg C (sublimes) [Weast, R.C. (ed.) Handbook of Chemistry and Physics, 68th ed. Boca Raton, Florida: CRC Press Inc., 1987-1988.,p. B-73].

Fate.Detail: Detailed Information on Fate, Transport, Persistence, and/or Pathways:

Aquatic Fate of Arsenic: Arsenic as a free element (0-oxidation state) is rarely encountered in natural waters. Soluble inorganic arsenate (+5-oxidation state) predominates under normal conditions since it is thermodynamically more stable in water than arsenite (+3 oxidation state). [USEPA; Ambient Water Quality Criteria Doc: Arsenic p.A-1 (1980) EPA 440/5-80-021] [366].

Soils can be contaminated with inorganic arsenic or methylated arsenical contained in herbicides [604]. Methylated arsenical are eventually degraded (half-lives = 0.5 to 2.9 years) to carbon dioxide and arsenate by soil microorganisms [604].

Additional information on arsenic fate from 1990 report entitled: "Fish and Wildlife Resources and Agricultural Drainage in the San Joaquin Valley, California," quoted word for word with the permission of Senior Author Stephen Moore, Fish and Wildlife Service, Portland Oregon, Regional Office (see original publication for embedded references) [445]:

> The estimated ratio of natural to anthropogenic inputs of arsenic into the atmosphere is 60:40. Copper smelting accounts for 40% of the anthropogenic input, while low temperature volatilization and volcanic activity account for 97% of natural emissions (Chilvers and Peterson, 1987). In addition, biological reduction of arsenicals by microorganisms in soil and water releases volatile arsines (e.g., dimethylarsine [(CH3)2AsH] and trimethylarsine [(CH3)3As]) to the atmosphere (NRC of Canada, 1978). Most atmospheric arsenic exists as dispersed fine particulate matter, composed primarily of inorganic arsenic oxides and arsenates (NRC of Canada, 1978). Arsenic is removed from the air as the oxide dust settles or is washed out by rain water (Dickerson, 1980) [445].
>
> The precipitation of naturally occurring arsenic in the atmosphere contributes relatively less arsenic to the aquatic environment than natural weathering of crustal rocks (NRC of Canada, 1978). The aquatic environment

also receives a large amount of arsenic from many different anthropogenic sources. Ferguson and Gavis (1972) estimated that the annual anthropogenic input to surface waters is nearly 3 times that contributed from natural weathering. Arsenic generally remains in low concentrations in natural waters because of its ability to adsorb onto clays, coprecipitate with hydrous iron oxide, or bind with sulfide in reduced bottom mud (Hem, 1985). Inorganic forms of arsenic prevail in most natural waters (NRC of Canada, 1978) and rivers seem to cleanse themselves of soluble arsenic (Dickerson, 1980). Organic forms can also be present as a result of microbial transformation, but they are usually volatilized to the air or oxidized back to oxides which readsorb to the sediment (Woolson, 1983) [445].

Arsenic mobility in the soil is affected by natural processes and by changes induced by anthropogenic activities. Arsenic is volatilized from the soil as arsine, which is produced through chemical reduction by soil microorganisms. Arsenic is also lost from surface soils through leaching. The amount removed by leaching is related to the solubility of arsenic, which is greater in sandy or low-clay soils; the solubility of arsenic is reduced by the adsorption of arsenic onto organic matter and charged surfaces of clays, and the binding of arsenic to metallic compounds. Additionally, deep plowing of contaminated soils can dilute the surface content and expose arsenic to additional sites for fixation (NRC of Canada, 1978) [445].

In general, organisms can have a significant influence on the distribution of arsenic in the environment by accumulating, transporting, and transforming it. Some of the transformations, such as oxidation and reduction, are probably catalyzed by the presence of organisms, but occur in their absence; other processes, such as methylation, occur only in the presence of organisms (Ferguson and Gavis, 1972)[445].

In three River Nile coastal lakes in Egypt (areas away from pollution sources), As(V) is the predominant dissolved As species constituting between 85 and 95% of total dissolved As (TDAs). Near local sewage discharge points As(III) appeared constituting between 14 and 33% (of TDAs) at low and high water discharge periods. Dimethylarsenic is the dominant organic As form reaching 22% of total dissolved As while the maximum concentration of monomethylarsenic (1.0 mu g L super(-1)) constituted about 8% of TDAs. Particulate As is mostly partitioned among reducible and detrital phases while the organic phase appeared dominant in eutrophic areas (> 30% of particulate As). About 413 t yr super(-1) As entered the northern delta lakes via agricultural drains and waste water discharge. Phosphate fertilizers,

detergents, herbicides, loamy Nile deposits are the main As sources to the drainage system. 52% of the total As derived to the Nile delta lakes is transported to the Mediterranean coastal seawater through the lakes' inlets (Abdel-moati, A.R. 1990. Speciation and behavior of arsenic in the Nile Delta lakes. Water Air Soil Pollut. 51:107-132).

Laboratory and/or Field Analyses:

Many methods have been used to monitor for arsenic [716,861,1001,1003,1004,1005,1006]. EPA methods recommended depend on the application: whether for drinking water, NPDES permits, CERCLA, RCRA, or water-quality based permitting [861,1001,1003,1004,1005,1006, 40 CFR 136 and 40 CFR Part 141]. If one simply wants to know whether or not the concentration exceeds criteria or benchmarks for fish and wildlife, it is not always too clear which "EPA standard method" is optimum, although some might argue that the new EPA methods 1632 and 1669 (see details below) should apply.

Low concentration criteria and benchmarks (especially those for cancer) usually require that either Hydride Generation or Graphite Furnace Atomic Absorption (AA) be used with lower detection limits than ICP analyses would allow. Detection limits should be no higher than comparison benchmarks or criteria for various media (water, sediments, soil, tissues, etc), some of which are low (see sections above). Otherwise, the detection limits should usually not exceed the following default concentrations often recommended by the Fish and Wildlife Service and the National Park Service: 0.50 ppm dry weight in tissues, sediments, and soils, 0.005 ppm (mg/L) in water (Roy Irwin, National Park Service, Personal Communication, 1996).

If needed for comparison with low benchmarks or criteria, lab detection limits can be as low as:

0.1 ppm in solids [716].

2 ng/L (ppt) [716,1003] for water. For arsenic analyses using EPA lab method 1632, EPA recommends a detection limit of 0.002 ug/L using a hydride AA technique; the lowest EPA water quality criterion is 0.018 ug/L [1001].

In another place, EPA states the method detection limit (MDL; 40 CFR 136, Appendix B) for total inorganic arsenic has been determined to be 3 ng/L for water when no background elements or interferences are present [1003]. Low detection levels are sometimes needed since ambient water quality criteria are as low as 18 ng/L [1003].

Acceptable containers (after proper cleaning per EPA protocols) for Antimony, Arsenic, Cadmium, Copper, Lead, Nickel, Selenium, Silver, Thallium, and Zinc: 500-mL or 1-L fluoropolymer, conventional or linear polyethylene, polycarbonate, or polypropylene containers with lid [1003].

Drinking water methods have included: Atomic

absorption/furnace technique (EPA 206.2; SM 304); atomic absorption/gaseous hydride (EPA 206.3; SM 303E; ASTM D-2972-78B) [893].

Hair analyses: Hair samples collected from common hare (Lepus europaeus), common vole (Microtus arvallis), and wood mouse (Apodemus sylvaticus) were subjected to instrumental neutron activation analysis (INAA). Up to 18 elements (arsenic, gold, bromine, cesium, cobalt, chromium, copper, iron, mercury, potassium, lanthanum, sodium, antimony, scandium, selenium, samarium, thorium and zinc) were detected in each hair sample. Animal hair samples from areas polluted by thermal power plants burning coal were taken and compared with hair samples from the animals living in relatively nonpolluted control areas. Animal hair samples from areas with higher levels of pollution contain usually higher concn of toxic and essential elements as As, Co, Cr, Fe, and Se. Muride rodents can be used for more detailed monitoring of environmental exposure than the hare. Moreover, the hair of the common vole shows usually highest levels of contamination as compared with the wood mouse, which could be explained by different components of feed. Animal hair was a rather sensitive indicator of environmental exposure and INAA proved to be a suitable analytical tool for this purpose (Obrusnik I, Paukert J; J Radioanal Nucl Chem 83 (2): 397-406 (1984)[940].

Notes on total vs. acid soluble vs. dissolved metals:

> Although most of the lab tests done to develop water quality criteria and other benchmarks were originally based on "total" values rather than "dissolved" values, the lab settings were typically fairly clean and the numbers generated by the lab tests are therefore often even more comparable to field "dissolved" values than to field "total" values (Glen Suter, Oak Ridge National Lab, Personal Communication, 1995). As of January 1995, the U.S. EPA was recommending that states use dissolved measurements in water quality standards for metals, in concert with recommendations EPA previously made for the Great Lakes [672]. The conversion factor recommended by EPA for converting total recoverable arsenic III to dissolved concentrations in the January 1997 draft EPA Guidelines for 5 year 305(B) assessments was 1.00. However, both total and dissolved concentrations should be checked at new locations before relying on generic conversion factors (Pat Davies, Colorado Division of Wildlife, personal communication, 1997).

Contaminants data from different labs, different states, and different agencies, collected by different people, are often not very comparable (for more discussion, see disclaimer section at the top of this entry). As of 1997, the problem of lack of data comparability (not only for water methods but also for soil, sediment, and tissue methods) between different "standard methods" recommended by different agencies seemed to be getting worse, if anything, rather than better. The trend in quality assurance seemed to be for various agencies, including the EPA and others, to

insist on quality assurance plans for each project. In addition to quality control steps (blanks, duplicates, spikes, etc.), these quality assurance plans call for a step of insuring data comparability [1015,1017]. However, the data comparability step is often not given sufficient consideration. The tendency of agency guidance (such as EPA SW-846 methods and some other new EPA methods for bio-concentratable substances) to allow more and more flexibility to select options at various points along the way, makes it harder in insure data comparability or method validity. Even volunteer monitoring programs are now strongly encouraged to develop and use quality assurance project plans [1015,1017].

At minimum, before using contaminants data from diverse sources, one should determine that field collection methods, detection limits, and lab quality control techniques were acceptable and comparable. The goal is that the analysis in the concentration range of the comparison benchmark concentration should be very precise and accurate.

It should be kept in mind that quality control field and lab blanks and duplicates will not help in the data quality assurance goal as well as intended if one is using a method prone to false negatives. Methods may be prone to quality assurance problems due to the use of detection limits that are too high, the loss or addition of contaminants through inappropriate handling, or the use of inappropriate methods.

Additional discussion on sources of potential variation in contaminants data:

Variation in concentrations of contaminants may sometimes be due to differences in how individual investigators treat samples in the field and lab rather than true differences in environmental concentrations. It was recognition that collectors and labs often contaminate samples that led EPA to develop the 1600 series of water protocols for low detection limit applications [1001,1002,1003,1004]. Contaminants data from different labs, different states are often not very comparable. They may be as different as apples and oranges since:

1) Different Agencies (EPA, USGS, NOAA, and various State Agencies) publish different lab and field protocols. Each of these protocols is different and has typically changed over time.

Note: Even "Standard EPA Methods" which are supposedly widely used by consultants, industry, and academia, have been variable over time and between application category (Drinking Water vs. NPDES, vs. RCRA, vs. CERCLA, vs. Water-Quality Based permits, etc.).

Preservation and other details of various EPA lab and field protocols have changed over the years, just as they have at USGS and various

States and other agencies. USGS data from 30 years ago may be different than USGS data today due to differences (drift) in lab and field protocols rather than differences in environmental concentrations.

2) Independent labs and field investigators are not always using "the latest and greatest methods," and it is difficult for them to keep up with all the changes from various agencies in the midst of their "real world" busy lives. Updates are not always convenient to obtain. For example, EPA changes are scattered through various proposed Federal Register Notices, various updates of CFRs, and numerous publications originating in many different parts of EPA and their contractors. The wording is sometimes imprecise and is often inconsistent between EPA methods for different applications.

3) The details of the way one person collects, filters, and acidifies water samples in the field may be different than the way another does it. Sources of potential variation include the following:

A) The protocol phrases "As soon as practical or as soon as possible." Different situations can change the elapsed time considered by the field collector to be "as soon as practical." It may take different amounts of time to get to a safe or otherwise optimum place to filter and/or acidify and cool the samples. In one case precipitation and other changes could be going on in the collection bottle while the bottle is on the way to filtration and acidification. In other cases, the field collector filters and acidifies the samples within minutes. Weather, safety concerns, and many other factors could play a role.

B) Differences in numerous other details of the method used can drastically change the results. Some cold, wet, hurried, or fire ant-bitten collectors might decide that it is not "practical" to filter and acidify quite so immediately in the field, and may decide the shore, a vehicle, a motel room, or even a remote lab are more "practical" locations. Filtering and acidifying in the field immediately has been thought of as a better option for consistency (see copper and silver entries for examples of what can happen if there is a delay). However, in recent methodology designed to prevent some the

contamination and variability listed above, EPA has recently suggested that waiting until the sample arrives at the lab before acidifying is OK [1003].

C) What kind of .45 micron filter was used? The flat plate filters that were used for years tended to filter .45 micron sizes at first and then smaller and smaller sizes as the filtering proceeded and the filter loaded up with particulate matter. As the filter clogged, the openings grew smaller and colloids and smaller diameter matter began to be trapped on the filter. For this reason, both the USGS and EPA 1600 series protocols have gone to tortuous-path capsule filters that tend to filter .45 micron sizes more reliably over time. Example of specifications from EPA method 1669:

> Filter—0.45-um, 15-mm diameter or larger, tortuous-path capsule filters, Gelman Supor 12175, or equivalent [1003].

D) "Normally 3 mL of (1+1) of nitric acid per liter should be sufficient to preserve the (water) sample" (40 CFR Part 136, Appendix C, pertaining to ICP analyses using method 200.7, 1994 edition of CFR Part 40). Sometimes it is not, depending on alkalinity and other factors. What field collectors sometimes (often?) do is just use pop tabs of 3 mL of nitric acid and hope for the best rather than checking to see that the acidity has been lowered to below a pH of two. EPA CFR guidelines just call for a pH of below two, whereas samples meant to be "acid soluble" metals call for a pH of 1.5 to 2.0 [25]. See also, various USEPA 1984 to 1985 Ambient Water Quality Criteria Documents for individual metals.

> Note: Some shippers will not accept samples with a pH of less than 1 for standard shipping (John Benham, National Parks Service Personal Communication, 1997).

E) One person might use triple distilled concentrated nitric acid rather than reagent grades of acid to avoid possible contamination in the acid, while another may not. When using very low detection limits, some types of acid may introduce contamination and influence the results. Using a 10% dilution of nitric

acid as called for by EPA [1003] is another potential source of contamination, since the dilution water and/or containers may be contaminated. Sometimes people may be incorrectly determining that background concentrations are high due to contamination sources such as these (Pat Davies, Colorado Division of Wildlife, personal communication, 1997).

> Note: Just using triple distilled nitric acid may not be the total answer to potential contamination. The key issue to be sure that the acid used is free of the metals being analyzed. In guidance for EPA method 1669, the use of "ultrapure nitric acid; or Nitric acid, dilute, trace-metal grade" is specified [1003]. In guidance for EPA method 1638, the use of "Nitric acid—concentrated (sp gr 1.41), Seastar or equivalent" is specified [1003].

F) Holding times can strongly influence the results and there can be quite a bit of variation even within EPA recommended 6 month limits (see Silver entry for details). Holding times recommended for EPA for water samples of metals other than mercury or chromium VI have usually been listed as 6 months (Federal Register, Volume 49, No. 209, Friday, October 28, 1984, page 43260). In the 1994 version of the CFR, NPDES holding times for mercury and Chromium VI are the same ones listed in 1984, but no EPA holding times are given for other metals (40 CFR, Part 136.3, Table 2, page 397, 1994). EPA sources stated this was a typo, that no one else brought it to their attention in the last 3 years, that 6 months is still an operable holding time for "other metals" including this one, and that 6 months is actually an artifact from the days when 6 month composite samples were used for NPDES permits rather than having been originally scientifically derived.

> Counterpoint: Although some information suggests that 6 months is probably too long for some contaminants in some scenarios (see silver and copper entries), not all of the information in the literature casts the 6 month metals holding time in such questionable light. In one study, two EPA research chemists found that preservation under certain

conditions of drinking water (EPA Method 200.8) metals samples to a pH of less than 2 effectively stabilized the metal concentrations for 6 months. They found that trace metal standards in the 10 to 50 ug/L concentration could be held in 1% nitric acid if a 5% change of concentration was acceptable [1009]. Some metal concentrations changed more than 5% (Zinc up to 24%, Selenium up to 23%) [1009]. Vanadium, Manganese and Arsenic changed up to 5-7% [1009]. In some of the trials, metals were higher after 6 months due to leaching from containers, while in some they were lower [1009]. The changes were nevertheless considered not of great consequence related to drinking water MCLs and EPA method 200.8 [1009]. However, it is not clear that the careful measures utilized (like rechecking to make sure the pH was less than 2, the use of particular kinds of water samples, the use of particular acids, etc.) in this one study replicates what goes on in day to day ("real world") contaminants lab work around the country.

Some EPA sources state that 6 months should be OK if the sample bottle is vigorously shaken and re-acidified in the lab prior to lab analyses, a practice not universally or even particularly commonly done in labs today. The degree to which a water sample is re-acidified, re-checked for pH, shaken before analysis, and the length of time it sits before and after these steps, seems to vary a lot between laboratories, and EPA guidance for various methods is not consistent. Some labs recheck pH, some don't. Some shake, some don't, etc. For drinking water, preservation is considered complete after the sample is held in pH of less than 2 for at least 16 hours [1007]. New EPA Method 1638 specifies:

> "Store the preserved sample for a minimum of 48 h at 0–4°C to allow the acid to completely dissolve the metal(s) adsorbed on the container walls. The sample pH should be verified as <2 immediately before withdrawing an aliquot for processing or direct analysis. If, for some reason such as high

alkalinity, the sample pH is verified to be >2, more acid must be added and the sample held for sixteen hours until verified to be pH <2" [1003].

For many other methods, the minimum holding time in acid is not stated or is different (see various EPA and other Agency methods).

G) If present, air in head space can cause changes in water sample concentrations (Roy Irwin, National Park Service, Personal Communication, based on several discussions with EPA employees and various lab managers in February 1997).

Note: air from the atmosphere or in headspace can cause oxidation of anaerobic groundwater or anaerobic sediment samples. This oxidation can cause changes in chemical oxidation states of contaminants in the sample, so that the results are not typical of the anaerobic conditions which were present in the environment prior to sampling (John Benham, National Park Service, Personal Communication, 1997).

H) When is the sample shaken in the lab or the field? If the filter is acidified in the field, it will be shaken on the way back to the lab. If lab acidified, how much and when is the sample shaken and then allowed to sit again for various times periods before analyses? Many methods treat this differently, and what many field collectors and labs actually do before analyzing samples is different as well. For EPA method 1638, the word shake appears in the "Alternate total recoverable digestion procedure":

"..Tightly recap the container and shake thoroughly" [1003].

I) If one field filters and acidifies, one often changes metal concentrations and colloidal content compared to samples not treated in this manner. Acidifying effects microbial changes. If one holds the samples a while before filtering and acidifying, the situation changes. In collection bottles, there are potential aging effects: temperature changes, changes in basic water chemistry as

oxygen and other dissolved gasses move from the water into the headspace of air at the top, potential aggregation of colloidal materials, precipitation of greater sizes over time, development of bigger and more colloids, and more sorption (Roy Irwin, National Park Service, personal communication, 1997).

4) The guidance of exactly where to take water samples varies between various state and federal protocols. Taking water samples at the surface microlayer tends to increase concentrations of various contaminants including metals. Other areas of the water column tend to produce different concentrations. Large quantities of anthropogenic substances frequently occur in the surface microlayer at concentrations ranging from 100 to 10,000 times greater than those in the water column [593]. These anthropogenic substances can include plastics, tar lumps, PAHs, chlorinated hydrocarbons, as well as lead, copper, zinc, and nickel [593]. Sometimes a perceived trend can be more the result of the details of the sample micro-location rather than real changes in environmental concentrations (Roy Irwin, National Park Service, personal communication, 1997). The new EPA method 1669 mentions the microlayer, and states that one can use a fluoropolymer closing mechanism, threaded onto the bottle, to open and close a certain type of bottle under water, thereby avoiding surface microlayer contamination [1003]. However, even this relatively new EPA method 1669 also gives recommendations for ways to sample directly at the surface, and does not discourage the use of surface samples.

5) Although the above examples are mostly related to water samples, variability in field and lab methods can also greatly impact contaminant concentrations in tissues, soil, and sediments. Sediment samples from different microhabitats in a river (backwater eddy pools vs. attached bars, vs. detached bars, vs. high gradient riffles vs. low gradient riffles, vs. glides, etc.) tend to have drastically different concentrations of metals as well as very different data variances (Andrew Marcus, Montana State University, personal communication, 1995). Thus, data is only optimally comparable if both data collectors were studying the same mix of microhabitats, a stratified sampling approach which would be unusual when comparing random data from different investigators.

6) Just as there are numerous ways to contaminate, store, ship, and handle water samples, so are there

different agency protocols and many different ways to handle samples from other media. One investigator may use dry ice in the field, another may bury the samples in a large amount of regular ice immediately after collection in the field, while a third might place samples on top of a small amount of ice in a large ice chest. The speed with which samples are chilled can result in different results not only for concentrations of organics, but also for the different chemical species (forms) of metals (Roy Irwin, National Park Service, personal communication, 1997).

7) In comparing contaminants metals data, soil and sediment contaminant concentrations should usually be (but seldom has been) normalized for grain size, total organic carbon, and/or acid volatile sulfides before biologically-meaningful or trend-meaningful comparisons are possible (Roy Irwin, National Park Service, Personal Communication, 1997).

8) There has been tremendous variability in the precautions various investigators have utilized to avoid sample contamination. Contamination from collecting gear, clothes, collecting vehicles, skin, hair, collector's breath, improper or inadequately cleaned sample containers, and countless other sources must carefully be avoided when using methods with very low detection limits [1003].

More Detailed Discussion of Filtration and Acidification of Water Samples:

For ICP water samples for metals, EPA recommends the following (40 CFR Part 136, Appendix C, pertaining to ICP analyses using method 200.7, 1994 edition of CFR Part 40):

1) For samples of "total or total recoverable elements," samples should be acidified to a pH of two or less at the time of collection or as soon as possible thereafter. However, other EPA guidance before and after 1994 has been different:

In previous 1991 guidance for this same method 200.7, one method which has been used for arsenic, EPA stated that if field acidification was not done because of sampling limitations or transport restrictions, that the sample should be acidified upon receipt in the laboratory and held in pH of less than 2 for at least 16 hours prior to analysis [1005].

In more recent (1996) guidance related to the more rigorous method 1669, EPA clarified (some would say confused or added data variability) the issue of when to acidify by stating:

"Preservation recommendations for Antimony, Arsenic, Cadmium, Copper, Lead, Nickel, Selenium, Silver, Thallium, and Zinc: Add 5 mL of 10% HN03 to 1-L sample; preserve on-site or immediately upon laboratory receipt" [1003].

Note: the nitric acid (triple distilled or not?) and dilution water (contaminated or not?) and containers (proper type, cleaned correctly or not?) used are all potential sources of contamination (see more detailed note below related to data variation factors). For arsenic analyses using method 1632, EPA has specified the use of a particular kind of nitric acid, "Nitric acid—concentrated (sp gr 1.41), Seastar or equivalent" [1003].

2) For determination of dissolved elements, the samples must be filtered through a 0.45 micron membrane filter as soon as soon as practical after collection, using the first 50-100 ml to rinse the filter flask. Acidify the filtrate with nitric acid to a pH of 2 or less. Normally 3 mL of (1+1) of nitric acid per liter should be sufficient to preserve the sample.

3) For determination of suspended elements, the samples must be filtered through a 0.45 micron membrane filter as soon as soon as practical after collection. The filter is then transferred to a suitable container for storage and shipment, with no preservation required.

Highlights from EPA Method 1632: Inorganic Arsenic in Water by Hydride Generation Quartz Furnace AA:

This method is for determination of total inorganic arsenic (As) in filtered and unfiltered water by hydride generation and quartz furnace atomic absorption detection [1003]. The method is for use in EPA's data gathering and monitoring programs associated with the Clean Water Act [1003]. The method is based on a contractor-developed method and on peer-reviewed, published procedures for the determination of arsenic in aqueous samples [1003].

This method is accompanied by Method 1669: Sampling Ambient Water for Determination of Trace Metals at EPA Water Quality Criteria Levels (Sampling Method) [1002,1003]. Highlights from method 1669 are detailed below near the end of this section. The Sampling Method is necessary to preclude contamination during the sampling process [1002,1003].

As of March 1997, the EPA 1600 series methods had not yet been officially approved in 40 CFR for use in NPDES permits, but the improvements in these methods were suggested by EPA staff to be wise practice when attempting low detection limit analyses for metals.

This method is designed for measurement of dissolved and total arsenic in the range of 10–200 ng/L [1003]. This method is not intended for determination of arsenic at concentrations normally found in treated and untreated discharges from industrial facilities [1003]. Existing regulations (40 CFR Parts 400–500) typically limit concentrations in industrial discharges to the part-per-billion (ppb) range, whereas ambient arsenic concentrations are normally in the low part-per-trillion (ppt) range [1003].

The detection limits and quantitation levels in this method are usually dependent on the level of background elements rather than instrumental limitations [1003]. The method detection limit (MDL; 40 CFR 136, Appendix B) for total inorganic arsenic has been determined to be 3 ng/L when no background elements or interferences are present [1003]. The minimum level (ML) has been established at 10 ng/L [1003]. Ambient water quality criteria are as low as 18 ng/L [1003].

The ease of contaminating water samples with the metal(s) of interest and interfering substances cannot be overemphasized [1003]. This method includes suggestions for improvements in facilities and analytical techniques that should maximize the ability of the laboratory to make reliable trace metals determinations and minimize contamination [1003].

Additional suggestions for improvement of existing facilities may be found in EPA's Guidance for Establishing Trace Metals Clean Rooms in Existing Facilities, which is available from the National Center for Environmental Publications [1003].

The method calls for many handling precautions

[1003]. For example:

> Do not dip pH paper or a pH meter into the sample; remove a small aliquot with a clean pipet and test the aliquot [1003].
>
> The sample is either field or laboratory preserved by the addition of 5 mL of pretested 10% HNO3 per liter of sample, depending on the time between sample collection and arrival at the laboratory [1003].
>
> Store the preserved sample for a minimum of 48 h at 0–4°C to allow the acid to completely dissolve the metal(s) adsorbed on the container walls [1003].

Highlights from EPA Method 1669 for Sampling Ambient Water for Trace Metals at EPA Water Quality Criteria Levels [1003]:

> As of March 1997, the 1600 series methods had not yet been officially approved in 40 CFR for use in NPDES permits, but the improvements in these methods were suggested by EPA staff to be wise practice when attempting low detection limit analyses for metals.
>
> This "field method details" protocol is for the collection and filtration of ambient water samples for subsequent determination of total and dissolved Antimony, Arsenic, Cadmium, Copper, Chromium III, Chromium VI, Lead, Mercury, Nickel, Selenium, Silver, Thallium, and Zinc, at low (Water Quality Criteria Range) concentrations [1003]. It is designed to support the implementation of water quality monitoring and permitting programs administered under the Clean Water Act [1003].
>
> This method is not intended for determination of metals at concentrations normally found in treated and untreated discharges from industrial facilities [1003]. Existing regulations (40 CFR Parts 400–500) typically limit concentrations in industrial discharges to the mid to high part-per-billion (ppb) range, whereas ambient metals concentrations are normally in the low part-per-trillion (ppt) to low ppb range [1003]. This guidance is therefore directed at the collection of samples to be measured at or near the water quality criteria levels [1003]. Often these methods will be necessary in a water quality criteria-based approach to EPA permitting [1001]. Actual concentration ranges to which this guidance is applicable will be dependent on the sample matrix, dilution levels, and other laboratory operating conditions [1003].
>
> The ease of contaminating ambient water samples with the metal(s) of interest and interfering substances cannot be

overemphasized [1003]. This method includes sampling techniques that should maximize the ability of the sampling team to collect samples reliably and eliminate sample contamination [1003].

Clean and ultraclean—The terms "clean" and "ultraclean" have been used in other Agency guidance [1004] to describe the techniques needed to reduce or eliminate contamination in trace metals determinations [1003]. These terms are not used in this sampling method due to a lack of exact definitions [1003]. However, the information provided in this method is consistent with summary guidance on clean and ultraclean techniques [1004].

Preventing ambient water samples from becoming contaminated during the sampling and analytical process is the greatest challenge faced in trace metals determinations [1003]. In recent years, it has been shown that much of the historical trace metals data collected in ambient water are erroneously high because the concentrations reflect contamination from sampling and analysis rather than ambient levels [1003]. Therefore, it is imperative that extreme care be taken to avoid contamination when collecting and analyzing ambient water samples for trace metals [1003].

There are numerous routes by which samples may become contaminated [1003]. Potential sources of trace metals contamination during sampling include metallic or metal-containing sampling equipment, containers, labware (e.g. talc gloves that contain high levels of zinc), reagents, and deionized water; improperly cleaned and stored equipment, labware, and reagents; and atmospheric inputs such as dirt and dust from automobile exhaust, cigarette smoke, nearby roads, bridges, wires, and poles [1003]. Even human contact can be a source of trace metals contamination [1003]. For example, it has been demonstrated that dental work (e.g., mercury amalgam fillings) in the mouths of laboratory personnel can contaminate samples that are directly exposed to exhalation [1003].

For dissolved metal determinations, samples must be filtered through a 0.45-um capsule filter at the field site [1003]. The filtering procedures are described in this method [1003]. The filtered samples may be preserved in the field or transported to the laboratory for preservation [1003].

This document is intended as guidance only [1003]. Use of the terms "must," "may," and "should" are included to mean that EPA believes that these procedures must, may, or should be followed in order to produce the desired results when using

this guidance [1003]. In addition, the guidance is intended to be performance-based, in that the use of less stringent procedures may be used so long as neither samples nor blanks are contaminated when following those modified procedures [1003]. Because the only way to measure the performance of the modified procedures is through the collection and analysis of uncontaminated blank samples in accordance with this guidance and the referenced methods, it is highly recommended that any modifications be thoroughly evaluated and demonstrated to be effective before field samples are collected [1003].

The method includes a great many details regarding prevention of field contamination of samples, including clothing needed, clean hands vs. dirty hands operations, and numerous other details [1003].

Surface sampling devices—Surface samples are collected using a grab sampling technique [1003]. Samples may be collected manually by direct submersion of the bottle into the water or by using a grab sampling device [1003]. Grab samplers may be used at sites where depth profiling is neither practical nor necessary [1003].

An alternate grab sampler design is available [1003]. This grab sampler is used for discrete water samples and is constructed so that a capped clean bottle can be submerged, the cap removed, sample collected, and bottle recapped at a selected depth [1003]. This device eliminates sample contact with conventional samplers (e.g., Niskin bottles), thereby reducing the risk of extraneous contamination [1003]. Because a fresh bottle is used for each sample, carryover from previous samples is eliminated [1003].

Subsurface sampling devices—Subsurface sample collection may be appropriate in lakes and sluggish deep river environments or where depth profiling is determined to be necessary [1003]. Subsurface samples are collected by pumping the sample into a sample bottle [1003]. Examples of subsurface collection systems include the jar system device or the continuous-flow apparatus [1003].

Advantages of the jar sampler for depth sampling are (1) all wetted surfaces are fluoropolymer and can be rigorously cleaned; (2) the sample is collected into a sample jar from which the sample is readily recovered, and the jar can be easily recleaned; (3) the suction device (a peristaltic or rotary vacuum pump, is located in the boat, isolated from the sampling jar; (4) the sampling jar can be continuously flushed with sample, at sampling depth, to equilibrate the system; and (5) the

sample does not travel through long lengths of tubing that are more difficult to clean and keep clean [1003]. In addition, the device is designed to eliminate atmospheric contact with the sample during collection [1003].

Selection of a representative site for surface water sampling is based on many factors including: study objectives, water use, point source discharges, non-point source discharges, tributaries, changes in stream characteristics, types of stream bed, stream depth, turbulence, and the presence of structures (bridges, dams, etc.) [1003]. When collecting samples to determine ambient levels of trace metals, the presence of potential sources of metal contamination are of extreme importance in site selection [1003].

Ideally, the selected sampling site will exhibit a high degree of cross-sectional homogeneity [1003]. It may be possible to use previously collected data to identify locations for samples that are well mixed or are vertically or horizontally stratified [1003]. Since mixing is principally governed by turbulence and water velocity, the selection of a site immediately downstream of a riffle area will ensure good vertical mixing [1003]. Horizontal mixing occurs in constrictions in the channel [1003]. In the absence of turbulent areas, the selection of a site that is clear of immediate point sources, such as industrial effluents, is preferred for the collection of ambient water samples) [1003].

To minimize contamination from trace metals in the atmosphere, ambient water samples should be collected from sites that are as far as possible (e.g., at least several hundred feet) from any metal supports, bridges, wires or poles [1003]. Similarly, samples should be collected as far as possible from regularly or heavily traveled roads [1003]. If it is not possible to avoid collection near roadways, it is advisable to study traffic patterns and plan sampling events during lowest traffic flow [1003].

The sampling activity should be planned to collect samples known or suspected to contain the lowest concentrations of trace metals first, finishing with the samples known or suspected to contain the highest concentrations [1003]. For example, if samples are collected from a flowing river or stream near an industrial or municipal discharge, the upstream sample should be collected first, the downstream sample collected second, and the sample nearest the discharge collected last [1003]. If the concentrations of pollutants is not known and cannot be estimated, it is necessary to use precleaned sampling equipment at each sampling location [1003].

One grab sampler consists of a heavy fluoropolymer collar fastened to the end of a 2-m-long polyethylene pole, which serves to remove the sampling personnel from the immediate vicinity of the sampling point [1003]. The collar holds the sample bottle [1003]. A fluoropolymer closing mechanism, threaded onto the bottle, enables the sampler to open and close the bottle under water, thereby avoiding surface microlayer contamination [1003]. Polyethylene, polycarbonate, and polypropylene are also acceptable construction materials unless mercury is a target analyte [1003]. Assembly of the cleaned sampling device is as follows:

Sample collection procedure—Before collecting ambient water samples, consideration should be given to the type of sample to be collected, the amount of sample needed, and the devices to be used (grab, surface, or subsurface samplers) [1003]. Sufficient sample volume should be collected to allow for necessary quality control analyses, such as matrix spike/ matrix spike duplicate analyses [1003].

It is recommended that 1 mL of ultrapure nitric acid be added to each vial prior to transport to the field to simplify field handling activities [1003].

Preservation of aliquots for metals other than trivalent and hexavalent chromium—Using a disposable, precleaned, plastic pipet, add 5 mL of a 10% solution of ultrapure nitric acid in reagent water per liter of sample [1003]. This will be sufficient to preserve a neutral sample to pH <2 [1003].