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

by
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TABLES

Number

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Abstract. Ecological and toxicological aspects of silver (Ag) and silver salts in the environment are briefly summarized with an emphasis on natural resources. Subtopics include sources and uses, chemistry and metabolism, concentrations in field collections, lethal and sublethal effects, and recommendations for the protection of natural resources. Elevated silver concentrations in biota occur in the vicinities of sewage outfalls, electroplating plants, mine waste sites, and silver-iodide seeded areas; in the United States, the photography industry is the major source of anthropogenic silver discharges into the biosphere. Maximum concentrations recorded in field collections, in milligrams total silver/kilogram dry weight (tissue), were 1.5 in mammals (liver), 6 in fish (bone), 14 in plants (whole), 30 in annelid worms (whole), 44 in birds (liver), 110 in mushrooms (whole), 185 in bivalve mollusks (soft parts), and 320 in gastropods (whole); humans afflicted with silver poisoning (argyria) contained 72 mg total Ag/kg dry weight skin and 1,300 mg total Ag/kg fresh weight whole body. Silver and its compounds are not known to be mutagenic, teratogenic, or carcinogenic. Under normal routes of exposure, silver does not pose serious environmental health problems to humans at less than 50 µg total Ag/L drinking water or 10 µg total Ag/m^3 air. Free silver ion, however, was lethal to representative species of sensitive aquatic plants, invertebrates, and teleosts at nominal water concentrations of 1.2 to 4.9 µg/L; at sublethal concentrations, adverse effects were significant between 0.17 and 0.6 µg/L. No data were found on effects of silver on avian or mammalian wildlife; all studied effects were on poultry, small laboratory animals, and livestock. Silver was harmful to poultry at concentrations as low as 1.8 mg total Ag/kg whole egg fresh weight by way of injection, 100 mg total Ag/L in drinking water, or 200 mg total Ag/kg in diets; sensitive mammals were adversely affected at total silver concentrations as low as 250 µg/L in drinking water, 6 mg/kg in diets, or 13.9 mg/kg whole body. Proposed criteria for the protection of living organisms from silver are listed and discussed.

Key words: Silver, plants, fish, wildlife, invertebrates, mammals, ecotoxicology.

Silver (Ag) found in the body of mammals (including humans) has no known biological purpose and is suspected of being a contaminant (Smith and Carson 1977). Silver, as ionic Ag^+, is one of the most toxic metals known to aquatic organisms in laboratory testing, although large industrial losses to the aquatic environment are probably infrequent because of its economic value as a recoverable resource (National Association of Photographic Manufacturers [NAPM] 1974; Nebeker et al. 1983). Silver, however, is of concern in various aquatic ecosystems because of the severity of silver contamination in the water column, sediments, and biota. San Francisco Bay, for example, is impacted from discharges of silver in wastewater outfalls and from the diagenic remobilization of silver from contaminated sediments in the estuary (Luoma and Phillips 1988; Rivera-Duarte and Flegal 1993).

The principal industrial use of silver is as silver halide in the manufacture of photographic imaging materials; other products include jewelry, coins, indelible inks, and eating utensils (Klaassen et al. 1986). In medicine, silver salts are used as caustics, germicides, antiseptics, and astringents; the use of silver nitrate for prophylaxis of ophthalmia neonatorum in the eyes of newborn infants is a legal requirement in some states (Klaassen et al. 1986). Long-term industrial or medical exposure to silver and its compounds may increase blood concentrations of silver to levels which can have toxic effects, such as induction of sarcomas, anemia, and enlargement of the

heart (Aoki et al. 1993). Repeated occupational handling of silver objects, especially after repeated minor injuries, may result in localized argyria—a bluish-gray discoloration of the skin at the exposed site (Fowler and Nordberg 1986). In humans, the most common noticeable effects of chronic exposure to silver and its compounds are generalized argyria, localized argyria, and argyrosis (argyria of the eye, usually; Smith and Carson 1977). Generalized argyria consists of a slate-gray pigmentation of the skin and hair caused by deposition of silver in the tissues, a silver coloration of the hair and fingernails, and a blue halo around the cornea and in the conjunctiva. Acute toxic effects in humans have resulted only from accidental or suicidal overdoses of medical forms of silver (Smith and Carson 1977).

Ecological and toxicological aspects of silver are reviewed by Smith and Carson (1977), the U.S. Environmental Protection Agency [EPA] (1980, 1987), Lockhart (1983), the U.S. Public Health Service [PHS] (1990), Andren et al. (1993, 1994), and Andren and Bober (1995). The present report is another in a series on hazards of selected contaminants to plants and animals, with an emphasis on fishery and wildlife resources. It was prepared in response to requests for information on silver from environmental specialists of the U.S. Fish and Wildlife Service.

Sources and Uses

General

About 2.47 million kg of silver are lost each year to the domestic biosphere, mostly (82%) as a result of human activities. As discussed later, the photography industry accounts for about 47% of all silver discharged into the environment from anthropogenic sources. In 1990, about 50% of the refined silver consumed domestically was used to manufacture photographic products; 25% in electrical and electronic products; 10% in electroplated ware, sterlingware, and jewelry; 5% in brazing alloys; and 10% in other products and processes.

Sources

Silver is a rare but naturally occurring metal, often found deposited as a mineral ore in association with other elements (PHS 1990). Argentite is the main ore from which silver is extracted by cyanide, zinc reduction, or electrolytic processes (Fowler and Nordberg 1986). Silver is frequently recovered as a byproduct from smelting of nickel ores in Canada, from lead-zinc and porphyry copper ores in the United States, and from platinum and gold deposits in South Africa (Smith and Carson 1977). About 12-14% of the domestic silver output is recovered from lead ores and about 4% from zinc ores. Secondary sources of silver comprise new scrap generated in the manufacture of silver-containing products; coin and bullion; and old scrap from electrical products, old film and photoprocessing wastes, batteries, jewelry, silverware, and bearings (Smith and Carson 1977).

Silver is produced in 68 countries, but most (75%) of the world's silver (excluding the former Soviet bloc) is mined in the United States, Mexico, Canada, Australia, and Japan; the United States produces about 50% of the world's supply of refined silver (Smith and Carson 1977). The primary silver mines of the United States are in the Coeur d'Alene mining district in the northern Idaho panhandle (PHS 1990). Between 1949 and 1970, the United States consistently produced less than 15% of the global silver production and consumed 34-64% (Heyl et al. 1973). Since 1951, silver consumption in the United States has exceeded its extraction from ore (PHS 1990). In 1979, about 95% of the silver in domestic production was from Idaho, Nevada, Arizona, Colorado, Utah, Montana, and Missouri (U.S. Bureau of Mines 1980). End use categories of silver consumed domestically in 1979 included photography (39%), electrical and electronic components (25%), sterlingware and electroplated materials (15%), and brazing alloys and solders (8%). In 1979, the photographic industry was located mainly in New York, and most other end use manufacturers were in Connecticut, New York, Rhode Island, and New Jersey (U.S. Bureau of Mines 1980).

World production of silver increased from 7.40 million kg in 1964 to 9.06 million kg in 1972 and to 9.67 million kg in 1982 (Fowler and Nordberg 1986). In 1986, 13.06 million kg of silver were produced globally; the United States produced 1.06 million kg in 1986 but consumed 3.94 million kg (PHS 1990). In 1990, the estimated world mine production of silver was 14.6 million kg; major producers were Mexico with 17% of the total, the United States with 14%, Peru with 12%, the former Soviet Union with 10%, and Canada with 9% (Reese 1991). In the United States during 1990, about 160 mines produced silver worth an estimated value of $320 million; most (71%) of the 1990 mine production was in Nevada (32%), Idaho (21%), Montana (11%), and Arizona (7%). In 1990, 22 major refiners of commercial grade silver and more than 5,000 silver fabricating and manufacturing firms were located primarily in the northeastern states. Of the silver imported into the United

States in 1990, 44% came from Mexico, 34% from Canada, 5% from Peru, and 4% from Chile. Most was exported after transformation to sterlingware, coinage, and other finished products. Melting and refining old scrap silver in 1990 accounted for 500,000 kg of silver (Reese 1991).

Emissions from smelting operations, manufacture and disposal of certain photographic and electrical supplies, coal combustion, and cloud seeding are some of the anthropogenic sources of silver in the biosphere (Freeman 1979). Fallout from cloud seeding with silver iodide is not always confined to local precipitation; silver residuals have been detected several hundred kilometers downwind of seeding events (Freeman 1979). In 1978, the estimated loss of silver to the environment in the United States was 2.47 million kg, mostly to terrestrial and aquatic ecosystems; the photography industry alone accounted for about 47% of all silver discharged into the environment from anthropogenic sources (Smith and Carson 1977; Table 1). In California, anthropogenic sources contributed 50% more silver to sediments of coastal basins than did natural sources, as judged by sedimentary basin fluxes of 0.09 µg/cm^2 in anthropogenic sources of silver and 0.06 µg/cm^2 in natural sources (Bruland et al. 1974). Sometimes, liquid effluents from the nuclear industry contained significant quantities of radiosilver-110m (Berthet et al.1992). In Lake Michigan, storms contribute a large fraction of the annual load of tributary-derived silver; concentrations of particle-bound silver in many rivers during storms were more than 0.1 µg/L (Shafer et al. 1995).

Table 1. Estimated release of silver to the environment in the United States in 1978 (U.S. Public Health Service 1990).

Compartment and source category	Metric tons[a]
Atmosphere	
Metals production	30
Urban refuse combustion	10
Coal and petroleum combustion	9
Iron and steel production	7
Cloud seeding	3
Cement manufacture	2
Other	30
Aquatic	
Soil erosion (natural source)	438
r uban runoff	72
Sewage treatment plants	70
Photographic developing	65
Photographic manufacture	54
Other	6
Terrestrial	
Photographic industry	630
Urban refuse	445
Sewage treatment	220
Metals production	165
Electrical contacts and conductors	150
Alloys and solders	60
Other	5

[a]Of the total silver released to the domestic environment in 1978 (2,470,700 kg), about 3.7% entered the atmosphere, 28.5% the aquatic environment, and 67.8% the terrestrial ecosystem.

Most of the silver lost to the environment each year enters terrestrial ecosystems where it is immobilized in the form of minerals, metal, or alloys; agricultural lands may receive as much as 80,000 kg of silver from photoprocessing wastes in sewage sludge. An estimated 150,000 kg of silver enter the aquatic environment every year from the photography industry, mine tailings, and electroplating (Smith and Carson 1977). During processing of photographic paper and film, silver is generally solubilized as the tightly bound thiosulfate complex. Silver thiosulfate in secondary biological waste treatment plants is converted to insoluble silver sulfide,

which is removed in the sludge; only trace amounts of complexed and adsorbed silver are discharged into the aquatic environment. The silver incorporated into the sludge is immobile and should not restrict the use of sludge for the enrichment of soils (Dagon 1973; Bard et al. 1976; Cooley et al. 1988). The atmosphere receives 300,000 kg of silver each year from a variety of sources, but atmospheric concentrations are not known to exceed the occupational threshold limit value of 10 µg total Ag/m^3 (Smith and Carson 1977).

Daily intake of total silver from all sources by humans in the United States ranges from 70 to 88 µg; diet accounted for 35-40 µg daily (EPA 1980). Sources of elevated dietary silver include seafood from areas near sewage outfalls or industrial sources and crops grown in areas with high ambient levels of silver in the air or soil (PHS 1990). Most occupational exposures to silver occur through inhalation of silver-containing dusts or dermal exposure to photographic compounds. Dermal routes of human exposure to silver include handling of silver-containing processing solutions used in radiographic and photographic materials, dental amalgams, and silver sulfadiazine cream and solutions for treating burns (PHS 1990).

Uses

Silver has been used for ornaments and utensils for almost 5,000 years, and as a precious metal, a monetary medium, and a basis of wealth for more than 2,000 years. Until the late 1960's, it was used extensively for coinage (Heyl et al. 1973). Since 1970, U.S. coinage has not contained silver, although minting of as many as 45 million silver-clad subsidiary coins has been authorized (Smith and Carson 1977). Industrial consumption of silver in the United States between 1966 and 1972 totaled 4.67 million kg, primarily in the manufacture of photographic materials, electrical contacts and conductors, and sterlingware (Table 2). In 1973, silver was used mainly in photographic materials (29%), electrical and electronic components (22%), sterlingware (20%), electroplated ware (10%), brazing wares (20%), dental and medical products, catalysts, bearings, and jewelry (9%; Heyl et al. 1973). In 1986, photographic materials accounted for 45% of the silver consumption in the United States; electrical and electronic components, 25%; jewelry, sterlingware, and electroplated ware, 11%; alloys and solders, 5%; and mirrors, dental amalgam, medical supplies, chemicals, water purification, and cloud seeding, 14% (PHS 1990). Silver, as silver iodide, is used in the United States for weather modification, including rain and snow making and hail suppression; as much as 3,110 kg of silver is used for this purpose annually (Smith and Carson 1977). Silver nitrate in hair dyes has been in use regularly for almost 200 years (EPA 1980), although its use may lead to argyria (Smith and Carson 1977). In 1990, about 50% of the refined silver consumed domestically was used to manufacture photographic and x-ray products; 25% in electrical and electronic products; 10% in electroplated ware, sterlingware, and jewelry; 5% in brazing alloys; and 10% in other uses (Reese 1991).

Table 2. Industrial domestic use of silver during 1966-72. Total silver used was 4.67 million kg (Smith and Carson 1977).

Category	Approximate percent of total
Photographic materials	28.1
Contacts and conductors	20.2
Sterlingware	17.1
Brazing alloys and solders	10.1
Electroplated ware	10.0
Batteries	5.1
Jewelry	3.3
Miscellaneous	2.4
Dental and medical supplies	1.5
Mirrors	1.2
Bearings	0.4

Because of its bacteriostatic properties, silver compounds are used in filters and other equipment to purify water of swimming pools and drinking water and in the processing of foods, drugs, and beverages (Smith and Carson 1977; EPA 1980; PHS 1990). Activated charcoal filters coated with metallic silver to yield water concentrations of 20-40 µg Ag/L are used in filtering systems of swimming pools to control bacteria (EPA 1980). Silver may also function as an algicide in swimming pools if chlorine, bromine, and iodine are absent; it prevents growth of blue-green algae at 80-140 µg Ag/L (Smith and Carson 1977). Aboard orbiting Russian space stations and spaceships, potable water is routinely treated with 100-200 µg Ag/L to eliminate microorganisms; sterilization is usually complete in 20 min (Smith and Carson 1977). Silver-containing ceramic water filters are used to purify potable water in Swiss ski resorts, German breweries, British ships, oil tankers, drilling rigs, U.S. home consumption, and more than half the world's airlines. Monovalent and metallic silver compounds are considered excellent disinfectants; however, Ag^{2+} and Ag^{3+} are about 50 to 200 times more effective than Ag^{+} or Ag^{o} (Antelman 1994), possibly because of their higher oxidation states (Kirschenbaum 1991).

Silver nitrate was used for many years as eye drops in newborns to prevent blindness caused by gonorrhea (PHS 1990). Laws in many states still require that a few drops of a 1-2% silver nitrate solution be applied to the conjunctiva of the eyes of newborn infants to prevent ophthalmia neonatorum by transmittal of gonorrhea from the mother (EPA 1980; PHS 1990). This treatment is still required in Denmark but not in Japan or Australia (EPA 1980). Silver nitrate is not used in many U.S. hospitals because of the dangers of chemical conjunctivitis and has been replaced by antibiotics (EPA 1980). In the United States, several silver-containing pharmaceuticals were used topically on skin or mucous membranes to assist in healing burn patients and to combat skin ulcers (Smith and Carson 1977; EPA 1980). Oral medicines containing silver include silver acetate-containing antismoking lozenges; breath mints coated with silver; and silver nitrate solutions for treating gum disease (PHS 1990). The widespread medical use of silver compounds for topical application to mucous membranes and for internal use became nearly obsolete in the past 50 years because of the fear of argyria and the development of sulfonamide and antibiotic microbials (Smith and Carson 1977).

Chemistry and Metabolism

General

Silver occurs naturally in several oxidation states, the most common being elemental silver (Ag^{o}) and the monovalent ion (Ag^{+}). Soluble silver salts are in general more toxic than insoluble salts; in natural waters, the soluble monovalent species is the form of environmental concern. Sorption is the dominant process that controls silver partitioning in water and its movements in soils and sediments. As discussed later, silver enters the animal body through inhalation, ingestion, and movement across mucous membranes and broken skin. The interspecies differences in the ability of animals to accumulate, retain, and eliminate silver are large. Almost all of the total silver intake is usually excreted rapidly in feces; less than 1% of the total silver intake is absorbed and retained in tissues, primarily liver, through precipitation of insoluble silver salts. In mammals, silver usually interacts antagonistically with selenium, copper, and vitamin E; in aquatic environments, ionic or free silver interferes with calcium metabolism in frogs and marine annelids and with sodium and chloride uptake in gills of fishes.

Physical and Chemical Properties

Silver is a white, ductile metal occurring naturally in the pure form and in ores (EPA 1980). Silver has the highest electrical and thermal conductivity of all metals. Some silver compounds are extremely photosensitive and are stable in air and water except for tarnishing readily when exposed to sulfur compounds (Heyl et al. 1973). Metallic silver is insoluble in water but many silver salts, such as silver nitrate, are soluble in water to 1,220 g/L (Table 3). In natural environments, silver occurs primarily in the form of the sulfide or is intimately associated with other metal sulfides, especially those of lead, copper, iron, and gold, which are all essentially insoluble (EPA 1980; PHS 1990). Silver readily forms compounds with antimony, arsenic, selenium, and tellurium (Smith and Carson 1977). Silver has two stable isotopes (^{107}Ag and ^{109}Ag) and 20 radioisotopes; none of the radioisotopes of silver occurs naturally, and the radioisotope with the longest physical half-life (253 days) is ^{110m}Ag. Several compounds of silver are potential explosion hazards: silver oxalate decomposes explosively when heated; silver acetylide (Ag_2C_2) is sensitive to detonation on contact; and silver azide (AgN_3) detonates spontaneously under certain conditions (Smith and Carson 1977).

Table 3. Some properties of silver and silver nitrate (Lockhart 1983; U.S. Public Health Service 1990).

Variable	Silver	Silver nitrate
Alternate names	Argentum, argentum crede CI 77820, shell silver, silver atom, silver colloidal, silflake, silpowder, silber	Lunar caustic fused silver nitrate, molded silver nitrate argenti, nitras, nitric acid silver (I) salt, nitric acid silver (1+) salt, silver (1+) nitrate
CAS number	7440-22-4	7761-88-8
Chemical formula	Ag	$AgNO_3$
Molecular weight	107.87	169.89
Physical state	Solid metal	Solid crystalline
Boiling point	2,212° C	Decomposes at 440 C
Solubility	Insoluble in water; soluble in nitric acid but not sulfuric acid	Soluble in water to 1,220 g/L; soluble in ethanol and acetone
Density	10.5	4.35

Silver occurs naturally in several oxidation states, usually as Ag^0 and Ag^+; other possible oxidation states of silver are Ag^{2+} and Ag^{3+} (PHS 1990). In surface fresh water, silver may be found as the monovalent ion; in combination with sulfide, bicarbonate, or sulfate; as part of more complex ions with chlorides and sulfates; and adsorbed onto particulate matter (PHS 1990). Soluble silver salts are more toxic than insoluble salts, and soluble silver ion (Ag^+) is the most toxic chemical species. In natural waters, the soluble monovalent species is the form of environmental concern (EPA 1980). The argentous ion (Ag^+) does not hydrolyze appreciably in solution and is considered to be a mild oxidizing agent (Smith and Carson 1977). Hypervalent silver species, such as Ag^{2+} and Ag^{3+}, are significantly more effective as oxidizing agents than Ag^0 and Ag^+ (Kouadio et al. 1990; Kirschenbaum 1991; Sun et al. 1991) but are unstable in aqueous environments, especially at water temperatures near 100^0 C (Smith and Carson 1977). In natural waters, silver may exist as metalloorganic complexes or adsorb to organic materials (EPA 1980). In fresh water and soils, the primary silver compounds under oxidizing conditions are bromides, chlorides, and iodides; under reducing conditions the free metal and silver sulfide predominate (PHS 1990). In river water, one study showed silver present as the monovalent ion (Ag^+) at 53-71% of the total silver, as silver chloride (AgCl) at 28-45%, and as silver chloride ion ($AgCl_2^-$) at 0.6-2.0% (PHS 1990). Increasing salinity of brackish and marine waters increase concentrations of silver chloro complexes ($AgCl^0$, $AgCl_2^-$, $AgCl_3^{2-}$, $AgCl_4^{3-}$); these chloro complexes retain some silver in dissolved form, and relatively small anthropogenic quantities can substantially enrich the environment (Luoma 1994; Andren et al. 1995). In the open ocean, the principal dissolved form of silver is $AgCl_2^-$, but the most bioavailable form may be the neutral monochloro complex AgCl (Bryan and Langston 1992).

Sorption is the dominant process that controls silver partitioning in water and its movement in soils and sediments (EPA 1980; PHS 1990). Silver may leach from soils into ground water; the leaching rate increases with decreasing pH and increasing drainage (PHS 1990). Silver adsorbs to manganese dioxide, ferric compounds, and clay minerals, and these compounds are involved in silver deposition into sediments; sorption by manganese dioxide and precipitation with halides reduce the concentration of dissolved silver, resulting in higher concentrations in sediments than in the water column (EPA 1980). Under reducing conditions, adsorbed silver in sediments may be released and subsequently reduced to metallic silver or it may combine with reduced sulfur to form the insoluble silver sulfide (EPA 1980). Sediments may be a significant source of silver to the water column. In one study, anoxic sediments containing 1.0-27.0 g Ag/kg dry weight (DW) and 10 mmoles of acid volatile sulfide/kg DW were resuspended in oxygenated seawater for several hours to days. The seawater in contact with sediment containing 10.8 g/kg had 20 µg Ag/L; seawater in contact with sediments containing 27

g Ag/kg had about 2,000 µg Ag/L, which seems to be the solubility of silver in seawater (Crecelius and Phillips 1995).

The global biogeochemical movements of silver are characterized by releases to the atmosphere, water, and land by natural and anthropogenic sources, long-range transport of fine particles in the atmosphere, wet and dry deposition, and sorption to soils and sediments (PHS 1990). The chief source of silver contamination of water is silver thiosulfate complexes in photographic developing solutions that photofinishers discard directly to sewers (Smith and Carson 1977). Secondary waste treatment converts most of the silver thiosulfate complex to insoluble silver sulfide and forms some metallic silver (Lytle 1984). About 95% of the total silver is removed in publicly owned treatment works from inputs containing municipal sewage and commercial photoprocessing effluents, and effluents contain less than 0.07 µg ionic silver/L (Lytle 1984). Silver in sewage treatment plant effluents may be associated with suspended particles or be present as thiosulfate complex, colloidal silver complex, colloidal silver chloride, silver sulfide, or soluble organic complexes (Smith and Carson 1977). Silver on suspended matter and in colloidal forms and insoluble salts ultimately settles out in the sediments. At the water treatment plant, most of the silver is precipitated after treatment with lime or adsorbed after treatment with alum-flocculent. Chlorination converts some silver to silver chloride or to a soluble silver chloride complex (Smith and Carson 1977).

Forms of silver in atmospheric emissions are probably silver sulfide, silver sulfate, silver carbonate, silver halides, and metallic silver (Smith and Carson 1977). About 50% of the silver released into the atmosphere from industrial operations is transported more than 100 km and is eventually deposited in precipitation (PHS 1990). Minute amounts of ^{110m}Ag detected in natural waters are attributed to atmospheric fallout from nuclear explosions (Smith and Carson 1977).

A variety of spectrographic, colorimetric, polarographic, and other analytical techniques are used for routine measurement of silver in biological and abiotic samples. The detection limit of silver in biological tissues with scanning electron microscopy and x-ray energy spectrometry is 0.02 µg/kg and sometimes as low as 0.005 µg/kg. In air, water, and soil samples, the preferred analytical procedures include flame and graphite furnace atomic absorption spectrometry, plasma emission spectroscopy, and neutron activation (Fowler and Nordberg 1986; PHS 1990). Sensitive voltammetry techniques using anodic stripping have recently been developed to measure free silver ion in solution at concentrations as low as 0.1 µg/L (Schildkraut 1993; Song and Osteryoung 1993).

Metabolism

The acute toxicity of silver to aquatic species varies drastically by the chemical form and correlates with the availability of free ionic silver (Wood et al. 1994). In natural aquatic systems, ionic silver is rapidly complexed and sorbed by dissolved and suspended materials that are usually present. Complexed and sorbed silver species in natural waters are at least one order of magnitude less toxic to aquatic organisms than the free silver ion (Rodgers et al. 1994). Thus, silver nitrate—which is strongly dissociated—is extremely toxic to rainbow trout (*Oncorhynchus mykiss*); the 7-day LC50 value is 9.1 µg/L. Silver thiosulfate, silver chloride, and silver sulfide are relatively benign (7-day LC50 values >100,000 µg/L), presumably because of the abilities of the anions to remove ionic silver from solution (Wood et al. 1994, 1996b; Hogstrand et al. 1996). The probable cause of hyperventilation in rainbow trout exposed to silver nitrate was a severe metabolic acidosis manifested in decreased arterial plasma pH and HCO_3^- levels. Lethality of ionic silver to trout is probably due to surface effects at the gills—disrupting Na^+, Cl^-, and H^+—causing secondary fluid volume disturbance, hemoconcentration, and eventual cardiovascular collapse (Wood et al. 1994, 1995, 1996a, 1996b). Morgan et al. (1995) suggest that the sites of action of silver toxicity in rainbow trout may be inside the cells of the gill epithelium rather than at the external surface, and that they are linked to carbonic anhydrase—a gill enzyme involved in Na^+ and Cl^- transport. Silver concentrations and metallothionein levels in gills and livers of rainbow trout increased with increasing exposure to silver; internal toxicity associated with increased silver accumulations may be lessened by the formation of silver-induced metallothioneins (Hogstrand et al. 1996). In seawater, silver nitrate is less toxic to biota than in fresh water (Wood et al. 1995). This difference is probably due to the low concentration of free Ag^+ (the toxic moiety in freshwater) in seawater and to the high levels of chloride and negatively charged Ag-chloro complexes in seawater. However, high levels of silver nitrate are

toxic to marine invertebrates despite the absence of Ag^+, and this is attributed to the bioavailability of Ag-chloro complexes; mechanisms of silver toxicity in marine fishes are still unknown (Wood et al. 1995).

Ionic silver interferes with calcium metabolism of frogs and marine polychaete worms. Silver ions cause muscle fibers of frogs (*Rana* spp.) to deteriorate by allowing excess calcium to enter the cell. Studies with frog skeletal muscle fibers exposed to 1.08 mg/L showed that silver activated the calcium ion channel by acting on sulfhydryl groups in a calcium ion channel protein (Aoki et al. 1993). In marine polychaetes contaminated with silver, the calcium content of nephridial cells was reduced, although silver was not detected in the calcium vesicles (Koechlin and Grasset 1988). Silver binds with protein sulfhydryl groups and this process protects the worm against silver poisoning (Koechlin and Grasset 1988). In marine mollusks, however, sulfide anion was the ligand of silver (Truchet et al. 1990). In marine gastropods (*Littorina littorea*), silver was stored in the basement membranes of the digestive system; in clams (*Scrobicularia plana*), it was stored in the basement membranes of the outer fold of the mantle edge and in the amoebocytes (Truchet et al. 1990). The availability of free silver in marine environments was strongly controlled by salinity because of the affinity of silver for the chloride ion (Sanders et al. 1991). Silver sorbs readily to phytoplankton and to suspended sediments. As salinity increases, the degree of sorption decreases. Nearly 80% of silver sorbed to suspended sediments at low salinities desorbs at higher salinities, but desorption does not occur when silver is associated with phytoplankton. Thus, silver incorporation in or on cellular material increases the retention of silver in the estuary, reducing the rate of transport (Sanders and Abbe 1987).

Silver may enter the body of mammals through inhalation, ingestion, and movement across mucous membranes or broken skin (Smith and Carson 1977; EPA 1980; Klaassen et al. 1986; PHS 1990). In most cases of occupational argyrosis, absorption occurs via the respiratory tract or at the eyes (Smith and Carson 1977; EPA 1980). Silver is retained by all body tissues; tissue concentrations are related to the dose, form of administered silver, and route of exposure. Silver also accumulates in mammalian tissues with increasing age of the individual, even if none is administered intentionally. Inside the body, silver is transported mainly in the protein fractions of plasma, presumably as silver albuminate or silver chloride (Smith and Carson 1977; EPA 1980). In mammals, the highest concentrations of silver are usually found in the liver and spleen and to some extent in the muscles, skin, and brain (Fowler and Nordberg 1986). The primary sites of silver deposition in the human body are the liver, skin, adrenals, lungs, muscle, pancreas, kidney, heart, and spleen; silver is also deposited in blood vessel walls, the trachea, and bronchi (Smith and Carson 1977). Dogs exposed to silver by inhalation accumulated most of the administered dose in the liver; concentrations in the lung, brain, skin, and muscle were lower (EPA 1980; Fowler and Nordberg 1986). Intravenous injection of silver produces accumulations in the spleen, liver, bone marrow, lungs, muscle, and skin (Klaassen et al. 1986). Intestinal absorption of silver by rodents, canids, and primates has been recorded at 10% or less after ingestion of radioactive silver; a value of 18% was estimated in a single human given radiosilver acetate (EPA 1980; Klaassen et al. 1986) and about 3-10% of the absorbed silver is retained in the tissues (Smith and Carson 1977). In a human given radioactive silver, more than 50% of the whole-body burden of silver was found in the liver after 16 days (Fowler and Nordberg 1986)

Deposition of silver in tissues of warm-blooded animals results from precipitation of relatively insoluble silver salts, such as silver chloride and silver phosphate (PHS 1990). These insoluble salts may be transformed into soluble silver sulfide albuminates that bind or complex with RNA, DNA, and proteins, or may be reduced to metallic silver by ascorbic acid or catecholamines. In humans with argyria, the blue or gray skin discoloration is caused by the photoreduction of silver chloride to metallic silver during exposure to ultraviolet light. Metallic silver, in turn, is oxidized and bound as black silver sulfide (PHS 1990). Silver sulfide (Ag_2S) is localized in extracellular structures such as basement membranes and in macrophageous cells (Baudin et al. 1994). Before storage as a stable mineral combination, silver binds to proteins that contain a large proportion of sulfhydryl groups such as metallothioneins (Fowler and Nordberg 1986). The last stage in the catabolic pathway of these proteins leads to storage of silver after reaction with a sulfur ligand (Baudin et al. 1994). These mechanisms explain why the liver, the most important organ for protein synthesis, shows the highest capacity for silver accumulation. High concentrations of silver in the digestive tract are linked to the numerous basement membranes contained in its tissues. Interspecies differences in the ability to accumulate, retain, and eliminate silver are large (Baudin et al. 1994).

The enzyme-inhibiting action of silver ions may be due to the binding of sulfhydryl groups of some enzymes. Binding, in certain enzymes, is probably at a histidine imidazole group; in the case of glucose oxidase, silver ions compete with molecular oxygen as a hydrogen acceptor (Smith and Carson 1977). About 60% of the silver in liver and kidneys of silver-injected rats was in the cytosol fractions bound to the high molecular weight proteins and metallothionein fractions; however, in spleen and brain only 30% of the total tissue silver was found in the cytosol fractions (Fowler and Nordberg 1986). At moderate doses (0.4 mg Ag/kg body weight [BW]) in rats, the liver handles most of the absorbed silver from the body in the bile; at higher doses, silver deposits are markedly increased in the skin (EPA 1980). In house sparrows (*Passer domesticus*), a silver-binding protein was identified in liver after radiosilver-110m injection; the protein was heat-stable, resistant to low pH, and of low molecular weight (Kumar and Bawa 1979). The properties of the hepatic silver-binding protein in birds were similar to other studied metallothioneins, but more research is needed to distinguish differences from mammalian metallothioneins (Kumar and Bawa 1979).

Most absorbed silver is excreted into the intestines by way of the liver into the bile and subsequently excreted in feces; urinary excretion of silver is generally very low (EPA 1980; Fowler and Nordberg 1986; Klaassen et al. 1986; PHS 1990). Rodents, monkeys, and dogs given radioactive silver salts by oral and other routes excreted more than 90% of the absorbed dose in the feces (Fowler and Nordberg 1986). Rats injected intravenously with radioactive silver nitrate excreted silver in bile mainly bound to a low molecular weight complex similar to glutathione (Fowler and Nordberg 1986). Excretion was faster and percentages excreted by mice, rats, monkeys, and dogs were larger when silver was administered orally than by intravenous or intraperitoneal injection (Smith and Carson 1977).

Among mammals, low doses of ingested silver were eliminated from the body within 1 week (PHS 1990). In rats, mice, and rabbits, about 99% of a single oral dose of silver was eliminated within 30 days (EPA 1980). Time for 50% clearance of silver in rats, mice, monkeys, and dogs after oral ingestion was about 1 day; this short half-time is due, in part, to fecal elimination of unabsorbed silver; the half-times were longer (1.8-2.4 days) after intravenous injection (Fowler and Nordberg 1986). Rodents dosed with silver accumulated high initial concentrations in the liver, which greatly decreased within 10 days; however, silver concentrations in spleen and brain were retained for longer periods. The biological half-time of radiosilver in rats given a single intraperitoneal injection was 40 h in whole blood, plasma, kidney, and liver; 70 h in spleen; and 84 h in brain. After exposure by inhalation, dogs cleared 59% of an administered dose of radiosilver-110m from the lungs in 1.7 days and from the liver in 9 days (Fowler and Nordberg 1986). The mean daily intake of silver in humans is about 88 µg; about 60 µg is excreted daily in the feces (Smith and Carson 1977). In humans, the whole-body effective half-time of persistence was 43 days (EPA 1980). The biological half-time of silver in the lungs of an exposed person was about 1 day; in liver it was 52 days (Fowler and Nordberg 1986). In humans, 80% of the retained silver in lung was cleared in about 1 day; 50% of the remainder was usually cleared in 3 days (EPA 1980). In persons who had accidentally inhaled radiosilver-110m, most of the inhaled silver had a half-time persistence of about 1 day, probably because of rapid mucociliary clearance, swallowing, and fecal excretion; most of the absorbed radiosilver translocated to the liver (EPA 1980).

Silver interacts competitively with selenium, vitamin E, and copper and induces signs of deficiency in animals fed adequate diets or aggravates signs of deficiency when diets lack one or more of these nutrients; antagonistic effects of silver have been described in dogs, pigs, rats, sheep, chicks, turkey poults, and ducklings (EPA 1980). Conversely, the addition of selenium, copper, or vitamin E to diets of turkey poults decreased the toxicity of diets containing 900 mg Ag/kg (Fowler and Nordberg 1986). Dietary administration of silver acetate antagonized selenium toxicity; silver prevented growth depression and death in chicks fed diets containing excess selenium (EPA 1980). The addition of selenium to the diets of rats exposed to silver in drinking water prevented growth retardation but increased the concentration of silver in liver and kidneys (Fowler and Nordberg 1986). Silver deposits in rat liver, kidneys, and other internal organs were in the form of sulfides; under high selenium exposure, the sulfur can be replaced with selenium (PHS 1990) and formation of silver selenide deposits in the liver may be considered a silver detoxification process (EPA 1980).

Concentrations in Field Collections

General

Silver is comparatively rare in the earth's crust—67th in order of natural abundance of elements; the crustal abundance is an estimated 0.07 mg/kg and predominantly concentrated in basalt (0.1 mg/kg) and igneous rocks (0.07 mg/kg; Heyl et al. 1973). Silver concentrations in nonbiological materials tend to be naturally elevated in crude oil and in water from hot springs and steam wells. Anthropogenic sources associated with the elevated concentrations of silver in nonliving materials include smelting, hazardous waste sites, cloud seeding with silver iodide, metals mining, sewage outfalls, and especially the photoprocessing industry. Silver concentrations in biota were greater in organisms near sewage outfalls, electroplating plants, mine wastes, and silver-iodide seeded areas than in conspecifics from more distant sites.

Nonbiological Materials

Maximum concentrations of total silver recorded in selected nonbiological materials were 36.5 ng/m^3 in air near a smelter in Idaho; 2.0 µg/m^3 in atmospheric dust; 0.1 µg/L in oil well brines; 4,500 ng/L in precipitation from clouds seeded with silver iodide; 6.0 µg/L in groundwater near a hazardous waste site; 8.9 µg/L in seawater from Galveston Bay, Texas; 260 µg/L in the Genesee River, New York—the recipient of photoprocessing wastes; 300 µg/L in steam wells; 300 ng/L in treated photoprocessing wastewaters; 31 mg/kg in some Idaho soils; 43 mg/L in water from certain hot springs; 50 mg/kg in granite; as much as 100 mg/kg in crude oils; 150 mg/kg in some Genesee River sediments; and 27,000 mg/kg in some solid wastes from photoprocessing effluents (Table 4). It is emphasized that only a small portion of the total silver in each of these compartments is biologically available. For example, typical publicly owned treatment works receiving photoprocessing effluents show silver removal efficiencies greater than 90%; the mean concentration of free silver ion present in the effluents from these plants ranged from 0.001 to 0.07 µg/L (Lytle 1984; Bober et al. 1992).

Table 4. Silver concentrations in representative nonbiological materials.

Table 4. Material, units of concentration, and other variables	**Concentration[a]**	**Reference[b]**
Air, ng/m^3		
Chicago, 1969	4.3	1
Heidelberg, Germany; April 1971	4.2	1
Indiana, industrialized area	1-5	2
Industrialized areas	7.0	3
Kellogg, Idaho, near smelter, 1977	10.5; Max. 36.5	1, 2
Niles, Michigan; June 1969	1.0	1
Rural areas		
Silver-iodide cloud-seeding area	1.0	1
Areas not cloud-seeded	0.04-0.17	2,3
San Francisco, 1970	0.15	1
U.S. national parks	0.012-0.19	2
Vicinity of lead smelters	Max. 175	3
Vicinity of silver iodide ground-based cloud-seeding generator		

Table 4. Material, units of concentration, and other variables	**Concentration[a]**	**Reference[b]**
At generator site	>10,000	1
>50 m from site	0.1	1
Washington, D.C. 1974	1.1	1
Atmospheric dust, µg/m^3		
Northern hemisphere	2.0	4
Drinking water, solid residues, mg/kg		
United States	0.08 (0.01-0.20)	1
Fossil fuels, mg/kg		
Coal fly ash	Max. 10-15	2, 5
Crude oil	Max. 100	3
Fuel oil, residual	Max. 0.12	3
Freshwater, µg/L		
Amazon River, South America	0.23	3
Genesee River, New York; receives photoprocessing wastes; 1973		
June	90-260	1
Winter	20	1
Patuxent River, Maryland	0.08-0.1	6
Rhone River, Europe	0.38	3
United States		
Dissolved	Usually <0.2	1, 3
Rivers	0.3 (0.09-0.55)	1, 3, 5
Surface waters	2.6	3
Tap water	2.2 (0.3-5.0)	1, 5
Tap water	Max. 26	3
Groundwater, µg/L		
Near hazardous waste site	6.0	2
Noncontaminated site	<0.5	4
Hot springs, µg/L	Max. 43,000	1
Oil well brines, µg/L	0.1	1
Precipitation, ng/L		
From seeding clouds with silver iodide	Usually 10-300; Max. 4,500	1, 2
From non-seeded clouds	Usually 0.0-20; Max. 216	1, 2

Table 4. Material, units of concentration, and other variables	**Concentration[a]**	**Reference[b]**
Rock, mg/kg		
Granite, igneous	Max. 50	5
Seawater, µg/L		
Near shore	0.25 (0.06-2.9)	1, 2, 5
Open ocean	0.00004-0.0025	3, 7
Galveston Bay, Texas, 1989		
Dissolved	3.2 (0.2-8.9)	8
Particulate	2.8 (0.7-5.9)	8
Sediments, mg/kg		
Ireland, Cork Harbour; intertidal sites, February 1990	<0.05	9
United Kingdom		
19 estuaries	0.07-4.1	7
Contaminated vs. uncontaminated estuaries	>1 vs. <0.1	7
United States		
Marine sediments, near Pacific coast cities	1.5-3.5	4
New York, Genesee River, 1973; receives photoprocessing effluents	150	1
Puget Sound, Washington, August, 1982		
0-20 cm depth	Max. 0.67	10
51-75 cm depth	Max. 0.55	10
110-175 cm depth	Max. 0.27	10
195-265 cm depth	Max. 0.07	10
San Francisco Bay	Max. >10	7
Southern California coastal basins, contaminated by wastewater	14-20	5
Soils, mg/kg		
Canada	0.l3	2
Earth's crust	0.1	2
Hazardous waste site	4.5	2
Kellogg, Idaho	20.0 (3.2-1.0)	2
Michigan		

Table 4. Material, units of concentration, and other variables	**Concentration[a]**	**Reference[b]**
Agricultural	0.19	2
Industrial	0.37	
Residential	0.13	2
Solid wastes, mg/kg		
Municipal wastes	3.0 (<3-7)	2
Municipal and industrial wastes	15-120	2
Photoprocessing effluents	450-27,000	2
Sewage sludge	225-960	2
Steam wells		
Water, µg/L	Max. 300	1
Residue, mg/L	Max. 13,000	1
Wastewater, µg/L		
Agricultural drainage water	Max. 1	1
Entering southern California coastal basins	Max. 30	5
Municipal wastewater	0.05-45.0	1
Sewage sludge, United States	5-150	5
Photoprocessing wastes, treated	70.0 (20-300)	2, 11

[a]Concentrations are shown as mean, range (in parentheses), and maximum (Max.).

[b]1, U.S. Environmental Protection Agency (EPA) 1980; 2, U.S. Public Health Service (PHS) 1990; 3, Smith and Carson 1977; 4, Freeman 1979; 5, Fowler and Nordberg 1986; 6, Connell et al. 1991; 7, Bryan and Langston 1992; 8, Morse et al. 1993; 9, Berrow 1991; 10, Bloom and Crecelius 1987; 11, National Association of Photographic Manufacturers 1974.

Silver is usually found in extremely low concentrations in natural waters because of its low crustal abundance and low mobility in water (EPA 1980). One of the highest silver concentrations recorded in fresh water, 38 µg/L, occurred in the Colorado River at Loma, Colorado, downstream of an abandoned gold-copper-silver mine, an oil shale extraction plant, a gasoline and coke refinery, and a uranium processing facility (EPA 1980). The maximum recorded value of silver in tap water in the United States was 26 µg/L—significantly higher than finished water from the treatment plant (maximum of 5.0 µg/L)—because of the use of tin-silver solders for joining copper pipes in the home, office, or factory (EPA 1980).

In general, silver concentrations in surface waters of the United States decreased between 1970-74 and 1975-79, although concentrations increased in the north Atlantic, Southeast, and lower Mississippi basins (PHS 1990). About 30 to 70% of the silver in surface waters may be ascribed to suspended particles (Smith and Carson 1977), depending on water hardness or salinity. For example, sediments added to solutions containing 2 µg Ag/L had 74.9 mg Ag/kg DW sediment after 24 h in freshwater, 14.2 mg/kg DW at 1.5% salinity and 6.9 mg/kg DW at 2.3% salinity (Sanders and Abbe 1987). Riverine transport of silver to the ocean is considerable: suspended materials in the Susquehanna River, Pennsylvania—containing as much as 25 mg silver/kg—result in an estimated transport of 4.5 metric tons of silver to the ocean each year (EPA 1980).

Emissions of silver from coal-fired power plants may lead to accumulations in nearby soils (Fowler and Nordberg 1986). Silver in soils is largely immobilized by precipitation to insoluble salts and by complexation or adsorption by organic matter, clays, and manganese and iron oxides (Smith and Carson 1977).

Silver can remain attached to oceanic sediments for about 100 years under conditions of high pH, high salinity, and high sediment concentrations of iron, manganese oxide, and organics (Wingert-Runge and Andren 1994). Estuarine sediments that receive metals, mining wastes, or sewage usually have higher silver concentrations (>0.1 mg/kg DW) than noncontaminated sediments. Silver is tightly bound by sewage sludge, and elevated silver concentrations in sediments are often characteristic of areas near sewage outfalls. In the absence of sewage, silver in oxidized sediments is associated with oxides of iron and with humic substances (Bryan and Langston 1992). Sediments in the Puget Sound, Washington, were significantly enriched in silver, in part from human activities; concentrations were higher in fine-grained particles (Bloom and Crecilius 1987). Marine annelids and clams accumulate dissolved and sediment-bound forms of silver. Uptake of silver from sediments by marine polychaete annelids decreased in sediments with high concentrations of humic substances or copper but increased in sediments with elevated concentrations of manganese or iron (Bryan and Langston 1992).

Plants and Animals

Maximum concentrations of total silver recorded in field collections of living organisms (Table 5), in milligrams silver per kilogram dry weight, were 1.5 in liver of marine mammals, 2 in liver and 6 in bone of trout from ecosystems receiving precipitation from silver-iodide seeded clouds, 7 in kidneys and 44 in liver of birds from a metals-contaminated area, 14 in marine algae and macrophytes, 30 in whole annelid worms from San Francisco Bay, 72 in skin of humans afflicted with argyria, 110 in whole mushrooms, 133 to 185 in soft parts of clams and mussels near sewage and mining waste outfalls, and 320 in whole gastropods from South San Francisco Bay. Silver concentrations in conspecifics from areas remote from anthropogenic contamination were usually lower by one or more orders of magnitude (Table 5).

Silver is a normal trace constituent of many organisms (Smith and Carson 1977). In terrestrial plants, silver concentrations are usually less than 1.0 mg/kg ash weight (equivalent to less than 0.1 mg/kg DW) and are higher in trees, shrubs, and other plants near regions of silver mining; seeds, nuts, and fruits usually contain higher silver concentrations than other plant parts (EPA 1980). Silver accumulations in marine algae (max. 14.1 mg/kg DW) are due mainly to adsorption rather than uptake; bioconcentration factors of 13,000 to 66,000 are not uncommon (PHS 1990).

Silver concentrations in mollusks vary widely between closely related species and among conspecifics from different areas (Bryan 1973; Eisler 1981; Table 5). The inherent differences in ability to accumulate silver among bivalve mollusks are well documented (oysters >> scallops >> mussels; Brooks and Rumsby 1965; Eisler 1981). The highest silver concentrations in all examined species of mollusks were in the internal organs, especially in the digestive gland and kidneys (Eisler 1981; Miramand and Bentley 1992; Table 5). Elevated concentrations of silver (5.3 mg/kg DW) in shells of limpets from uncontaminated sites suggest that silver may actively participate in carbonate mineral formation (Navrot et al. 1974), but this needs verification. In general, silver concentrations were elevated in mollusks collected near port cities and in the vicinities of river discharges (Fowler and Oregioni 1976; Berrow 1991), electroplating plant outfalls (Eisler et al. 1978; Stephenson and Leonard 1994), ocean dump sites (Greig 1979), and urban point sources including sewage outfalls (Alexander and Young 1976; Smith and Carson 1977; Martin et al. 1988; Anderlini 1992; Crecelius 1993) and from calcareous sediments rather than detrital organic or iron oxide sediments (Luoma and Jenne 1977). Season of collection (Fowler and Oregioni 1976; Sanders et al. 1991) and latitude (Anderlini 1974) also influenced silver accumulations. Seasonal variations in silver concentrations of Baltic clams (*Macoma balthica*) were associated with seasonal variations in soft tissue weight and frequently reflected the silver content in the sediments (Cain and Luoma 1990). Oysters from the Gulf of Mexico vary considerably in whole body concentrations of silver and other trace metals. Variables that modify silver concentrations in oyster tissues include the age, size, sex, reproductive stage, general health, and metabolism of the animal; water temperature, salinity, dissolved oxygen, and turbidity; natural and anthropogenic inputs to the biosphere; and chemical species and interactions with other compounds (Presley et al. 1990). Silver concentrations in whole American oysters (*Crassostrea virginica)* from the Chesapeake Bay were reduced in summer, reduced at increasing water salinities, and elevated near sites of human activity; chemical forms of silver taken up by oysters included the free ion (Ag^+) and the uncharged

$AgCl^0$ (Sanders et al. 1991; Daskalakis 1995). Declines in tissue silver concentrations of the California mussel (*Mytilus californianus*) were significant between 1977 and 1990; body burdens decreased from 10-70 mg/kg DW to less than 2 mg/kg DW and seem to be related to the termination of metal plating facilities in 1974 and decreased mass emission rates by wastewater treatment facilities (Stephenson and Leonard 1994).

Table 5. Silver concentrations (milligrams of silver per kilogram fresh weight [FW], dry weight [DW], or ash weight [AW]) in field collections of selected plants and animals.

Table 5. Taxonomic group, organism, and other variables	**Concentration[a] (mg/kg)**	**Reference[b]**
Algae, macrophytes, and higher plants		
Marine algae and macrophytes, 24 species		
9 species	<0.1-2.0 DW	1
11 species	2.1- 10.0 DW	1
4 species	10.1-14.1 DW	1
Higher terrestrial plants	0.2-<1.0 AW	2, 3
Cnidarians		
Corals, 34 species	<1.0 DW	1
Various, 10 species	Max. 0.1 DW	1
Mollusks		
Cephalopods, 7 species; digestive gland	3.0-46.0 DW	4
Cephalopods, French coast of English Channel, October 1987		
Octopus, *Eledone cirrhosa*		
Digestive gland	2.0-4.4 DW	4
Digestive tract	0.5 DW	4
Other tissues	<0.3 DW	4
Whole	0.8 DW	4
Cuttlefish, *Sepia officinalis*		
Digestive gland	4.9-7.4 DW	4
Kidney	0.7 DW	4
Other tissues	<0.3 DW	4
Whole	0.7 DW	4
Clam, *Corbicula* sp.; San Francisco Bay, 1983-86; soft parts	0.07-0.2 DW	5
Eastern oyster, *Crassostrea virginica*; soft parts		
Connecticut	6.1 FW	6

Table 5. Taxonomic group, organism, and other variables	Concentration[a] (mg/kg)	Reference[b]
East coast	0.3-5.0 DW	7, 55
Georgia	28.0-82.0 DW	8
Gulf coast	0.6-6.0 DW; Max. 7.0 DW	7, 9, 10
Louisiana	5.5 DW	11
Maryland, Chesapeake Bay, 1986-88	2.0-6.0 DW	12
Northeast coast	0.8-2.3 FW	13
Red abalone, *Haliotis rufescens*; California		
Digestive gland	14.0-60.0 DW	14
Foot	1.0-44.0 DW	14
Gills	13.0-129.0 DW	14
Mantle	16.0-54.0 DW	14
Periwinkle, *Littorina littorea*; soft parts; Looe estuary, U.K. vs. uncontaminated site,1988	10.7 (3.1-17.4) DW vs. 4.1 (3.4-5.0) DW	15
Baltic clam, *Macoma balthica*; soft parts; San Francisco Bay		
Near sewage outfall	32.0-133.0 DW	11, 16
Reference site	<1.0 DW	11
Mollusks, marine; edible tissues; 18 species		
10 species	<0.1 FW	17
5 species	0.1-0.3 FW	17
3 species	0.3-0.7 FW	17
Mollusks; south San Francisco Bay, 1982; soft parts		
Mud snail, *Nassarius obsoletus*	Max. 320.0 DW	16
Bent-nose macoma (clam), *Macoma nasuta*	Max. 5.1 DW	16
Softshell clam, *Mya arenaria*	Max. 34.0 DW	16
Clam, *Tapes japonica*	Max. 65.0 DW	16
California mussel, *Mytilus californianus;* soft parts		
Bodega Bay, California; 1976-78 vs. 1986-88	0.15 DW vs. 0.1 DW	18
California coast, 1977-81 vs. 1989-91	Max. 10.0-70.0 DW vs. <2.0 DW	19
San Diego Bay near municipal wastewater outfall vs. reference sites in Baja California and northern California	59.0 DW vs. 0.08-0.22 DW	20
San Francisco Bay, 1982; North Bay vs. South Bay	0.04-0.16 DW vs. 0.7-2.9 DW	16

Table 5. Taxonomic group, organism, and other variables	Concentration[a] (mg/kg)	Reference[b]
Common mussel, *Mytilus edulis*		
Shell	0.1-6.3 DW	21, 22, 23
Soft parts		
Europe	0.1-6.0 DW	1
Ireland, February 1990; contaminated sites (Cork Harbour) vs. reference sites (east coast of Ireland)	0.8-4.3 DW vs. <0.05-1.0 DW	24
Rhode Island, Narragansett Bay, 1976-78 vs. 1986-88	0.20 DW vs. 0.22 DW	18
United States	0.04-2.3 DW	1
East coast; rural sites vs. urban areas	0.3 DW vs. Max. 2.0 DW	55
West coast; rural sites vs. urban areas	0.1 DW vs. Max. 5.0 DW	55
Mussel, *Mytilus edulis aoteanus*; soft parts; New Zealand, 1986-87; various distances from sewage outfall		
50 m	5.3-7.7 DW	25
100 m	4.2 DW	25
200 m	3.9 DW	25
750 m	3.5-4.1 DW	25
1,500 m	2.9 DW	25
3,000 m	2.7-3.4 DW	25
Oyster, *Ostrea equestris*; soft parts, United States		
East coast	18.9 DW	7
Gulf coast	0.7-1.6 DW	7
Oyster, *Ostrea sinuata*; New Zealand		
Foot, gills, soft parts	0.7-1.1 DW	26
Gonad, mantle	0.2 DW	26
Intestine	2.9 DW	26
Kidney	4.8 DW	26
Muscle, shell	<0.1 DW	26
Limpet, *Patella vulgata*; Israel, 1973; soft parts vs. shell		
Near sewage outfall	6.7 DW vs. 5.7 DW	27
Reference site 80 km north of outfall	1.2 DW vs. 5.3 DW	27

Table 5. Taxonomic group, organism, and other variables	Concentration[a] (mg/kg)	Reference[b]
Mussel, *Perna canaliculus*; soft parts; New Zealand, 1986-87; distance from sewage outfall		
200 m	35.0-113.0 DW	25
750 m	49.0-85.0 DW	25
1,500-3,000 m	8.0-13.0 DW	25
False quahog, *Pitar morrhuanus* (formerly Widgeon clam, *Pitar morrhuana*); soft parts; near Rhode Island electroplating plant	1.2-4.6 DW	28
Sea scallop, *Placopecten magellanicus*; soft parts		
Ocean disposal site	Max. 9.1 DW	11
Reference site	<0.1 DW	11
Clam, *Potamocorbula amurensis*; San Francisco Bay (0.006 µg Ag/L); 1991-92; soft parts	2.2 (0.3-7.0) DW; BCF of about 366,000	57
Oysters, *Saccostrea* spp.; Australia, 1980-83; soft parts	Max. 0.4 FW	29
Clam, *Scrobicularia plana*		
Digestive gland	0.8 DW	4
Kidney	0.4 DW	4
Soft parts		
Reference sites	0.2-1.5 (0.03-2.1) DW	15, 30
Silver-contaminated estuary	4.0-5.8 (1.1- 185.0) DW	15, 31
Bryozoans		
Bryozoan, *Victorella* sp; whole; Chesapeake Bay, Maryland	11.5 DW	32
Crustaceans		
Amphipods, whole; Antarctica, February- March 1989	1.2 (0.7-1.4) DW	33
Rock crab, *Cancer irroratus*		
Digestive gland	2.1-3.4 FW; 6.3 DW	34, 35
Muscle	0.2-0.8 FW; 0.2 DW	34, 35
Crustaceans, edible tissues, 16 species		
8 species	<0.1 FW	17
5 species	0.1-0.2 FW	17
3 species	0.3-0.5 FW	17
Barnacle, *Elminius modestus*; pyrophosphate granules	10.5 (9.7-11.3) DW	36

Table 5. Taxonomic group, organism, and other variables	Concentration[a] (mg/kg)	Reference[b]
American lobster, *Homarus americanus*; muscle	0.4-0.5 DW	37
Shrimps, unidentified		
Exoskeleton	1.1 DW	38
Muscle	0.2 DW	38
Annelids		
Polychaete annelid, *Marphysa sanguinea*; whole; San Francisco Bay, 1982	Max. 5.5 DW	16
Sandworm, *Nereis diversicolor*; whole	5.2 (0.7-30.0) DW	31
Echinoderms		
Various, 9 species	Usually <0.3 DW; Max. 0.6 DW	1
Starfish, *Luidia clathrata*; Tampa Bay, Florida vs. Gulf of Mexico; 1992		
Body wall	0.26-0.84 DW vs. 0.67 DW	39
Pyloric caeca	0.4-1.1 DW vs. 0.17 DW	39
Tunicates		
Whole, 2 species; New Zealand	Max. 0.03 DW; Max. 0.005 FW	1
Tunicate, *Cynthia claudicans*; soft parts, Greece	0.9 FW; 4.8 DW	1
Fishes and Elasmobranchs		
Blackfin icefish, *Chaenocephalus aceratus*; Antarctica, February-March 1989		
Liver	0.05 (0.04-0.05) DW	33
Muscle	0.01 (0.008-0.012) DW	33
Freshwater fishes, whole;United States, 1975-79	0.225 (0.004-1.9) FW	11
Atlantic cod, *Gadus morhua*; Newfoundland, November 1990-March 1991; females		
Liver	Max. 1.49 DW; Max. 0.44 FW	40
Muscle	Max. 0.3 DW; Max. 0.02 FW	40
Ovaries	Max. 0.32 DW; Max. 0.04 FW	40
Marine fishes		
Liver		
66 species	<0.01 FW	17
12 species	(0.1-0.3) FW	17
4 species	(0.3-0.6) FW	17
Muscle		

Table 5. Taxonomic group, organism, and other variables	Concentration[a] (mg/kg)	Reference[b]
158 species	<0.1 FW	17
1 species	(0.1-0.2) FW	17
Scales, 7 species	(0.1-0.3) DW	41
Whole		
10 species	<0.1 FW	17
7 species	(0.1-0.2) FW	17
Striped bass, *Morone saxatilis*		
Liver	0.08 FW	42
Muscle	0.003 FW	42
Smooth dogfish, *Mustelus canis*; New York Bight		
Liver	Max. 0.3 FW	43
Muscle	<0.1 FW	43
Hump rock cod, *Notothenia gibberifrons*; Antarctica, February-March 1989; muscle	0.014 (0.012-0.016) DW	33
Winter flounder, *Pleuronectes americanus*		
Liver	<0.1-0.8 FW	43
Muscle	<0.1 FW	43
Windowpane flounder, *Scophthalmus aquosus*		
Liver	<0.1-0.5 FW	35
Muscle	<0.1 FW	35
Birds		
Antarctica, February-March 1989		
Arctic giant-petrel, *Macronectes giganteus*; muscle	0.018 (0.017-0.02) DW	33
Imperial shag, *Phalacrocorax atriceps*; muscle	0.01 DW	33
Adelie penguin, *Pygoscelis adeliae*		
Liver	0.02 DW	33
Muscle	0.01 DW	33
Chinstrap penguin, *Pygoscelis antarctica*		
Feces	0.18 (0.13-0.22) DW	33
Liver	0.05 DW	33
Muscle	0.009 DW	33

Table 5. Taxonomic group, organism, and other variables	Concentration[a] (mg/kg)	Reference[b]
Gentoo penguin, *Pygoscelis papua*		
Liver	0.43 (0.41-0.46) DW	33
Muscle	0.01 DW	33
Greater scaup, *Aythya marila*		
San Francisco Bay, March-April 1982; liver	1.0 (0.4-3.1) DW	44, 45
British Columbia, Canada		
Diet	0.006-0.029 FW	46
Liver	0.04-0.32 FW	46
Ruffed grouse, *Bonassa umbellus*; primary feathers; Virginia, 1977-79		
Adults	<0.01 DW	47
Immatures	<0.01 DW	47
Lesser black-backed gull, *Larus fuscus*; Norway; metals-contaminated area		
Kidney	1.0 DW	22
Liver	2.0 DW	22
Muscle	3.0 DW	22
Surf scoter, *Melanitta perspicillata*		
British Columbia		
Diet	0.004-0.026 FW	46
Liver	0.03-0.14 FW	46
San Francisco Bay, March-April 1982		
Kidney	Max. 3.7 DW	44
Liver	0.9 (0.3-3.7) DW	44
Common eider, *Somateria mollissima*; Norway; metals-contaminated area		
Eggs	1.0 DW	22
Kidneys	7.0 DW	22
Liver	44.0 DW	22
Muscle	2.0 DW	22
Mammals		
Human, *Homo sapiens*		
Daily intake, 70-kg individual, whole body		

Table 5. Taxonomic group, organism, and other variables	Concentration[a] (mg/kg)	Reference[b]
All sources (35-88 µg)	0.0005-0.00125 FW	11
Air (0.023 µg)	0.0000033 FW	11
Drinking water (20-100 µg)	0.00285-0.00143 FW	11
Food (4.5 µg)	0.000064 FW	11
Diet		
Beef liver	0.005-0.194 FW	3
Beef muscle	0.004-0.024 FW	3, 11
Cereals and grains	0.008 (0.0-140.0) FW	11
Cigarettes; filter vs. nonfilter	0.27 FW vs. 0.18 FW	11
Crustaceans	2.0 DW	3
Dairy products	<0.06 FW	11
Fruits	<0.05 FW	11
Leafy vegetables	0.007 (0.0-0.04) FW	11
Meat, fish, poultry	0.01 (0.0-87.0) FW	11
Milk (cow)	0.027-0.059 FW	3, 11
Mushrooms	"Up to several hundred" DW	3
Oils and fats	<0.03 FW	11
Pork and mutton	0.006-0.012 FW	3, 11
Sugar	0.002-0.03 FW	3
Tea	0.2-2.0 DW	3, 11
Trout	0.48-0.68 DW	3
Typical diet	0.0091 DW	11
Wheat	0.5 DW	3
Tissues and organs		
Abnormal (argyria)		
Skin	63.0-72.0 DW	48
Normal		
Kidney	0.001 FW; 0.4 DW	48
Liver	0.006 FW; 0.7 DW	48
Lung	0.0001 FW	48
Skin	0.035 DW	48

Table 5. Taxonomic group, organism, and other variables	Concentration[a] (mg/kg)	Reference[b]
Spleen	2.7 DW	48
Whole body	0.05 FW; <10.0 DW	2
Leopard seal, *Hydrurga leptonyx*; Antarctic, February-March 1989		
Kidney	0.15 DW; Max. 0.24 DW	49
Liver	0.99 DW; Max. 1.55 DW	49
Muscle	0.01 DW; Max. 0.017 DW	49
Stomach contents	0.22 (0.20-0.24) DW	49
Weddell seal, *Leptonychotes weddelli*; Antarctic, February-March 1989		
Kidney	0.10 DW; Max. 0.29 DW	49
Liver	0.73 DW; Max. 0.94 DW	49
Muscle	Max. 0.012 DW	49
Crabeater seal, *Lobodon carcinophagus*; Antarctic, February-March 1989		
Kidney	0.06 DW; Max. 0.17 DW	49
Liver	0.81 DW; Max. 1.36 DW	49
Muscle	0.01 DW; Max. 0.022 DW	49
Terrestrial mammals, various species, liver	<50.0 AW	2
Polar bear, *Ursus maritimus*; Northwest Territories, Canada, 1984; liver	0.21-0.54 DW	50
California sea lion, *Zalophus californianus*; recent mothers; liver		
Mothers with normal pups	0.5 DW	51
Mothers giving birth to premature pups	0.4 DW	51
Integrated Studies		
Alpine lake, Colorado, 1973-74. Silver iodide (43 kg), equivalent to 19.7 kg silver, released into system from local cloud-seeding practices between 1963 and 1973		
Lake water, 1973 vs. 1974		
Bottom	0.00022 (Max. 0.00063) FW vs. 0.00044 (Max. 0.00122) FW	52
Surface	0.00031 (Max. 0.0009) FW vs. 0.00071 (Max. 0.00134) FW	52
Cutthroat trout, *Oncorhynchus clarki*; age 1year vs. age 3 years		
Bone	5.8 DW vs. 2.6 DW	52

Table 5. Taxonomic group, organism, and other variables	Concentration[a] (mg/kg)	Reference[b]
Liver	2.3 DW vs. 1.4 DW	52
Muscle	0.1 DW vs. 0.4 DW	52
Skin	0.2 DW vs. 0.4 DW	52
Arabian Sea, near Pakistan, 1987-88		
Sediments	0.53 DW	53
Water	0.000015 FW; Max. 0.000033 FW	53
Seaweeds, whole, 4 species	0.40-0.76 FW	53
Shrimps, edible portions, 2 species	0.25-0.29 FW	53
Fish, muscle, 3 species	0.29-0.53 FW	53
Calcasieu River, Louisiana		
Periphyton, whole	2.1 DW	54
Hooked mussel, *Ischadium recurvum* (formerly *Brachidontes exustus)* soft parts	0.4 DW	54
American oyster, *Crassostrea virginica*; soft parts	1.0 DW	54
Zooplankton, whole	0.8 DW	54
Blue crab, *Callinectes sapidus*; muscle	0.1 DW	54
Shrimps, 2 species; muscle	0.04 DW	54
Fish, 7 species: muscle	0.1 DW	54
Poland, 1989-92		
Mushrooms, *Agaricus campestris*, whole	9-62 (6-110) DW	56
Soils	0.1-0.95 DW; Max. 1.4 DW; BCF values for silver by mushrooms from soils ranged between 60 and 330	56

[a]Concentrations are shown as mean, range (in parentheses), and maximum (Max.).

[b]1, Eisler 1981; 2, Smith and Carson 1977; 3, U.S. Environmental Protection Agency 1980; 4, Miramand and Bentley 1992; 5, Luoma et al. 1990; 6, Thurberg et al. 1974; 7, Goldberg et al. 1978; 8, Windom and Smith 1972; 9, Morse et al. 1993; 10, Presley et al. 1990; 11, U.S. Public Health Service 1990; 12, Sanders et al. 1991; 13, Greig and Wenzloff 1978; 14, Anderlini 1974; 15, Truchet et al. 1990; 16, Luoma and Phillips 1988; 17, Hall et al. 1978; 18, Lauenstein et al. 1990; 19, Stephenson and Leonard 1994; 20, Martin et al. 1988; 21, Segar et al. 1971; 22, Lande 1977; 23, Graham 1972; 24, Berrow 1991; 25, Anderlini 1992; 26, Brooks and Rumsby 1965; 27 Navrot et al. 1974; 28, Eisler et al. 1978; 29, Talbot 1985; 30, Bryan and Uysal 1978; 31, Bryan and Hummerstone 1977; 32, Connell et al. 1991; 33, Szefer et al. 1993; 34, Greig et al. 1977a; 35, Greig et al. 1977b; 36, Pullen and Rainbow 1991; 37, Greig 1975; 38, Bertine and Goldberg 1972; 39, Lawrence et al. 1993; 40, Hellou et al. 1992; 41, Papadopoulu and Kassimati 1977; 42, Heit 1979; 43, Greig and Wenzloff 1977a; 44, Ohlendorf et al. 1986; 45, Bryan and Langston 1992; 46, Vermeer and Peakall 1979; 47, Scanlon et al. 1980; 48, Fowler and Nordberg 1986; 49, Szefer et al. 1994; 50, Braune et al. 1991; 51, Martin et al. 1976; 52, Freeman 1979; 53, Tariq et al. 1993; 54, Ramelow et al. 1989; 55, Crecelius 1993; 56, Falandysz and Danisiewicz 1995; 57, Brown and Luoma 1995.

Among arthropods, pyrophosphate granules isolated from barnacles have the capability to bind and effectively detoxify silver and other metals under natural conditions (Pullen and Rainbow 1991). In a Colorado alpine lake, silver concentrations in caddisflies and chironomid larvae usually reflected silver concentrations in sediments; seston, however, showed a high correlation with lakewater silver concentrations from 20 days earlier (Freeman 1979).

In other studies, silver concentrations in fish muscles rarely exceeded 0.2 mg/kg DW and usually were less than 0.1 mg/kg fresh weight (FW); livers contained as much as 0.8 mg/kg FW, although values greater than 0.3 mg/kg FW were unusual; and whole fish contained as much as 0.2 mg/kg FW (Table 5). Livers of Atlantic cod (*Gadus morhua*) contained significantly more silver than muscles or ovaries; a similar pattern was evident in other species of marine teleosts (Hellou et al. 1992; Szefer et al. 1993; Table 5). Accumulations of silver in offshore populations of teleosts is unusual, even among fishes collected near dump sites impacted by substantial quantities of silver and other metals. For example, of seven species of marine fishes from a disposal site in the New York Bight that were examined for silver content, concentrations were highest (0.15 mg/kg FW) in muscle of blue hake (*Antimora rostrata*; Greig et al. 1976). Similarly, the elevated silver concentration of 0.8 mg/kg FW in liver of winter flounder (*Pleuronectes americanus*; Table 5) was from a specimen from the same general area (Greig and Wenzloff 1977b).

Silver concentrations in muscles of Antarctic birds were low (0.01 mg/kg DW) when compared to livers (0.02-0.46 mg/kg DW) or feces (0.18 mg/kg DW; Szefer et al. 1993; Table 5). Silver concentrations in avian tissues, especially in livers, were elevated in the vicinity of metals-contaminated areas and in diving ducks from the San Francisco Bay (Table 5). Birds with elevated concentrations of silver in tissues—as much as 44 mg/kg DW in liver in the common eider (*Somateria mollissima*)—seemed outwardly unaffected (Bryan and Langston 1992).

Silver in mammalian tissues is usually present at low or nondetectable concentrations (Klaassen et al. 1986). The concentration of silver in tissues of three species of seals collected in the Antarctic during 1989 was highest in liver (1.55 mg/kg DW; Table 5) and lowest in muscle (0.01 mg/kg DW); intermediate in value were kidney (0.29 mg/kg DW) and stomach contents (0.24 mg/kg DW; Szefer et al. 1994). The mean concentration of silver in livers from normal female California sea lions (*Zalophus calfornianus*), with normal pups, was 0.5 mg/kg DW (Martin et al. 1976; Table 5). Mothers giving birth to premature pups had only 0.4 mg Ag/kg DW liver. In general, *Zalophus* mothers delivering premature pups had lower concentrations in liver of silver, cadmium, copper, manganese, mercury, and zinc than did mothers delivering normal pups (Martin et al. 1976). Silver concentrations in tissues of Antarctic seals were related to, and possibly governed by, concentrations of other metals (Szefer et al. 1994). In muscle, silver inversely correlated with zinc; in liver, silver positively correlated with nickel, copper, and zinc; and in kidney, correlations between silver and zinc and between silver and cadmium were negative (Szefer et al. 1994). In humans, EPA (1980) states that silver is present in placentas and fetal livers, that silver concentrations in tissues increase with age, and that variations in tissue concentrations of silver are wide. The average maximum daily intake of silver from all sources by humans is 88 µg (Table 5), but very little of the silver ingested from nontherapeutic sources is retained (Smith and Carson 1977).

Lethal and Sublethal Effects

General

As discussed and documented later, free silver ion is lethal to representative species of sensitive aquatic plants, invertebrates, and teleosts at water concentrations of 1.2-4.9 µg/L. Adverse effects occur on development of trout at concentrations as low as 0.17 µg/L and on phytoplankton species composition and succession at 0.3 to 0.6 µg/L. Aquatic organisms accumulate silver from environmental sources. No data were found on effects of silver on avian or mammalian wildlife and all studied effects were on poultry and small laboratory mammals. Silver was not mutagenic, carcinogenic, or teratogenic to tested animals by normal routes of exposure. Adverse effects of silver on poultry occur at 1.8 mg/kg FW whole egg by way of injection (reduced survival), 10 mg/kg in copper-deficient diets (reduced hemoglobin), and 200 mg/kg in copper-adequate diets (growth suppression), or when the birds are given drinking water containing 100 mg Ag/L (liver necrosis). Effects of silver on sensitive species of mammals include death at 13.9-20.0 mg/kg BW by intraperitoneal injection, histopathology of kidney and brain at 250-450 µg Ag/L drinking water, tissue accumulations at 6 mg/kg diet, and

liver necrosis when fed diets containing more than 130 mg/kg. In humans, generalized argyria seems to be declining, which may be due to improved work conditions.

Terrestrial Plants

Smith and Carson (1977) report that sprays containing 9.8 mg dissolved Ag/L kill corn (*Zea mays*), and sprays containing 100-1,000 mg dissolved Ag/L kill young tomato (*Lycopersicon esculentum*) and bean (*Phaseolus* spp.) plants. Hirsch et al. (1993) planted seeds of corn, lettuce (*Lactuca sativa*), oat (*Avena sativa*), turnip (*Brassica rapa*), soybean (*Glycine max*), spinach (*Spinacia oleracea*), and Chinese cabbage (*Brassica* spp.) in soils amended with silver sulfide and sewage sludge to contain 10, 50, or 100 mg Ag/kg DW soil. All plants germinated and most grew normally at the highest soil concentration of silver tested. But growth of Chinese cabbage and lettuce was adversely affected at 10 mg Ag/kg DW soil and higher. Silver concentrations in edible portions from all plants at all soil levels of silver tested, except lettuce, were less than 80 µg/kg DW. Lettuce grown in soil containing 100 mg Ag/kg DW had about 1.2 mg Ag/kg DW (Hirsch et al. 1993).

Aquatic Organisms

In fish and amphibian toxicity tests with 22 metals and metalloids, silver was the most toxic tested element as judged by acute LC50 values (Birge and Zuiderveen 1995). In solution, ionic silver is extremely toxic to aquatic plants and animals (Nehring 1976; Nelson et al. 1976; Calabrese et al. 1977a; Gould and MacInnes 1977; Smith and Carson 1977; EPA 1980; Buhl and Hamilton 1991; Bryan and Langston 1992), and water concentrations of 1.2-4.9 µg/L killed sensitive species of aquatic organisms, including representative species of insects, daphnids, amphipods, trout, flounders, sticklebacks, guppies, and dace (Table 6). At nominal water concentrations of 0.5-4.5 µg/L, accumulations in most species of exposed organisms were high and had adverse effects on growth in algae, clams, oysters, snails, daphnids, amphipods, and trout; molting in mayflies; and histopathology in mussels (Table 6). Among all tested species, the individuals most sensitive to silver were the poorly nourished and young and those exposed to low water hardness or salinity (Smith and Carson 1977; EPA 1980; Le Blanc et al. 1984; Table 6). It is emphasized that silver-induced stress syndromes vary widely among animal classes. Among marine organisms, for example, silver ion was associated with respiratory depression in marine gastropods and cunners (*Tautagolabrus adspersus*), a teleost; however, silver ion increased oxygen consumption in six species of bivalve mollusks (Gould and MacInnes 1977).

Sensitive aquatic plants accumulated silver from water containing as little as 2 µg Ag/L to whole-cell burdens as high as 58 mg Ag/kg DW; grew poorly at 3.3-8.2 µg Ag/L during exposure for 5 days; and died at concentrations greater than 130 µg Ag/L (Table 6). Some metals seem to protect aquatic plants against adverse effects of silver. Algae in small lakes that contained elevated concentrations of metals, especially copper and nickel, had higher tolerances to silver than conspecifics reared in the laboratory under conditions of depressed concentrations of heavy metals (EPA 1980). Species composition and species succession in Chesapeake Bay phytoplankton communities were significantly altered in experimental ecosystems continuously stressed by low concentrations (0.3-0.6 µg/L) of silver (Sanders and Cibik 1988; Sanders et al. 1990). At higher concentrations of 2-7 µg/L for 3 to 4 weeks, silver inputs caused disappearance of *Anacystis marina*, a mat-forming blue-green alga; increased dominance by *Skeletonema costatum*, a chain-forming centric diatom; and increased silver concentrations in various species of phytoplankton to 8.6-43.7 Ag mg/kg DW (Sanders and Cibik 1988). Dissolved silver speciation and bioavailability were important in determining silver uptake and retention by aquatic plants (Connell et al. 1991). Silver availability was controlled by the concentration of free silver ion (Ag^+) and the concentrations of other silver complexes, such as AgCl (Sanders and Abbe 1989). Silver uptake by phytoplankton was rapid, in proportion to silver concentration, and inversely proportional to water salinity. Silver incorporated by phytoplankton was not lost as the salinity increased, and silver associated with cellular material was largely retained in the estuary (Sanders and Abbe 1989). Diatoms (*Thalassiosira* sp.), for example, readily accumulated silver from the medium. Once incorporated, silver was tightly bound to the cell membrane, even after the cells were mechanically disrupted (Connell et al. 1991).

Table 6. Effects of silver on representative aquatic plants and animals. Concentrations are in micrograms of free silver (Ag^+) per liter of medium added at start unless indicated otherwise.

Table 6. Taxonomic group, organism, silver concentration (ppb), and other variables	Effects	Reference[a]
Bacteria, algae, and macrophytes		
Bacteria, freshwater		
Escherichia coli		
1,000-2,000 (as Ag+3)	All dead in 0.5-5.0 min	1
Streptococcus faecalis		
1,000-2,000 (as Ag^{+3})	All dead in 0.5-5.0 min	1
Various species		
100-200	All dead in 15-20 min	2
Bacteria, marine		
Isolated from tubes of deep-sea polychaete annelids		
3,000	Lowest concentration tested that inhibited growth in 50% of strains during 10-day exposure	3
20,000	Silver-resistant strains (55% of all strains tested) survived at least 24 h	3
40,000	Some strains survived during 10-day exposure	3
Alga, *Chlorella* spp.		
50-100	Growth inhibition	4
Waterweed, *Elodea canadensis*		
100	Respiration inhibited	4
Freshwater algae and macrophytes, 13 species		
30-7,500	Adverse effects	4
Freshwater plants		
26	Bioconcentration factor (BCF) of X200	2
Duckweed, *Lemna minor*		
270	Phytotoxic	4
Marine algae, 3 species		
2	After 24 h algae contained 27.8-58.6 mg silver/kg dry weight (DW) at 1% salinity, 16.4-33.4 mg/kg at 1.5% salinity, and 9.8-25.2 mg Ag/kg DW at 2.0% salinity	5

Table 6. Taxonomic group, organism, silver concentration (ppb), and other variables	Effects	Reference[a]
Marine algae, 4 species; various concentrations	BCF between X13,000 and X66,000 at equlibrium	40
Freshwater alga, *Phormidium inundatum*		
80-140	Controls growth in swimming pools	2
Marine alga, *Prorocentrum mariae-lebouriae*		
3.3	50% growth reduction in 5 days at 0.75% salinity	6
6.7	50% growth reduction in 5 days at 1.5-2.25% salinity	6
8.2	50% growth reduction in 5 days at 3% salinity	6
Marine algae, various species		
Radiosilver-110m at 3.3 microcuries/L	BCF of X1,600 to X2,800 in 38 days	7
Marine plants		
60	BCF of X200	2
Alga, *Scendesmus* spp.		
100-200	100% growth inhibition	4
Marine diatom, *Skeletonema costatum*		
5.9	50% growth reduction in 5 days at 0.75% salinity	6
15.4	50% growth reduction in 5 days at 1.5% salinity	6
20.0	50% growth reduction in 5 days at 2.25-3.0% salinity	6
130-170	50% reduction in cell numbers in 96 h	4
Protozoans		
Ciliate, *Fabrea salina*; held in seawater solution containing radiosilver-110m	Bioconcentration factor (volume/volume basis) of 7,000 to 40,000 within 24 h	39
Protozoan, *Spirostomum ambiguum*		
8.8	LC50(24 h) at 2.8 mg $CaCO_3$/L	8
15.3	LC50(24 h) at 250 mg $CaCO_3$/L	8
Nematodes		
Free-living nematode, *Caenorhabditis elegans*		
102 (95% confidence interval [CI] of 10 to 4,980)	LC50(96 h)	9
5,000, (95% CI of 3,000 to 10,000)	LC50(96 h)	9

Table 6. Taxonomic group, organism, silver concentration (ppb), and other variables	Effects	Reference[a]
Mollusks		
Bay scallop, *Argopecten irradians*, juveniles		
22	Oxygen consumption elevated after 96 h	10
33	LC50(96 h);survivors with elevated silver concentrations	10
Freshwater snail, *Australorbis* sp.		
30-100	Inhibited feeding and coordination	2
Bivalves, 4 species		
10	Elevated oxygen consumption after exposure for 30-90 days	10, 11
Scallop, *Chlamys varia*, adults		
20	After 14 days soft parts contained 18 mg Ag/kg DW vs. 1.7 in controls	12
100	LC50(115 h)	12
Asiatic clam, *Corbicula fluminea*		
4.5	Adverse effects on growth after exposure for 21 days; residues of 1.65 mg Ag/kg FW soft parts were associated with reduced growth	13
7.8	No deaths in 21 days	13
26.0	Some deaths in 21 days	13
155 (116-208); exposed for 96 h then transferred to uncontaminated media for 96 h	LC50 for juveniles at end of observation period	13
Pacific oyster, *Crassostrea gigas*		
2-10	5% to 8% of embryos exposed for 48 h were abnormal (retarded shell growth, reduced size, erratic swimming behavior) vs. 1% in controls; no significant effect on embryogenesis	14
13.5-15.5	Significant effect on embryogenesis; 25-37% of embryos developed abnormally	14
18-32	95-98% of embryos exposed for 48 h were abnormal	14
20	Soft parts of adults exposed for 28 days contained 188.0 mg Ag/kg DW vs. 3.0 mg/kg DW in controls	12
Juveniles held in 20 µg Ag/L for 14 days then transferred to uncontaminated seawater for 23	No histopathology. During exposure, but not depuration, glycogen storage capacity was diminished. During depuration, silver concentrations	15

Table 6. Taxonomic group, organism, silver concentration (ppb), and other variables	Effects	Reference[a]
days	decreased from 31.3 mg/kg DW soft parts to 12.8 vs. <10.0 in controls. Most of the insoluble accumulated silver was sequestered as Ag_2S in amoebocytes and basement membranes	
100	LC50(209 h), adults	12
American oyster, *Crassostrea virginica*		
0.1 (controls) vs. 2; adults exposed for 14 days in large enclosures with natural phytoplankton assemblages	In controls, phytoplankton had 0.03 mg Ag/kg DW and oyster soft parts 0.8 mg/kg DW. In the 2.0 µg/L group, phytoplankton had 8.6 mg Ag/kg DW and oysters 2.8 mg/kg DW; oyster growth rate significantly reduced	16
5 or 7; conditions as above	Phytoplankton had 24-44 mg Ag/kg DW and oysters 4.8- 6.6 mg Ag/kg DW	16, 17
5.8	LC50(48 h), embryos	10
10	LC100(48 h), embryos	4
25	LC50(12 days), juveniles	10
500-1,000	Adults exposed for 96 h had 12.4-14.9 mg Ag/kg FW in body and 34-38 mg Ag/kg FW in gills	11
Slipper limpet, *Crepidula fornicata*		
1, 5, or 10; exposed for 24 months and observed for effects on growth, reproduction, histology and accumulations	Growth reduced in the 5 and 10 µg/L groups and reproduction inhibited in the 10 µg/L group. All test groups showed deposition of silver in connective tissues and basement membranes. Maximum silver concentrations (mg/kg FW soft parts) were recorded for the controls at 12 months (2.8), for the 1.0 µg/L group at 12 months (34.0), for the 5.0 µg/L group at 6 months (54.1), and for the 10.0 µg/L group at 6 months (86.7). After 24 months, silver-exposed groups contained between 5.4 and 8 mg Ag/kg FW soft parts	18
Zebra mussel, *Dreissena polymorpha*, adults		
400	No deaths in 28 days; soft parts at 28 days contained 147-184 mg Ag/kg DW vs. 0.02-1.8 mg/kg DW in controls	12
Quahog clam, *Mercenaria mercenaria*		
21.0	LC50(48 h), embryos	10
32.4	LC50(10 days), juveniles	10
500-1,000	Adults exposed for 96 h had 0.8-1.0 mg Ag/kg FW in soft parts and 6.9-7.6 mg Ag/kg FW in gills. Controls	11

Table 6. Taxonomic group, organism, silver concentration (ppb), and other variables	Effects	Reference[a]
	(<1.5 µg/L) had 0.4 mg Ag/kg FW soft parts and 1.6 mg/kg FW in gills	
Softshell clam, *Mya arenaria*		
100	Increased oxygen consumption after 96 h	11
500	After exposure for 96 h adults had 10.4 mg Ag/kg FW soft parts vs. 0.3 mg Ag/kg FW in controls	11
1,000	All adults died within 96 h	11
Common mussel, *Mytilus edulis*		
Exposed continuously for 21 months to 0.0 (control), 1, 5, or 10 µg Ag/L from age 2.5 months (4.5 mm in shell length) and observed for growth, accumulations, and histopathology	No effect on growth. Silver concentrations (mg/kg FW soft parts) for controls ranged between 0.2 and 0.7; maximum concentrations in the 1 µg/L group (9.1) occurred at 18 months; for the 5 µg/L group, residues were highest (11.9) at 18 months; for the 10 µg/L group, concentrations were highest (15.3) at 12 months. All silver-exposed groups had histopathology of basement membranes and connective tissues	19
Juveniles (16.1 mm shell length) and adults (53.4 mm shell length) were continuosly exposed for 12 months to 0.0 (control), 5, 25, or 50 µg Ag/L and observed for growth and accumulations	Growth inhibition of the 50 µg/L group after 6 months but growth normal after 12 months; growth of other groups as in controls. At 12 months residues, in mg Ag/kg FW soft parts, for juveniles (adults) were 0.2 (0.1) in controls, 9.9 (2.0) in the 5 µg/L group, 8.0 (2.0) in the 25 µg/L group, and 10.7 (3.0) in the 50 µg/L group	19
100	Increasing oxygen consumption with increasing water salinity	11
500 or 1,000	Adults exposed for 96 h had 3.7-5.2 mg Ag/kg FW soft parts	11
Radiosilver-110m	BCF values after 1 day were X860 in soft parts and X8 in shell; after 9 days these values were X2,550 in soft parts and X11 in shell	20
Mussel, *Mytilus galloprovincialis*, adults		
10	Growth normal after 21-month exposure	12
20	After 28 days soft parts contained 15 mg Ag/kg DW vs. 0.08 mg Ag/kg DW in controls (<0.1 µg Ag/L)	12
25	Growth depressed after 21-month exposure	12
50	Severe tissue histopathology after 14 days	12
100	LC50(110 h)	12
Mud snail, *Nassarius obsoletus*		

Table 6. Taxonomic group, organism, silver concentration (ppb), and other variables	Effects	Reference[a]
1.0	Inhibition of embryonic development	21
Clam, *Potamocorbula amurensis*; exposed for 14 days at 1.8% salinity to 0.1, 0.2, 0.5, 1.0, or 2.0 µg Ag^+/L	Silver concentrations in soft parts rose in a dose-dependent manner from <1.0 mg Ag/kg DW to about 14.0 mg/kg Ag/kg DW	42
Clam, *Scrobicularia plana*, adults		
20	Normal after 14 days	12
50	No deaths in 16 days; severe histopathology	12
100	LC50(250 h)	12
200	LC50(96 h)	12
Surf clam, *Spisula solidissima*		
10	Elevated oxygen consumption in juveniles after 96 h	10
14; 1-h exposure 60 min after fertilization	50% of embryos developed abnormally in 48 h	21
100	Lethal to juveniles in 96 h	10
Bryozoans		
Bryozoan, *Victorella* sp.		
0.18 (control), 2, or 10	Silver concentrations, in mg/kg DW whole animal, after 24 h were 11.5 in controls, 38.3 in the 2 µg/L group, and 180.0 in the 10 µg/L group	22
Arthropods		
Copepod, *Acartia tonsa*		
36	LC50(96 h)	4
Daphnid, *Daphnia magna*		
0.4-15.0	LC50(96 h) at 38-75 mg $CaCO_3$/L	4
0.9	50% of starved daphnids immobilized in 48h	23
1.6-19.4	MATC[b]	23
3.5	50% reduction in growth of nonstarved daphnids in 21 days	23
4.1	Reduced survival during 21-day exposure	23
10.5	Reproduction inhibited during 21-day exposure	23
12.5	50% of nonstarved daphnids immobilized in 48 h	23
45-49	LC50(96 h) at 255 mg $CaCO_3$/L	4

Table 6. Taxonomic group, organism, silver concentration (ppb), and other variables	Effects	Reference[a]
Daphnids, *Daphnia* spp.		
10 (0.25-49.0)	LC50(96 h)	9
Mayfly, *Ephemerella grandis*		
<1.0	LC50(14 days), naiads	24
4.0-8.8	LC50(7-15 days), adults	4
60 or 120	On death, whole mayflies contained 25.3 and 28.7 mg Ag/kg DW	24
Scud (amphipod), *Gammarus pseudolimnaeus*		
4.5 (3.7-5.5)	LC50(96 h) at 44 mg $CaCO_3$/L	25
American lobster, *Homarus americanus*		
6.0	Altered enzyme activity after 30 days but no effect on survival, oxygen consumption, or osmoregulation	10
Amphipod, *Hyalella azteca*		
0.95	No observable effects after 21-day exposure	13
1.4	Reduced growth after 20 days	13
1.9 (1.4-2.3)	LC50(96 h)	13
Mayfly, *Isonychia bicolor*		
1.6	Molting inhibited after 20 days	13
6.8 (5.5-7.8)	LC50(96 h)	13
Stonefly, *Leuctra* sp.		
0.69	Adverse effects after 12 days	13
2.5 (1.7-3.2)	LC50(96 h)	13
Mysid shrimp, *Mysidopsis bahia*		
250.0	LC50(96 h)	4
Grass shrimp, *Palaemonetes pugio*		
2.0	After 2 weeks whole shrimps contained 0.5 mg Ag/kg DW vs. 0.36 in controls	22
5.0	After 2 weeks whole shrimps had 3.7 mg Ag/kg DW	22
10.0	Whole shrimps contained 4.5 mg Ag/kg DW after exposure for 2 weeks	22
For 2 weeks shrimp ate *Artemia*	Silver concentration (mg/kg DW whole body) in	22

Table 6. Taxonomic group, organism, silver concentration (ppb), and other variables	Effects	Reference[a]
nauplii containing 0.72 mg Ag/kg DW, or bryozoans (*Victorella* sp.) containing elevated silver burdens (38-180 mg Ag/kg DW), or control bryozoans (11.5 mg Ag/kg DW)	shrimp on *Artemia* diet was 0.19 vs. 0.09 in silver-free *Artemia* diet; 0.26-0.62 in the high-silver bryozoan diet and 0.36 in the control bryozoan diet	
Stonefly, *Pteronarcys californica*		
4.0-9.0	LC50(96 h)	24
50.0	On death, whole stoneflies contained 9.1 mg Ag/kg DW	24
105.0	Dead stoneflies had 13.2 mg Ag/kg DW	24
Mayfly, *Stenonema* sp.		
3.9 (2.5-5.7)	LC50(96 h)	13
Midge, *Tanytarsus dissimilis*		
3,160 (2,490-4,010)	LC50(48 h) at 44 mg $CaCO_3$/L	25
Annelids		
Marine polychaete, *Sabella pavonina*		
Adults immersed in seawater containing 50 µgAg/L for 8 weeks then transferred to silver-free media for 8weeks	During immersion, the maximum whole-body silver concentration was 22.1 mg/kg DW vs. 0.8 in controls; main sites of accumulation were the connecting tissues of nephridia and gut. No histopathology. A constant elimination of silver in urine occurs simultaneously with silver accumulation. During depuration, new connective tissue formed and silver concentrations were reduced by 88%	26
Echinoderms		
Sea urchin, *Arbacia lixula*		
0.5	Reduced embryo development after 52 h	4
Fishes		
Mottled sculpin, *Cottus bairdi*		
5.3	LC50(96 h) at 30 mg $CaCO_3$/L	4
14.0	LC50(96 h) at 250 mg $CaCO_3$/L	4
Sheepshead minnow, *Cyprinodon variegatus*		
1,400	LC50(96 h), juveniles	4
Common carp, *Cyprinus carpio*		

Table 6. Taxonomic group, organism, silver concentration (ppb), and other variables	Effects	Reference[a]
Held in radiosilver-110m solutions for 41 days then transferred to uncontaminated media for 42 days	Whole body BCF during immersion rose rapidly and progressively to X51 at day 19, then more slowly to X73 at day 41. At day 41, BCF for liver was X866, for digestive tract X560, for kidneys X299, for spleen X155, and for air bladder X109. During depuration, 68% of the silver was eliminated-- about 31% during the first 3 days and 37% in the next 39 days. In both uptake and depuration phases, 71-77% of all radiosilver was present in liver and digestive tract	27
Mummichog, *Fundulus heteroclitus*		
30-40	Inhibited liver enzyme activity after 4 days	2, 4
Mosquitofish, *Gambusia affinis*		
23.5 (17.2-27.0)	LC50(96 h), juveniles	13
Threespine stickleback, *Gasterosteus aculeatus*		
3	Lethal in 10-30 days	2
4	Lethal in 7 days	2
10	Lethal in 96 h	2
Flagfish, *Jordanella floridae*		
9.2 (8.0-10.7)	LC50(96 h) at 44 mg $CaCO_3$/L	25
Bluegill, *Lepomis macrochirus*		
31.7 (24.2-48.4)	LC50(96 h)	13
70.0	Survival as in controls after 6 months; whole body contained 0.3 mg Ag/kg ash weight	28
Atlantic silverside, *Menidia menidia*		
110.0	LC50(96 h), larvae	4
400.0	LC50(96 h), juveniles	4
Largemouth bass, *Micropterus salmoides*		
7.0	Survival of young of year as in controls after continuous exposure for 6 months. After 4 months, viscera contained 0.6 mg Ag/kg ash weight, gills 0.38, and carcass 0.016 mg Ag/kg ash weight	28
70.0	All dead within 24 h. Prior to death, bass had reddened gills, body tremors, and erratic swimming	28
Chum salmon, *Oncorhynchus keta*		
Eggs were exposed in freshwater to	At end of study, experimentals had a small, but	29

Table 6. Taxonomic group, organism, silver concentration (ppb), and other variables	Effects	Reference[a]
4 µg Ag/L for 14 days prior to hatch. After hatching, larvae were exposed to 4 µg Ag/L in saltwater for 14 days during yolk-sac resorption. Fry were then transferred to uncontaminated saltwater and fed a high metals diet for 64 days (diet contained--in µg metal/kg feed DW--2 Ag, 255 Cd, 6,670 Cu, 1,320 Pb, and 96,000 Zn)	significant, decline in survival. Just prior to transfer to uncontaminated saltwater, experimentals contained 62 µg Ag/kg whole body DW vs. 4 in controls. After 64 days in uncontaminated saltwater, whole alevins contained 18 µg Ag/kg DW vs. 4 in controls	
Coho salmon, *Oncorhynchus kisutch*		
11.1 (7.9-15.7)	LC50(96 h), alevins	30
12.5 (10.7-14.6)	LC50(96 h), juveniles	30
Rainbow trout, *Oncorhynchus mykiss*		
0.03-0.06	MATC[b] after 13-month exposure beginning at eyed embryo stage	4
0.09-0.17	MATC[b] after exposure of eyed eggs and subsequent developmental stages for 18 months in soft water	31
0.18-0.40	MATC[b]; 10-month exposure starting with newly-fertilized embryos	4
0.6	All eyed eggs survived 10-week exposure	31
1.2	40% of eyed eggs dead in 39 days	31
2.0	Inhibition of Na^+ and Cl^- influx across gills in mature trout after 72 h	44
2.2	All eyed eggs dead in 60 days	31
4.8-8.9	LC50(144 h); juveniles	13, 44
5.3-8.1	LC50(96 h); water hardness 20-31 mg $CaCO_3$/L	31
7.6-10.9	LC50(96 h)	4, 23, 41
10.0	LC50(28 days) at 93-105 mg $CaCO^3$/L	4
13.0	LC50(96 h); water hardness 350 mg $CaCO_3$/L	31
16.1 (12.8-20.2)	LC50(96 h); alevins	30
19.2 (16.0-23.1)	LC50(96 h); juveniles	30
Steelhead trout, *Oncorhynchus mykiss*		

Table 6. Taxonomic group, organism, silver concentration (ppb), and other variables	Effects	Reference[a]
0.1-1.1	Growth reduced during chronic exposure from egg through swimup fry	23
0.5 and 1.3	Survival reduced at low dose when exposed continuously from egg through swimup; all dead at high dose	23
9.2	LC50(96 h)	23
100.0	LC50(62 h); eyed embryos	32
200.0	LC50(96 h); eyed embryos	32
Chinook salmon, *Oncorhynchus tshawytscha*		
33.0	Fry survived exposure for 48 h	2
40.0-44.0	Lethal to fry in 48 h	2
Summer flounder, *Paralichthys dentatus*		
4.7	LC50(96 h); larvae	4
8.0-48.0	LC50(96 h); embryos	4
Fathead minnow, *Pimephales promelas*		
5.3-20.0	LC50(96 h) at water hardness of 25-75 mg $CaCO_3$/L	4
5.6-7.4	LC50(96 h); flow-through tests	23
9.4-9.7	LC50(96 h); static tests	23
10.7 (10.6-10.8)	LC50(96 h) at 44 mg $CaCO_3$/L	25
29.0	LC100(96 h)	33
110.0-270.0	LC50(96 h) at water hardness of 255 mg $CaCO_3$/L	4
Guppy, p*oecilia reticulata*		
4.3	Lethal	2
Winter flounder, Pleuronectes americanus		
10.0	Depressed liver transaminase activity after 60 days	10
54, 92, 180, or 386	No significant effect of lowest dose on growth or survival during exposure for 18 days of embryo through yolk-sac absorption. At 92 µg/L, 31% died; at 180 µg/L, 97% died; at 386 µg/L, hatch was reduced 24% and all larvae died	34
200.0-450.0	LC50(96 h); embryos	4

Table 6. Taxonomic group, organism, silver concentration (ppb), and other variables	Effects	Reference[a]
Speckled dace, *Rhinichthys osculus*		
4.9	LC50(96 h) in soft water	4
14.0	LC50(96 h) in hard water	4
Brown trout, *Salmo trutta*		
Fingerlings exposed to radiosilver-110m for 57 days then transferred to clean freshwater for 28 days	After 57 days the whole-body BCF was X2.7 with about 70% of total radiosilver concentrated in the liver (BCF for liver was X282); during depuration, 23% of whole fish radiosilver was excreted but concentration in liver was unchanged	35
Fingerlings were fed a diet containing radiosilver-110m for 34 days then fed a clean diet for 27 days	After 34 days, about 12% of the silver fed was retained; liver contained 63% of the total radioactivity. After 27 days of depuration 31% of the radiosilver was lost from whole trout, but liver contained 79% of the total radioactivity	36
Cunner, *Tautogolabrus adspersus*		
120, 250, or 500; after 96 h, gill tissues were excised and gill oxygen consumption monitored for 4 h	Gill-tissue oxygen consumption was significantly lower than controls at all silver concentrations tested in a dose-dependent manner	37
500	After 96 h, gill tissue respiration was reduced and liver enzyme activity altered. Similar effects seen for silver nitrate and silver acetate	38
>500	Lethal after 96 h	37
Arctic grayling, *Thymallus arcticus*		
6.7 (5.5-8.0)	LC50(96 h); alevins	30
11.1 (9.2-13.4)	LC50 (96 h); juveniles	30
Amphibians		
Early life stages exposed to silver nitrate from fertilization through 4 days after hatching		
Leopard frog, *Rana pipiens*		
0.7-0.8	10% mortality or abnormal development of embryos and larvae	43
10.0	50% mortality or gross terata of embryos and larvae	43
5 species		
1-34	10% mortality or gross terata of embryos and larvae	43
10-240	50% mortality or abnormal development of embryos and larvae	43

[a]1, Antelman 1994; 2, Smith and Carson 1977; 3, Jeanthon and Prieur 1990; 4, U.S. Environmental Protection Agency 1980; 5, Sanders and Abbe 1987; 6, Sanders and Abbe 1989; 7, Eisler 1981; 8, Nalecz-Jawecki et al. 1993; 9, Williams and Dusenbery 1990; 10, Calabrese et al. 1977b; 11, Thurberg et al. 1974; 12, Berthet et al. 1992; 13, Diamond et al. 1990; 14, Coglianese and Martin 1981; 15, Berthet et al. 1990; 16, Sanders et al. 1990; 17, Abbe and Sanders 1990; 18, Nelson et al. 1983; 19, Calabrese et al. 1984; 20, Nolan and Dahlgaard 1991; 21, Bryan and Langston 1992; 22, Connell et al. 1991; 23, Nebeker et al. 1983; 24, Nehring 1976; 25, Lima et al. 1982; 26, Koechlin and Grasset 1988; 27, Baudin et al. 1994; 28, Coleman and Cearley 1974; 29, Buell 1991; 30, Buhl and Hamilton 1991; 31, Davies et al. 1978; 32, Rombough 1985; 33, LeBlanc et al. 1984; 34, Klein-MacPhee et al. 1984; 35, Garnier et al. 1990; 36, Garnier and Baudin 1990; 37, Thurberg and Collier 1977; 38, Gould and MacInnes 1977; 39, Fisher et al. 1995; 40, Fisher et al. 1984; 41, Hogstrand et al. 1996; 42, Brown and Luoma 1995; 43, Birge and Zuiderveen 1995; 44, Morgan et al. 1995.

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

The ability to accumulate dissolved silver from the medium ranges widely between species. Some reported bioconcentration factors (mg Ag per kg FW organism/mg Ag per liter of medium) are 210 in diatoms, 240 in brown algae, 330 in mussels, 2,300 in scallops, and 18,700 in oysters (EPA 1980). Silver is the most strongly accumulated of all trace metals by marine bivalve mollusks (Luoma 1994). Studies with radiosilver-110m suggest that the half-time persistence of silver is 27 days in mussels, 44-80 days in clams, and more than 180 days in oysters (Fisher et al. 1994). In oysters and other bivalve mollusks, the major pathway of silver accumulation was from dissolved silver; uptake was negligible from silver adsorbed onto suspended sediments or algal cells, and oysters eliminated adsorbed silver in the feces (Abbe and Sanders 1990; Sanders et al. 1990). Sometimes, benthic bivalve mollusks accumulated silver from certain sediments. Sediment-bound silver was taken up by the Baltic clam (*Macoma balthica*) at 3.6 to 6.1 times the concentration in calcite sediments but less than 0.85 times from manganous, ferrous, and biogenic $CaCO_3$ sediments (EPA 1980). In oysters, silver associated with food was unavailable for incorporation, which may be due to the ability of silver to adsorb rapidly to cell surfaces and to remain tightly bound despite changes in pH or enzymatic activity (Connell et al. 1991). Silver concentrations in American oysters (*Crassostrea virginica)* held in seawater solutions containing 1.0 mg Ag/L for 96 h rose from 6.1 mg/kg FW soft parts to 14.9 mg/kg FW; in gills, these values were 5.9 and 33.9 mg/kg FW (Thurberg et al. 1974). A similar pattern was evident in common mussels (*Mytilus edulis*) and quahog clams (*Mercenaria mercenaria*; Thurberg et al. 1974). Adults of surf clams (*Spisula solidissima*) immersed for 96 h in seawater containing 10 µg Ag/L had 1.0 mg Ag/kg FW soft tissues versus 0.08 mg/kg in controls (Thurberg et al. 1975). Oysters accumulated radiosilver-110m from the medium by factors of 500 to 32,000 (Pouvreau and Amiard 1974); uptake of dissolved silver by oysters was higher at elevated temperatures in the range of 15-25^{o} C (Abbe and Sanders 1990). American oysters maintained near a nuclear power plant in Maryland that discharged radionuclides on a daily basis into the Chesapeake Bay accumulated radiosilver-110m; accumulations were higher in summer and fall than in winter and spring (Rose et al. 1988).

Marine gastropods exposed to concentrations as low as 1.0 µg Ag/L for as long as 24 months showed histopathology and accumulations as high as 34 mg Ag/kg FW soft parts; higher exposure concentrations of 5 and 10 µg Ag/L were associated with inhibited reproduction and whole-body burdens as high as 87 mg Ag/kg FW (Nelson et al. 1983). Histopathological findings in silver-exposed mussels (*Mytilus edulis*) were typical of argyria in humans and other mammals that have absorbed organic or inorganic silver compounds (Calabrese et al. 1984). Juvenile Pacific oysters (*Crassostrea gigas*) exposed for 2 weeks to solutions containing 20 µg Ag/L had high silver accumulations in tissues and a reduced capacity to store glycogen; however, after 30 days of depuration, glycogen storage capacity was restored and 80% of the soluble silver and 27% of the insoluble forms were eliminated, suggesting recovery to a normal physiological state (Berthet et al. 1990). About 70% of the insoluble silver in Pacific oysters was sequestered as Ag_2S, a stable mineral form that is not degradable, thereby limiting the risk of silver transfer through the food chain (Berthet et al. 1990). Most (69-89%) of the silver accumulated from the medium in soft tissues of oysters and clams was sequestered in amoebocytes and basement membranes; in scallops and mussels, silver was stored in basement membranes and pericardial gland. In all species of bivalve mollusks, sequestered silver was in the form of silver sulfide (Berthet et al. 1992). American oysters excreted about 60% of their accumulated silver in soft tissues within 30 days of transfer to silver-free seawater; soluble forms were preferentially eliminated and insoluble forms retained (Berthet et al.

1992). Interspecies differences in ability to retain silver among bivalve mollusks are large, even among closely related species of crassostreid oysters. For example, the half-time persistence of silver was about 149 days in American oysters but only 26 days in Pacific oysters (PHS 1990).

Among arthropods, grass shrimp (*Palaemonetes pugio*) rapidly incorporate silver dissolved in brackish water in proportion to its concentration but not from planktonic or detrital food sources containing elevated silver burdens (Connell et al. 1991). Variations in ability of decapod crustaceans to accumulate radiosilver-110m from seawater are large, as judged by concentration factors that ranged from 70 to 4,000 (Pouvreau and Amiard 1974). The reasons for this variability are unknown but may be associated with hepatopancreas morphology. It is generally acknowledged that hepatopancreas or digestive gland is the major repository of silver in decapods (Greig 1975; Greig et al. 1977a, 1977b). Aquatic insects concentrate silver in relative proportion to environmental levels (Nehring 1976), and more efficiently than most fish species (Diamond et al. 1990). Whole-body bioconcentration factors (BCF = mg total Ag per kg fresh weight tissue divided by mg total Ag per liter of medium) of silver in three species of aquatic insects ranged from 21 to 240 in water containing 30-65 mg $CaCO_3$/L during exposure of 3-15 days; in bluegill sunfish (*Lepomis macrochirus*), this value was less than 1 after exposure for 28 days (EPA 1980). Molt frequency of a mayfly (*Isonychia bicolor*) was a sensitive indicator of silver stress over time, and 1.6 µg total Ag/L over a 20-day period inhibited molting (Diamond et al. 1990).

Silver ion (Ag^+) was the most toxic chemical species of silver to fishes. Silver ion was 300 times more toxic than silver chloride to fathead minnows (*Pimephales promelas*), 15,000 times more toxic than silver sulfide, and more than 17,500 times more toxic than silver thiosulfate complex; in all cases, toxicity reflected the free silver ion content of tested compounds (LeBlanc et al. 1984); a similar pattern was noted in rainbow trout (Hogstrand et al. 1996). Silver was less toxic to fathead minnows under conditions of increasing water hardness between 50 and 250 mg $CaCO_3$/L, increasing pH between 7.2 and 8.6, and increasing concentrations of humic acid and copper; starved minnows were more sensitive to ionic silver than minnows fed regularly (Brooke et al. 1994). Eggs of rainbow trout exposed continuously to silver concentrations as low as 0.17 µg/L had increased embryotoxicity and hatched prematurely; resultant fry had a reduced growth rate (Davies et al. 1978). Removal of the egg capsule of eyed embryos of steelhead trout (*Oncorhynchus mykiss*) significantly lowered the resistance of the embryos to salts of silver, copper, and mercury but not zinc and lead (Rombough 1985). Silver accumulation in gills of juvenile rainbow trout exposed to 11 µg Ag/L for 2-3 h was significantly inhibited by various cations (Ca^{2+}, Na^+, H^+) and complexing agents (dissolved organic carbon, thiosulfate, chloride); these variables must be considered when constructing predictive models of silver binding to gills (Janes and Playle 1995).

Largemouth bass (*Micropterus salmoides*) and bluegills accumulated silver from the medium; accumulations increased with increasing concentrations of ionic silver and increasing duration of exposure (Coleman and Cearley 1974). Bioconcentration factors of radiosilver-110m for various species of teleosts were as high as 40 after 98 days (Pouvreau and Amiard 1974). However, plaice (*Pleuronectes platessa*; a marine flounder), and thornback rays (*Raja clavata*) fed nereid polychaete worms labeled with radiosilver-110m retained about 4.2% of the ingested dose after 3 days (Pentreath 1977), which suggests that the high silver concentration factors reported by Pouvreau and Amiard (1974) may have been due to loosely bound adsorbed silver. Flounders (*Pleuronectes* sp.) held for 2 months in seawater solutions containing 40 µg Ag/L had elevated silver concentrations in the gut (0.49 mg Ag/kg FW) but less than 0.05 mg/kg in all other examined tissues (Pentreath 1977). Similarly exposed rays (*Raja* sp.) contained 1.5 mg Ag/kg FW in liver, 0.6 in gut, 0.2 in heart, and 0.05-0.18 mg/kg FW in spleen, kidney, and gill filament (Pentreath 1977); liver is usually considered the major repository of silver in teleosts (Garnier et al. 1990).

Food chain biomagnification of silver in aquatic systems is unlikely at silver concentrations normally encountered in the environment (Connell et al. 1991), although regular ingestion of fish from contaminated waters may significantly affect dietary silver intake in humans (EPA 1980). Silver—as thiosulfate-complexed silver at nominal concentrations of 500 or 5,000 µg Ag/L—was concentrated and magnified over a 10-week period in freshwater food chains of algae, daphnids, mussels, and fathead minnows (Terhaar et al. 1977), although the mechanisms of accumulation in this study were imperfectly understood.

Birds and Mammals

No data were found on the effects of silver compounds on avian or mammalian wildlife. All controlled studies with silver were with domestic poultry, livestock, or small laboratory mammals. Signs of chronic silver ion intoxication in tested birds and mammals included cardiac enlargement, vascular hypertension, hepatic necrosis, anemia, lowered immunological activity, altered membrane permeability, kidney pathology, enzyme inhibition, growth retardation, and a shortened life span (Smith and Carson 1977; Freeman 1979; Fowler and Nordberg 1986; PHS 1990).

Silver affects turkeys (*Meleagris gallopavo*) and domestic chickens (*Gallus* spp.). Turkey poults on diets containing 900 mg Ag/kg feed for 4 weeks had enlarged hearts and reduced growth, hemoglobin, and hematocrit (EPA 1980). Chicken eggs injected with silver nitrate at 0.1 mg Ag/egg (equivalent to about 1.8 mg Ag/kg egg FW) had a 50% reduction in survival but no developmental abnormalities (Ridgway and Karnofsky 1952). Adverse effects of silver were reported in normal chicks fed diets containing 200 mg Ag/kg ration (growth suppression) or given drinking water containing 100 mg Ag/L (liver necrosis; Smith and Carson 1977). Chicks on copper-deficient diets had adverse effects at 10 mg Ag/kg ration (reduced hemoglobin; reversible when fed copper-adequate diet) and at 50-100 mg Ag/kg ration (growth suppression and increased mortality). Chicks that were deficient in vitamin E experienced reduced growth when given drinking water containing 1,500 mg Ag/L. Chickens infected with pathogenic strains of *Salmonella* sp. and *Escherichia coli* were cured with aerosol treatments containing 10 µg Ag/L air (Smith and Carson 1977).

Studies with small laboratory mammals—which require verification—show that long-term exposure to high levels of silver nitrate in drinking water may result in sluggishness and enlarged hearts; however, these effects have not been observed in silver-exposed humans (PHS 1990). Concentrations as high as 200 µg Ag/L in drinking water of test animals for 5 months had no significant effect on animal health or metabolism (EPA 1990). But 400 µg Ag/L for 5 months caused kidney damage, and 500 µg/L for 11 months was associated with impaired conditioned-reflex activities, immunological resistance, and altered brain nucleic acid content (EPA 1980). Diets deficient in vitamin E or selenium caused rapidly fatal hepatocellular necrosis and muscular dystrophy to rats if they contained the dietary-intake equivalent of 130 mg Ag/kg BW daily, a comparatively high silver ion intake (Smith and Carson 1977; PHS 1990).

The extent of absorption of an administered dose of silver depends on silver speciation, the presence and extent of silver-binding proteins, and other variables. But absorption is dependent mainly on the transit time through the gastrointestinal tract: the faster the transit time is, the less silver is absorbed. Transit times ranged from about 8 h in mice and rats to about 24 h in monkeys, dogs, and humans (PHS 1990). Route of administration affected the excretion rate of silver. Clearance of silver from mammals 2 days after silver was administered intravenously ranged from 15% in dogs to 82% in mice; clearance rates were intermediate in monkeys and rats. When silver was administered orally, clearance was more rapid, and extended from 90.4% in dogs to 99.6% in mice (PHS 1990). The half-time persistence of silver administered orally to mice was 0.1 day for the short-lived component and 1.6 days for the long-lived component. Other species of tested laboratory animals had biphasic or triphasic whole-body silver-excretion profiles that differed significantly from mice. Monkeys, for example, had a biphasic excretion profile with peaks at 0.3 and 3.0 days; rats had a triphasic profile with peaks at 0.1, 0.7, and 5.9 days; and dogs had half-time persistence peaks at 0.1, 7.6, and 33.8 days (PHS 1990).

Ionic silver is lethal to mice (*Mus* spp.) at 13.9 mg/kg BW by intraperitoneal injection, to rabbits (*Oryctolagus* spp.) at 20 mg/kg BW intraperitoneally, to dogs (*Canis familiaris)* at 50 mg/kg BW by intravenous injection, to humans at greater than 166 mg/kg BW in a single dose, and to rats (*Rattus* spp.) at 1,586 mg/L drinking water for 37 weeks (Table 7). Sublethal effects are reported in rabbits given 250 µg Ag/L drinking water (brain histopathology), in rats given 400 µg Ag/L drinking water for 100 days (kidney damage), in mice given 95 mg Ag/L drinking water for 125 days (sluggishness), in guinea pigs (*Cavia* sp.) given 81 mg Ag/cm^2 skin applied daily for 8 weeks (reduced growth), and in rats given diets containing 6 mg Ag/kg for 3 months (high accumulations in kidneys and liver) or 130-1,110 mg/kg (liver necrosis; Table 7).

The connections between human cancers and silver as a causal agent are tenuous (EPA 1980). All available evidence is negative or inconclusive regarding silver's ability to induce cancer, mutagenicity, or birth defects in animals by normal routes of exposure (EPA 1980; PHS 1990). Silver pellets, however, implanted

under the skin of rodents, have caused sarcomas, malignant fibrosarcomas, fibromas, fibroadenomas, and invasions of muscle with connective tissue; in these cases, silver seems to act as a nonspecific irritant rather than as a specific carcinogen (Smith and Carson 1977; EPA 1980). Intratumoral injections of colloidal silver promotes cancer growth in rats, possibly by producing an area of lowered tissue resistance that allows resistant cancer cells to grow freely (Smith and Carson 1977); however, silver nitrate seems to be a tumor inhibitor in mice (EPA 1980).

In humans, acute toxic effects of silver have resulted only from accidental or suicidal overdoses of medical forms of silver. Symptoms of acute silver poisoning in patients dying after intravenous administration of Collargo (silver plus silver oxide) included gastrointestinal disturbances, pulmonary edema, tissue necrosis, and hemorrhages in bone marrow, liver, and kidney (Smith and Carson 1977; EPA 1980). High sublethal doses of silver nitrate taken orally cause some patients to experience violent abdominal pain, abdominal rigidity, vomiting, and severe shock; systemic effects among recovering patients are unlikely, although degenerative liver changes may occur (EPA 1980). In humans, skin contact with silver compounds may cause mild allergic reactions such as rash, swelling, and inflammation (PHS 1990); industrial and medicinal exposures to silver may cause lesions of the kidneys and lungs, and arteriosclerosis (Klaassen et al. 1986); colloidal silver compounds may interfere with nasal ciliary activity (Smith and Carson 1977); and exposure to dust containing high levels of silver compounds, such as silver nitrate or silver oxide, may cause breathing problems, lung and throat irritation, and stomach pain (PHS 1990).

Table 7. Effects of silver on selected mammals.

Table 7. Organism, route of administration, dose, and other variables	**Effects**	**Reference[a]**
Domestic dog, *Canis familiaris*		
Inhalation route. Anaesthetized dogs exposed to metallic silver particles about 0.5 um in diameter; total dose deposited of 25 µg	About 3% (0.8 µg) of the deposited silver was found in liver and blood 6 h after exposure. Clearance from the lung to the blood was triphasic, with half-times of 1.7, 8.4, and 40 days	1
Intratracheal route. Elemental silver deposited in lungs	After 6 h, 96.9% remained in lungs, 2.4% in liver, 0.35% in blood, 0.14% in gall bladder and bile, 0.1% in intestines, 0.06% in kidneys, and 0.02% in stomach. After 225 days, 0.49% of the initial dose was detected in liver, and 0.01-0.03% each in brain, gall bladder, intestines, lungs and trachea, bone, stomach and contents, heart, and muscle. If lung is excluded, liver contained 77% of the total-silver body burden between 6 h and 225 days postexposure	1
Intravenous injection route		
0.003 µg/kg body weight (BW) daily	15% cleared in 2 days	1
500 mg (estimated at 50 mg/kg BW), single injection	All dead within 24 h with hemolysis and lung edema	2
Oral route; 0.005 µg/kg BW daily	90.4% cleared in 2 days	1

Table 7. Organism, route of administration, dose, and other variables	Effects	Reference[a]
Guinea pig, *Cavia* sp.		
Dermal application of 81 mg silver nitrate to 3.1 cm^2 of skin daily for 8 weeks	Growth rate reduced 10-20%	1
Humans, *Homo sapiens*		
External route		
0.25% silver nitrate solution in eyes for 3 weeks	Argyrosis	2
3-5% colloidal silver compounds in eyes for 5-10 weeks	Argyrosis	2
Inhalation route		
0.25 mg/m^3 air	Possibility of generalized argyria in 20 years	2
1-2 mg/m^3 air; occupational exposure	Argyrosis of cornea and conjunctiva	3
Clearance half-time		
Feces	>300 days	1
Lung	Biexponential profile; 1 day and 52 days	1
Urine	<54 days	1
Oral route		
80 µg/kg BW, single dose	21% of dose retained in body after 1 week	1
0.7 mg silver weekly in diet	Possibility of generalized argyria	2
2-30 g of silver nitrate, single dose	At dosages >10 g (equivalent to >166 mg Ag/kg BW for a 60-kg person), death usually occurs within a few hours to a few days	2, 3
50-260 g of metallic silver	Gastric fullness, anorexia, gastric pain, diarrhea	2, 3
>600 g over 1.2 years; given as silver nitrate to treat epilepsy and GI symptoms	Generalized argyria evident 2 years after last dose	2
Monkeys, various		
Intravenous administration of 0.01 µg/kg BW daily	44.1% excreted in 2 days	1
Oral administration of 0.01 µg/kg BW daily	94.3% cleared in 2 days	1

Table 7. Organism, route of administration, dose, and other variables	Effects	Reference[a]
Domestic mouse, *Mus* spp.		
Drinking water route; 95 mg/L for 125 days	Sluggishness	1
Intraperitoneal route		
13.9 mg/kg BW, single injection	LD50(30 days)	3
35.0 mg/kg BW, single injection; pretreated with single injection of 3.5 mg Ag/kg BW 24 h earlier	Only 3 of 10 pretreated mice died within 7 days vs. 8 of 10 nonpretreated mice	3
Intravenous injection; 1.0 µg/kg BW daily	82% cleared in 2 days	1
Oral route; 1.1 µg/kg BW daily	99.6% cleared in 2 days	1
Rabbit, *Oryctolagus* sp.		
Drinking water route		
Equivalent to 2.5 µg Ag/kg BW daily	No observable adverse effects	2
Equivalent to 25 or 250 µg Ag/kg BW daily for 11 months	Brain histopathology; altered conditioned reflexes	2
Equivalent to 500 or 5,000 µg Ag/kg BW daily for 11 months	Lowered immunological activity; pathology of vascular, nerve, brain, and spinal cord tissues	3
Intraperitoneal injection route; 20 mg/kg BW, single injection	All dead within 2 h. Silver granules in liver parenchyma and kidney tubules	3
Laboratory white rat, *Rattus* spp.		
Diet		
Equivalent to 6 mg/kg BW daily	After 12 weeks, kidneys and liver were impregnated with silver	2
130-1,110 mg/kg ration	Liver necrosis which could be prevented by adding Vitamin E	2
Drinking water route		
50 µg/L (equivalent to 0.0025 mg Ag/kg BW daily) for 11 months	Normal in all variables measured (conditioned reflex activity, gastric secretion, blood serum enzymes, histology)	3
200 µg/L for 6 months	Normal conditioned reflex activity	3
<400, 400, 700, or 1,000 µg/L for 100 days	At <400 µg/L, rats seemed healthy with normal tisssue histology. At 400 µg/L, hemorrhages noted	3

Table 7. Organism, route of administration, dose, and other variables	Effects	Reference[a]
	in kidney. At 700 µg/L, kidney and liver histopathology was evident. At 1,000 µg/L, spleen was pigmented and kidney and liver damage more pronounced	
500 µg/L (equivalent to 0.025 mg Ag/kg BW daily) for 6-11 months	Abnormal conditioned reflex activity; increased liver weight and liver RNA concentration	3
5 mg/L for 6 months	Signs of intoxication beginning at days 25-27	3
20 mg/L for 3 months	Decreased growth; abnormal liver; increased levels of blood amino acids	3
20 mg/L for 5 months	Pathology in stomach, small intestine, and liver; altered blood serum enzyne activity; growth depressed 36%	3
20 mg/L for 6 months	Increased brain DNA and RNA	2
129 mg/L (about 18 mg/kg BW daily) for 17.8 weeks	Silver accumulations in brain and central nervous system; reduced motor activity	1
634 mg/L (89 mg Ag/kg BW daily) for 2 years	No effect on male fertility; no silver deposits in testes	1
635-660 mg/L; lifetime exposure beginning shortly after weaning	Lifespan normal; hypertrophy of left ventricle, suggesting vascular hypertension; skin normal but internal organs and eyes darkened by silver deposits	3
1,200 mg/L for several months	Degenerative kidney changes	3
1,500 mg/L for 2-4 weeks; vitamin E-deficient rats	Liver necrosis and death. Prevented by adding vitamin E	2
1,586 mg/L for 37 weeks	Some deaths beginning at week 23; survivors weighed about 50% less than controls	1
2,500 mg/L for 3 months, then silver-free water for 16 months	At end of exposure livers had 6.7-7.0 mg Ag/kg FW and kidneys 3.7-7.1 mg/kg FW; 16 months after exposure livers had 1.6 mg/kg FW and kidneys 6.0 mg/kg FW	3
2,589 mg/L, (equivalent to 362 mg Ag/kg BW daily) for 2 weeks	25% died; water intake decreased beginning at day 1; survivors poorly groomed and listless	1
Intramuscular injection route		
Given radiosilver-110m alone or in combination with 4.0 mg Ag/kg BW daily for 6 days. Percent of		

Table 7. Organism, route of administration, dose, and other variables	Effects	Reference[a]
tracer dose recovered vs. percent tracer plus 4.0 mg/kg BW		
Blood	0.5 vs. 3.0	1
Bone	0.2 vs. 2.2	1
Feces	96.6 vs. 37.3	1
GI tract	1.1 vs. 8.2	1
Heart, lungs	0.06 vs. 0.6	1
Kidney	0.07 vs. 0.6	1
Liver	0.4 vs. 33.7	1
Muscle	0.2 vs. 2.4	1
Skin	0.2 vs. 7.4	1
Spleen	0.01 vs. 2.7	1
Urine	0.6 vs. 1.8	1
Intravenous injection route		
0.2 µg/kg BW daily	70.7% excreted in 2 days vs. 98.4% in 2 days when administered orally	1
Isolated hepatocytes exposed for 20 h		
<275 µg/L	Not cytotoxic	4
500, 2,000, or 5,000 µg/L	Silver accumulated in nuclear fraction of hepatocytes at all concentrations; DNA repair synthesis was stimulated at 2,000 µg/L; moderately cytotoxic at 5,000 µg/L	5
1,100 µg/L	Protein synthesis activity inhibited 50%	4
1,980 µg/L	Almost complete inhibition of protein synthesis activity	4
Subcutaneous injection route		
7 mg/kg BW, single injection	Adverse effects on spermatogenesis and on testes histology	2,3
Oral route		

Table 7. Organism, route of administration, dose, and other variables	Effects	Reference[a]
123 mg/kg BW, as silver cyanide	Acute oral LD50	6
200-400 mg/kg BW, as silver arsenate	Acute oral LD50	6
500-800 mg/kg BW, as silver nitrate	Acute oral LD50	6
Caribou, *Rangifer tarandus*		
Ratio of silver concentration (mg/kg FW) in tissues to silver concentration (mg/kg FW) in lichen diet		
Bone	Bioconcentration factor (BCF) of 3.0	3
Kidney	BCF of 1.3	3
Liver	BCF of 80.0	3
Muscle	BCF of 0.3	3

[a]1, U.S. Public Health Service 1990; 2, Smith and Carson 1977; 3, U.S. Environmental Protection Agency 1980; 4, Denizeau et al. 1990; 5, Denizeau and Marion 1989; 6, Lockhart 1983.

Chronic exposure of humans to silver or silver compounds has frequently resulted in generalized argyria (slate-gray pigmentation of the skin and hair caused by deposition of silver), localized argyria (limited areas of pigmentation usually associated with medicinal silver applications), or argyrosis (argyria of the eye). Every silver compound in common chemical use has caused generalized argyria, usually from medical and occupational exposures (Smith and Carson 1977; EPA 1980). In generalized argyria, skin pigmentation was highest in light-exposed areas, although silver concentrations in light-exposed and dark-exposed skin were the same (EPA 1980). In severe cases of argyria, the skin may become black with a metallic luster, the eyes affected to the point that vision is disturbed, and the respiratory tract impaired (Klaassen et al. 1986). Individual variability in susceptibility to argyria is great and this is probably explained by the variability in absorption and retention of silver (Smith and Carson 1977). Generalized argyria as an occupational disease is unusual but has been reported in workers that make silver nitrate or are involved in mirror plating, glass bead silvering, silver Christmas cracker manufacturing, photographic plate manufacturing, and silver mining. Generalized argyria was also associated with chronic inhalation or ingestion of silver fulminate, silver nitrate, silver albuminate, and silver cyanide (Smith and Carson 1977). Improved workplace ventilation and sanitation among silver nitrate workers effected a decline in general argyria (EPA 1980).

Localized argyria is rare and usually occurs when silver compounds contact broken skin or mucous membranes (Smith and Carson 1977). Localized argyria has been reported in workers who handle metallic silver in filing, drilling, polishing, turning, engraving, forging, soldering, or smelting operations. Silver polishers exposed for 25 years or more (range 2-38 years) sometimes exhibit increased densities in their lung x-rays due to silver impregnation of the elastic membranes of the pulmonary vessels. In one case, an Italian physician who dyed his facial hair with a silver dye for 25 years developed argyria in the conjunctiva of both eyes (Smith and Carson 1977).

Recommendations

Most measurements of silver concentrations in natural waters prior to the use of clean techniques are considered inaccurate. Until analytical capabilities that exceed the dissolved-particulate classification are developed, it will be necessary to rely on laboratory and theoretical modeling studies to fully understand chemical speciation of silver in natural waters (Andren et al. 1995).

Factors governing the environmental fate of silver are not well characterized, including silver transformations in water and soil and the role of microorganisms (PHS 1990). Food chain transfer of silver requires more current information on sources and forms of silver and data on concentrations in field collections of flora and fauna, especially near hazardous waste sites (PHS 1990). Although silver in sewage sludge is mostly immobilized, data are limited on the uptake by vegetation of silver from soils amended with silver-contaminated sewage sludge and on silver concentrations in flesh and milk of livestock pastured or fed grains raised on soils amended with sewage sludge (Smith and Carson 1977). Data are needed on partition coefficients and vapor pressures of silver compounds (PHS 1990) and on silver concentrations in emissions from cement producers and smelters and refineries of copper, lead, zinc, silver, iron, and steel (Smith and Carson 1977). Also, technology to recapture silver from waste media before it reaches the environment must be improved (PHS 1990).

In aquatic environments, more research is needed on the chemical speciation of silver to evaluate risk to the organism and its consumers (EPA 1987; Berthet et al. 1992). Most silver criteria formulated for the protection of aquatic life are now expressed as total recoverable silver per liter (Table 8). But total silver measurements do not provide an accurate assessment of potential hazard. Silver ion (Ag^{+}), for example, is probably the most toxic of all silver chemical species and must be accurately measured in the assessment of silver risks in aquatic environments (LeBlanc et al. 1984), perhaps as acid-soluble silver (EPA 1987). Little is known of the biocidal properties of Ag^{2+} and Ag^{3+} that are the active ingredients in disinfectants and used increasingly in water purification systems of drinking water and swimming pools (Antelman 1994). The effects of these silver species on organism health clearly must be researched (PHS 1990). Silver interactions with other metals and compounds in solution are not well defined. For example, mixtures of salts of silver and copper markedly increased the survival of oyster embryos, but only when copper concentrations were less than 6 µg/L and total silver less than 11 µg/L (Coglianese and Martin 1981).

Table 8. Proposed silver criteria for the protection of natural resources and human health.

Table 8. **Resource, criterion, and other variables**	**Effective silver concentration**	**Reference[a]**
Agricultural crops		
Soils	<100 mg total silver/kg dry weight soil for most species; <10 mg/kg for sensitive species	7
Freshwater aquatic life protection		
Acute exposure		
Total recoverable silver	<1.32 µg/L	1
Acid-soluble silver[b]	4-day average shall not exceed 0.12 µg/L more than once every three years; 1-h average not to exceed 0.92 µg/L more than once every 3 years	8
Acute exposure		
Total recoverable silver, in µg/L, should not exceed $e^{(1.72[\ln(\text{hardness})]-6.52}$ at any time. Examples follow		
50 mg $CaCO_3$/L	<1.2 µg/L	2

Table 8. Resource, criterion, and other variables	Effective silver concentration	Reference[a]
100 mg $CaCO_3$/L	<4.1 µg/L	2
200 mg $CaCO_3$/L	<13.0 µg/L	2
Chronic exposure	<0.12-<0.13 µg total recoverable silver/L	1, 2
Tissue residues		
Adverse effects on growth of the Asiatic clam, *Corbicula fluminea*	>1.65 mg total silver/kg soft tissues, fresh weight basis	1
Marine life protection		
Acute exposure		
Total recoverable silver	<2.3 µg/L at any time	2
Acid-soluble silver[b]	4-day average concentration not to exceed 0.92 µg/L more than once every 3 years on average and the 1-h concentration not to exceed 7.2 µg/L more than once every 3 years	8
Tissue residues		
Marine clams, soft parts		
Normal	<1 mg total silver/kg dry weight	3
Stressful or fatal	>100 mg total silver/kg dry weight	3
Human health		
Air, United States		
Current level of exposure, nationwide	100 µg total silver daily per person	2
Short-term exposure limit (15 min; up to 4 times daily with 60-min intervals at <0.01 mg Ag/m^3 air)	<0.03 mg total silver/m^3	2
Threshold limit value (8 h daily, 5 days weekly)		
Aerosol silver compounds	<0.01 mg total silver/m^3	2, 4, 5
Metallic silver dust	<0 .1 mg total silver/m^3	5
Diet, United States		
Current level of exposure	35 to 40 µg daily per person	2
Drinking water		
United States		
Long-term exposure (>10 days)	<50 µg total silver/L	2, 5, 6
Proposed long-term exposure	<90 µg total silver /L	5

Table 8. Resource, criterion, and other variables	Effective silver concentration	Reference[a]
Short-term exposure (1-10 days)	<1,142 µg total silver/L	5
California	<10 µg/L	2
Germany	<100 µg/L	2
Space vehicles		
Former Soviet Union	Max. 200 µg total silver/L	2
United States	100 to Max. 200 µg total silver/L	2
Switzerland	<200 µg total silver/L	2
Groundwater	<50 µg total silver/L	5

[a]1, Diamond et al. 1990; 2, U.S. Environmental Protection Agency (EPA) 1980; 3, Bryan and Langston 1992; 4, Smith and Carson 1977; 5, U.S. Public Health Service (PHS) 1990; 6, Fowler and Nordberg 1986; 7, Hirsch et al. 1993; 8, EPA 1987.

[b]Silver that passes through a 0.45-um membrane after the sample has been acidified to a pH between 1.5 and 2.0 with nitric acid.

The proposed human drinking water criteria of 50 to <200 µg total Ag/L do not seem to represent a hazard to human health, although much lower concentrations adversely affect freshwater and marine organisms (Smith and Carson 1977; Table 8). Proposed silver criteria for the protection of freshwater aquatic life during acute exposure now range from 1.2 to 13.0 µg total recoverable silver per liter (Table 8). If all total recoverable silver were in the ionic form, these proposed criteria would overlap the 1.2 to 4.9 µg/L range found lethal to sensitive species of aquatic plants and animals and indicates that the proposed freshwater acute silver criteria need to be reexamined. For freshwater aquatic life protection during chronic exposure, the proposed criterion of less than 0.13 µg total recoverable silver per liter (Table 8) is probably sufficient. But the proposed silver criterion of 2.3 µg total silver/L to protect marine life (Table 8) needs to be reconsidered because phytoplankton species composition and succession are significantly altered at 0.3-0.6 µg total silver/L and because some species of marine algae and mollusks show extensive accumulations at 1.0-2.0 µg total silver/L. Limited but insufficient data were available on correlations between tissue residues of silver with health of aquatic organisms (Table 8); additional research seems needed on the significance of silver residues in tissues.

Silver criteria in aquatic ecosystems are under constant revision by regulatory agencies. For example, total recoverable silver is no longer recommended by the U.S. Environmental Protection Agency in silver criteria formulation and should be replaced by dissolved silver (EPA 1995a). Dissolved silver more closely approximates the bioavailable fraction of silver in the water column than does total recoverable silver (EPA 1995b). Dissolved silver criteria recommended are about 0.85 times those of total recoverable silver under certain conditions but may vary considerably depending on other compounds present in solution (EPA 1995b).

No studies have been conducted with silver and avian or mammalian wildlife, and it is unreasonable to extrapolate the results of limited testing with domestic poultry and livestock to wildlife to establish criteria or administratively enforced standards. Research on silver and avian and terrestrial wildlife merits the highest priority in this subject area. No silver criteria are available for the protection of avian and mammalian health, and all criteria now proposed are predicated on human health (Table 8). As judged by the results of controlled studies with poultry and small laboratory mammals, safe concentrations of silver ion were less than 250 µg/L in drinking water of mammals, less than 100 mg/L in drinking water of poultry, less than 6 mg/kg in diets of mammals, less than 10 mg/kg in copper-deficient diets of poultry, less than 200 mg/kg in copper-adequate diets of poultry, and less than 1.8 mg/kg in chicken eggs. The proposed short-term (10-day) allowable limit of 1,142 µg Ag/L in drinking water for human health protection (Table 8) should be reconsidered because it is 4.6 times higher than the value that produced adverse effects in sensitive laboratory mammals. Additional animal studies

are needed to elucidate the effects of silver and silver compounds on reproduction, development, immunotoxicity, neurotoxicity, absorption, distribution, metabolism, and excretion; and on oral, dermal, and inhalation routes of exposure (PHS 1990). In animals, there is also the need to establish a target organ for intermediate exposures to silver, to establish suitable biomarkers of silver exposures and effects, and to measure effects of chronic silver exposures on carcinogenicity (PHS 1990). These studies should be implemented with suitable sentinel organisms including waterfowl, aquatic mammals, and other species of wildlife.

It is emphasized that silver and its compounds do not pose serious environmental health problems to humans from 50 µg/L in drinking water and 10 µg/m^3 in air (Smith and Carson 1977). The only proven effect of chronic exposure to silver is argyria from occupational or therapeutic exposure to much larger amounts of silver (minimum necessary absorption of 910 µg, equivalent to about 15 µg/kg BW) than can feasibly be ingested or inhaled from environmental sources. Regular ingestion of fish, meat, and plants from silver-contaminated areas probably does not cause argyria (Smith and Carson 1977). Humans at special risk to argyria include those treated with silver-containing medicinals and people marginally deficient or deficient in copper, selenium, or vitamin E (EPA 1980). There is no recognized effective treatment for argyria, although the condition seems to be relatively stationary when exposure to silver is discontinued (Fowler and Nordberg 1986). Absorption and retention of silver from food and medicinals is imperfectly understood (Smith and Carson 1977), suggesting the need for additional animal studies.

Finally, alternatives exist to the use of silver in various materials and processes. These include substitution of aluminum and rhodium for silver in mirrors and other reflecting surfaces; tantalum replacement of silver in surgical plates, pins, and sutures; using stainless steel as an alternative material to silver in the manufacture of table flatware; and, in photography, using film with reduced silver content (Reese 1991).

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Cited Literature

Abbe, G. R., and J. G. Sanders. 1990. Pathways of silver uptake and accumulation by the American oyster (*Crassostrea virginica*) in Chesapeake Bay. Estuarine, Coastal and Shelf Science 31:113-123.

Alexander, G. V., and D. R. Young. 1976. Trace metals in southern California mussels. Marine Pollution Bulletin 7:7-9.

Anderlini, V. 1974. The distribution of heavy metals in the red abalone, *Haliotis rufescens,* on the California coast. Archives of Environmental Contamination and Toxicology 2:253-265.

Anderlini, V. C. 1992. The effect of sewage on trace metal concentrations and scope for growth in *Mytilus edulis aoteanus* and *Perna canaliculus* from Wellington Harbour, New Zealand. The Science of the Total Environment 125:263-288.

Andren, A. W., and T. W. Bober, organizers. 1995. Transport, fate and effects of silver in the environment. Abstracts of 3rd international conference, 6-9 August, 1995, Washington, D.C. University of Wisconsin Sea Grant Institute, Madison. 35 pp.

Andren, A. W., T. W. Bober, E. A. Crecelius, J. R. Kramer, S. N. Luoma, J. H. Rodgers, and A. Sodergren, organizers. 1993. Proceedings of the first international conference on transport, fate, and effects of silver in the environment. University of Wisconsin Sea Grant Institute, Madison. 120 pp.

Andren, A. W., T. W. Bober, J. R. Kramer, A. Sodergren, E. A. Crecelius, S. N. Luoma, and J. H. Rodgers, organizers. 1994. Transport, fate and effects of silver in the environment. Proceedings of the 2nd international conference. University of Wisconsin Sea Grant Institute, Madison. 223 pp.

Andren, A. W., B. Wingert-Runge, and D. Sedlak. 1995. The occurrence and behavior of silver in natural waters. *In* A. W. Andren and T. W. Bober, organizers. Transport, fate and effects of silver in the environment. Abstracts of 3rd international conference, 6-9 August, 1995, Washington, D.C. University of Wisconsin Sea Grant Institute, Madison.

Antelman, M. S. 1994. Silver (II, III) disinfectants. Soap/Cosmetics/Chemical Specialties 70:52-59.

Aoki, T., K. Nihonyanagi, and T. Oba. 1993. Involvement of thiols in the induction of inward current induced by silver in frog skeletal muscle. Experientia 49:792-794.

Bard, C. C., J. J. Murphy, D. L. Stone, and C. J. Terhaar. 1976. Silver in photoprocessing effluents. Journal Water Pollution Control Federation 48:389-394.

Baudin, J. P., J. Garnier-Laplace, and A. Lambrechts. 1994. Uptake from water, depuration and tissue distribution of ^{110m}Ag in a freshwater fish *Cyprinus carpio* L. Water, Air, and Soil Pollution 72:129-141.

Berrow, S. D. 1991. Heavy metals in sediments and shellfish from Cork Harbour, Ireland. Marine Pollution Bulletin 22:467-469.

Berthet, B., J. C. Amiard, C. Amiard-Triquet, M. Martoja, and A. Y. Jeantet. 1992. Bioaccumulation, toxicity and physico-chemical speciation of silver in bivalve molluscs: ecotoxicological and health consequences. The Science of the Total Environment 125:97-122.

Berthet, B., C. Amiard-Triquet, and R. Martoja. 1990. Effets chimiques et histologiques de la decontamination de l'huitre *Crassostrea gigas* Thurberg prealablement exposee a l'argent. Water, Air, and Soil Pollution 50:355-369.

Bertine, K. K., and E. D. Goldberg. 1972. Trace elements in clams, mussels, and shrimp. Limnology and Oceanography 17:877-884.

Birge, W. J., and J. A. Zuiderveen. 1995. The comparative toxicity of silver to aquatic biota. *In* A. W. Andren and T. W. Bober, organizers. Transport, fate and effects of silver in the environment. Abstracts of 3rd international conference, 6-9 August 1995, Washington, D.C. University of Wisconsin Sea Grant Institute, Madison.

Bloom, N. S., and E. A. Crecilius. 1987. Distribution of silver, mercury, lead, copper and cadmium in central Puget Sound sediments. Marine Chemistry 21:377-390.

Bober, T. W., T. J. Dagon, and H. E. Fowler. 1992.Treatment of photographic processing wastes. Pages 173-227 *in* L. K. Wang and M. H. S. Wang, editors. Handbook of industrial waste treatment. Volume 1. Marcel Dekker, New York.

Braune, B. M., R. J. Norstrom, M. P. Wong, B.T. Collins, and J. Lee. 1991. Geographical distribution of metals in livers of polar bears from the Northwest Territories, Canada. The Science of the Total Environment 100:283-299.

Brooke, L.,R. Erickson, M. Kahl, F. Vande-Venter, S. Harting, T. Markee, C. Stephan, amd R. Spehar. 1994. Effects of laboratory test conditions on the toxicity of silver to aquatic organisms. Pages 119-122 *in* A. W. Andren, T. W. Bober, J. R. Kramer, A. Sodergren, E. A. Crecelius, S. N. Luoma, and J. H. Rodgers, organizers. Transport, fate and effects of silver in the environment. Proceedings of the 2nd international conference. University of Wisconsin Sea Grant Institute, Madison.

Brooks, R. R., and M. G. Rumsby. 1965. The biogeochemistry of trace element uptake by some New Zealand bivalves. Limnology and Oceanography 10:521-527.

Brown, C. L., and S. N. Luoma. 1995. Use of the euryhaline bivalve *Potamocorbula amurensis* as a biosentinel species to assess trace metal contamination in San Francisco Bay. Marine Ecology Progress Series 124:129-142.

Bruland, K. W., K. Bertine, M. Koide, and E. D. Goldberg. 1974. History of metal pollution in southern California coastal zone. Environmental Science and Technology 8:425-432.

Bryan, G. W. 1973. The occurrence and seasonal variation of trace metals in the scallops *Pecten maximus* (L.) and *Chlamys opercularis* (L.). Journal of the Marine Biological Association of the United Kingdom 53:145-166.

Bryan, G. W., and L. G. Hummerstone. 1977. Indicators of heavy metal contamination in the Looe estuary (Cornwall) with particular regard to silver and lead. Journal of the Marine Biological Association of the United Kingdom 57:75-92.

Bryan, G. W., and W. J. Langston. 1992.Bioavailability, accumulation and effects of heavy metals in sediments with special reference to United Kingdom estuaries: a review. Environmental Pollution 76:89-131.

Bryan, G. W., and H. Uysal. 1978. Heavy metals in the burrowing bivalve *Scrobicularia plana* from the Tamar estuary in relation to environmental levels. Journal of the Marine Biological Association of the United Kingdom 58:89-108.

Buell, J. W. 1991. Consequences of episodic exposure of chum salmon eggs/alevins to expected A-J mine tailing pond concentrations of Ag, Cd, Cu, Pb, and Zn. Pages 208-237 *in* P. Chapman, F. Bishay, E. Power, K. Hall, L. Harding, D. McLeary, M. Nassichuk, and W. Knapp, editors. Proceedings of the seventeenth annual aquatic toxicity workshop, 5-7 November 1990, Vancouver, B.C. Volume 1, Canadian Technical Report of Fisheries and Aquatic Sciences No. 1774 (Vol. 1).

Buhl, K. J., and S. J. Hamilton. 1991. Relative sensitivity of early life stages of Arctic grayling, coho salmon, and rainbow trout to nine inorganics. Ecotoxicology and Environmental Safety 22:184-197.

Cain, D. J., and S. N. Luoma. 1990. Influence of seasonal growth, age, and environmental exposure on Cu and Ag in a bivalve indicator, *Macoma balthica,* in San Francisco Bay. Marine Ecology Progress Series 60:45-55.

Calabrese, A., J. R. MacInnes, D. A. Nelson, R.A. Greig, and P. P. Yevich. 1984. Effects of long-term exposure to silver or copper on growth, bioaccumulation and histopathology in the blue mussel *Mytilus edulis.* Marine Environmental Research 11:253-274.

Calabrese, A., J. R. MacInnes, D. E. Nelson, and J. E. Miller. 1977a. Survival and growth of bivalve larvae under heavy-metal stress. Marine Biology 41:179-184.

Calabrese, A., F. P. Thurberg, and E. Gould. 1977b. Effects of cadmium, mercury, and silver on marine animals. Marine Fisheries Review 39:5-11.

Coglianese, M. P., and M. Martin. 1981. Individual and interactive effects of environmental stress on the embryonic development of the Pacific oyster, *Crassostrea gigas.* I. The toxicity of copper and silver. Marine Environmental Research 5:13-27.

Coleman, R. L., and J. E. Cearley. 1974. Silver toxicity and accumulation in largemouth bass and bluegill. Bulletin of Environmental Contamination and Toxicology 12:53-61.

Connell, D. B., J. G. Sanders, G. F. Riedel, and G. R. Abbe. 1991. Pathways of silver uptake and trophic transfer in estuarine organisms. Environmental Science and Technology 25:921-924.

Cooley, A. C., T. J. Dagon, P. W. Jenkins, and K. A. Robillard. 1988. Silver and the environment. Journal of Imaging Technology 14:183-189.

Crecelius, E. A. 1993. The concentration of silver in mussels and oysters from NOAA National Status and Trends Mussel Watch sites. Pages 65-66 *in* A. W. Andren, T. W. Bober, E. A. Crecelius, J. R. Kramer, S. N. Luoma, J. H. Rodgers, and A. Sodergren, organizers. Proceedings of the first international conference on transport, fate, and effects of silver in the environment. University of Wisconsin Sea Grant Institute, Madison.

Crecelius, E., and D. Phillips. 1995. Uptake and release of silver from marine sediment. *In* A. W. Andren and T. W. Bober, organizers. Transport, fate and effects of silver in the environment. Abstracts of 3rd international conference, 6-9 August 1995, Washington, D.C. University of Wisconsin Sea Grant Institute, Madison.

Dagon, T. J. 1973. Biological treatment of photo processing effluents. Journal Water Pollution Control Federation 45:2123-2135.

Daskalakis, K. D. 1995. Silver in oyster soft tissue: relations to site selection and sampling size. *In* A. W. Andren and T. W. Bober, organizers. Transport, fate, and effects of silver in the environment. Abstracts of 3rd international conference, 6-9 August 1995, Washington, D.C. University of Wisconsin Sea Grant Institute, Madison.

Davies, P. H., J. P. Goettl, Jr., and J. R. Sinley. 1978. Toxicity of silver to rainbow trout. Water Research 12:113-117.

Denizeau, F., and M. Marion. 1989. Genotoxic effects of heavy metals in rat hepatocytes. Cell Biology and Toxicology 5:15-25.

Denizeau, F., M. Marion, M. Chtaib, and J. P. Schmit. 1990. Toxicity of heavy metals in cultured hepatocytes. Environmental Toxicology and Chemistry 9:737-743.

Diamond, J. M., D. G. Mackler, M. Collins, and D. Gruber. 1990. Derivation of a freshwater silver criteria for the New River, Virginia, using representative species. Environmental Toxicology and Chemistry 9:1425-1434.

Eisler, R. 1981. Trace metal concentrations in marine organisms. Pergamon Press, Elmsford, New York. 687 pp.

Eisler, R., M. M. Barry, R. L. Lapan, Jr., G. Telek, E. W. Davey, and A. E. Soper. 1978. Metal survey of the marine clam *Pitar morrhuana* collected near a Rhode Island (USA) electroplating plant. Marine Biology 45:311-317.

Falandysz, J., and D. Danisiewicz. 1995. Bioconcentration factors (BCF) of silver in wild *Agaricus campestris.* Bulletin of Environmental Contamination and Toxicology 55:122-129.

Fisher, N. S., M. Bohe, and J. L. Teyssie. 1984. Accumulation and toxicity of Cd, Zn, Ag, and Hg in four marine phytoplankters. Marine Ecology Progress Series 18:201-213.

Fisher, N. S., V. T. Breslin, and M. Levandowsky. 1995. Accumulation of silver and lead in estuarine microzooplankton. Marine Ecology Progress Series 116:207-215.

Fisher, N.S., and J.R. Reinfelder. 1995. The trophic transfer of metals in marine systems. Pages 363-406 *in* A. Tessier and D.R. Turner, editors. Metal speciation and bioavailability in aquatic systems. John Wiley & Sons, New York.

Fisher, N. S., W. Wang, J. R. Reinfelder, and S.N. Luoma. 1994. Bioaccumulation of silver in marine bivalves. Pages 139-140 *in* A. W. Andren, T. W. Bober, J. R. Kramer, A. Sodergren, E. A. Crecelius, S. N. Luoma, and J. H. Rodgers, organizers. Transport, fate and effects of silver in the environment. Proceedings of the 2nd international conference. University of Wisconsin Sea Grant Institute, Madison.

Fowler, B. A., and G. F. Nordberg. 1986. Silver. Pages 521-530 *in* L. Friberg, G. F. Nordberg, and V. B. Vouk, editors. Handbook on the toxicology of metals. Volume II: specific metals. Elsevier, New York.

Fowler, S. W., and B. Oregioni. 1976. Trace metals in mussels from the N. W. Mediterranean. Marine Pollution Bulletin 7:26-29.

Freeman, R. A. 1979. Ecological kinetics of silver in an alpine lake ecosystem. Pages 342-358 *in* L. L. Marking and R. A. Kimerle, editors. Aquatic toxicology. Proceedings of the second annual symposium on aquatic toxicology. ASTM Special Technical Publication 667. American Society for Testing and Materials, Philadelphia.

Garnier, J., and J. P. Baudin. 1990. Retention of ingested ^{110m}Ag by a freshwater fish, *Salmo trutta* L. Water, Air, and Soil Pollution 50:409-421.

Garnier, J., J. P. Baudin, and L. Foulquier. 1990. Accumulation from water and depuration of ^{110m}Ag by a freshwater fish, *Salmo trutta* L. Water Research 24:1407-1414.

Goldberg, E. D., V. T. Bowen, J. W. Farrington, G. Harvey, J. H. Martin, P. L. Parker, R. W. Risebrough, W. Robertson, and E. Gamble. 1978. The mussel watch. Environmental Conservation 5:101-125.

Gould, E., and J. R. MacInnes. 1977. Short-term effects of two silver salts on tissue respiration and enzyme activity in the cunner (*Tautoglolabrus adspersus*). Bulletin of Environmental Contamination and Toxicology 18:401-408.

Graham, D. L. 1972. Trace metal levels in intertidal mollusks of California. Veliger 14:365-372.

Greig, R. A. 1975. Comparison of atomic absorption and neutron activation analyses for determination of silver, chromium, and zinc in various marine organisms. Analytical Chemistry 47:1682-1684.

Greig, R. A. 1979. Trace metal uptake by three species of mollusks. Bulletin of Environmental Contamination and Toxicology 22:643-647.

Greig, R. A., A. Adams, and D. R. Wenzloff. 1977a. Trace metal content of plankton and zooplankton collected from the New York Bight and Long Island Sound. Bulletin of Environmental Contamination and Toxicology 18:3-8.

Greig, R. A., and D. R. Wenzloff. 1977a. Final report on heavy metals in small pelagic finfish, euphausid crustaceans and apex predators, including sharks, as well as on heavy metals and hydrocarbons (C_{15+}) in sediments collected at stations in and near deepwater dumpsite 106. Pages 547-564 *in* Vol. III. Contaminant inputs and chemical characteristics. Baseline report of the environmental conditions on deepwater dumpsite 106. U.S. Department of Commerce, NOAA, Rockville, Md.

Greig, R. A., and D. R. Wenzloff. 1977b. Trace metals in finfish from the New York Bight and Long Island Sound. Marine Pollution Bulletin 8:198-200.

Greig, R. A., and D. R. Wenzloff. 1978. Metal accumulation and depuration by the American oyster, *Crassostrea virginica.* Bulletin of Environmental Contamination and Toxicology 20:499-504.

Greig, R. A., D. R. Wenzloff, B. Nelson, and C. Shelpuk. 1977b. Trace metals in organisms from ocean disposal sites of the middle eastern United States. Archives of Environmental Contamination and Toxicology 6:395-409.

Greig, R. A., D. R. Wenzloff, and J. B. Pearce. 1976. Distribution and abundance of heavy metals in finfish, invertebrates, and sediments collected at a deepwater disposal site. Marine Pollution Bulletin 7:185-187.

Hall, R. A., E. G. Zook, and G. M. Meaburn. 1978. National Marine Fisheries Service survey of trace elements in the fishery resources. U.S. Department of Commerce NOAA Technical Report NMFS SSRF-721. 323 pp.

Heit, M. 1979. Variability of the concentrations of seventeen trace elements in the muscle and liver of a single striped bass, *Morone saxatilis.* Bulletin of Environmental Contamination and Toxicology 23:1-5.

Hellou, J., W. G. Warren, J. F. Payne, S. Belkhode, and P. Lobel. 1992. Heavy metals and other elements in three tissues of cod, *Gadus morhua* from the northwest Atlantic. Marine Pollution Bulletin 24:452-458.

Heyl, A. V., W. E. Hall, A. E. Weissenborn, H. K. Stager, W. P. Puffett, and B. L. Reed. 1973. Silver. Pages 581-603 *in* D. A. Brobst and W. P. Pratt, editors. United States mineral resources. U.S. Geological Survey Professional Paper 820.

Hirsch, M., M. Ritter, K. Roser, P. Garrisi, and S. Forsythe. 1993. The effect of silver on plants grown in sludge-amended soils. Pages 69-73 *in* A. W. Andren, T. W. Bober, E. A. Crecelius, J. R. Kramer, S. N. Luoma, J. H. Rodgers, and A. Sodergren, organizers. Proceedings of the first international conference on transport, fate, and effects of silver in the environment. University of Wisconsin Sea Grant Institute, Madison.

Hogstrand, C., F. Galvez, and C. M. Wood. 1996. Toxicity, silver accumulation and metallothionein induction in freshwater rainbow trout during exposure to different silver salts. Environmental Toxicology and Chemistry 15; 1102-1108.

Janes, N., and R. C. Playle. 1995. Modeling silver binding to gills of rainbow trout (*Oncorhynchus mykiss*). Environmental Toxicology and Chemistry 14:1847-1858.

Jeanthon, C., and D. Prieur. 1990. Susceptibility to heavy metals and characterization of heterotrophic bacteria isolated from two hydrothermal vent polychaete annelids, *Alvinella pompejana* and *Alvinella caudata*. Applied and Environmental Microbiology 56:3308-3314.

Kirschenbaum, L. J. 1991. Higher oxidation states in group IB: the chemistry of hypervalent silver. Pages 1-29 *in* C. P. Murthy, editor. T. N. Rao commemorative celebration. Osmania University, Hyderabad, India.

Klaassen, C. D., M. O. Amdur, and J. Doull, editors. 1986. Casarett and Doull's toxicology. The basic science of poisons. Third edition. Macmillan, New York. 974 pp.

Klein-MacPhee, G., J. A. Cardin, and W. J. Berry. 1984. Effects of silver on eggs and larvae of the winter flounder. Transactions of the American Fisheries Society 113:247-251.

Koechlin, N., and M. Grasset. 1988. Silver contamination in the marine polychaete annelid *Sabella pavonina* S.: a cytological and analytical study. Marine Environmental Research 26:249-263.

Kouadio, I., L. J. Kirschenbaum, and R. N. Mehrota. 1990. The oxidation of iodide by the tetrahydroxoargentate (III) ion in aqueous alkaline media. Journal of the Chemical Society, Dalton Transactions 1990:1928-1933.

Kumar, R., and S. R. Bawa. 1979. Hepatic silver binding protein (Ag BP) from sparrow (*Passer domesticus*). Experientia 35:1621-1623.

Lande, E. 1977. Heavy metal pollution in Trondheimsfjorden, Norway, and the recorded effects on the fauna and flora. Environmental Pollution 12:187-198.

Lauenstein, G. G., A. Robertson, and T. P. O'Connor. 1990. Comparison of trace metal data in mussels and oysters from a mussel watch programme of the 1970s with those from a 1980s programme. Marine Pollution Bulletin 21:440-447.

Lawrence, J. M., W. D. Mahon, W. Avery, and M. Lares. 1993. Concentrations of metals in *Luidia clathrata* and *Luidia senegalensis* (Echinodermata: Asteroidea) in Tampa Bay and the nearshore Gulf of Mexico, Florida. Comparative Biochemistry and Physiology 105C:203-206.

LeBlanc, G. A., J. D. Mastone, A. P. Paradice, B. F. Wilson, H. B. Lockhart, Jr., and K. A. Robillard. 1984. The influence of speciation on the toxicity of silver to fathead minnow (*Pimephales promelas*). Environmental Toxicology and Chemistry 3:37-46.

Lima, A. R., C. Curtis, D. E. Hammermeister, D. J. Call, and T. A. Felhaber. 1982. Acute toxicity of silver to selected fish and invertebrates. Bulletin of Environmental Contamination and Toxicology 29:184-189.

Lockhart, H. B., Jr. 1983. Silver compounds. Pages 16-32 *in* Encyclopedia of chemical technology, third edition. John Wiley & Sons, New York.

Luoma, S. N. 1994. Fate, bioavailability and toxicity of silver in estuarine environments. Pages 151-155 *in* A. W. Andren, T. W. Bober, J. R. Kramer, A. Sodergren, E. A. Crecelius, S. N. Luoma, and J. H. Rodgers, organizers. Transport, fate and effects of silver in the environment. Proceedings of the 2nd international conference. University of Wisconsin Sea Grant Institute, Madison.

Luoma, S. N., R. Dagovitz, and E. Axtmann. 1990. Temporally intensive study of trace metals in sediments and bivalves from a large river-estuarine system: Suisan Bay/Delta in San Francisco Bay. The Science of the Total Environment 97/98:685-712.

Luoma, S. N., and E. A. Jenne. 1977. The availability of sediment-bound cobalt, silver, and zinc to a deposit-feeding clam. Pages 213-230 *in* H. Drucker and R. E. Wildung, editors. Biological implications of metals in the environment. ERDA Symposium Series 42. Available as CONF-750929 from the National Technical Information Service, Springfield, Va.

Luoma, S. N., and D. J. H. Phillips. 1988. Distribution, variability, and impacts of trace elements in San Francisco Bay. Marine Pollution Bulletin 19:413-425.

Lytle, P. E. 1984. Fate and speciation of silver in publicly owned treatment works. Environmental Toxicology and Chemistry 3:21-30.

Martin, J. H., P. D. Elliott, V. C. Anderlini, D. Girvin, S. A. Jacobs, R. W. Risebrough, R. L. Delong, and W. G. Gilmartin. 1976. Mercury-selenium bromine imbalance in premature parturient California sea lions. Marine Biology 35:91-104.

Martin, M., M. D. Stephenson, D. R.Smith, E. A. Gitierrez-Galindo, and G. F. Munoz. 1988. Use of silver in mussels as a tracer of domestic wastewater discharge. Marine Pollution Bulletin 19:512-520.

Miramand, P., and D. Bentley. 1992. Concentration and distribution of heavy metals in tissues of two cephalopods, *Eledone cirrhosa* and *Sepia officinalis,* from the French coast of the English Channel. Marine Biology 114:407-414.

Morgan, L. J., F. Galvez, R. F. Munger, C. M. Wood, and R. Henry. 1995. The physiological effects of acute silver exposure in rainbow trout (*Oncorhynchus mykiss*). *In* A. W. Andren and T. W. Bober, organizers. Transport, fate and effects of silver in the environment. Abstracts of 3rd international conference. 6-9 August 1995, Washington, D.C. University of Wisconsin Sea Grant Institute, Madison.

Morse, J. W., B. J. Presley, R. J. Taylor, G. Benoit, and P. Santschi. 1993. Trace metal chemistry of Galveston Bay: water, sediments, and biota. Marine Environmental Research 36:1-37.

Nalecz-Jawecki, G., K. Demkowicz-Dobrzanski, and J. Sawicki. 1993. Protozoan *Spirostomum ambiguum* as a highly sensitive bioindicator for rapid and easy determination of water quality. The Science of the Total Environment, Supplement 2:1227-1234.

National Association of Photographic Manufacturers [NAPM]. 1974. Environmental effect of photoprocessing chemicals. Volumes I and II. NAPM, Harrison, N.Y. 324 pp. plus appendices.

Navrot, J., A. J. Amiel, and J. Kronfeld. 1974. *Patella vulgata*: a biological monitor of coastal metal pollution—a preliminary study. Environmental Pollution 7:303-308.

Nebeker, A. V, C. K. McAuliffe, R. Mshar, and D. G. Stevens. 1983. Toxicity of silver to steelhead and rainbow trout, fathead minnows and *Daphnia magna*. Environmental Toxicology and Chemistry 2:95-104.

Nehring, R. B. 1976. Aquatic insects as biological monitors of heavy metal pollution. Bulletin of Environmental Contamination and Toxicology 15:147-154.

Nelson, D. A., A. Calabrese, R. A. Greig, P. P. Yevich, and S. Chang. 1983. Long-term silver effects on the marine gastropod *Crepidula fornicata*. Marine Ecology Progress Series 12:155-165.

Nelson, D. A., A. Calabrese, B. A. Nelson, J. R. MacInnes, and D. R. Wenzloff. 1976. Biological effects of heavy metals on juvenile bay scallops, *Argopecten irradians*, in short-term exposures. Bulletin of Environmental Contamination and Toxicology 16:275-282.

Nolan, C., and H. Dahlgaard. 1991. Accumulation of metal radiotracers by *Mytilus edulis*. Marine Ecology Progress Series 70:165-174.

Ohlendorf, H. M., R. W. Lowe, P. R. Kelly, and T. E. Harvey. 1986. Selenium and heavy metals in San Francisco Bay diving ducks. Journal of Wildlife Management 50:64-71.

Papadopoulu, C., and E. M. Kassimati. 1977. Stable elements in skeletal formations of fish species from Greek waters. Thalassia Jugoslavica 13:187-192.

Pentreath, R. J. 1977. The accumulation of ^{110m}Ag by the plaice, *Pleuronectes platessa* L. and the thornback ray, *Raja clavata* L. Journal of Experimental Marine Biology and Ecology 29:315-325.

Pouvreau, B., and J. C. Amiard. 1974. Etude experimentale de l'accumulation de l'argent 110m chez divers organismes marine. Commission Energie Atomique France, Report CEA-R-4571. 19 pp.

Presley, B. J., R. J. Taylor, and P. N. Boothe. 1990. Trace metals in Gulf of Mexico oysters. The Science of the Total Environment 97/98:551-593.

Pullen, J. S. H., and P. S. Rainbow. 1991. The composition of pyrophosphate heavy metal detoxification granules in barnacles. Journal of Experimental Marine Biology and Ecology 150:249-266.

Ramelow, G. J., C. L. Webre, C. S. Mueller, J. N. Beck, J. C. Young, and M. P. Langley. 1989. Variations of heavy metals and arsenic in fish and other organisms from the Calcasieu River and Lake, Louisiana. Archives of Environmental Contamination and Toxicology 18:804-818.

Reese, R. G. 1991. Silver. Pages 148-149 *in* Mineral commodity summaries 1991. U.S. Bureau of Mines, Washington, DC 20241.

Ridgway, L. P., and D. A. Karnofsky. 1952. The effects of metals on the chick embryo: toxicity and production of abnormalities in development. Annals of the New York Academy of Sciences 55:203-215.

Rivera-Duarte, I., and A. R. Flegal. 1993. Pore water fluxes of silver in estuaries. Pages 37-39 *in* A. W. Andren, T. W. Bober, E. A. Crecelius, J. R. Kramer, S. N. Luoma, J. H. Rodgers, and A. Sodergren, organizers. Proceedings of the first international conference on transport, fate, and effects of silver in the environment. University of Wisconsin Sea Grant Institute, Madison.

Rodgers, J. H., Jr., E. Deaver, and P. Rogers. 1994. Evaluations of the bioavailability and toxicity of silver in sediment. Pages 131-137 *in* A. W. Andren, T. W. Bober, J. R. Kramer, A. Sodergren, E. A. Crecelius, S. N. Luoma, and J. H. Rodgers, organizers. Transport, fate and effects of silver in the environment. Proceedings of the 2nd international conference. University of Wisconsin Sea Grant Institute, Madison.

Rombough, P. J. 1985. The influence of the zona radiata on the toxicities of zinc, lead, mercury, copper and silver ions to embryos of steelhead trout *Salmo gairdneri.* Comparative Biochemistry and Physiology 82C:115-117.

Rose, K. A., J. K. Summers, R. I. Mclean, and S. L. Domotor. 1988. Radiosilver (Ag-110m) concentrations in Chesapeake Bay oysters maintained near a nuclear powerplant: a statistical analysis. Environmental Monitoring and Assessment 10:205-218.

Sanders, J. G., and G. R. Abbe. 1987. The role of suspended sediments and phytoplankton in the partitioning and transport of silver in estuaries. Continental Shelf Research 7:1357-1361.

Sanders, J. G., and G. R. Abbe. 1989. Silver transport and impact in estuarine and marine systems. Pages 5-18 *in* G. W. Suter II and M. A. Lewis, editors. Aquatic toxicology and environmental fate. Vol. 11. American Society for Testing and Materials, Special Technical Publication 1007, Philadelphia.

Sanders, J. G., G. R. Abbe, and G. F. Riedel. 1990. Silver uptake and subsequent effects on growth and species composition in an estuarine community. The Science of the Total Environment 97/98:761-769.

Sanders, J. G., and S. J. Cibik. 1988. Response of Chesapeake Bay phytoplankton communities to low levels of toxic substances. Marine Pollution Bulletin 19:439-444.

Sanders, J. G., G. F. Riedel, and G. R. Abbe. 1991. Factors controlling the spatial and temporal variability of trace metal concentrations in *Crassostrea virginica* (Gmelin). Pages 335-339 *in* M. Elliott and J. P. Ducrotoy, editors. Estuaries and coasts: spatial and temporal comparisons. ECSA Symposium 19 (University of Caen, France, 1989). Olsen & Olsen, Fredensborg, Denmark.

Scanlon, P. F., R. G. Oderwald, T. J. Dietrick, and J. L. Coggin. 1980. Heavy metal concentrations in feathers of ruffed grouse shot by Virginia hunters. Bulletin of Environmental Contamination and Toxicology 25:947-949.

Schildkraut, D. E. 1993. Application of analytical voltammetric methods to the determination of silver at sub-ng/mL levels. Pages 5-7 *in* A. W. Andren, T. W. Bober, E. A. Crecelius, J. R. Kramer, S. N. Luoma, J. H. Rodgers, and A. Sodergren, organizers. Proceedings of the first international conference on transport, fate, and effects of silver in the environment. University of Wisconsin Sea Grant Institute, Madison.

Segar, D. A., J. D. Collins, and J. P. Riley. 1971. The distribution of the major and some minor elements in marine animals. Part II. Molluscs. Journal of the Marine Biological Association of the United Kingdom 51:131-136.

Shafer, M. M., D. E. Armstrong, and J. T. Overdier. 1995. Silver levels and partitioning in effluent-receiving streams and rivers tributary to Lake Michigan, and a preliminary mass balance for silver in the Lake Michigan basin. *In* A. W. Andren and T. W. Bober, organizers. Transport, fate and effects of silver in the environment. Abstracts of 3rd international conference, 6-9 August 1995, Washington, D.C. University of Wisconsin Sea Grant Institute, Madison.

Smith, I. C., and B. L. Carson. 1977. Trace metals in the environment. Vol. 2: Silver. Ann Arbor Science Publishers, Ann Arbor, Mich. 469 pp.

Song, S., and J. G. Osteryoung. 1993. Determination of silver (I) ion by anodic stripping in flow injection system. Pages 109-111 *in* A. W. Andren, T. W. Bober, E. A. Crecelius, J. R. Kramer, S. N. Luoma, J. H. Rodgers, and A. Sodergren, organizers. Proceedings of the first international conference on transport, fate, and effects of silver in the environment. University of Wisconsin Sea Grant Institute, Madison.

Stephenson, J. D., and G. H. Leonard. 1994. Evidence for the decline of silver and lead and the increase of copper from 1977 to 1990 in the coastal marine waters of California. Marine Pollution Bulletin 28:148-153.

Sun, Y., L. J. Kirschenbaum, and I. Kouadio. 1991. Kinetics and mechanism of the multi-step oxidation of ethylenediaminetetracetate by $[Ag(OH)_4]^-$ in alkaline media. Journal of the Chemical Society, Dalton Transactions 1991:2311-2315.

Szefer, P., J. Pempkowiak, B. Skwarzec, R. Bojanowski, and E. Holm. 1993. Concentration of selected metals in penguins and other representative fauna of the Antarctica. The Science of the Total Environment 138:281-288.

Szefer, P., K. Szefer, J. Pempkowiak, B. Skwarzec, R. Bojanowski, and E. Holm. 1994. Distribution and coassociations of selected metals in seals of the Antarctic. Environmental Pollution 83:341-349.

Talbot, V. 1985. Heavy metal concentrations in the oysters *Saccostrea cucullata* and *Saccostrea* sp. from the Dampier Archipelago, Western Australia. Australian Journal of Marine and Freshwater Research 36:169-175.

Tariq, J., M. Jaffar, M. Ashraf, and M. Moazzam. 1993. Heavy metal concentrations in fish, shrimp, seaweed, sediment, and water from the Arabian Sea, Pakistan. Marine Pollution Bulletin 26:644-647.

Terhaar, C. J., W. S. Ewell, S. P. Dziuba, W. W. White, and P. J. Murphy. 1977. A laboratory model for evaluating the behavior of heavy metals in an aquatic environment. Water Research 11:101-110.

Thurberg, F. P., W. D. Cable, M. A. Dawson, J. R. MacInnes, and D. R. Wenzloff. 1975. Respiratory response of larval, juvenile, and adult surf clams, *Spisula solidissima,* to silver. Pages 41-52 *in* J. J. Cech, Jr., D. W. Bridges, and D. B. Horton, editors. Respiration of marine organisms. TRIGOM Publishers, South Portland, Maine.

Thurberg, F. P., A. Calabrese, and M. A. Dawson. 1974. Effects of silver on oxygen consumption of bivalves at various salinities. Pages 67-78 *in* F. J. Vernberg and W. B. Vernberg, editors. Pollution and physiology of marine organisms. Academic Press, New York.

Thurberg, F. P., and R. S. Collier. 1977. Respiratory response of cunners to silver. Marine Pollution Bulletin 8:40-41.

Truchet, M., R. Martoja, and B. Berthet. 1990. Consequences histologiques de la pollution metallique d'un estuairesur deux mollusques, *Littorina littorea* L. et *Scrobicularia plana* da costa. Comptes rendus de l'Academie des Sciences. Serie III, 311:261-268.

U.S. Bureau of Mines. 1980. Mineral commodity summaries 1980. Available from the U.S. Government Printing Office: 1979-603-002/128.

U.S. Environmental Protection Agency (EPA). 1980. Ambient water quality criteria for silver. U.S. Environmental Protection Agency Report 440/5-80-071. 212 pp.

U.S. Environmental Protection Agency (EPA). 1987. Ambient aquatic life water quality criteria for silver. Draft. Available from the National Technical Information Service, Springfield, Va. 104 pp.

U.S. Environmental Protection Agency (EPA). 1995a. Stay of water quality criteria for metals. Federal Register 60 (86):22227-22229.

U.S. Environmental Protection Agency (EPA). 1995b. Water quality standards; establishment of numeric criteria for priority toxic pollutants; states' compliance—revision of metals criteria. Federal Register 60 (86):22229-22237.

U.S. Public Health Service (PHS). 1990. Toxicological profile for silver. Agency for Toxic Substances and Disease Registry TP-90-24. 145 pp.

Vermeer, K., and D. B. Peakall. 1979. Trace metals in seaducks of the Fraser River delta intertidal area, British Columbia. Marine Pollution Bulletin 10:189-191.

Williams, P. L., and D. B. Dusenbery. 1990. Aquatic toxicity testing using the nematode, *Caenorhabditis elegans*. Environmental Toxicology and Chemistry 9:1285-1290.

Windom, H. L., and R. G. Smith. 1972. Distribution of iron, magnesium, copper, zinc, and silver in oysters along the Georgia coast. Journal of the Fisheries Research Board of Canada 29:450-452.

Wingert-Runge, B., and A. W. Andren. 1994. Desorption behavior of silver from natural sediments under freshwater and marine conditions. Pages 89-93 *in* A. W. Andren, T. W. Bober, J. R. Kramer, A. Sodergren, E. A. Crecelius, S. N. Luoma, and J. H. Rodgers, organizers. Transport, fate and effects of silver in the environment. Proceedings of the 2nd international conference. University of Wisconsin Sea Grant Institute, Madison.

Wood, C. M., C. Hogstrand, F. Galvez, and R. S. Munger. 1996a. The physiology of waterborne silver toxicity in freshwater rainbow trout (*Oncorhynchus mykiss*): 1. The effects of ionic Ag^{+}. Aquatic Toxicology. 35:93-109.

Wood, C. M., C. Hogstrand, F. Galvez, and R. S. Munger. 1996b. The physiology of waterbone silver toxicity in freshwater rainbow trout (*Oncorhynchus mykiss*): 2. The effects of silver thiosulfate. Aquatic Toxicology. 35:111-125.

Wood, C. M., I. Morgan, F. Galvez, and C. Hogstrand. 1995. The toxicity of silver in fresh and marine waters. *In* A. W. Andren and T. W. Bober, organizers. Transport, fate and effects of silver in the environment. Abstracts of 3rd international conference, 6-9 August 1995, Washington, D.C. University of Wisconsin Sea Grant Institute, Madison.

Wood, C. M., S. Munger, F. Galvez, and C. Hogstrand. 1994. The physiology of silver toxicity in freshwater fish. Pages 109-114 *in* A. W. Andren, T. W. Bober, J. R. Kramer, A. Sodergren, E. A. Crecelius, S. N. Luoma, and J. H. Rodgers, organizers. Transport, fate and effects of silver in the environment. Proceedings of the 2nd international conference. University of Wisconsin Sea Grant Institute, Madison.