MINISTRY OF ENVIRONMENT, LANDS AND PARKS
PROVINCE OF BRITISH COLUMBIA

**AMBIENT WATER QUALITY CRITERIA FOR**

# SILVER

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February, 1996

ACKNOWLEDGEMENT

Thanks to Alasdair I. Beattie, who, as a student in the University of Victoria's Biology Co-op program, carried out most of the literature survey on which this report is based, and also wrote the first draft of the chapter on aquatic life.

## *Summary*

This document is one in a series that establishes ambient water quality criteria for British Columbia. It includes an overview which is followed by the main body of the report. This document sets criteria for silver to protect freshwater and marine aquatic life.

Criteria were not set for human, livestock or wildlife drinking water, recreational waters, irrigation water or industrial water uses, since, either suitable data documenting the effects of silver for these uses were lacking, or the criteria would have been about 1000 times higher than the aquatic life criteria and therefore redundant.

Silver is most toxic to microscopic organisms or larval forms of aquatic animals. There is no evidence that silver is naturally transformed to a hazardous biologically-available form (such as mercury into methyl mercury). Ionic silver is more toxic to aquatic organisms than silver compounds. Thiosulphate-complexed silver breaks down to silver sulphide which is less toxic than the silver ion. Silver criteria are summarized in the

chapter on Recommended Criteria. A more detailed discussion of the criteria is presented in the main body of the report.

***Preface***

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## Establishing the Criteria

The Ministry of Environment, Lands and Parks is developing ambient water quality criteria for British Columbia. This work has two goals:

· to provide criteria for the evaluation of data on water, sediment and biota
· to provide criteria for site-specific ambient water quality objectives

The criteria represent safe conditions or safe levels of a substance, and are set to protect various water uses. A criterion is defined as "A maximum and/or a minimum value for a physical, chemical or biological characteristic of water, sediment or biota, which should not be exceeded to prevent specified detrimental effects from occurring to a water use, including aquatic life, under specified environmental conditions."

The criteria are applied province-wide, but they are use-specific, and are being developed for these water uses:

· raw drinking water, public water supply and food processing*
· aquatic life and wildlife
· agriculture (livestock watering and irrigation)
· recreation and aesthetics **
· industrial water supplies

The criteria are established after considering the scientific literature, criteria from other jurisdictions and environmental conditions in British Columbia. The scientific literature provides information about the effects of toxicants on various life forms. This information is not always conclusive because it is usually based on laboratory work that, at best, only approximates field conditions. To compensate for this uncertainty, and applying the "precautionary principle", the criteria have built-in safety factors that are conservative, but reflect the natural background in the province. The criteria are subject to review and revision as new information becomes available or as other circumstances dictate.

## Using the Criteria to Set Objectives

The criteria are used to set ambient water quality objectives for specific waterbodies. The objectives are also based on present and future uses, waste discharges, hydrology, limnology, oceanography, and on existing background water quality.

In most cases, the objectives will be the same as the criteria. However, when natural background levels exceed the criteria, the objectives could be less stringent than the criteria. In rare instances-for example, if the resource is unusually valuable or of special provincial significance-the safety factor could be increased by using objectives that are more stringent than the criteria. Another approach in special cases would be to develop site-specific objectives by conducting toxicity experiments in the field. However, because this approach is costly and time consuming, it is seldom used.

Neither the criteria nor the objectives derived from them have any legal standing. Objectives can be used to calculate waste discharge limits.

These limits are outlined in waste management permits which do have legal standing. (Objectives are not usually incorporated as conditions of a permit.) Objectives are also used in the preparation of waste management orders and approvals. These documents also have legal standing.

***Introduction***

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## silver toxicity

A natural or man-made chemical present in the environment does not always lead to human or animal exposure. Exposure requires contact with substances containing the chemical. Exposure itself is not necessarily harmful; several other factors determine whether contact leads to harmful effects and the type and severity of these effects. These factors include the dose (how much), the duration (how long), the timing (when in the life cycle), the route of exposure (injection, inhalation, ingestion, contact) and individual characteristics and lifestyles (sex, age, health, habits, fitness, genetic predisposition).

Populations that are unusually susceptible to toxic effects from silver are those with dietary deficiencies of vitamin E or selenium, or those with genetically-based deficiencies in the metabolism of these essential nutrients. Those populations with damaged livers and those exposed to very high selenium levels in their diet are also at higher risk. Some people may exhibit an allergic response to silver.

## silver speciation

Most toxicological studies have been conducted with silver in the free, elemental or 0 oxidation state, and with the +1 monovalent silver ion. The rarer +2 and +3 oxidation states have not been studied adequately. The majority of silver resulting from photo-processing occurs in an insoluble form. Theoretical calculations of organic and inorganic silver complexes indicate that, due to the low solubility of silver sulphide and the high affinity of silver for sulphide, little free silver would occur at equilibrium, in effluents or surface waters that contained any sulphide.

Bioassays have demonstrated that, although 'free' silver caused the death of fathead minnows at relatively low silver concentrations, silver thiosulphate and silver sulphide salts had no effect at over 1000 times the `free' silver concentration.

The Aquatic Ecosystem Objectives Committee of IJC agrees that 'free' silver is a better measure of toxicity than total silver, The committee does not recommend the adoption of an objective based on 'free' silver for the following reasons:

-near an effluent, silver may not yet be in equilibrium with all the available complexing agents.

-sulphide, which is the only reactant likely to reduce silver adequately, is readily oxidized to sulphate when oxygen is available. The half-life of sulphide is about 50 hours but oxidation rates may be increased five-fold by metals such as calcium. Sulphide has not been found, even at very low detection limits, in the Great Lakes.

-weaker organic complexing agents for silver still permit 'free' silver to exist at concentrations near the objective level.

-the method presented by Kodak for measuring silver ion activity is not adequate for low environmental concentrations; there are difficulties with reproducibility, dependability and comparability. The method is only reliable at levels of 'free' silver higher than the criteria.

Until a reliable method is developed to measure 'free' silver at concentrations below 0.1 micrograms per litre, silver objectives should be expressed as total silver. This level is the routine total or dissolved silver detection limit for water samples analyzed in labs used by the British Columbia Ministry of Environment for analysis of ambient water samples.

## *Recommended Criteria*

The following criteria are based on information presented in the technical appendix, and are summarized in the tables below. The Canadian Council of Ministers of the Environment (CCME) is considering similar guidelines for silver.

## aquatic life

Summary Table

recommended criteria for the protection of
marine and freshwater aquatic life

| | Environment | Criteria as total Silver | Conditions |
|---|---|---|---|
| | fresh water | 0.05 µg/L as a 30-day mean | hardness 100 mg/L |
| | | 0.1 µg/L maximum | hardness 100 mg/L |
| | | 1.5 µg/L as a 30-day mean | hardness > 100 mg/L |
| | | 3.0 µg/L maximum | hardness > 100 mg/L |
| | marine water | 1.5 µg/L as a 30-day mean | open coast and estuaries |
| | | 3.0 µg/L maximum | open coast and estuaries |

## drinking water and food processing industries

For human, laboratory animal, wildlife and livestock drinking water, and for food processing industries where water is incorporated into the product, no silver criterion appears to be necessary. The aquatic life criteria are more than adequate for any such uses. The level used by Health and Welfare Canada in 1987, and by Australia in 1992, for human drinking water was 50 µg/L. Health and Welfare Canada deleted silver from the 1989 Guidelines for Canadian Drinking Water Quality as the value was very conservative and had no defensible, scientific basis.

## recreation and aesthetics

Silver is not volatile, has no offensive odour and does not cause any colour or other visual effects in water, therefore it is not a concern for aesthetics. For recreation, levels of concern would be at least as high as, or higher, than the drinking water criterion. Therefore, no silver criterion appears to be necessary for these uses of the water. The aquatic life criteria are more than adequate for any such uses.

## industrial

Industries, such as solid state electronics and photofinishing, which may have stringent silver requirements, may need to reduce silver concentrations in-house to levels suitable for their processes.

## *Application of Criteria for Aquatic Life*

Silver is a disinfectant for non-spore forming bacteria at concentrations about 1000 times lower than the levels at which it is toxic to mammalian life. This extreme mammalian-to-bacterial toxicity differential is the definition of an oligodynamic material. The low concentration necessary for oligodynamic activity allows silver or one of its insoluble salts to be used indefinitely in contact with sterile liquids without silver levels building up to concentrations harmful to people.

The biological effects of silver are apparently due to reversible bonds with enzymes and other active molecules on the surface of cells. Due to its sulphydryl binding propensity, biologically-available silver disrupts membranes, disables proteins and inhibits enzymes. The ionic form of silver is necessary for biological activity and the lipid phase of the membrane appears to be important in adsorbing silver ions to living cells. The active sites on enzymes which are affected by silver are apparently the electron-rich functional groups such as-SH groups.

Silver combines with plasma proteins, is removed by the liver and over 90% is eliminated in the bile; most of this in the feces with very little in the urine. That silver which is not excreted is deposited in the skin and mucous tissues. Tissue deposition of silver results from precipitation of insoluble salts such as silver chloride and silver phosphate. These may be transformed to soluble silver sulphide albuminates and bind with amino or carboxyl groups in proteins and nucleic acids. They may also be oxidized to metallic silver by ascorbic acid or catecholamines.

Argyria, silver deposition, occurs in all organs. Common deposition sites for people who have no history of therapeutic use are the liver, skin, pancreas, adrenals, glomeruli of the kidney, brain, bone marrow, walls of the blood vessels, thyroid, mesenteric glands, choroid plexus, spleen and testes. Generalized argyria is indicated by slate-gray skin and hair colouring, silver finger nails, a blue halo around the cornea and in the conjunctiva of the eye, disturbance of dark adaptation and turbidity of the anterior lens capsule. The tissue content and distribution pattern of silver deposition is a function of the intake route, quantity and chemical form.

The discoloured skin in argyric patients exposed to ultraviolet radiation is likely caused by photoreduction of silver chloride to metallic silver, which is then oxidized to black silver sulphide and bound by tissues. If the diet is high in selenium, the silver sulphide is converted to silver selenide which may result in higher silver deposition rates than with silver sulphide.

Silver is tightly bound by sewage sludge and elevated levels of silver are often associated with sewage outfalls receiving minimal treatment. In the absence of sewage, silver associates with iron oxides and humic substances. The relative bioavailability of either silver-inorganic complexes or silver-organic complexes appears to depend on the individual compounds. Silver-inorganic complexes are probably the most common in the marine environment. Silver-chlorides are generally not bioavailable except for silver chloride. Silver-iron oxides or silver-magnesium complexes in sediments increase the availability to bottom feeding organisms. Activated sludge organisms may bioaccumulate silver at 100 times the concentration in the effluent.

Silver has low toxicity to vertebrate animals and is eliminated rapidly when ingested orally. It is not a cumulative toxin. Since surface waters in Canada generally contain low levels of silver and there are few data on chronic silver toxicity to animals, no criteria seem to be justified at this time for wildlife, livestock or laboratory animal drinking water. Wildlife, free range and confined livestock and laboratory animals can safely drink water which meets the aquatic life criteria.

Maximum and 30-day average criteria have been set despite the paucity of data for marine fish since fish are not sensitive to silver at concentrations over 10 times the levels that affect most invertebrates and algae. Criteria are set to protect the most sensitive lifestage of the most sensitive species. The literature indicates that the most sensitive organisms are phytoplankton and the embryonic and larval stages of invertebrates. Since the sensitivity of invertebrates and phytoplankton to silver is much greater than that of fish, and because a great deal of literature is available concerning

their sensitivies, the requirements for marine fish data were considered to be superfluous and were waived.

The life stage, size of the organisms, length of time of exposure, species sensitivity and salinity all contribute to the variation in toxicity reported in the literature. The relative proportion of the free silver+ ion to the total silver content in the oceans is a function of the salinity. In the ocean, most of the silver at any one time is present as chloride complexes; of these complexes only the mono-chloro complex silver chloride is biologically available. At a salinity of 25 parts per thousand, which is almost the natural concentration in the sea, only about 1 part in 16 000 parts of the silver would exist as the free ion.

This lack of free silver ion is generally not accounted for in the literature where most values are given as total silver and the actual toxic forms of the silver would be a considerably smaller value. This factor accounts for much of the wide variation in toxicity values reported in the literature, for while salinity is generally reported, it is rarely considered as a factor affecting toxicity. The total silver measurements reflect the worst-case scenario, which should be considered when setting any water quality criteria in order to protect the most sensitive life stages of the most sensitive species. This worst-case scenario is particularly important for estuaries, where salinity can fluctuate quite dramatically.

Silver is one of the most toxic of the heavy metals to freshwater micro-organisms. Water hardness, length of exposure, size of the organism and life stage of the organism all affect the toxicity values. Reports of the validity of static versus flow-through tests within the literature are variable; however static tests with renewal of test water appear to be as accurate as flow-through bioassays. Invertebrates and embryos of fish are generally much more sensitive than juvenile and adult fish.

The effect of speciation on the acute and chronic toxicity of silver was compared using the fathead minnow as the test organism. Silver sulfide, silver thiosulfate and silver chloride were compared to the silver ion, added as silver nitrate. The tests were flow-through in soft water at 25C. Silver chloride was found to be 300 times less toxic, silver sulfide was 15,000 times less toxic, and silver thiosulfate was 17,500 times less toxic than silver nitrate.

Most existing silver criteria, objectives or regulated amounts are not based on the free ionic monovalent ion, which is acutely toxic to aquatic life. Instead they are based on total silver which includes the metal, complexes and precipitates, all of which are very much less toxic than the monovalent ion. Thus, these existing regulations and criteria

are often overprotective. A method of measuring the biologically-available forms of silver is needed so that the criteria and the risk are correlated.

Regulations should reflect the appropriate risk but the problem is that there is no monovalent ion specific measurement. In addition, some non biologically-available silver may be in forms that are in equilibrium with monovalent silver and thus much of the silver pool becomes ultimately available as the monovalent silver is taken up. Benthic organisms will take up some insoluble forms of silver as they graze, and thus more than just the monovalent form is available to them. Therefore, total silver is recommended.

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# 1. Introduction

## *1.1 silver toxicity determination*

A natural or man-made chemical present in the environment does not always lead to human or animal exposure. Exposure requires contact by touching, breathing, ingesting or injecting substances which contain the chemical. Exposure itself is not necessarily harmful; several other factors determine whether contact leads to harmful effects and the type and severity of these effects (Anon. 1990). These factors include the dose (how much), the duration (how long), the timing (when in the life cycle), the route of exposure (injection, inhalation, ingestion, contact) and individual characteristics and lifestyles (sex, age, health, habits, fitness, genetic predisposition).

Populations that are unusually susceptible to toxic effects from silver are those with dietary deficiencies of vitamin E or selenium, or those with genetically-based deficiencies in the metabolism of these essential nutrients. Those populations with damaged livers and those exposed to very high selenium levels in their diet are also at higher risk. Some people may exhibit an allergic response to silver (Anon. 1991).

## *1.2 silver speciation for criteria*

Most toxicological studies have been conducted with silver in the free, elemental or 0 oxidation state, and with the +1 monovalent silver ion. The rarer +2 and +3 oxidation states have not been studied adequately (Anon. 1990, Taylor 1964, MRI 1975, Thompson 1973, Boyle 1968, Mulvey 1978, Bertine *et al.* 1971, DeMayo *et al.* 1979, Klein 1978 and Presant *et al.* 1965).

The majority of silver resulting from photo-processing occurs in an insoluble form. Theoretical calculations of organic and inorganic silver complexes indicate that, due to the low solubility of silver sulphide and the high affinity of silver for sulphide, little free silver, usually <10 to 12 µg/L, would occur at equilibrium, in effluents or surface waters that contained any sulphide (IJC 1982). Bioassays have demonstrated that 'free' silver caused the death of fathead minnows at 5 to 16 µg/L, but the salts, silver thiosulphate and silver sulphide, had no effect at 11 000 to 21 000 µg/L (LeBlanc *et al.* 1984).

A method for measuring 'log silver ion activity' (pAg) with a specific ion electrode has been developed to measure 'free' silver in surface waters. Applying this method to Lake Ontario surface waters yielded pAg values in the 8.5 to 11.1 range, mostly 9.2 to 9.4. These values were claimed to be equivalent to 'free' silver ion levels of 0.340 to 0.00079 µg/L, primarily 0.054 to 0.63 µg/L (Lockhart 1980, Bionomics 1980a, Bionomics 1980b and Chudd 1979).

The authors indicate that the information in the prior paragraph supports an aquatic life objective of about 0.1 µg/L 'free' silver in order to limit that fraction of the total silver that is biologically active or toxic. The Aquatic Ecosystem Objectives Committee of IJC agrees that 'free' silver is a better measure of toxicity than total silver but does not recommend the adoption of an objective based on 'free' silver for the following reasons:

-near an effluent, silver may not yet be in equilibrium with all the available complexing agents.

-sulphide, which is the only reactant likely to reduce silver adequately, is readily oxidized to sulphate when oxygen is available (Chen *et al.* 1972). The half-life of sulphide is about 50 hours but oxidation rates may be increased fivefold by metals such as calcium. Sulphide has not been found, even at detection limits of 0.0001 µg/L, in the Great Lakes.

-weaker organic complexing agents for silver still permit 'free' silver to exist at concentrations near the objective level of 0.1 µg/L.

-the method presented by Kodak for measuring silver ion activity (Chudd 1979) is not adequate for low environmental concentrations; there are difficulties with reproducibility, dependability and comparability. The method is reliable at levels of 'free' silver higher than 11 µg/L.

Until a reliable method is developed to measure 'free' silver at concentrations below 0.1 µg/L, silver objectives should be expressed as total silver (IJC 1982). This is still the routine total or dissolved silver detection limit for water samples analyzed in labs used by the British Columbia Ministry of Environment for analysis of ambient water samples.

In this report the statement `water of hardness 230' indicates water with a hardness equivalent to that caused by 230 mg/L of $CaCO_3$.

# 2. Occurence

## *2.1 anthropogenic*

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### 2.1.1 uses

Silver is used as a plating or as an alloy for jewelry, flatware, dental amalgams and caps, electrical contacts, batteries, catalysts, coins, solders, brazing alloys, mirror backings, silvering of glass beads, etching ivory, as a catalyst in hydrogenation and oxidation, as an antimicrobial disinfectant in drinking water, to treat warts and burns, and as a photographic or radiographic emulsion. Silver is also used as a cloud seeding agent to promote rainfall.

Breath mints coated with silver, silver acetate anti-smoking lozenges, silver nitrate drops to prevent blindness in newborns exposed to gonorrhea, skin creams containing silver nitrate or silver sulphadiazine, and silver nitrate solutions for treating gum disease are the common medical or personal hygiene uses of silver (Anon. 1990, Anon. 1991, MRI 1975).

The reduction of silver salts by electromagnetic radiation is the basis of photographic and x-ray image capture. These are major industries that use large quantities of silver. The images are used for diagnostic purposes, information storage, transmission and aquisition, personal momentoes, scientific work and business advertising.

### 2.1.2 production

Canadian production declined for the fourth year in a row in 1992, to 1147 tonnes from 1261 tonnes the previous year (Keating 1992, Giancola 1994) In 1974 and 1975, of the over 1000 tonnes produced, most was refined silver, the remaining being silver ores and concentrates (Boyle 1968). In 1992 Canada was listed as the fifth largest producing nation in the world (Keating 1992, Giancola 1994), compared with being number one in 1974, at the time being responsible for 14% of the silver produced in the world, more than any other individual country. Forty percent of this came from Ontario and 15% from BC. Most of the silver production in the world is a by-product of lead-zinc, copper and gold mines; little is from silver-only mines (Boyle 1968).

### 2.1.3 consumption

Canadian consumption of silver was 341.5 tonnes in 1989, 464.0 tonnes in 1990 and 335.1 tonnes in 1991. Exports for 1991 and 1992 were 1070 tonnes and 1135 tonnes, respectively. In comparison, Canada's use of silver in 1974 and 1975 was just over 300 tonnes. However, Canada exported almost the entire production for those years as silver, or as ores and concentrates, and imported silver from the USA and Britain. The USA was the largest consumer at over 5500 tonnes in 1974, and 30% of this was used for photographic emulsions (George 1976, Keating 1992, Giancola 1994).

### 2.1.4 discharges

Silver is released to the environment as a waste product from mining and smelting activities (Anon. 1990). The 1993 summary report on the National Pollutant Release Inventory reports that in 1993 a total of 4.848 tonnes in 33 releases were made to the environment. This broke down to 0.203 tonnes in water, 3.016 tonnes underground and 0.011 tonnes on land. The facility with the largest reported release was Cominco Ltd. (Trail, British Columbia) with 3 reports of releases totalling 3 tonnes of silver; all released in the water. In New Brunswick one release was reported that was less than 1 tonne and in Quebec 3 reported releases totalled 0.962 tonnes. A total of 6 releases made as transfers to off-site locations totalled 26.376 tonnes. Most of this was in recovery, re-use and recycling (25.764 tonnes), and the remainder was in sewage (0.291 tonnes) and containment (0.324 tonnes). Of the 6 reports made, 5 were in Ontario where 25.764 tonnes was in recovery, re-use and recycling, 0.291 in sewage and 0.268 tonnes in containment with a total release of 26.320 tonnes. The other release was reported in Quebec with 0.056 tonnes reported for containment (Anon. Env. Can. 1995).

About 600 to 700 kg of AgI were released in cloud seeding over western Canada in 1977 (Chisholm) out of a total of about 3 tonnes worldwide (Klein and Mulvey 1978). In the USA about 25% of the annual consumption is estimated to be discarded annually.

This discarded silver consists of about 10% unrecycled photographic silver sent directly to the water, 30% silver deposited in sewage sludge and 60% silver contained in combustible refuse and discarded appliances. An estimated 327 tonnes were released to the atmosphere with 54% from the iron and steel industry, 29% from the cement industry and 12% from burning coal (MRI 1975).

The total consumption of coal in the world in 1974 was about 3 billion tonnes. At an average silver content of 100 µg/kg, the amount of silver present was 300 tonnes. Only about half of the coal produced was burned to produce energy, resulting in only 10% of the silver (15 tonnes) being released to the atmosphere in fly ash. The total crude oil production of the world in 1976, 2.47 billion tonnes, contained only about 0.2 tonnes of silver; this is a relatively negligible source of silver release to the atmosphere (Bertine and Goldberg 1971 and McMullen 1976).

Silver thiosulphate complexes from waste photographic solutions are the main form in which the 500 or so tonnes of silver enter the environment each year in the USA. Probably 75% of this is trapped in sewage sludge (MRI 1975). The estimated total amount of silver released to the environment through anthropogenic activities around the world is 2500 tonnes annually; while smaller than the release of 11 000 tonnes due to natural weathering, this is still a significant amount (Klein and Mulvey 1978).

## *2.2 natural*

### 2.2.1 rocks

Silver is a valuable, naturally occurring, metallic element. It is rarely found as elemental silver but commonly occurs as a nitrate, chloride, sulphide or oxide in rocks and soils, and is often associated with other metals such as copper, zinc, iron and antimony (Taylor *et al.* 1980). In rocks, the mean silver concentration ranges from about 0.04 to 0.12 mg/kg and the mean crustal abundance is about 0.07 mg/kg. Granitic igneous rock in Nevada contains up to 50 mg/kg of silver (Seiler *et al.* 1988). Coal contains between 40 and 2000 µg/kg of silver but the level in crude oil is much lower, about 100 ng/kg.

### 2.2.2 air

Volcanic activity emits silver sulphides, sulphates, chlorides and ammonium salts. The background concentration of silver in air is estimated at <0.1 ng/$m^3$ (Boyle 1968, Mulvey 1978).

### 2.2.3 soils

Normal soils contain silver levels of about 0.3 mg/kg, with a range of 0.1 to 5 mg/L, but near lead, zinc, and silver deposits this may rise to 10 mg/kg. Well-drained New Brunswick podzols contained 0.1 to 7.8 mg/kg of silver. Silver tends to concentrate in the surface layers of soils at a pH greater than 4, particularly in organic soils with high levels of humic colloids. Silver also binds to clay minerals. Adding 0.5 to 5 mg/L silver iodide solutions to soils resulted in surface concentrations of ten times the solution strength and equal concentrations were reached at 5 cm. Soils amended with sewage sludge can have up to 10 times as much silver as normal soils (USEPA 1980a).

### 2.2.4 water

2.2.4.1-rain water

Rainfall in a remote area of Ontario contained 2.8 to 30 ng/L of silver compared to rainfall in Germany that ranged from 70 to 9000 ng/L (Merritt 1976). In the USA, silver in rain, snow and hail ranged from 0 to 1200 ng/L (Mulvey 1978). The normal silver level in rainfall in Alberta is 2.5 to 5 ng/L but it rose to 46 to 62 ng/L after cloud seeding with 240 g of AgCl. These effects could be detected 100 km from the site of the seeding; the peak silver levels occurred an hour after seeding (Warburton 1973, Zacharuk *et al.* 1977).

2.2.4.2-drinking water

Silver levels in 380 samples of finished drinking water in the USA ranged from <1 to 5 µg/L (Nordberg *et al.* 1988). Finished water from the 100 largest USA cities had a median silver level of 2.3 µg/L with a maximum of 7 µg/L (Durfor *et al.* 1964). Other set of 380 samples of finished drinking water had silver levels of 0.3 to 5 µg/L, with a mean of 2.2 µg/L (Kopp *et al.* 1967). Only 6% of the 380 samples contained any silver and data from 1577 samples of well and surface waters from 130 points in the USA indicated silver levels above the detection limit of 0.1 µg/L in only 104 of the samples. The levels ranged from 0.1 to 38 µg/L with a median of 2.6 µg/L (Kopp *et al.* 1967). A later report on drinking water supplies in the US, both ground water and surface water, showed silver at levels up to 80 µg/L with up to one-third of these sources containing over 30 µg/L (Anon. 1990).

2.2.4.3-fresh water

Acidic waters tend to contain more silver than neutral or alkaline waters and silver is often associated with chloride, sulphate, bicarbonate, organic material, manganese and

iron. Uncontaminated lakes and rivers in non-industrialized areas may contain about 0.2 to 2.0 µg/L from natural sources (Anon. 1990, Boyle 1968). Raw water from Lake Ontario contained 1 µg/L (Bard *et al.* 1976). In the Environment Canada computerized data storage system, NAQUADAT, over 90% of the silver records were below the various detection limits which ranged between 4 and 10 µg/L. However, in heavily industrialized regions, or areas where heavy-metal mining and smelting occur, Sudbury and Trail for example, contamination by silver can result in stream concentrations up to 38 µg/L (Stokes *et al.* 1973, MRI 1975).

The seasonal fluctuation of silver in various components of a small lake in Colorado was monitored by Freeman in 1975. In the summer, silver is associated with terrestrial and aquatic communities, which increases the silver concentration in the soil and lowers its concentration in the water. At the end of the growing season, when massive die-off occurs in terrestrial organisms and chemical changes occur in the soil, silver concentrations in surface and ground water rise. In the lake, die-off resulted in higher silver levels in the sediment which remained high over the winter under the ice.

Redox reactions with iron and biological fallout contributed to high sediment silver levels. Plankton levels were low in the winter. In the spring, surface and ground waters contained about the same level of silver as at freeze-up. During spring thaw, as biological activities intensified, the cycle repeated and silver moved from the sediment into the biotic communities.

2.2.4.4-marine water

The natural erosion of rocks and soils releases a large amount of silver into the environment. Weathering processes are estimated to result in 11 000 tonnes of silver per year entering the oceans. Silver is apparently delivered to the oceans primarily (up to 90%) as dissolved species. Estimates of silver in seawater vary from 0.15 to 2.9 µg/L and the concentration tends to increase with depth in highly productive areas (Boyle 1968, MRI 1975, Thompson 1973, Bowen 1966, Weast 1977).

## *2.3 sediments*

Adsorption seems to be the dominant process involved in partitioning silver onto sediments. Lake sediment silver levels correlate with organic content. Lake sediment silver concentrations have been reported as being 100 times the level in the overlying water (Freeman 1977). Magnesium dioxide, ferric compounds and clay minerals all have an affinity for silver but manganese dioxide appears to have the strongest affinity.

Stream sediments in mineralized areas may average around 930 µg/kg while sediments for non-mineralized areas are about 140 µg/kg (Boyle 1968). Analyses of some US stream sediments gave a range of 300 to 1500 µg/kg with the high values being from industrialized mining areas (Turekian *et al.* 1967).

Sediment enrichment by silver is accompanied by enrichment of barium, strontium, lead, arsenic and antimony. Silver enrichment is highest in sediments with high manganese (Boyle 1968), and enrichment increases as particle size decreases (McDuffie *et al.* 1976). This is typical of many metals due to the increased surface area available for adsorption.

Deep sea sediments average 110 µg/kg of silver (Turekian *et al.* 1961). The silver adsorbed to manganese and other suspended river sediments is virtually all released on contact with seawater (Kharker *et al.* 1968, Dyck 1968, Chao *et al.* 1974). Silver is readily available from many types of sediments and shellfish concentrations correlate well with most non-crystalline silver extractions (Luoma and Jenne 1976). Silver is reported at 14 to 20 mg/kg in the bottom sediments of California coastal basins (Friberg *et al.* 1986).

## *2.4 tissues*

On a wet-weight basis, the silver levels in human soft tissues such as brains, kidneys, lymph nodes, lungs, muscles, testes and ovaries, ranged from 1 to 4 µg/kg. Hard tissues, such as rib bones, had 101 mg/kg (Klein 1978). On a dry-weight basis some values for normal tissues were spleen, 2.68 mg/kg, and kidney, 0.36 mg/kg. On an ash-weight basis levels were liver, 0.7 to 1.70 mg/kg, brain, 0.5 to 1.7 mg/kg and skin, 1.3 mg/kg (MRI-1975). Placental levels of 5 µg/kg and umbilical cord blood levels of 5 µg/L were measured; the maternal blood level was measured as 4 µg/L (Creason *et al.* 1976).

Molluscs have been found to contain 0.1 to 10 mg/kg dry-weight in their tissues and crustaceans 2 mg/kg dry-weight. Trout from Lake Cayuga, NY contained 0.48 to 0.68 mg/kg dry-weight (USEPA 1980a). Molluscs from coastal areas of the North Sea contained up to 2 mg/kg (Friberg *et al.* 1986).

Levels of heavy metals in oranges and avocados from Ghana, in gold mining and in control areas, were reported by Golow *et al.* in 1994. Soil levels of silver were not measured. Konongo and Obuasi soils were rich in gold but Kumasi soils did not contain gold. The data shown in Table 2.1 indicate slightly higher silver levels in the fruit from

gold-rich soils than from the gold-poor soil, and slightly higher levels in the peels or rinds than in the flesh except for the avocados from Obuasi which had higher silver levels in the flesh.

# 3. Reactions

### *3.1 chemistry*

Elemental silver, in the 0 oxidation state, is fairly common as is the +1 monovalent argentous silver ion. Most toxicological studies have been conducted on these two forms of silver. The argentic +2 and rarer +3 oxidation states have not been well studied. The +2 state is generated by oxidizing acidified solutions of +1 ions with persulphate, fluorine or ozone. Once formed, the +2 and +3 states are fairly stable in acidic solutions, especially phosphoric acid; reduction to the +1 state occurs slowly. Tables 3.1 to 3.6 give the physical and chemical properties of silver and some of its salts.

In acidic solutions the +1 state is soluble and mobile but silver precipitates as the solution becomes alkaline, and at a pH between 7.5 and 8.0 silver hydrolyzes as the oxide or a basic salt. The precipitation of silver from alkaline solutions is also dependent on the other ions present. If ammonia or some other complexing agent is present, the silver may stay in solution. Silver will precipitate as a halide if $Cl^-$, $Br^-$, or $I^-$ ions are introduced, as $Ag_2S$ if $H_2S$ or $S^{2-}$ is present, and as a thiosulphate, phosphate, chromate or arsenate in the presence of $S_2O_3^-$, $PO_4^{3-}$, $CrO_4^{2-}$ or $AsO_4^{3-}$, respectively (Anon. 1990, Boyle 1968, MRI 1975, Kharkar *et al.* 1968, Thompson 1973).

Studies using radiosilver released from nuclear power stations show that in fresh water at around pH 8.0, at least 50% of the silver precipitated, and within 48 hours 80% was adsorbed onto sediments. In marine and estuary conditions where the chloride ion is prevalent, silver is rapidly desorbed, 70% in 24 hours, and remains in solution (Murray and Murray 1973, Fukai and Murray 1973).

In natural waters, silver is in the +1 state as a sulphate, bicarbonate or nitrate. Silver chloride complexes, such as $Na(AgCl_2)$, form when NaCl or KCl are present. This is the most likely form of silver in seawater. In areas of high biological activity, such as reef complexes in the ocean, $[Ag(S_2O_3)_2]^{3-}$ is probably only stable at low temperatures for a short time near the surface. It is soluble in alkaline and neutral solutions but decomposes to S, $Ag_2S$ and $Ag_2SO_4$ in acidic conditions (Taylor 1980).

Complex sulphide or polysulphide ions or hydrosulphides may form in water with high levels of $H_2S$ or $S^{2-}$. Silver may also be dissolved as double complexes of sulphur, arsenic, antimony, tellurium and selenium. Silver may exist in colloidal form in water as an integral part of, or adsorbed onto, various humic complexes as AgCl, $Ag_2S$, $Ag_2Se$ and $Ag_3AsS_3$, or dissolved as acetates, tartrates and other organic compounds. It may also be adsorbed onto plankton or inside the tissues of micro-organisms (Taylor 1980).

Theoretical equilibrium calculations indicate that $Ag^+$ and AgCl are the dominant forms of silver in aqueous solution; at 1 µg/L, 60% is $Ag^+$. Adding organic complexing agents such as NTA, citrate, glycine and cysteine to the equilibrium calculations has little effect on the outcome. Model calculations for an estuary with a dissolved silver concentration of 0.04 µg/L and an $H_2S$ and $HS^-$ concentration of 0.01 µg/L, have shown that at the river input end of the estuary AgHS was the most prevalent form of silver followed by $Ag^+$ and AgCl. At the marine end of the estuary, $AgCl^-$ was dominant followed by AgHS, $AgCl_3^{2-}$ and $AgCl_4^{3-}$ (Morel *et al.* 1973, Hem 1977, Jenne *et al.* 1978).

In photographic film development the silver bromide grains dispersed in the emulsion are `activated', by a poorly understood process, and thus become more susceptible to reduction by mild reducing agents in the `developer'. These exposed grains are reduced to black metallic silver. To prevent the unexposed grains from reduction when subsequently exposed to light, they are dissolved in `hypo' or `fixer' which contains $S_2O_3^{2-}$ or thiosulphate ions. This results in the formation of a silver thiosulphate complex, $[Ag(S_2O_3)_2]^{3-}$, which is washed out of the emulsion leaving behind only the black metallic silver deposits in areas where the light struck the emulsion (Sienko and Plane 1966).

The majority of silver from photoprocessing occurs in an insoluble form. Theoretical calculations of organic and inorganic silver complexes indicate that due to the low solubility of silver sulphide, and the high affinity of silver for sulphide, little free silver, <10 to 12 µg/L, would occur at equilibrium in effluents or surface waters that contained any sulphide (Lockhart 1980).

There is no evidence that silver is naturally transformed to a hazardous biologically available form (such as mercury into methyl mercury). Ionic silver is more toxic to aquatic organisms than silver compounds. Thiosulphate-complexed silver breaks down to silver sulphide which is less toxic than the silver ion.

### *3.2 wastewater treatment*

Treating effluent by the lime coagulation process at pH 11 removed 97% of the silver leaving a residual of <4.6 µg/L. When this effluent was spiked with silver to 54.6 µg/L, sand filtration removed 11.6% of the silver. Subsequent sequential treatments increased the removal efficiency; activated carbon achieved 97.1%, followed by cation exchange to give 98.8% and anion exchange for a final 99.4% removal. A lime precipitation-activated carbon treatment removed 98% of the silver with a residual of 10 µg/L, ferric chloride-activated carbon removed 99.1% and alum-activated carbon removed 99.2% to achieve a residual of 5 µg/L. A secondary effluent with silver levels up to 10 µg/L had no further silver reduction after additional treatment (Linstedt *et al.* 1971, Cohen 1977, Argo and Culp 1972).

### *3.3 oligodynamics*

Silver is a disinfectant for non-spore forming bacteria at concentrations three or four orders of magnitude below the levels at which it is toxic to mammalian life (about 1 gm/day for humans or 500 mg/L in their drinking water). This extreme mammalian-to-bacterial toxicity differential is the definition of an oligodynamic material. Sterilizing contaminated water occurs at 40 to 200 µg/L and 250 µg/L is strongly germicidal to gram-positive and gram-negative bacteria. The low concentration necessary for oligodynamic activity allows silver or one of its insoluble salts to be used indefinitely in contact with sterile liquids without silver levels building up to concentrations harmful to people (Thompson 1973). Russian spacecraft used silver as a germicide to convert polluted wastewater to potable water (MRI 1975).

### *3.4 metabolism*

The biological effects of silver are apparently due to reversible bonds with enzymes and other active molecules on the surface of cells. Due to its sulphydryl binding propensity, biologically-available silver disrupts membranes, disables proteins and inhibits enzymes. The ionic form of silver is necessary for biological activity and the lipid phase of the membrane appears to be important in adsorbing silver ions to living cells. The active sites on enzymes which are affected by silver are apparently the electron-rich functional groups such as-SH groups. Silver was similar in toxicity to mercury in *in vitro* experiments on the activity of glutamic oxaloacetic transaminase (GOT), and lactic dehydrogenase (LDH), obtained from the blood plasma of the white sucker, *Catostomus commersoni*. The GOT was the most sensitive and was completely inhibited at 50mg/L (Cooper and Jolly 1970, Christienson 1971/72). Interference with

active transport and cyclic adenosine monophosphate are reported, as is binding to bases, riboses and phosphates on DNA (Klein 1978).

Silver combines with plasma proteins, is removed by the liver and over 90% is eliminated in the bile; most of this in the feces with very little in the urine. That which is not excreted is deposited in the skin and mucous tissues (MRI 1975, Cooper and Jolly 1970). In 1986, George *et al.* reported a metallothionein which complexes silver and copper.

Tissue deposition of silver results from precipitation of insoluble salts such as silver chloride and silver phosphate. These may be transformed to soluble silver sulphide albuminates and bind with amino or carboxyl groups in proteins and nucleic acids. They may also be oxidized to metallic silver by ascorbic acid or catecholamines.

Experiments with radiosilver indicate that it is mainly associated with the reticulo-endothelial system. The silver is concentrated in the basement membrane of the skin, in the elastic fibres around sweat glands and the eyes; generally in areas of the skin exposed to the sun. The discoloured skin in argyric patients exposed to ultraviolet radiation is likely caused by photoreduction of silver chloride to metallic silver, which is then oxidized to black silver sulphide and bound by tissues. If the diet is high in selenium, the silver sulphide is converted to silver selenide which may result in higher silver deposition rates than with silver sulphide (Danscher 1981, Berry and Galle 1982, Buckley *et al.* 1965).

## *3.5 bioaccumulation*

### 3.5.1 general

Literature on the bioaccumulation of silver was generally inadequate. Often the ambient concentrations of silver were not reported or measured for field collected organisms. Some studies compare sites as contaminated or uncontaminated, but only assume this to be so. Silver bioaccumulates at low concentrations because most silver compounds are only sparingly soluble in water. Planktonic concentrations are correlated with water levels and benthos concentrations are correlated with sediment levels (Freeman 1977).

### 3.5.2 marine waters

3.5.2.1-general

Table 3.7 gives some data on the bioaccumulation of silver by marine invertebrates. The review of silver included ambient water concentrations and speciation, ambient sediment level and complexation with organic compounds and their availability. Ambient ocean concentrations ranged from 0.04 to 2.5 µg/L silver, principally in the form of $AgCl_2$, while the most bioavailable form is AgCl. Sediment concentrations were usually three to five orders of magnitude greater than concentrations in the the overlying water and ranged from 0.1 to 1.0 µg/g silver.

Silver is tightly bound by sewage sludge and elevated levels of silver are often associated with sewage outfalls with minimal treatment. In the absence of sewage, silver associates with iron oxides and humic substances. The relative bioavailability of either silver-inorganic complexes or silver-organic complexes appears to depend on the individual compounds. Silver-inorganic complexes are probably the most common in the marine environment. Silver-chlorides are generally not bioavailable except for AgCl. Silver-iron oxides or silver-magnesium complexes in sediments increase the availability to bottom feeding organisms.

Silver uptake by the worm *Nereis diversicolor* , decreased in sediments with high humic content, but increased in sediments with high iron or magnesium concentrations. In an experiment with the clam, *Macoma balthica*, Harvey *et al.* 1985) showed that silver uptake increased if silver-iron oxide particles were coated with an extra-cellular polymer produced by bacteria commonly associated with sewage outfall areas. This is important since most high silver tissue levels observed in the field are associated with sewage outfall areas, which are areas of high bacterial activity, and would suggest that some organic complexes increase rather than decrease bioavailability. Evidence from the literature (Nelson 1978, Greig *et al.* 1975, 1977 and 1983, Gilfillan *et al.* 1985 and Robinson *et al.* 1985) is not conclusively in support of this theory.

Accumulation appears to occur mainly as granular deposits in the kidneys or at the basal membranes of tissues and organs and is likely not toxic to the organisms themselves nor subject to either trophic transfer or biomagnification. Although fish, invertebrates and algae can all accumulate silver, there does not appear to be magnification up the trophic food chain to top carnivores.

3.5.2.2-estuaries

In 1992 Bryan and Langston conducted a review on bioavailability of heavy metals in sediments with special reference to the estuaries. They discussed some of the processes which affect the concentrations and bioavailability of metals, such as mobilization of metals to interstitial water and chemical speciation, transformation, control exerted by major sediment components, competition between metals for uptake

sites and influence of bioturbation, salinity, redox or pH on the above processes. Synergistic effects of metals were also discussed, and it was found that attributing deleterious effects of a single metal to an organism in the field is rare.

Tolerance and the induction of metal detoxification involving the formation of granules or metal binding proteins may lead to tissue concentrations which are unusually high but without deleterious effects unequivocally attributable to the metal. It was also noted that the only consistent evidence of biomagnification involves methyl mercury.

Young *et al.* (1981) measured silver in several marine species, commonly used as seafood, near a large municipal sewage outfall in California. It was found that fish species did not accumulate silver significantly with respect to controls. In contrast, benthic invertebrates accumulated three to five times as much silver.
3.5.2.3-molluscs

Cain and Luoma (1985), conducted an *in situ* study comparing silver accumulation in two populations of clams, *Macoma balthica*, one resident in a contaminated area, and the other transplanted from a relatively pristine area. Results indicated that accumulation of silver in the hard and soft tissues of the transplanted clams was half that of the resident population; however, the transplanted clams retained 90% of the silver accumulated, whereas in the resident clams the loss of silver from the soft tissue was equivalent to the gain. Shell closure was observed to occur earlier in the transplanted clams. Also noted were seasonal fluctuations in the levels of ambient silver, and an increased bioavailability of silver in the winter months compared to the summer months. Tissue weights changed seasonally for both populations, which was shown to bias measured tissue metal concentrations if not accounted for. This might have lead to spurious conclusions in some of the literature regarding tissue metal concentrations.

The effects of long term silver exposure on growth, bioaccumulation and histopathology of laboratory and field collected blue mussels, *Mytilus edulis*, were examined. After 12 months, significant accumulation of silver occurred only with laboratory raised mussels exposed to 10 µg/L silver; however, at 18 and 21 months, mussels exposed to 1, 5, and 10 µg/L silver all showed significant accumulation. Significant levels of copper were also accumulated from the ambient levels in the test seawater. In field-collected juvenile and adult mussels, silver accumulation was significant over the first 12 month period but the rate of accumulation was lower for the adults. Growth was inhibited at 6 months, but at 12 months the rate of growth was equal to the controls. Seasonal differences in the rate of accumulation were also noted (Calabrese *et al.* 1984).
Species related variations of silver bioaccumulation were investigated in a static renewal assay with the Pacific oyster, *Crassostrea gigas*, the mussel, *Mytilus*

*galloprovincialis*, and the scallop, *Chlamys varia*. Exposures to silver occurred through phytoplankton containing 20 µg/g silver, water containing 20 µg/L silver, or food+water containing a total of 20 µg/L silver. Exposure via food only resulted in a rate of uptake of <10 mg/kg, while exposure via the water only or via the water+food resulted in a rate of uptake >100 mg/kg. Uptake via exposure through the food was still considered significant. Heavy accumulation of AgS in the glandular cells, which secrete the byssal threads in the mussel, resulted in a significant percent detachment from the container walls. The scallop and the oyster were observed to retain more silver than the mussel on a quantity of metal available per unit weight of filter feeding organism (Metayer *et al.* 1990).

In a 28 day study, juvenile Pacific oysters, *Crassostrea gigas*, were exposed to silver either through 20 µg/L dissolved in solution, or by 59.7 µg/g in the phytoplankton (Majorta *et al.* 1988). A plateau, where the body burden of silver no longer increased, was reached within 14 days of the study. The major storage of silver was as the sulphide in amoebocytes and basal membranes of tissues and organs. Elimination of the amoebocytes probably resulted in the accumulation plateau. The fact that the major storage of silver occurs as silver sulphide is important as this compound is very stable and unlikely to be a vector for trophic transfer or biomagnification of silver.

Nelson *et al.* in 1983 reported silver uptake in two succeeding generations of the marine snail, *Crepidula fornicata*. In the parent generation a very significant uptake of silver was observed for the first six months at all treatment levels. After 12 months only the lowest exposure level, 1µg/L silver, continued to show any significant increase and after 24 months body burdens at all treatment levels were significantly reduced. Males accumulated silver faster than the females for the first 12 months; in the same period females showed a significant reduction in metal body burden while the males did not. Copper accumulation increased as silver accumulation decreased, which may indicate some competition for binding sites. The F1 progeny also accumulated significant amounts of silver in much the same pattern. Silver was mainly deposited on the basal membranes of cells in tissues and organs, bound primarily to sulfhydryl groups, and therefore probably not toxic.

3.5.2.4-worms

The rates of silver accumulation in two populations of the polychaete, *Neanthes virens*, one from a local polluted area, and one from a relatively pristine area were compared (Pereira and Kanungo 1981). Transplanted worms accumulated more than twice as much silver in 24 hours of exposure to 1.0 µg/L silver. Increasing levels of the ambient silver and time of exposure increased the levels of silver accumulated. A significant decrease in oxygen consumption was observed in transplanted worms with a metal

body burden >113 mg/kg, and a significant decrease in water content was observed in worms with a body burden >88 mg/kg. Worms resident to the contaminated area did not show any adverse effects.

These results are in contrast to those of Bryan and Hummerstone in 1977, which indicated only low levels of silver accumulation in *Nereis diversicolor*, perhaps indicating that silver accumulation is mainly a function of water, rather than sediment, concentrations; this result may, however, only indicate species variability. Though this study provides evidence of a tolerance of organisms indigenous to a polluted area, it is not clear how this relates to the lowered rate of accumulation of these same animals.
3.5.2.5-crustaceans

The environmental parameters affecting trace metal uptake and toxicity in estuaries were examined in some detail. The complexation of silver in freshwater was discussed as freshwater input to the estuarine environment is often important. In the estuarine environment, silver is likely to be bound to inorganic material due to a high affinity for the chloride ion. Increasing salinity will further decrease silver-organic complexing because calcium and magnesium will compete for chelation sites. In a study with the grass shrimp, *Palaemonetes pugio*, silver uptake was most closely related to the concentration of silver chloride (r2=0.90) than any other expected silver complex. It was suggested that this was partly due to a potentially greater permeability of silver chloride across cell membranes due to its neutral charge, which generally allows for permeability several orders of magnitude greater than for charged species. Despite this, the rate of uptake of all silver and chloride complexes decreased with the increasing salinity.

In 1985, Cain and Luoma suggested that, because the initial burden of silver in transplanted animals is low, the influx of silver is high, until whatever regulating systems that are utilized in detoxifying metals become accustomed to the change in environmental parameters and are able to operate efficiently. The body burden of silver was measured in the spot prawn, *Pandalus platyceros*, from two locations in BC waters (Whyte and Boutillier 1991). Levels of silver were measured in the abdomen and the carapace of immature males, mature males and mature females in both areas. In general there was no correlation between size, age or sex of the prawn and silver levels. The greatest accumulation was in the hepatopancreas, 8.27 µg/g silver, followed by the carapace tissue, 1.16 µg/g silver, and the abdomen, 0.80 µg/g silver. Body burdens in the abdomen tissue were the same at both locations, suggesting that the diet was the same.

Carapace levels were significantly different suggesting that silver was incorporated into the carapace due to ambient levels in the surrounding water, rather than deposited through metabolic activity.

3.5.2.6-phytoplankton

In 1989, Sanders and Abbe reported on a study and review on silver transport and impact in estuarine systems, with particular reference to phytoplankton. Uptake of silver was rapid, but was inversely proportional to salinity. Bioavailability, especially at high salinities, appeared to be controlled by the free monovalent silver ion and possibly the silver chloride complex. Accumulation rates were similar in all phytoplankton species monitored.

### 3.5.3 fresh waters

3.5.3.1-fish

In a six month static bioassay, accumulation of silver in the largemouth bass, *Micropterus salmoides*, and the bluegill sunfish, *Lepomis macrochirus*, was measured (Coleman and Cearley 1974). Silver accumulation was significant for the first two months only. By then an equillibrium had developed between the silver levels in the bass tissues and in the water. At 7 µg silver per litre, accumulation in the bass was highest in the gills, 200 times, followed by the internal organs, 12 times, then the whole rest of the body, 9 times. The eventual concentration of silver was greater in the internal organs, 600 µg/kg, than in the gills, 370 µg/kg or the rest of the body, 17 µg/kg. Zinc uptake was observed to decrease as silver uptake increased. The data in this paper should be used with caution since the water contained inordinately high levels of chloride ion which would result in much of the silver being complexed to unavailable forms (Davies and Goettl 1978).

table 3.8

bioconcentration of silver in freshwater fish.

| bcf | exposure duration | silver nitrate in the water | fish species | tissues |
|---|---|---|---|---|
| 11 | 120 days | 1 mg/L | *Micropterus salmoides*<br>-largemouth bass | fillet |
| 19 | 120 days | 10 mg/L | *Micropterus* | fillet |

| | | | *salmoides*<br>-largemouth bass | |
|---|---|---|---|---|
| 15 | 180 days | 10 mg/L | *Lepomis macrochirus*<br>-bluegill sunfish | whole |
| 150 | 180 days | 100 mg/L | *Lepomis macrochirus*<br>-bluegill sunfish | whole |

Coleman and Cearley 1974.

The fathead minnow, *Pimephales promelas*, concentrated silver from the water but less so than Daphnia. It was thought that the fish did not accumulate the silver from the food since the fish had less silver per unit weight; no biomagnification occurred (Terhaar *et al.* 1977). Bard *et al.* in 1976 also discussed this lack of biomagnification in fish. Fish in the St Lawrence River had silver levels ranging from 10 to 30 µg/kg fresh weight while the river levels ranged from 1 to 6 µg/L (Tong *et al.* 1972). Silver levels in Lake Michigan fish muscle, on a wet-weight basis, ranged from 28 µg/kg in yellow perch to 44 µg/kg in brown trout. Whole fish analyses gave 34 µg/kg in alewife and 39 µg/kg in rainbow smelt (Copeland *et al.* 1973).
3.5.3.2-cladocerans and Algae

A study with *Daphnia magna* and *Euglena*, or mixed algal species, with respect to accumulation and biomagnification was reported by Cowgill and Burns in 1975. Algae concentrated silver from water by a factor of 7.5, but *D. magna*, fed the algae in their diet, did not accumulate silver significantly. In another study by Terhaar *et al.* in 1977, the alga, *Scenedesmus*, and the cladoceran, *Daphnia magn*a, absorbed silver from the water. The concentration factor was 26 for the *Scenedesmus* and 96 for the *Daphnia*, in water with 500 µg silver per litre.

3.5.3.3-insects

In 1976 Nehring reported on the accumulation of silver by two aquatic insects, the mayfly, *Ephemeralla grandi*, and the stonefly, *Pteronarcys californica*. Both accumulated silver by a factor of 100 or greater. The level of silver accumulated appeared to be correlated with the level of exposure, which suggests these species are good indicators of silver contamination.

In 1975 and 1977 Freeman studied silver levels in the sediments and in chironomid and caddis fly larvae in a simple ecosystem where sampling may have affected the

distribution of biomass. Sediment and tissue levels were well correlated in 1977 but less so in 1975. In 1975 the silver levels in the sediment, chironomids and caddis flies were 510, 2600 and 400 µg/kg, respectively. The respective levels for 1977 were 290, 560 and 120 µg/kg. The lake water contained 1 µg/L; sediment and both species of insect larvae concentrated silver relative to the water but only the chironomids concentrated silver relative to the sediment. This is to be expected given the differential feeding behaviour and habitat preferences of caddis fly and chironomid larvae. The top predator, cutthroat trout, had silver levels of 3690, 1810 and 290 µg/kg in their bone, liver and muscle, respectively.3.5.3.4-plants

In water with a silver level of 0.4 µg/L, *Lemna minor* (duckweed) accumulated 33 µg/kg dry-weight (Hutchinson and Czyrska 1975). *Nymphaea odorata* (waterlily) contained 60 to 2300 µg/kg silver, with a mean of 500. *Rhopalosiphum nymphaeae* (aphids) feeding on the lilies contained 420 µg/kg. The plants accumulated only twice the level of the substrate. The silver level in the lake water was 12 ng/L. Presumably the silver could not diffuse from the sediment which contained iron sulphide (Cowgill 1973).

In 1985, Jones *et al*. reported on the levels of silver in three aquatic bryophytes, *Scapania undulata, Hygrohypnum luridum* and *Polytrichum commune*, from streams in the lead mining district of Wales. All had higher levels of silver in their tissues than the surrounding water; site specific ambient levels were not reported. *S. undulata* was considered the best pollution monitor since there were strong correlations between tissue levels of silver and those of lead, zinc, copper and cadmium.

3.5.3.5-microorganisms

Activated sludge organisms bioaccumulate silver at 100 times the concentration in the effluent (Chin 1973).

## ***3.6 tolerance***

There is ample evidence in the literature for tolerance to elevated levels of silver by marine invertebrates. Resident clams, *Macoma balthica*, accumulated only half as much silver as clams transplanted from a pristine area. Transplanted clams retained 90% of the accumulated silver in their tissues while the resident clams lost as much as they gained, and shell closure in the transplanted clams was observed to occur earlier relative to the resident clams (Cain and Luoma 1985).

*Mytilus edulis* collected from the field and grown in the laboratory in 5, 25 and 50µg/L of silver, grew slower after being exposed for six months, but by 12 months their growth rates equalled that of the controls (Calabrese *et al.* 1984). A transplanted polychaete, *Neanthes virens*, from a pristine area accumulated twice as much silver as a population from a contaminated area; transplanted worms also exhibited reduced oxygen consumption, changes in the ionic balance of the coelomic fluid and loss of water proportional to increasing body burdens of silver while the local population did not (Pereira and Kanungo 1981).

In 1992 Bryan and Langston noted that there was no clear evidence that metal body burden is proportional to toxicity of the metal. They cited evidence for tolerance to elevated levels of metals as given by Koechlin and Grasset in 1988 who showed that exposure of the polychaete, *Sabella pavonina*, to 50 µg/L silver eventually resulted in the deposition of silver into granules where it was associated with sulfhydryls. The granules were then excreted and thus not of toxicological concern.

In 1987 Flemming studied the effects of silver on bacteria growing on ion exchangers. The addition of silver suppressed bacterial growth until a tolerant population developed. Bacteria growing near the area where the silver was applied received sublethal concentrations promoting the development of tolerant populations. Tolerance was shown to fluctuate from 5 to 50 µg/L silver. Many microorganisms can acquire resistance to silver and it appears that the evolution of heavy-metal resistant enzymes is primarily restricted to microorganisms (Klein 1978).

Only two studies were found which indicated that fish could develop a tolerance to silver. In 1974, Coleman and Cearley noted, in a study with largemouth bass, *Micropterus salmoides*, and bluegill sunfish, *Lepomis macrochirus*, that silver accumulation was only significant for the first two months of the study, after which a plateau was reached, and no significant mortality was observed except at the highest level of silver exposure in the largemouth bass.

In 1984 Birge *et al.* studied the effects of time of acclimatization and deacclimatization with the fathead minnow, *Pimephales promelas.* Acclimatization of the minnow to 1.5 µg/L or 15 µg/L silver for 7 to 14 days significantly increased the $LC_{50}$ values; however, deacclimatization for >7 days lowered the $LC_{50}$ value to a level similar to the unacclimatized minnows.

Lab strains of the algae*Scenedesmus* and *Chlorella* were inhibited by 100 µg silver/L and 30 µg silver/L, respectively. The *Scenedesmus* grew at 50 µg silver/L. Strains from lakes in the Sudbury area were much more tolerant. Lake *Chlorella* grew at 50 µg silver/L and were not completely inhibited until 100 µg silver/L. Lake *Scenedesmus* was

not completely inhibited until 200 µg silver/L. The lab strains exhibited a complete cutoff of growth but growth in lake strains gradually slowed as silver levels rose.

These lake strains were also more tolerant to other metals (Stokes *et al.* 1973). *Chlorella* from lakes containing high levels of nickel were nickel tolerant but were also silver tolerant. The lake silver levels were between 1 and 37 µg silver/L (Hutchinson and Stokes 1975).

### *3.7 synergism*

Voyer *et al.* noted, in 1982, that silver reduced cadmium toxicity in an acute test with embryos of the winter flounder, *Pseudopleuronectes americanus*, exposed to mixtures of cadmium and silver. In contrast, the clam, *Mercenaria mercenaria*, exposed to cadmium in the diet showed reduced uptake of silver into the kidney and digestive gland (Robinson *et al.* 1985). Similarly, Chou *et al.* in 1987 observed that addition of cadmium to the diet of the American lobster, *Homarus americanus*, decreased uptake of silver in the digestive gland, disrupting a significant relationship between the levels of copper and silver that were otherwise present.

Several studies have also indicated a relationship between copper and silver levels. In 1981, Coglianese and Martin showed that the toxicity of silver was increased by low levels of copper. In 1981 Chou *et al.* noted that an increase of copper in the diet of *Homarus americanus* resulted in an increased uptake of silver. In contrast, Nelson *et al.* in 1983 observed that copper and silver levels in the marine snail, *Crepidula fornica*, increased together until a critical point, at which time the levels of silver dropped off while the levels of copper in the tissues continued to increase. Similarly in 1976, Luoma and Jenne noted that high ambient copper levels decreased silver uptake in the polychaete, *S. plana*. Zinc uptake was observed to decrease as silver uptake increased (Davies and Goettl 1978).

### *3.8 carcinogenicity*

Tumors may form at injection sites as a result of solid state effects but no chronic, oral, dermal or inhalation study has shown any increase in tumors, or carcinogenicity to people (Clayton and Clayton 1981-1982).

### *3.9 mutagenicity*

Ames assays by McCoy and Rosenkranz in 1978 with silver sulfadiazine, on several strains of *Salmonella typhimurium* did not demonstrate any gene mutations. *Bacillus subtilis* subjected to silver chloride by Nishioka in 1975 showed no indications of mutagenicity. Beta-galactosidase and alkaline phosphatase activities in *Escherichia coli* did not change after treatment with silver nitrate (Olivier and Marzin 1987). These are all indications of no mutagenicity by silver.

### *3.10 genotoxicity*

Silver will bind with DNA and can cause DNA strands to break and affect replication (Goff and Powers 1975, Loeb *et al.* 1977, Luk *et al.* 1975, Mauss *et al.* 1980, Robison *et al.* 1982, Scicchitano and Pegg 1987, Anon. USEPA).

### 3.11 *immunotoxicity*

Inhaled silver may elicit a mild contact dematitis or allergic response; there is no other known immunological effect of silver (USEPA. 1990).

### *3.12 neurotoxicity*

There are no known neurological effects attributable to silver.

# 4. Animal Life

### *4.1 general*

Silver has low toxicity to vertebrate animals, is eliminated rapidly when ingested orally, and is not a cumulative toxin (Cooper and Jolly 1970). Since surface waters in Canada generally contain low levels of silver and there are few data on chronic silver toxicity to animals, no criteria seem to be justified at this time for wildlife, livestock or laboratory

animal drinking water. Wildlife and free range livestock can safely drink water which meets the aquatic life criteria. Confined livestock and laboratory animals should drink water which meets human drinking water criteria.

The role of silver in animal metabolism is still unknown (Boyle 1968). Generally, the health effects on animals of short-term or long-term exposure to specific concentrations of silver in the air or the food are not known. Little information is available in the literature on the effects of chronic exposure of silver in farm animals. No studies were located documenting cancer in animals after exposure to silver or silver compounds (Anon. 1990).

## *4.2 exposure routes*

Silver may enter the body through the digestive tract (ingestion) when eating or drinking substances which contain silver. It may also be absorbed through the skin, epidermis, (dermal) if part of the body is immersed in silver-containing fluids or is in contact with silver-containing powders. Silver and its compounds may also be breathed in (inhalation) and deposited in, or absorbed via, the lungs (Anon. 1990). Intravenous or intraperitoneal injections also introduce silver into the body and silver may be released from silver amalgam dental fillings in unlined cavities (USEPA 1980a).

### 4.2.1 absorption

The absorption of silver may occur from the lungs after inhalation, from the gastrointestinal tract after ingestion or directly through the epidermis after contact with liquids, solids or gases containing silver or its compounds. Studies evaluating the therapeutic uses of silver in people and the effects of occupational exposure, indicate that silver is readily absorbed after ingestion or inhalation when in the colloidal form (Hill and Pilsbury 1939, Newton and Holmes 1966, Dequidt *et al.* 1974). Absorption rates are a very low percentage of the applied dose for most silver compounds or for elemental silver.

Absorption is a function of transit time in the gastrointestinal tract; there is a lower absorption rate with faster transit times. Transit times range from about 8 hours in rats and mice to 24 hours in dogs and primates (Furchner *et al.* 1968). One could therefore expect more uptake from herbivores with their long intestinal tract and less from carnivores with their short intestines. People retain about 21% of an oral dose of silver after 1 week (East *et al.* 1980, MacIntyre *et al.* 1978).

Whatever the method of silver introduction, it eventually enters the bloodstream and most is removed by the liver and sent to the bile for excretion. After a reasonable period of time has elapsed to allow mobilization from the site of intake, the remaining silver has the same distribution pattern in other organs regardless of the route of uptake. This distribution is not dependent upon the original exposure route.

**4.2.2 oral uptake**

4.2.2.1-laboratory animals

Silver is widely distributed in rats following silver nitrate intake in drinking water at 88.9 mg/kg/day silver (Olcott 1948). Silver is found in many tissues: liver, spleen, bone marrow, lymph nodes, skin, kidney, tongue, teeth, salivary glands, thyroid, parathyroid, heart, pancreas, gastrointestinal tract, adrenals and brain. Within these tissues the silver particles are found in the basement membrane of the glomeruli, the walls of blood vessels between the kidney tubules, the portal vein and other parts of the liver, the choroid plexus of the brain and the choroid layer of the eye (Olcott 1948, Moffat and Creasey 1972, Walker 1971).

Death was observed in rats after very high oral doses of colloidal and inorganic silvers. In the drinking water of rats 2.6 g/L is fatal. In mice 95 mg/L of silver in the drinking water for 125 days caused sluggish behaviour, and in rats 1.6 g/L of silver in the drinking water for 37 weeks caused decreased weight gain (Anon. 1990). Experimental rats receiving 222 mg/kg/day of $AgNO_3$ for long periods, began to die after 23 weeks. Weight loss also began to show up after 23 weeks and surviving animals averaged 50% less weight than controls drinking distilled water (Matuk *et al.* 1981). Rats receiving $AgNO_3$ daily for 2 weeks in their drinking water did not die at 181 mg/kg/day but 3 of 12 died at 362 mg/kg/day (Walker 1971). Some rats given 1.7 g/kg/day of colloidal silver for 4 days died (Dequidt *et al.* 1974).

Liver necrosis in rats is caused by 130 mg/L of silver in the drinking water or food. This toxicity is prevented by 50 µg/L of selenium. Silver causes the same type of hepatic degeneration as selenium deficiency (Green and Bunyan 1969). Ingestion of $AgNO_3$ and $AgCl_2$ will also cause deposition of silver granules in the skin and eyes of animals and discolouration of the skin (Matuk *et al.* 1981, Olcott 1947, Rungby 1986). No loss of fertility was seen in male rats exposed to 89 mg silver/kg/day for two years, as $AgNO_3$ or $AgCl_2$ in their drinking water. No silver deposits were seen in the testes and the spermatozoa were normal in appearance (Olcott 1948).

Granular deposits were seen in the brains of 20 female mice exposed to $AgNO_3$ in their drinking water for four months. The mice were less active than controls and the

deposits were primarily in the areas of the brain involved in motor control, red nucleus, deep cerebellar nuclei and motor nuclei of the brain stem. There were lesser amounts in the basal ganglia, anterior olfactory nucleus and general cortex. The test lasted for 125 days and 18 mg/kg/day was administered daily (Rungby and Danscher 1984).

Long-term experiments with rats and rabbits indicated that 2.5 µg silver/kg body weight had no detrimental effects. Conditioned reflexes in the rats and immunological activity in the rabbits were affected at 25 µg/kg. At 25 and 250 µg/kg there were histopathological changes in rabbit vascular, nervous and glial tissues of the encephalon and medulla (Barkov and El'Piner 1968). Rats given drinking water containing silver at 1 g/L had shorter lifespans and increased hypertrophy of the left ventrical. Silver salts in the diet of animals apparently cause vascular hypertension. Experimental rats given 20 mg/L silver in their drinking water had short-term increases in the RNA and DNA of their brains but, after a year, levels dropped and brain dystrophy occurred (MRI 1975).

Adding 130 to 1000 mg/kg to the food or 1500 mg/L to the drinking water of weanling rats on a vitamin E deficient diet caused liver lesions and fatal liver necrosis after 2 weeks. Selenium at 50 to 100 µg/kg prevented necrosis at the 130 mg/kg diet level but not at higher doses (Grasso *et al.* 1969).

Wistar rats given colloidal silver at 1.7 g/kg for 4 days, or 420 mg/kg for 12 days, absorbed about 2%, and 5% of the dose, respectively (Dequidt *et al.* 1974). Rats given carrier-free radioactive silver <1 µg by stomach tube, eliminated 99% in the feces and 0.18% in the urine in 4 days (Scott and Hamilton 1950). Radioactively-labelled silver, as silver nitrate, was fed to, and injected intravenously into, rats, beagles and monkeys. Body burden and persistence increased with species size and was greater in the intravenous than the oral route. However, cumulative excretion was between 90 and 99% by the second day. Absorption was low from the gut and proportional to transit time (Furchner *et al.* 1966, 1968).

4.2.2.2-livestock

Protargol, a silver protein with 8% silver, inhibited the *in vitro* biosynthesis of prostaglandin in the seminal vescicles of bulls (Deby *et al.* 1973). Silver acts as an antagonist to copper metabolism in cattle and chickens (MRI 1975). In the absence of copper, the growth of chicks was retarded by 100 mg silver/kg in the diet. Chick mortality on a copper deficient diet was greater when silver was over 50 mg/kg (Hill *et al.* 1964). Silver is present in animals at the range of 1 to 10 mg/kg in the ash or 1 to 25 µg/kg wet-weight in beef meat (Klein 1978). Analyses of milk samples from 32 cows gave silver concentrations from 37 to 59 µg/L (mean of 47 µg/L). The mean silver

content of milk from different cities in the United States varied from 27 to 54 µg/L (Murthy and Rhea 1968).

4.2.2.3-man

Estimates of silver in human diets vary widely. There are estimates of 0.4 µg/day for three Italian populations and 10 to 44 µg/day (with a mean of 27 µg/day) in the UK (USEPA 1980a).

table 4.1

summary table: silver intake
by the average canadian city dweller.

| exposure | intake rates by age groups (ng/kg body weight/day) | | | | | |
|---|---|---|---|---|---|---|
| medium | up to 6 months 7 kg | 7 months to 4 years 13 kg | 5 yearsto 11 years 27 kg | 12 years to 19 years 57 kg | over 20 years 70 kg | totals in a 70 year life span |
| Air | 0.86 | 1.2 | 1.3 | 1.1 | 1.0 | 1.0 |
| Drinking water | 28.6 | 15.4 | 11.1 | 8.8 | 5.7 | 7.3 |
| Soil | 1.6 | 1.2 | 0.4 | 0.1 | 0.09 | 0.2 |
| Mushrooms | 22.9 | 160.8 | 141.1 | 113.7 | 95.1 | 104.6 |
| Fin Fish | 17.9 | 59.0 | 71.6 | 44.8 | 42.6 | 46.4 |
| Shellfish | 0 | 17.5 | 19.2 | 14.2 | 22.3 | 20.7 |
| all other foods | 2966 | 2284 | 1168 | 562 | 344 | 567 |
| Total | 3038 | 2540 | 1413 | 745 | 511 | 747 |

Data in the table are for **intake** only, absorption and retention may only be 10% to 20% of this figure. Data do not include dental amalgams or cigarette smoke. No breast feeding is assumed since no data are available on silver levels in human breast milk. From Mitchell, M. 1993.

There are many case studies and experiments which indicate that in humans a number of silver compounds, including salts and silver-protein colloids, are absorbed across mucous membranes in the mouth and nasal passages after ingestion. Silver acetate was readily absorbed after an oral dose of 80 µg/kg/day. Some of the silver was radio-active $Ag^{110}$. About 20% of the dose was retained in the body after 1 week (East *et al.* 1980, MacIntyre *et al.* 1978). A woman who repeatedly applied a $AgNO_3$ solution to her gums developed generalized argyria indicating absorption and translocation of the silver (Marshall and Schneider 1977). A man who used a silver-containing mouthwash for ten years absorbed about 88 gm of silver and had respiratory and kidney problems, proteinuria and elevated macroglobulins. A silver containing compound was used to fill the renal pelvis for an x-ray and resulted in severe gastrointestinal hemorrhaging and death (Zech *et al.* 1973 and Hill and Pilsbury 1939 *in*:USEPA 1980a).

### 4.2.3 inhalation

4.2.3.1-laboratory animals

Acute inhalation of an aerosol containing colloidal silver for two to eight hours led to ultrastructural damage and disruption of the cells of the tracheal epithelium in rabbits (Konradova 1968). A study in dogs gave an estimate of 1 µg/$cm^2$/day absorption rate for metallic silver from the lungs. Six hours after exposure, the silver was found mostly in the lungs (96.9%), but also in the liver (2.4%), and the blood (0.35%). The remaining silver was in the gall bladder and bile (0.14%), intestines (0.10%), kidneys (0.06%), and stomach (0.02%). After 225 days 0.49% of the deposited silver was in the liver and another 0.1% in other organs; the rest of the silver had been excreted. At both times, excluding the deposits in the lungs where it was introduced, 77% of the body burden was in the liver (Phalen and Morrow 1973).

4.2.3.2-man

Silver is taken up primarily by inhalation but also through cuts and injuries. Uptake is primarily by people engaged in occupations where silver is filed, drilled, hammered, turned, engraved, polished, forged, soldered, plated, smelted, mined, purified, cast, alloyed or brazed. Exposure also occurs during the manufacture of silver mirrors, silver nitrate, photographic emulsions, inks, dyes, porcelain, germicidals, antiseptics, caustics, analytical reagents and paints. Film processing, photographic, x-ray and astronomic emulsions, also provide an avenue of exposure.

Radioactive silver metal dust was inhaled during a nuclear reactor accident. Between two and six days after exposure 25% of the detectable silver was found in the liver; some silver was also found in the feces indicating absorption from the lungs (Newton

and Holmes 1966). In a $AgNO_3$ and AgO manufacturing plant, with airborne levels of silver ranging from 0.039 to 0.378 mg/m$^3$, 12 of the 30 workers had blood silver levels over 6 µg/L (Rosenman *et al.* 1979). Workers in a photographic materials manufacturing plant were exposed to airborne insoluble silver from 0.001 to 0.1 mg/m$^3$. Silver was found in 80% of the blood samples at a mean level of 11 µg/L and in all of the fecal samples at a mean of 15 µg/g. Two urine samples had a mean silver level of 9 ng/L. Control fecal silver levels were at 1.5 µg/g.

**4.2.4 dermal application**

4.2.4.1-laboratory animals

In guinea pigs, a solution of 41 g/L applied to the skin for 28 days caused reduced weight gain. Although no deaths were reported, guinea pigs stopped gaining weight when 2.0 mL of a 0.239 molar solution of silver nitrate in water was applied to 3.1 cm$^2$ of skin for 8 weeks (Wahlburg 1965).

4.2.4.2-man

Although the amount of absorption is low in intact skin, about 1% of the applied dose (Snyder *et al.* 1975), several silver compounds are absorbed by people. Silver thiosulphate penetrated the skin of a photochemical worker through sweat glands and caused localized argyria within 6 months (Buckley 1963). Silver nitrate (0.5%), and silver sulphadiazine cream are used as topical antibiotics for burned skin and silver is absorbed and distributed throughout the body through such damaged skin (Sano *et al.* 1982, Bader 1966).

**4.2.5 intravenous injection**

4.2.5.1-laboratory animals

Acute effects of silver in mammals is usually only associated with intravenous injection; silver has been used to produce pulmonary edema in dogs (Taylor *et al*. 1980).

4.2.5.2-man

When 10 cc of a 2% solution of silver (20 g/L or 200 mg/10 cc) were administered intravenously the patient died of pulmonary edema in 5 minutes (Hill and Pilsbury 1939).

**4.2.6 intraperitoneal injection**

4.2.6.1-laboratory animals

In pregnant rats intraperitoneal injection of silver lactate resulted in silver crossing the placenta and being found in fetal liver and brain tissues (Rungby *et al.* 1983).

## *4.3 translocation*

Translocation within the body is primarily in the blood plasma as silver chloride or silver albuminate (MRI 1975). About 10% of ingested silver is absorbed but only 4% retained in the tissues (Klein 1978). The mean silver intake rate from the diet is estimated at 88 µg/day (Kehoe *et al.* 1940). As indicated from the absorption studies mentioned above, silver is found in organs and areas of the body remote from the site of absorption, so translocation must occur. If the silver is introduced to the lungs by inhalation, some silver is cleared by the cilia and swallowed but most is dissolved and removed in the blood (Phalen and Morrow 1973).

## *4.4 deposition*

Argyria, silver deposition, occurs in all organs. Common deposition sites for people who have no history of therapeutic use are the liver, skin, pancreas, adrenals, glomeruli of the kidney, brain, bone marrow, walls of the blood vessels, thyroid, mesenteric glands, choroid plexus, spleen and testes. Generalized argyria is indicated by slate-gray skin and hair colouring, silver finger nails, a blue halo around the cornea and in the conjunctiva of the eye, disturbance of dark adaptation and turbidity of the anterior lens capsule. Argyria may also affect the respiratory system. The tissue content and distribution pattern of silver deposition is a function of the intake route, quantity and chemical form. The average silver content of the human body in the USA is estimated at 50 µg/kg wet-weight (MRI 1975).

Silver has a permanent cosmetic effect when the body burden exceeds about 1 g. Highest levels usually occur in the liver and spleen with lesser amounts in the muscles, skin and brain. Normal values, as mg/kg dry-weight, include 0.4 in the kidney, 0.7 in the liver and 2.7 in the spleen. Normal wet-weight values, as µg/kg, include 45 in the kidney, 32 in the liver and 60 in the lungs. In people with argyria, skin levels reach 50 to 71 mg/kg dry-weight in contrast to normal levels of 20 to 50 µg/kg. One case of 72 mg/kg wet-weight was reported (Seiler *et al.* 1988, Friberg *et al.* 1986).

Some reported levels of silver in human tissues from the UK include: 2 µg/kg in 8 kidney medulla, 6 µg/kg in 11 livers, 1 µg/kg in 6 lymph nodes, 2 µg/kg in 6 muscle samples, 2 µg/kg in 5 testes, 2 µg/kg in 6 ovaries, 1.1 µg/kg in 22 ribs from people living in areas with hard water, 4 µg/kg in 10 whole brains, 3 µg/kg in 2 brain frontal lobes, 4 µg/kg in 2 basal ganglia, 2 µg/kg in 11 lungs, 2 µg/kg in 8 kidneys and 1 µg/kg in 8 kidney cortex (USEPA 1980a).

## *4.5 excretion*

About 70% of the daily dietary ingestion or inhalation of silver in people is excreted in the feces within a week; little is found in the urine (Kehoe *et al.* 1940, Anon. 1990). Animal experiments with radio-silver show that mice, rats, monkeys and dogs excrete 99.6%, 98.4%, 94.4% and 90.4%, respectively, within 2 days of oral administration. Whole body retention of silver after one week is generally less than 1% of the initial dose in animals but rises to about 20% in man. Little, if any, is normally excreted in the urine but administration of calcium EDTA increases urinary excretion (Furchner *et al.* 1968). About 18% to 19% of a single oral dose of silver acetate was still in a human body 8 to 30 weeks later (East *et al.* 1980 and Macintyre *et al.* 1978). This is greater than the amount retained by dogs and most other animals.

Urine samples were taken from six men who had been employed from 7 to 23 years in jewelry handicrafting. Silver levels for 24 hours ranged from 5 to 261 µg/L. A mean value of 27 µg was found after a five-day shift for workers casting with oxyacetylene while the five-day mean was only 5 µg for those using an electromagnetic induction process (Minoia *et al.* 1987).

## *4.6 effects*

### 4.6.1 general

There is no known physiological function of silver in the human body and it is considered a contaminant when found in tissues. Toxicity is generally not seen until ingestion reaches about one gram per day. No studies are known documenting cancer or birth defects in humans due to silver exposure. Reproductive tissue damage due to silver nitrate injections is reversible.

Apart from some occupational exposure studies, the health effects on people of either short-term or long-term exposure to known specific concentrations of silver in the air, food or water are not known (Anon. 1990). Most silver exposure is incidental or anecdotal and unquantified.

### 4.6.2 inhalation

No studies are known documenting death or cardiovascular, musculoskeletal, immunological, neurological, developmental or genotoxic effects due to inhalation of silver or silver compounds in animals or people (Anon. 1990).The only study showing renal effects had co-exposure to cadmium, a known renal toxin, so the possible effects of silver are not known. Decreased night vision was also reported by these workers (Rosenman *et al*. 1987).

Dusts with a high silver oxide or silver nitrate content may cause gastrointestinal effects in occupationally-exposed workers. There was a burning abdominal pain reported by 10 of 30 workers which was relieved by antacids. Estimated exposure levels were between 39 and 378 µg silver per $m^3$ of air. Chemical forms and particle sizes were not known and employment duration ranged from <1 to >10 years. The symptoms were correlated with blood silver levels. Apart from one man with an elevated hemoglobin level, there were no hematological effects and blood counts were normal (Rosenman *et al*. 1979).

These same dusts also caused respiratory effects. Twenty-five of the above workers complained of upper respiratory irritation (runny nose, sneezing, sore throat or stuffiness) and 20 of these also had coughs, wheezing or chest tightness. Chest radiograms and respiratory function tests were normal (Rosenman *et al*. 1979). Further study of these same workers showed granular deposits in the conjunctiva and cornea of the eyes of 20 of the men. Subjective assessment of the degree of silver deposition showed correlation with the duration of employment, the reports of changes in skin colour and decreased night vision (Rosenman *et al*. 1979).

A man suffered respiratory problems 14 hours after working with molten silver ingots. Concentrations and the silver species were not known, nor was the prior history of exposure to silver. Breathing was noisy, pulse rapid, capillary blood low in oxygen and there were scattered thickenings in the lungs as seen in a chest radiogram. The patient suffered acute respiratory failure but eventually recovered fully (Forycki *et al*. 1983).

Silver reclamation workers chronically exposed to insoluble silver compounds (silver halides) showed marginal increases in mean corpuscular volume and marginal decreases in red blood cell counts (Pifer *et al*. 1989). The toxicological significance of

these changes, if any, is not known. Measured levels of liver enzymes (alanine amino transferase, aspartate amino transferase, gamma glutamyl transferase and alkaline phosphatase) found no significant differences between workers exposed to silver and insoluble silver salts and those with no history of silver exposure. Conjunctival and corneal silver deposits were found in about 25% of these workers and nasal-septal pigmentation in about 75% of them (Pifer *et al.* 1989).

### 4.6.3 dermal

No studies were located concerning death, respiratory, cardiovascular, neurological, developmental, genotoxic, gastrointestinal, hematological, musculoskeletal, hepatic, renal or ocular effects in people after dermal exposure to silver or silver compounds. Silver may cause mild allergic responses such as rashes, swellings and inflammation in some people. Silver is not likely to be a health hazard through skin contact.
Silver nitrate eye drops have been used for many years to prevent blindness in newborns exposed to gonorrhea. Many people have used skin creams containing silver nitrate or silver sulphadiazine for extended periods with no known detrimental health effects (Anon. 1990).

Medical case histories indicate that dermal exposure to silver or silver compounds for a long time can lead to localized skin discolouration similar to the generalized argyria which results from oral exposure. There is no quantification of this effect (Buckley 1963, McMahon and Bergfield 1983). Mild allergic responses are possible after repeated dermal contact with silver and silver compounds such as silver cyanide (6 months), radiographic processing solutions (10 years), and dental amalgams (20 years) (Catsakis and Sulica 1978, Heyl 1979, Marks 1966).

### 4.6.4 oral

Breath mints coated with silver, silver nitrate capsules for the relief of gastrointestinal discomfort, silver nitrate nose drops, silver acetate anti-smoking lozenges, and silver nitrate solutions for treating gum disease have also been used with no apparent adverse health effects, if taken as directed. There are case histories of people who have ingested excessive amounts of silver through abuse of these products. However, the data are mainly anecdotal, neither quantitative nor reliable, and cannot be used to set criteria or establish effect levels (Aeseth *et al.* 1981, Blumberg and Carey 1934, East *et al.* 1980, Landas *et al.* 1985, MacIntyre *et al.* 1978, Marshall and Schneider 1977, Shelton and Golding 1979, Shimamoto and Shimamoto 1987).

Gram amounts of silver-containing medicines taken over several months in small doses are needed to cause argyria and ingestion of gram levels would likely be required to

cause life-threatening conditions. Silver nitrate taken orally causes necrosis of the gastrointestinal tract. Doses of 10 to 1000 mg do not cause any symptoms but 2 to 30 g cause death in hours to days (MRI 1975, Sollman 1957)

Apart from the above, no studies were located documenting death or respiratory, hematological, immunological, musculoskeletal, cardiovascular, developmental, genotoxic, hepatic or renal effects in humans after oral exposure to silver or silver compounds. Silver may build up and be retained in the body under some conditions. Long exposures may cause a grey or blue-grey build-up of silver called argyria which is a permanent, but only a cosmetic, effect.

## *4.7 literature criteria*

### 4.7.1 drinking water

The USEPA suggested a level of silver in drinking water of not more than 50 µg/L to protect people from possible long-term health effects due to long exposure. In May 1989, a short-term exposure limit (from 1 to 10 days) of 1.142 mg/L was proposed. For occupational exposures OSHA set a legal limit of 10 $\mu g/m^3$ of silver in the workplace air (Anon. 1990). Health and Welfare Canada and the US Public Health Service recommended a maximum acceptable concentration of 50 µg/L of silver in drinking water (HWC 1978). At this concentration it would take 27 years to ingest the human toxic dose of 1 gram of silver, assuming no elimination and a 2 litre per day intake. This recommedation has since been dropped. In practice, elimination is over 90% of the ingested dose so it would take several life-times.

The Australian criteria for raw domestic water supplies are 50 µg/L (Hart 1974; Anon., Aust., 1992) and in 1980 the IWD, Water Quality Branch, also recommended this level of silver for water supplies which are used without treatment or with only simple filtration. When water received chemical treatment, the recommended silver concentrationwas 200 µg/L (Taylor *et al.* 1980). The latest guidelines, (CCME 1987; Anon., H. & W. Can., 1993), make no recommendation for silver in drinking water.

table 4.2

literature criteria for the protection of
drinking water.

| criteria* | jurisdiction | references |
| --- | --- | --- |
| 50 (MCL) | EPA-regulation | USEPA 1987 |
| 90 (SMCL) | EPA-proposed regulation | USEPA 1989 |
| 50 | FDA-permissible bottled water regulation | USFDA 1988 |
| 50 | EPA-recommended limit guideline | USEPA 1985 |
| 50 | EPA-ambient water criteria guideline | USEPA 1980b |
| 50 (maximum) | USA individual states-regulations | CELDS 1989 |
| 50 | Australia | ANON 1992 |

* in µg/L, mcl is maximum contaminant level and smcl is secondary maximum contaminant level.

**4.7.2 recreation and aesthetics**

The recommended level was 50 µg/L of total silver (Taylor *et al.* 1980). The aquatic life level is much lower so there is adequate protection for recreation and sport fishing in areas where aquatic life is being protected. The latest guidelines, (Anon., H. & W. Can., 1992), make no recommendation for silver in recreational water.

**4.7.3 wildlife and livestock**

There are no data to set a criterion for livestock or wildlife. Toxicity to animals is low and elimination is rapid. Surface waters have low silver levels and should not be a problem (Taylor *et al.* 1980). The aquatic life level is much lower than any wildlife or livestock criterion would need to be so there is adequate protection in areas where aquatic life is being protected. For confined livestock or laboratory animals, the human drinking water criterion is proposed.

## *4.8 proposed criteria*

#### 4.8.1 drinking water

No silver criterion appears to be necessary for human, laboratory animal, wildlife and livestock drinking water. The level used by Health and Welfare Canada in 1987 was 50 µg/L. Silver was dropped from the 1989 Guidelines for Canadian Drinking Water Quality since the 50 µg/L value was very conservative but was not scientifically defensible. A scientific risk assessment would likely determine a higher value as safe for drinking water, but such assessment has not been done.

#### 4.8.2 recreation and aesthetics

No silver criterion appears to be necessary for this use of the water. The aquatic life criteria are more than adequate for any such uses.

# 5. Irrigation

## *5.1 literature criteria*

There are not enough data to recommend a silver criterion for irrigation. Silver becomes toxic to plants at levels several orders of magnitude above those usually found in surface water, or levels recommended for aquatic life, so effects on plants are not likely to become an issue (Taylor *et al.* 1980).

## *5.2 general*

Although silver is widely distributed, it is not an essential element for plant growth (Vanselow 1966) and is not a normal constituent of plants (MRI 1975). Silver generally accumulates in the root zone of soils (Klein 1978). Factors influencing the silver concentration in plants and the uptake of silver are the plant species, soil composition, soil pH and the form of silver. Foliar applications of $AgNO_3$ at 150 mg silver/L protected pea seedlings from the effects of exogenous ethylene. This same protection was also seen in cotton, tomato and cucumber plants and the orchid *Cattleya*. There was no phytotoxicity at these concentrations (Beyer 1976).

### 5.3 silver speciation effects

Greenhouse studies have shown that silver is taken up much less readily from AgI than from $AgNO_3$. The silver concentrations in roots and stems of sugar beets, corn and kentucky blue grass grown in soil treated with 26 mg silver/kg as $AgNO_3$ were 4 to 24 times the concentrations in plants grown in soil treated with 12 mg silver/kg as AgI.

No increase in the silver concentrations of maize and soybean grown on loam or sand soils, and wheat grown on loam soils, treated with 1 gm/kg silver as AgI were noted compared to the control soils. Wheat grown on silver-treated sand soils showed a six fold increase in silver in the tissues. No increase in the silver concentrations of maize grown on loam or sand soils treated with 1 gm/kg silver as $AgNO_3$ were noted compared to the control soils (Klein 1978, Teller and Klein 1973, Weaver and Klarich 1973).

### 5.4 vegetation levels

The silver content of fruits and vegetables was 20 to 100 µg/kg and an average value for land plants was given as 60 µg/kg. High levels have been found in some plants, especially fungi, but no plants have been found which are regular silver concentrators. The mushroom, *Clavulina cinerea*, had 16 mg/kg and *Caulerpa prolifera* had 10 mg/kg. *Ephedra gerardiana* had a seasonal variation with 8 mg/kg in May and 20 mg/kg in September (Ewing *et al.* 1969, Horovitz *et al.* 1974, Cooper and Jolly 1970). Silver does not accumulate in the leaves of citrus plants grown in soil containing 75 mg silver/kg as soluble $AgNO_3$. The leaves had only 500 µg/kg (Vanselow 1966). In terrestrial herbs, shrubs and trees, the silver content of the ash is generally low, 200 µg/kg.

### 5.5 soil and vegetation interactions

Although higher silver levels in plants occur in regions of silver mineralization, there is no correlation between soil levels and plant levels of silver (Boyle 1968). The ratio of silver in plants to silver in soil has been given as 1:1.5 but one should use such a ratio with caution because the range is very wide, up to 1600 times (Lisk 1972, Horovitz *et al.* 1974).

In grass from a sub-alpine meadow community, the ratio of plant silver to soil silver went from 0.06 in treated soil to 5.04 in control soil. The treated soil contained 127.1 mg silver/kg after dosing with 100 mg/kg $AgNO_3$ and the control soil had 28 μg silver/kg (Klein and Giangiordano 1976).

Concentrations of 728 to 1015 mg silver/kg ash were found in roots of the experimental crops, corn, sugar beet and kentucky blue grass, grown on soils containing 26 mg silver/kg dry-weight. The silver was applied as $AgNO_3$. The ratio of silver in roots to that in soil was 28:1 to 39:1. If the soil was treated with AgI to contain 12 mg silver/kg dry-weight, the silver level in the roots was 43 to 176 mg/kg for a ratio of silver in roots to silver in soil of 4:1 to 15:1. In control soils with 90 μg silver/kg dry-weight, the silver levels in the roots were 1.2 to 3.4 mg/kg dry weight for a ratio of 13:1 to 38:1. The corresponding ratios for stems and leaves of control crops were similar, 13:1 to 17:1, but the ratios in the treated plants were smaller from 0.5:1 for AgI to 7:1 with $AgNO_3$. Silver thus concentrates in the roots and the silver in $AgNO_3$ is more available than the silver in AgI (Teller and Klein 1973).

## *5.6 plant toxicity*

Irrigation with 9.8 mg silver/L is toxic to maize and 4.9 mg/L is toxic to lupines (Cooper and Jolly 1970). There was no significant effect on wheat or maize at 460 mg/kg silver as AgI in sandy or loam soil but 640 mg/kg silver as AgI of soil inhibited germination of Engelmann spruce seeds (Klein 1978). Spraying a $AgNO_3$ solution at 9.5 mg silver/L caused damage to *Cattleya* orchids (Beyer 1976) and a decrease was noted in the growth rate of bean plants grown in a nutrient solution containing 9 μg silver as $AgNO_3$/L. Silver levels in the sediments or soils which exceed 25 to 50 mg silver/kg may have significant effects on the heterotrophic activities of the microbial flora (Sokol and Klein 1975).

## *5.7 hydroponics*

Fungal pathogens are a problem in hydroponic systems. Strains of such species as *Phytophtora nicotiana*, *Phytophtora cryptogea*, *Pythium aphanidermatum*, *Verticillum albo-atrum*, *Theilaviopsis basicola*, and *Fusarium oxysporum* are all pathogens of tomatoes. At 50 μg/L, silver ions rapidly killed zoospores. Silver is concentrated up to 2000 times in fungal conidia (Jain 1977, Miller *et al.* 1953, Slade and Pegg 1993).

# 6. Industrial

## *6.1 literature criteria*

The food industry, where water is a part of the final product, is one industry where silver levels were of concern and the recommended level was the drinking water level of 50 µg/L, as total silver, in water supplies which were not treated or were only filtered. If water treatment was carried out, then the level of the raw water could be up to 200 µg/L (Taylor *et al.* 1980).

## *6.2 recommended criteria*

For the food industry, where water is a part of the final product, no silver criterion appears to be necessary. Other industries, such as solid state electronics and photofinishing, which may have more stringent requirements, will need to have their own in house systems to reduce silver to required levels.

# 7. Aquatic Life

## *7.1 literature criteria*

### 7.1.1 marine water

A criterion of 2.3 µg/L of total recoverable silver, not to be exceeded at any time, was set by USEPA in 1980a. In 1976, their criterion was 0.01 of the 96 hour $LC_{50}$ of a resident sensitive species.

### 7.1.2 fresh water

Concentrations of total silver in an unfiltered sample should not exceed 0.1 µg/L to protect aquatic life (IJC 1982, Taylor *et al.* 1980, Ontario 1979 and 1984, CCME 1987). A provisional or preliminary criterion of 0.1 µg/L was set by Manitoba in 1983. The USEPA set ambient water quality criteria of 50 µg/L in 1980a and the water quality

criteria for 6 US states was reported as 50 µg/L by CELDS in 1989. In 1980a the USEPA set hardness dependent levels of 1.2 µg/L at 200 mg/L hardness, 4.1 µg/L at 400 mg/L hardness and 13 µg/L at 800 mg/L hardness. These 1980a USEPA criteria, which are not to be exceeded at any time, are derived from the equation: $e^{(1.72 [\ln(\text{hardness})] - 6.52)}$.

## *7.2 proposed criteria (µg/L total silver)*

table 7.1

recommended criteria for the protection of marine and freshwater aquatic life.

| Environment | Criteria as total Silver | Conditions |
|---|---|---|
| freshwaters | 0.05 µg/L as a 30-day average | hardness 100 mg/L |
| | 0.1 µg/L maximum | hardness 100 mg/L |
| | 1.5 µg/L as a 30-day average | hardness > 100 mg/L |
| | 3.0 µg/L maximum | hardness > 100 mg/L |
| marine waters | 1.5 µg/L as a 30-day average | open coast and estuaries |
| | 3.0 µg/L maximum | open coast and estuaries |

See Table 7.11 for the calculations leading to these criteria.

## *7.3 rationale for the criteria*

### 7.3.1 marine water

Maximum and 30-day average criteria have been set despite the paucity of data for temperate marine fish since they are generally not sensitive to silver below levels of 70

µg/L; a factor of 10 greater than the sensitivity of most invertebrate and algal species. Criteria are set to protect the most sensitive lifestage of the most sensitive species. The literature indicates the most sensitive organisms are phytoplankton, followed by the embryonic and larval stages of invertebrates. Since the sensitivity of invertebrates and phytoplankton to silver is much greater than that of fish, and because a great deal of literature is available concerning their sensitivies, the requirements for marine fish datawere considered to be superfluous and were waived.

The criteria set are in agreement with the literature value of 0.1 µg/L of total silver, in an unfiltered sample, set to protect aquatic life (IJC 1982, Taylor *et al.* 1980, Ontario 1979 and 1984, CCME 1987).

Phytoplankton are at the base of the marine food chain, thus an affect on them has the potential to produce an effect on almost all marine primary and secondary consumers. A fifty percent reduction in growth or biomass, a standard chronic response or $EC_{50}$ measure, represents a substantial drop in primary production. The open coast generally does not experience dramatic fluctuations in either salinity or temperature throughout the year and and it is relatively easy to set defensible and operationally functional criteria. Estuaries are a much more difficult situation. Silver toxicity has been shown to be inversely proportional to salinity (higher salinity results in lower toxicity) and in the estuarine environment both salinity and hardness fluctuate seasonally and with tidal changes. A criterion should reflect environmental fluctuations that can occur in the estuarine setting but it must also be operationally practical. We have chosen to leave the estuarine criteria the same as the marine and hard freshwater criteria recognizing that site specific situations may dictate that the low hardness criteria are more applicable in certain situations. The data support setting marine criteria the same as freshwater criteria in hard water, which is a reflection of the high hardness and high capacity of seawater to complex silver.

Site-specific criteria should be set for sewage outfall areas. Although a tolerance to elevated levels of silver apparently occurs, there is debate within the literature regarding whether accumulated levels of silver are toxic to the organisms. Detoxifying mechanisms, acclimation, and biomagnification potential are not well understood. Studies have shown that silver is tightly bound in sewage outfall areas, and that bacterial activity associated with sewage sludge may enhance the bioavailability of silver. Further research is needed in these areas to elucidate these processes. Complexation and speciation of silver in marine waters is not well understood. Silver may act synergisticly with copper, and cadmium may reduce silver toxicity. Field attempts to attribute an effect to the action of a single metal are often difficult, if not impossible. Experiments to determine the fate of silver, as well as that of other metals and their synergistic effects, are required. More good data are also needed on the

acute and chronic effects of silver on marine fish species, especially larval and embryo life stages.

### 7.3.2 fresh waters

Water hardness, organism size and lifestage, and length of exposure affect the toxicity of silver to freshwater organsisms. In 1980a, the USEPA determined criteria from an equation which included the effect of water hardness; however, the results did not protect the most sensitive species in water with hardness >100. From the literature, *Daphnia magna* appears to be the most sensitive organism, followed by embryos and larvae of fish species. Therefore, we propose both a maximum value, to protect *D. magna*, and a 30-day average value, to protect the embryos and larvae of fish species. Juvenile and adult fish, aquatic insects, and aquatic plants all seem to have a resistance to silver 10 to 1000 times greater than the organisms and lifestages upon which the criterion is based.

Further research is needed in order to determine the mechanism of toxicity of silver, its synergistic effects, and its environmental fate. No data were found concerning effects on anandromous salmonids, particularly to the fry in soft freshwater habitats where the toxicity of silver is expected to be much higher (Davies *et al*. 1978). Research should be undertaken in this area in order to determine if the criteria will protect both hatchery fry and wild fry.

See Table 7.11 for the safety factors used in deriving criteria from the lowest reliable effects levels found in the literature.

## *7.5 fresh waters*

### 7.5.1 general

The following listed tables provide some data on the effects of silver, under various conditions, on freshwater aquatic life.

Table 7.4-Acute effects of silver on freshwater organisms;
Table 7.5-Chronic effects of silver on freshwater organisms;
Table 7.6-Effects of acclimatization on silver toxicity in water of hardness 141 to *Pimephales promelas*, the fathead minnow;
Table 7.7-Comparison of the acute and chronic toxicity (to embryo/larvae) of different silver salts to the fathead minnow, *Pimephales promelas*;

Table 7.8-Effects of water hardness on the 96h $LC_{50}$ of silver nitrate to larval rainbow trout, *Oncorhynchus mykiss* and juvenile fathead minnows, *Pimephales promelas*, in static and flow through bioassays;
Table 7.9-Effects of hardness on the 48h $EC_{50}$ of silver nitrate to *Daphnia magna* in static bioassays and
Table 7.10-Mortality of fathead minnows exposed to measured concentrations of silver.

Silver is one of the most toxic of the heavy metals to freshwater micro-organisms, in several multi-metal toxicity tests it was placed first, second or third in the rank order of toxicity. Water hardness, as mg/L calcium, length of exposure, size of the organism and life stage of the organism all appear to affect the toxicity values. Variable reports of the validity of static versus flow-through tests within the literature exist; however static with renewal tests appear to be as accurate as flow-through bioassays. Invertebrates and embryos of fish are generally much more sensitive than juvenile and adult fish.

### 7.5.2 fish

Juvenile rainbow trout, *Oncorhynchus mykiss* (51 to 76 mm) were subjected to metal mining wastes (Hale 1977). In a continuous flow experiment, the trout were subjected to 5 to 80 µg/L of silver in water of hardness 100. The 96-hour $TL_{50}$ was determined to be 6.6 µg/L silver. The order of metal toxicity was cadmium > silver > lead.

In 1974 Coleman and Cearley studied silver accumulation and toxicity in the largemouth bass, *Micropterus salmoides*, and the bluegill, *Lepomis macrochirus*, in a 6 month static assay in water of hardness 180. Silver, as AgNO3, was tested at 0.9 µg/L, 7 µg/L and 70 µg/L silver exposure. Only the 70 µg/L silver exposure level had any significant effect. It was toxic to the bass within the first 24 hours; however, the bluegill tolerated this concentration for six months with no significant mortality. No effects on growth or weight gain were noted for either species at any treatment level. The water was hard and had a chloride concentration of 193 mg/L; much higher than most natural fresh waters. Davies *et al.* (1978) recommended that the results of this study be used with caution since the silver would be mostly complexed by the chloride, and would likely precipitate due to the low solubility of AgCl, (1.5 mg/L, Jones 1939) rather than be available as the free $Ag^+$ ion.
Davies *et al.* (1978) also reported the toxicity of silver to rainbow trout in hard water, hardness 350, and soft water, hardness 26. The $LC_{50}$ values for hard and soft water were 13.0 µg/L and 6.5 µg/L silver, respectively. An 18-month experiment gave a NOEL of 0.09 to 0.17 µg/L. At >017 µg/L silver, premature hatching, high mortality of larvae, and reduced growth rate were observed.

In the study with *O. mykiss*, Davies *et al.* (1978) examined the effect of silver iodide in soft water, hardness 30, in a six-week study with fry, a 13-month study initiated with eyed eggs, and a 10-month experiment started with green eggs. In the six-week study with fry, exposure was set at 0.88 µg/L silver, resulting in 94 percent mortality and retardation in development and growth in the surviving fish. The 13-month NOEL using eyed-eggs was 0.03 to 0.06 mg/L silver. No other effects on growth or development were evident. The 10-month NOEL using green eggs was 0.18 to 0.40 µg/L silver, a value much higher than the NOEL for the eyed-eggs. This may be due to embryonic acclimatization to silver in the three months exposure prior to swim up; it may also be due to genetic differences. No additional effects on growth or development were noted.

The toxicity of AgCl to the threespine stickleback, *Gasterosteus aculeatus*, was determined by Jones in 1939. The lethal limit was 3 µg/L silver, which was fatal in approximately 25 minutes. Silver was found to be the most toxic metal of a suite of metals tested. The apparent mode of action of silver was determined to be precipitation of gill secretions causing asphyxiation. Jones (1939) was apparently careful to determine that the levels of silver in solution were below the solubility limits for AgCl, but no water quality data were given.
The flagfish, *Jordanella floridae*, and the fathead minnow, *Pimephales promelas*, had 96-hour $LC_{50}$ values determined in a flow-through bioassay in water of hardness 44 and temperature of 24.7C (Lima *et al.* 1982). The values were 9.2 µg/L and 10.7 µg/L silver for the flagfish and the fathead minnow, respectively. Most fish died shortly after showing signs of stress, and fathead minnows had bright red gills at death. Lemke (1981) gives a 96-hour $LC_{50}$ value of 3.9 to 12 µg/L silver in water of hardness 40 to 49.

A static renewal assay in water of hardness 250 and temperature of 30C was used to determine the acute toxicity of silver to the fish *Puntius sophore, Channa punctatus* and *Lebistes reticulatus* (Khangarot *et al.* 1988b). Ninety-six hour $LC_{50}$ values were 7.55 µg/L, 18.89 µg/L and 6.44 µg/L silver, respectively. $LC_{50}$ values determined at 12, 24, 48 and 72 hours also indicate that silver toxicity increased with the time of exposure. Behavioural and pathological observations noted in early stages of exposure were erratic opercular movement, difficulty in respiration, convulsions and surfacing. In later stages of exposure, behaviour included loss of equilibrium, erratic body movement, upside-down swimming, irregular opercular movement and absence of shoaling. At death, observations made included copious mucus on skin and gills, light and dull color of gill filaments and shrinkage of gill lamellae and hemorrhaging of the mouth and caudal regions. Silver toxicity was inversely proportional to fish size, with greater toxicity in smaller fish. Cause of death was believed to be gill damage resulting in hypoxia, but was possibly also poisoning of enzymes related to gas exchange.

In 1983, Holcombe *et al.* reported on the acute toxicity of silver in both flow-through and static tests in water of hardness 40 to 45 and temperature of 23 to 25C, to the fathead minnow, *Pimephales promelas*, and the channel catfish, *Ictaluraus punctatus*. Chronic tests were also conducted on the larvae of the fathead minnow.
Ninety-six hour $LC_{50}$ values for the fathead minnow were 6.7 µg/L silver for the flow-through test, and 14.0 µg/L silver for the static test, indicating that flow-through tests were more sensitive. Lemke (1981) reported 96-hour $LC_{50}$ values for the fathead minnow in soft water ranging from 3.9 to 30 µg/L silver, and in hard water as ranging from 110 to 270 µg/L silver. The 96-hour $LC_{50}$ value for the channel catfish (under flow-through conditions) was 17.3 µg/L silver, which is much greater than the value for the fathead minnow. However, the catfish average weight was approximately 100 times that of the minnow. Silver toxicity increased with increasing period of exposure. The 28-day chronic assay with the fathead minnow larvae gave a NOEL of 0.37 µg/L silver and a maximum acceptable concentration (MATC) of 0.65 µg/L silver.

The effect of speciation on the acute and chronic toxicity of silver was compared using the fathead minnow as the test organism (LeBlanc *et al.* 1984). Silver sulfide, silver thiosulfate and silver chloride were compared to $Ag^+$, added as silver nitrate. The tests were flow-through in water of hardness 38 and temperature 25C. The 96-hour $LC_{50}$ value for $Ag^+$ (as silver nitrate) was 16 µg/L silver. Silver chloride was found to be 300 times less toxic, silver sulfide was 15,000 times less toxic, and silver thiosulfate was 17,500 times less toxic than $Ag^+$. The 30 day MATC's for silver sulfide and silver thiosulfate were determined to be >11,000 µg/L silver and 16,000 to 35,000 µg/L silver, respectively.

Nebeker *et al.* (1983) reported on a series of side by side (within the same compartmentalized container), flow-through and static acute tests with both steelhead and rainbow trout (*Oncorhynchus mykiss*) in water of hardness 26 to 42 and temperature of 9 to 12C; the fathead minnow, *Pimephales promelas* , in water of hardness 38 to 46 and temperature of 20 to 22C; and a 60-day chronic assay with embryo/larvae of the steelhead trout.

Four static tests were conducted with rainbow trout, two aerated and two non-aerated. The 96-hour $LC_{50}$ values were reported as 72.9 and 84.4 µg/L silver in the aerated tests and 10.9 and 8.5 µg/L silver in the non-aerated tests. A significant difference between the two was the much larger fish size in the aerated tests. The two flow-through tests conducted with the rainbow trout gave $LC_{50}$ values of 8.6 and 9.7 µg/L silver, and for the steelhead trout 9.2 µg/L silver. The results of the flow-through and the static tests were not significantly different, nor were the results of the steelhead and the rainbow flow-through tests. The static fathead minnow assays gave $LC_{50}$ values of 9.4 and 9.7 µg/L silver, and the flow-through assays gave $LC_{50}$ values of 5.6 and 7.4

μg/L silver, an average factor of 0.7 lower; however, the authors did not specify if this was significant. The result of the chronic study was a 60-day MATC of <0.1 μg/L silver, and an $LC_{100}$ value of 1.3 μg/L silver. The authors noted a significant decrease in percent survival at 0.5 μg/L silver and a significant reduction in growth at 0.1 μg/L silver.

A technique for simultaneous multi-species testing in freshwater of hardness 44 and temperature of 17C was developed by Holcombe *et al.* (1987). The authors believed the technique provided a more accurate direct comparison of species sensitivity and behavioural responses. Species tested for silver toxicity included the fathead minnow-*Pimephales promelas*, rainbow trout-*Oncorhynchus mykiss* and bluegill-*Lepomis macrochirus*. The 96-hour $LC_{50}$ values obtained were 9 μg/L of silver, 6 μg/L of silver and 13 mg/L of silver, respectively. An overall species sensitivity factor, defined as {[$LC_{50}$ (most resistant species)] ÷ [$LC_{50}$ (least resistant species)]}, of 622 was calculated. Though not as sensitive as *Daphnia magna* to silver, the rainbow trout was considered the most all-round sensitive species for toxicity testing. Fish, except for the rainbow trout, were less sensitive than *D. magna* by a factor of 10. Silver was the only metal tested, the others were organics, and was the most toxic of the test reagents to all species, on average.

In 1970 Jackim, *et al.* reported on the activity of five liver enzymes from the estuarine killifish, *Fundulus heteroclitus*, exposed to the toxic metals copper, lead, mercury, cadmium, silver and beryllium at concentrations approximating the 96-h $LC_{50}$. Silver did not affect all the enzymes to the same extent. Liver acid phosphatase was not affected *in vivo* although there was 50% inhibition by 10.8 mg/L *in vitro*. Mercury and silver were the most potent inhibitors of catalase *in vitro* and fish exposed to 40 μg/L had decreased liver catalase activity. One should use caution in extrapolating *in vitro* experiments to natural ecosystems since different chemical mechanisms may act under the two conditions and the chemicals may not reach the active sites in the whole animal.

Water hardness is an important factor for the toxicity of some heavy metals such as copper and zinc but appears to be less important for silver. The 96-h $LC_{50}$ for rainbow trout, *Oncorhynchus mykiss*, was 8.1 μg silver/L in water of hardness 26 and 13 μg silver /L in water of hardness 350 (Goettl *et al.* 1976). Developing eggs were exposed to silver nitrate and the no effect level lay between 90 and 170 ng silver /L. Silver concentrations over 1 μg/L caused the eyed-eggs to hatch before complete development and the fry soon died. Silver iodide was more toxic than siver nitrate and the no effect level was between 30 and 70 ng silver /L (Goettl *et al.* 1976).

In 1976, Davies recommended a concentration between 70 and 130 ng/L as being safe. After a one-year exposure to silver, the mortality of rainbow trout fry after swim-up decreased from 38% at 500 ng silver/L to 18% at 130 ng silver/L and 3% at 70 ng silver/L. There was no mortality in control fish. For the carp, *Cyprinus carpio*, the 96-h $LC_{50}$ was 3.8 µg $AgNO_3$/L in water of hardness 118 (Rao *et al.* 1975).

### 7.5.3 amphipods

*Crangonyx pseudogracilis*, a freshwater amphipod, was subjected to acute exposures of silver and other metals in a static assay in water of hardness 45 to 55 and temperature of 13C for 96 hours (Martin and Holdich 1986). Forty-eight and 96-hour $LC_{50}$ values were 6 µg/L of silver and 5 µg/L of silver, respectively. After 48-hours the increasing order of metal toxicity was silver > mercury > manganese (VII), but at 96 hours the order had changed slightly to mercury > silver >chromium (VI)> manganese (VII). The values were expressed as ppm in the paper, but the authors suggest that only molar equivalents are comparable.

### 7.5.4 rotifers

Rotifers are a major part of some fish diets and the effects of silver were reported by Buikema *et al.* in 1974. The 96-hour $EC_{50}$ to *Philodina acuticornis* in water of hardness 17 was 1.4 to 1.7 mg of silver per litre. The silver salt used was $AgNO_3$.

### 7.5.5 amphibians

Tadpoles of the toad, *Bufo melanostictus*, were tested for sensitivity to silver and other metals in water of hardness 185 and temperature of 31C (Khangarot and Ray 1987b). $LC_{50}$ values were calculated at 12, 24, 48, 72 and 96 hours. The 96-hour $LC_{50}$ value was 4.1 µg/L silver, an order of magnitude lower than the 12-hour $LC_{50}$ value. The rank order of toxicity of the metals to the toad was: silver > mercury > copper > cadmium > zinc > nickel > chromium.

### 7.5.6 cladocerans

A *D. magna* renewal life cycle test method was evaluated in a series of inter-laboratory tests involving different water quality parameters (Nebeker 1982). The assays were to determine the 48-hour $EC_{50}$, the 21-day $EC_{50}$, the NOEL (as number of young/female/day) and the MATC. The 48-hour $EC_{50}$ values ranged from 0.65 µg/L silver at a water hardness of 46, to 51.5 µg/L silver at a water hardness 255. The $EC_{50}$ values from water of hardness 60 were generally <1.0 µg/L silver, while the $EC_{50}$ values from water of hardness >60 were generally >10 µg/L silver. The 21-day $EC_{50}$

values ranged from 2.9 to 3.9 µg/L silver. The NOEL ranged from 1.6 to 8.8 µg/L silver in water of hardness 60, and from 10.5 to 20.0 µg/L silver in water of hardness >60. The MATC ranged from 3.1 to 19.4 µg/L silver in water of hardness of 60, and from 21.2 to 41 µg/L silver in water of hardness of >60. The chronic concentrations are greater than the acute concentrations, due to the addition of food which would complex the silver and reduce its bioavailability.

Anderson (1948) studied the effects of AgNO3 and other chlorides of metals on *Daphnia magna*. The test took place over 64 hours to ensure that ecdysis would occur. The toxic threshold was determined to be 5.1 µg/L silver and ecdysis was deemed to exert little effect on the toxicity of silver. Silver appeared to be one million times more toxic than the other metals, including mercury. In 1959, Bringmann and Kuhn reported the lethal level of silver nitrate to *D. magna* to be 30 µg/L.

Khangarot and Ray (1987a) determined the acute toxicity of silver to *D. magna* and attempted to find a correlation between *D. magna* $EC_{50}$ values and $LC_{50}$ values for the rainbow trout, *Oncorhynchus mykiss*. The 48- and 96-hour *D. magna* $EC_{50}$ values were 23 µg/L silver and 10 µg/L silver, respectively. The difference between the two values was significant.
A strong correlation between *D. magna* and rainbow trout sensitivity was found (straight line regression, r2 = 0.814) and therefore *D. magna* is presented as a pollution monitor species. The rank toxicity of metals to *D. magna* was: mercury > silver > copper > zinc > chromium = cadmium = lead > nickel.

A 48-hour toxicity test was conducted using three cladoceran species, *Daphnia pulex*, *Ceriodaphnia reticulata* and *Simocephalus vetulus* (Mount and Norberg 1984). The tests were static assays with food in the solution (50,000 to 100,000 bacterial cells per culture). It is not clear from the text whether the test used renewal techniques. The 48-hour $LC_{50}$ values for silver were: 14 µg/L silver for *D. pulex*, 11 µg/L silver for *C. reticulata* and 15 µg/L for *S. vetulus*. It is likely that these values are higher than they would be if there were no food in the solution.

In a study using several species and methods, Nebeker *et al.* (1983) determined the toxicity of silver to *D. magna* with and without food. The mean result of three acute 96-hour assays without food, at a hardness of 33 to 40 and temperature of 20C, was an $EC_{50}$ of 0.9 µg/L silver; one assay with food gave an $EC_{50}$ of 12.5 µg/L silver. To determine how much of the silver was lost to the food, water with 4 µg/L silver was added. When the concentration of silver in the water was measured, the average level was 4.2 µg/L silver. When the water was filtered before the silver content was measured, 59% of the silver was lost. When food was added to the water, but the water

was not filtered, 42% was lost. When food was added to the water and then the solution was filtered, 89% of the silver was lost.

### 7.5.7 worms

The tubificid worm, *Tubifex tubifex*, was exposed to several metals in a series of 96-hour acute assays at a hardness of 245 and temperature of 30C (Khangarot 1991). $EC_{50}$ values were calculated at 24, 48 and 96 hours. The 96-hour $EC_{50}$ value was 31 µg/L silver, an order of magnitude lower than the 24-hour $EC_{50}$ value. The rank order of toxicity of the metals to *Tubifex tubifex* was: mercury > silver > copper > zinc > nickel > cadmium > lead.

### 7.5.8 molluscs

A 96-hour assay determined the toxicity of silver and other heavy metals to the pulmonate snail, *Lymnaea luteola*, at a water hardness of 195 and temperature of 32C (Khangarot and Ray 1988a). $LC_{50}$ values were determined at 24, 48, 72 and 96 hours. The 96-hour $LC_{50}$ value was 4.2 µg/L silver, 10 times lower than the 24-hour $LC_{50}$ value. The highest mortality was observed to occur in the first 48 hours, after which it decreased. The rank order of metal toxicity to the snail was: silver > mercury > copper > nickel = cadmium = zinc > chromium.

A static-renewal test at a hardness of 50.4 and temperature of 25.5C was carried out using a number of aquatic organisms including the the snail, *Aplexa hypnorum* (Holcombe *et al.* 1983). In a 96-hour assay, with renewal taking place every 24 hours, the $LC_{50}$ value was 241 µg/L silver. The snails were generally the most resistant species for all the chemicals tested.

### 7.5.9 insects

Nehring (1976) investigated the sensitivity to silver of two aquatic insects, the mayfly, *Ephemerella grandis*, and the stonefly, *Pteronarcys californica*, in a series of flow-through assays at a hardness of 30 to 70 and temperature of 3 to 9C. The mayfly was more sensitive than the stonefly with a $TL_{50}$ value of <1 µg/L silver, compared to 4 to 9 µg/L silver for the stonefly. Accumulation of silver was also studied in both species of insect at several levels of exposure. The results indicated that both accumulated silver, with a concentration factor of 113 for the stonefly and 195 for the mayfly. Also noted was a strong correlation to accumulation with respect to exposure concentrations, indicating that the insects accumulated silver by some constant amount, which in turn suggests that they might be useful as a pollution monitor species. The 7-day $LC_{50}$ for

the immature stoneflies was below 4 µg/L but 9 µg/L killed 96% of the mayflies in 10 days (Goettl *et al.* 1976).

### 7.5.10 miscellaneous and mixed invertebrates

The 96-hour $LC_{50}$ for the scud, *Gammarus pseudolimnaeus*, and the 48-hour $LC_{50}$ for the midge, *Tanytarsus dissimilis*, were determined in water of hardness 44 in a test conducted under flowing conditions (Lima *et al.* 1982). The scud was much more sensitive than the midge, with a 48-hour $LC_{50}$ value of 4.7 µg/L silver and a 96-hour $LC_{50}$ value of 4.5 µg/L silver, as opposed to a 48-hour $LC_{50}$ value of 3160 µg/L for the midge. After 22 hours of exposure, the scuds exposed to the two highest concentrations of silver, 15.3 µg/L and 35.6 µg/L, were dead. Lemke (1981) is cited, giving a 96-hour $LC_{50}$ value of 0.39 to 2.9 µg/L silver at a hardness of 40 to 49 for *Daphnia magna*, indicating that *Daphnia* is more sensitive to silver than the scud. Holcombe *et al.* (1987) conducted a series of simultaneous tests with a large number of species using a flowing system and compartmentalized tanks at a water hardness of 44.7 and temperature of 17C. Invertebrates tested were: the cladoceran, *D. magna*, the leech, *Nephelopsis obscura*, the snail, *Aplexa hypnorum*, the midge, *Tanytarsus dissimilis* and the crayfish, *Orconectes immunis*.. The results are an $EC_{50}$ of 0.9 µg/L silver for *D. magna* , an $LC_{50}$ of 420 µg/L silver for the midge and an $LC_{50}$ of 560 µg/L silver for the crayfish. The snail had an $LC_{50}$ value of 83 µg/L silver, and the leech an $LC_{50}$ value of 29 µg/L silver. *D. magna* was the most sensitive species tested to silver.

### 7.5.11 plants

Brown and Rattigan (1979) carried out a 24-hour study to determine the effects of silver and other metals on photosynthesis, and a 28-day chronic study on phytotoxicity with the aquatic macrophyte *Elodea canadensis*. The test water was described as soft. Results of the acute study were given as the I50 or the I90 (inhibiting photosynthesis by 50% or 90%, respectively). The I50 and the I90 had values of 100 µg/L silver and 180 µg/L silver, respectively. The results of the chronic study were given as the concentration which caused 50% plant damage with respect to the control. For *E. canadensis* the value was 7500 µg/L silver, and for another aquatic macrophyte, *Lemna minor*, the value was 270 µg/L silver. It was noted that silver caused a significant increase in oxygen uptake, similar in magnitude to copper, just before complete cessation of photosynthesis.

Nasu and Kugimoto (1981) investigated the sensitivity of the duckweed, *Lemna paucicostata*, to silver. Using a static assay at 25C and two different growth media, the authors also attempted to determine if pH would have an effect. No pH effect was

noted; however, in one medium at pH 4.1 and low free silver ion concentrations, 100 to 1000 µg/L, *Lemna* flowered.

Flowering was not observed in the second media. Growth of new fronds was reduced 50% with respect to the controls for plants grown in 10 mg/L silver. Hutchinson and Czyrska in (1975) reported that silver was less toxic to the duckweed, *Lemna valdiviana*, and the aquatic fern, *Salvinia natans*, than to several species of algae. There was no growth inhibition at 50 µg/L but there was inhibition at 500 µg/L.

### 7.5.12 algae

*Chlorella vulgaris* growth was first slowed at 10 µg silver/L and growth was completely inhibited at 60 µg/L (Hutchinson and Stokes 1975). Bringmann and Kuhn (1959) reported the lethal level of silver nitrate to *Scenedesmus quadricauda* to be 50 µg/L. The toxicity thresholds for the onset of cell multiplication inhibition, for the blue-green alga, *Microcystis aeruginosa*, and the green alga, *Scenedesmus quadricauda*, were 0.7 and 9.5 µg silver/L, respectively. They were grown in stock nutrient solutions and distilled water (Bringmann and Kuhn 1978). Lab strains of *Scenedesmus* and *Chlorella* were inhibited by 100 µg silver/L and 30 µg silver/L, respectively. The *Scenedesmus* grew at 50 µg silver/L (Stokes *et al.* 1973).

### 7.5.13 bacteria

Silver thiosulphate and insoluble silver sulphide had no effect on activated sludge at 100 mg/L, but 10 mg/L silver nitrate or silver chloride caused 84% and 43%, respectively, inhibition of oxygen uptake (Bard *et al.* 1976). Freshwater bacteria community numbers were not affected but their heterotrophic activity was affected by 10 and 100 ng silver/L (Albright and Wilson 1974). Klein and Giangiordano (1976) showed that silver at 5, 10 and 100 µg/L caused an increase in growth delays at low temperatures in populations of *Escherichia coli* and *Hyphomicrobium*. Transferring the cells to higher temperatures, 9 to 16C, neutralized the effect of the silver. In *Pseudomonas aeruginosa,* silver sulfadiazine affected the DNA. Silver ion binding to the DNA was both chemically and biologically reversible. Inhibition of growth started at 0.32 µg silver/L and was complete at 5.4 µg/L. Cell death occurred at 21.6 µg/L (Modak and Fox 1973). Bringmann and Kuhn (1959) reported the lethal level of silver nitrate to *Escherichia coli* to be 40 µg/L. Bacterial and fungal spores are more resistant to silver than the vegetative stages and yeasts are more resistant than bacteria (Klein 1978).

## Research Needs

Most existing silver criteria, objectives or regulated amounts are not based on the free ionic monovalent ion, which is acutely toxic to aquatic life; they are based, instead, on total silver which includes the metal, complexes and precipitates all of which are very much less toxic than the monovalent ion. Thus, these existing regulations and criteria are often overprotective.

Regulations should reflect the appropriate risk but the problem is that there is no monovalent ion specific measurement. Therefore, total silver is measured to provide a margin of safety. In addition, some non biologically-available silver may be in forms that are in equilibrium with monovalent silver and thus much of the silver pool becomes ultimately available as the monovalent silver is taken up. Benthic organisms will take up some insoluble forms of silver as they graze, and thus more than just the monovalent form is available to them.

A method of measuring the biologically-available forms of silver is needed so that the criteria and the risk are well correlated.

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# 10. DATA TABLES

table 2.1

silver in oranges and avocados from gold-rich and gold-poor soils.

| soil type | silver levels in mg/kg-mean (range) | | | |
|---|---|---|---|---|
| of the | oranges | | avocados | |
| ghanian farms | flesh | peel | flesh | rind |
| Konongo: gold-rich | 1.4 (1.2-1.8) | 1.7 (1.0-2.3) | 1.2 (1.0-1.4) | 1.7 (1.7-1.8) |
| Obuasi: gold-rich | 2.2 (1.0-3.4) | 3.3 (1.0-5.0) | 3.5 (0.3-6.7) | 2.0 (1.0-2.9) |

| Kumasi: gold-poor | 0.8 (0.1-1.5) | 0.9 (0.1-1.7) | 0.7 (0.0-1.2) | 1.9 (1.3-2.4) |
|---|---|---|---|---|

table 3.1

physical and chemical properties and data: silver.

| parameters | values | references |
|---|---|---|
| Synonyms | silver, argentum, argentum crede, CI 77820, shell silver, silver atom, silver colloidal, silflake, silpowder, silber | CHEMLINE 1988<br>HSDB 1988<br>Weast 1977<br>Stokinger 1981 |
| formula | Ag | Grayson 1983<br>Windholz 1983 |
| structure | Ag | HSDB 1988 |
| CAS number | 7440-22-4 | HSDB 1988 |
| NIOSH RTECS | VW 3500000 | HSDB 1988 |
| EPA hazardous waste | D011 | HSDB 1988 |
| OHM/TADS | 7216881 | HSDB 1988 |
| HSDB | 5034 | HSDB 1988 |
| molecular weight | 107.868 | Weast 1988 |
| colour | white, lustrous | Weast 1988 |
| physical state | solid, soft, ductile metal | Grayson 1983 |
| valence states | 0, +1, +2, +3 | Windholz 1983 |
| melting point | 961.93C / 960.5C | Weast 1988 |
| boiling point | 2212C @760 mm Hg | Weast 1988 |
| density @20C | 10.53 g/cm$^3$, 10.43 g/cm$^3$ hard drawn, 10.49 g/cm$^3$ annealed | Weast 1988<br>Grayson 1983 |
| solubility in water @20C | insoluble; soluble in nitric acid but not in sulphuric acid and alkaline cyanide | Windholz 1983<br>ITII 1982 |

| | | |
|---|---|---|
| | solutions | |
| vapour pressure of liquid silver at 1865C | 100 mmHg | Weast 1988 |
| flammability | dust is moderately flammable | ITII 1982 |

table 3.2

physical and chemical properties and data:
silver nitrate.

| parameters | values | references |
|---|---|---|
| Synonyms | lunar caustic, fused silver nitrate, molded silver nitrate, argenti, nitras, nitric acid silver (I) salt, nitric acid silver (I+) salt, silver (I+) nitrate | Weiss 1986<br>HSDB 1988<br>Windholz 1983 |
| formula | $AgNO_3$ | Grayson 1983<br>Weiss 1986 |
| structure | $Ag^+ NO_3^-$ | HSDB 1988 |
| CAS number | 7761-88-8 | Grayson 1983<br>Weiss 1986 |
| NIOSH RTECS | VW 4725000 | HSDB 1988 |
| STCC | 49 187 42 | HSDB 1988 |
| OHM/TADS | 7216883 | HSDB 1988 |
| HSDB | 685 | HSDB 1988 |
| molecular weight | 169.89 | Weast 1988 |
| colour | colourless or white | Grayson 1983 |
| physical state | solid crystaline | Weast 1988 |
| melting point | 212C | Grayson 1983 |
| boiling point | decomposes at 440C | Grayson 1983 |
| density @19C | 4.35 g/cm$^3$<br>4.33 g/cm$^3$ | HSDB 1988<br>Weiss 1986 |

| | | |
|---|---|---|
| solubility in water @0C | 122 g/100 mL | HSDB 1988 |
| solubility in organics | ethanol and acetone | Grayson 1983 |
| flammability | not flammable | Weiss 1986 |

from: USEPA 1990, TP-90-24.

table 3.3

physical and chemical properties and data:
silver (i) oxide.

| parameters | values | references |
|---|---|---|
| Synonyms | argentous oxide, silver oxide, disilver oxide, silver (I+) oxide | Windholz 1983 |
| formula | $Ag_2O$ | Grayson 1983 Weiss 1986 |
| structure | $Ag^+ Ag^+ O_2^-$ | RTECS 1988 |
| CAS number | 20667-12-3 | Grayson 1983 |
| NIOSH RTECS | VW 4900000 | HSDB 1988 |
| molecular weight | 231.8 | Weast 1988 |
| colour | dark brown to black | Windholz 1983 |
| physical state | solid crystaline | Weast 1988 Weiss 1986 |
| melting point | decomposes at 230C | Weast 1988 |
| boiling point | decomposes at 230C | Weast 1988 |
| density @20C | 7.14 g/cm$^3$ | Weiss 1986 |
| solubility/water @25C | 22 mg/L | Grayson 1983 |
| flammability | not flammable | Weiss 1986 |

from: USEPA 1990, TP-90-24.

table 3.4

physical and chemical properties and data:
silver (ii) oxide.

| parameters | values | references |
|---|---|---|
| Synonyms | argentic oxide, silver suboxide, silver peroxide, divasil, silver (II+) oxide | Windholz 1983 |
| formula | AgO | Grayson 1983 |
| structure | $Ag^{2+}$ $O_2^-$ | Grayson 1983 |
| CAS number | 1301-96-8; 35366-11-1 | Grayson 1983 |
| molecular weight | 123.88 | Windholz 1983 |
| colour | charcoal gray powder<br>black crystal | Grayson 1983<br>Windholz 1983 |
| physical state | solid crystaline | Windholz 1983 |
| boiling point | decomposes above 100C | Windholz 1983 |
| solubility/water @20C | decomposes in water | Windholz 1983 |

from: USEPA 1990, TP-90-24.

table 3.5

physical and chemical properties and data:
silver sulfide.

| parameters | values | references |
|---|---|---|
| Synonyms | argentous sulfide, acanthite | Weast 1988<br>Windholz 1983 |
| formula | $Ag_2S$ | Grayson 1983 |
| structure | $Ag^+$ $Ag^+$ $S_2^-$ | Windholz 1983 |
| CAS number | 21548-73-2 | Grayson 1983 |
| molecular weight | 247.80 | Weast 1988 |
| colour | gray black | Weast 1988 |
| physical state | solid | Grayson 1983 |

| | | |
|---|---|---|
| boiling point | decomposes 810C | Grayson 1983 |
| density at 20C | 7.326 $g/cm^3$ | Weast 1988 |
| solubility/water @20C | 0.14 mg/L | Grayson 1983 |

from: USEPA 1990, TP-90-24.

table 3.6

physical and chemical properties and data:
silver chloride.

| parameters | values | references |
|---|---|---|
| Synonyms | silver (I) chloride, silver monochloride | RTECS 1988 |
| formula | AgCl | Grayson 1983 |
| structure | $Ag^+ Cl^-$ | RTECS 1988 |
| CAS number | 7783-90-6 | Grayson 1983 |
| NIOSH RTECS | VW 3563000 | RTECS 1988 |
| molecular weight | 143.34 | Windholz 1983 |
| colour | white | Windholz 1983 |
| physical state | solid | Windholz 1983 |
| melting point | 455C | Windholz 1983 |
| boiling point | 1550C | Windholz 1983 |
| density at 20C | 5.56 $g/cm^3$ | Windholz 1983 |
| solubility/water @25C | 1.93 mg/L | Windholz 1983 |

from: USEPA 1990, TP-90-24.

table 3.7

bioaccumulation of silver in marine invertebrates.

| baf | water source | species and tissues | sources |
|---|---|---|---|
| 30 | sewage outfall | *Haliotis cracherodii*- muscle abalone, µg/kg wet weight | Young *et al*. 1981 |
| <10 | clean (control) | *Haliotis cracherodii*- muscle abalone, µg/kg wet weight | Young *et al*. 1981 |
| 22 | sewage outfall | *Hinnites giganteus*- muscle scallop, µg/kg wet weight | Young *et al*. 1981 |
| 6 | clean (control) | *Hinnites giganteus*- muscle scallop, µg/kg wet weight | Young *et al*. 1981 |
| 50 | sewage outfall | *Panulirus interruptus*- muscle lobster, µg/kg wet weight | Young *et al*. 1981 |
| 10 | clean (control) | *Panulirus interruptus*- muscle lobster, µg/kg wet weight | Young *et al*. 1981 |
| 100 | sewage outfall | *Cancer anthony*- muscle crab, µg/kg wet weight | Young *et al.* 1981 |
| 220 | clean (control) | *Cancer anthonyl*- muscle crab, µg/kg wet weight | Young *et al* 1981 |
| <10 | sewage outfall | *Strongylocentrotus franciscanus*- muscle red sea urchin, µg/kg wet weight | Young *et al*. 1981 |
| <10 | clean (control) | *Strongylocentrotus franciscanus*- muscle red sea urchin, µg/kg wet weight | Young *et al*. 1981 |
| 20 | sewage outfall | *Genyonemus lineatus*- muscle white croaker, µg/kg wet weight | Young *et al*. 1981 |
| 20 | clean (control) | *Genyonemus lineatus*- muscle white croaker, µg/kg wet weight | Young *et al*. 1981 |
| <10 | sewage outfall | *Citharichthys sordidus*- muscle pacific sanddab, µg/kg wet weight | Young *et al*. 1981 |
| <10 | clean (control) | *Citharichthys sordidus*- muscle pacific sanddab, µg/kg wet weight | Young *et al*. 1981 |

| 20 | sewage outfall | *Scorpaena guttata*- muscle<br>california scorpionfish, µg/kg wet weight | Young<br>*et al*. 1981 |
|---|---|---|---|
| 20 | clean (control) | *Scorpaena guttata* - muscle<br>california scorpionfish, µg/kg wet weight | Young<br>*et al*. 1981 |
| baf | water source | species and tissues | sources |
| <10 | sewage outfall | *Paralichthys californicus*-muscle<br>california halibut, µg/kg wet weight | Young<br>*et al*. 1981 |
| <10 | clean (control) | *Paralichthys californicus*-muscle<br>california halibut, µg/kg wet weight | Young<br>*et al*. 1981 |
| <10 | sewage outfall | *Sebastes paucispinis*- muscle tissue<br>bocaccio, µg/kg wet weight | Young<br>*et al*. 1981 |
| <10 | clean (control) | *Sebastes paucispinis*- muscle tissue<br>bocaccio, µg/kg wet weight | Young<br>*et al*. 1981 |
| 6200 to 8000 | D/S of Pb/Ag mines | *Scapula undulata*<br>liverwort, µg/kg dry weight | Jones<br>*et al*. 1985 |
| 12 to 47 | U/S ambient <1ng/L | *Scapula undulata*<br>liverwort, µg/kg dry weight | Jones<br>*et al*. 1985 |
| 2.1 to 4.4 | clean | *Olivella biplicata*- all soft tissues<br>marine gastropod, µg/g dry weight | Nelson 1978 |
| 3.4 to 4.2 | clean | *Olivella biplicata*-shell<br>marine gastropod, µg/g dry weight | Nelson 1978 |
| 10.7 | dirty | *Olivella biplicata*- all soft tissues<br>marine gastropod, µg/g dry weight | Nelson 1978 |

| 3.5 | dirty | *Olivella biplicata*-shell marine gastropod, µg/g dry weight | Nelson 1978 |
|---|---|---|---|
| 2.4 | clean | *Pisaster brevispinus*-ray tissue sea star, µg/g dry weight | Nelson 1978 |
| 2.5 | dirty | *Pisaster brevispinus*-ray tissue sea star, µg/g dry weight | Nelson 1978 |
| 1.5 | clean | *Patella vulgata* -whole body limpet, µg/g dry weight | Greig *et al*. 1975 |
| 3.8 | clean | *Littorina littorea*-whole body winkle, µg/g dry weight | Greig *et al*. 1975 |
| 2.9 | unknown | *Haliotis rufescens*-whole body abalone, µg/g dry weight | Greig *et al*. 1975 |
| 1.4 | unknown | *Haliotis rufescens*-foot abalone, µg/g dry weight | Greig *et al*. 1975 |
| 7.0 | unknown | *Haliotis rufescens*-viscera including digestive gland abalone, µg/g dry weight | Greig *et al*. 1975 |
| baf | water source | species and tissues | sources |
| 8.1 | unknown | *Haliotis rufescens*-digestive gland abalone, µg/g dry weight | Greig *et al*. 1975 |
| 2.9 | unknown | *Haliotis rufescens*-whole body abalone, µg/g dry weight | Greig *et al*. 1975 |
| 1.2 | unknown | *Haliotis rufescens*-foot abalone, µg/g dry weight | Greig *et al*. 1975 |
| 7.7 | unknown | *Haliotis rufescens*-viscera including digestive gland abalone, µg/g dry weight | Greig *et al*. 1975 |
| 9.9 | unknown | *Haliotis rufescens*-digestive gland abalone, µg/g dry weight | Greig *et al*. 1975 |
| 0.28 | unknown | *Haliotis rufescens*-blood abalone, µg/g dry weight | Greig *et al*. 1975 |
| 0.35 | unknown | *Haliotis rufescens*-muscle | Greig |

| | | | |
|---|---|---|---|
| | | abalone, µg/g dry weight | *et al*. 1975 |
| 0.87 | unknown | *Haliotis rufescens*-gills<br>abalone, µg/g dry weight | Greig<br>*et al*. 1975 |
| 6.2 | unknown | *Haliotis rufescens*-kidney<br>abalone, µg/g dry weight | Greig<br>*et al*. 1975 |
| 0.08 | dirty | *Scophthalmus aquosus*-liver<br>windowpane flounder, µg/g wet weight | Greig<br>*et al*. 1983 |
| 0.14 | clean | *Mytilus edulis*-all soft tissues<br>mussel, µg/g dry weight | Gillfilan<br>*et al*. 1985 |
| 0.36 | dirty | *Mytilus edulis*-all soft tissues<br>mussel, µg/g dry weight | Gillfilan<br>*et al*. 1985 |
| <0.02 | clean | *Mercenaria mercenaria*-adductors<br>hard clam, 15 days, µg/g dry weight | Robinson<br>*et al*. 1985 |
| 0.8 ± 0.4 | clean | *Mercenaria mercenaria*-digestive gland<br>hard clam, 15 days, µg/g dry weight | Robinson<br>*et al*. 1985 |
| <0.4 | clean | *Mercenaria mercenaria*-gills<br>hard clam, 15 days, µg/g dry weight | Robinson<br>*et al*. 1985 |
| 7.6±3.7 | clean | *Mercenaria mercenaria*-kidneys<br>hard clam, 15 days, µg/g dry weight | Robinson<br>*et al*. 1985 |
| <0.2 | clean | *Mercenaria mercenaria*-mantle<br>hard clam, 15 days, µg/g dry weight | Robinson<br>*et al*. 1985 |
| baf | water source | species and tissues | sources |
| <0.2 | clean | *Mercenaria mercenaria*-viscera<br>hard clam, 15 days, µg/g dry weight | Robinson<br>*et al*. 1985 |
| <0.02 | dirty, 0.1 µg/L Cd present | *Mercenaria mercenaria*-adductors<br>hard clam, 15 days, µg/g dry | Robinson<br>*et al*. 1985 |

| | | weight | |
|---|---|---|---|
| 0.6 ± 0.2 | dirty, 0.1 µg/L Cd present | *Mercenaria mercenaria*-digestive gland<br>hard clam, 15 days, µg/g dry weight | Robinson *et al*. 1985 |
| <0.2 | dirty, 0.1 µg/L Cd present | *Mercenaria mercenaria*-gills<br>hard clam, 15 days, µg/g dry weight | Robinson *et al*. 1985 |
| 4.8 ± 2.5 | dirty, 0.1 µg/L Cd present | *Mercenaria mercenaria*-kidneys<br>hard clam, 15 days, µg/g dry weight | Robinson *et al*. 1985 |
| 0.2 ± 0.1 | dirty, 0.1 µg/L Cd present | *Mercenaria mercenaria*-mantle<br>hard clam, 15 days, µg/g dry weight | Robinson *et al*. 1985 |
| 0.2 ± 0.04 | dirty, 0.1 µg/L Cd present | *Mercenaria mercenaria*-viscera<br>hard clam, 15 days, µg/g dry weight | Robinson *et al*. 1985 |
| 0.26 ± 0.1 | clean | *Cancer irroratus*-flesh<br>rock crab, µg/g wet weight | Greig *et al*. 1977 |
| 2.6 ± 0.2 | clean | *Cancer irroratus*-digestive gland<br>rock crab, µg/g wet weight | Greig *et al*. 1977 |
| 0.80 ± 0.22 | clean | *Cancer irroratus*-gills<br>rock crab, µg/g wet weight | Greig *et al*. 1977 |
| 0.42 | ocean dumping site | *Cancer irroratus*-flesh<br>rock crab, µg/g wet weight | Greig *et al*. 1977 |
| 2.9 | ocean dumping site | *Cancer irroratus*-digestive gland<br>rock crab, µg/g wet weight | Greig *et al*. 1977 |
| 0.71 | ocean dumping site | *Cancer irroratus*-gills<br>rock crab, µg/g wet weight | Greig *et al*. 1977 |
| baf | water source | species and tissues | sources |
| <0.1 | clean | *Scophthalmus aquaosus* -flesh<br>windowpane flounder, µg/g wet | Greig *et al*. 1977 |

| | | weight | |
|---|---|---|---|
| <0.1 | clean | *Scophthalmus aquaosus* -liver windowpane flounder, µg/g wet weight | Greig *et al*. 1977 |
| <0.1 | ocean dumping site | *Scophthalmus aquaosus* -flesh windowpane flounder, µg/g wet weight | Greig *et al*. 1977 |
| <0.1 | ocean dumping site | *Scophthalmus aquaosus* -liver windowpane flounder, µg/g wet weight | Greig *et al*. 1977 |
| <0.1 | clean | *Spisula solidissima*-muscle surf clam, µg/g wet weight | Greig *et al*. 1977 |
| <0.2 | ocean dumping site | *Spisula solidissima*-muscle surf clam, µg/g wet weight | Greig *et al*. 1977 |
| <0.1 | clean | *Busycon canaliculatum*-muscle channeled whelk, µg/g wet weight | Greig *et al*. 1977 |
| 6.8 | clean | *Busycon canaliculatum*-digestive gland channeled whelk, µg/g wet weight | Greig *et al*. 1977 |
| <0.2 | ocean dumping site | *Busycon canaliculatum*-muscle channeled whelk, µg/g wet weight | Greig *et al*. 1977 |
| 13,5 | ocean dumping site | *Busycon canaliculatum*-digestive gland channeled whelk, µg/g wet weight | Greig *et al*. 1977 |
| 0.9 ± 0.7 | dirty | *Cancer irroratus*-gills rock crab, µg/g wet weight | Greig *et al*. 1982 |

No ambient levels of silver were given in the literature sources. The body burdens are shown as coming from contaminated or dirty, uncontaminated or clean or unknown water quality conditions.

table 7.2

acute effects of silver nitrate (µg/l) on marine organisms.

| $Ag^+$ | species | conditions | effects | sources |
|---|---|---|---|---|
| 4.7 | *Parlichthys dentatus* (summer flounder) | S, U, larvae | $LC_{50}$ | USEPA 1980a |
| 12.2 | *Crassostrea gigas* (american oyster) | static bioassay 20C, pH 8.2 salinity 22.7 | 48-h $LC_{50}$ | Sanders & Abbe 1989 |
| 32 | *Spisula solidissima* surf clam (embryo) | static bioassay 20C, pH 8 salinity 30 (development) | 48-h $LC_{100}$ | Eyster & Morse |
| 32.4 | *Mercenaria mercenaria* hard clam larvae | static bioassay lab seawater (growth) | 10-d $LC_{50}$ | Calabrese *et al*. |
| 55 ± 20.8 | *Cancer magister* (crab zoeae) | static bioassay pH 8.1 salinity 33.8 0/00 | 96-h $LC_{50}$ | Martin *et al.* 1981 |
| 100 | *Chlamys varia* (scallop) | seawater | 4.8-d TLm | Metayer *et al.* 1990 |
| 100 | (mussel) *Mytilus galloprovincialis* | seawater | 4.6-d TLm | Metayer *et al.* 1990 |
| 100 | *Crassostrea gigas* (american oyster) | seawater | 8.7-d TLm | Metayer *et al.* 1990 |
| 110 | *Menidia menidia* (silverside) | S, U, larvae | $LC_{50}$ | USEPA 1980a |
| 249 | *Mysidopsis bahia* mysid | flow through pH 8, 23C | 96-h $LC_{50}$ | Lussier *et al*. 1985 |
| 400 | *Menidia menidia* (silverside) | S, U, juveniles | $LC_{50}$ | USEPA 1980a |

| | | | | |
|---|---|---|---|---|
| >500 | *Tautogolabrus adspersus* (cunner) | salinity 24 ± 2 20C, pH 7.3-7.6 | 96-h $LC_{100}$< | Pereira & Kanungo 1981 |
| 500 | *Pseudopleuronectes americanus* (winter flounder) | S, U, larvae | $LC_{50}$ | USEPA 1980a |
| 550 | *Apoltes quadracus* (fourspine stickleback) | S, U, adults | $LC_{50}$ | USEPA 1980a |
| $Ag^+$ | species | conditions | effects | sources |
| 1000 | *Chlamys varia* (scallop) | seawater | <1-d TLm | Metayer *et al.* 1990 |
| 1000 | (mussel) *Mytilus galloprovincialis* | seawater | 3.3-d TLm | Metayer *et al.* 1990 |
| 1400 | *Cyprinodon varienotus* (sheepshead minnow) | S, M, juveniles | $LC_{50}$ | USEPA 1980a |
| 2000 | *Nassarius obsoletus* mud snail | static bioassay lab seawater 20C salinity 25 | 72-h $LC_{100}$ | MacInnes & Thurberg 1973 |

TLm is time to 50% mortality.

table 7.3

chronic and sub-lethal effects of silver on marine organisms.

| $Ag^+$ (µg/L) | species | conditions | effects | |
|---|---|---|---|---|
| 0.20 | *Perna perna* brown mussel | (filtering rate) | 1-h $EC_{50}$ | Watling & Watling |

| | | | | |
|---|---|---|---|---|
| 1.4 | *Chroomonas* | static bioassay 25C salinity 7.5 (yield) | $EC_{50}$ | Sanders & Abbe |
| 1.8 | *Chroomonas* | static bioassay 25C salinity 7.5 (growth) | $EC_{50}$ | Sanders & Abbe |
| 2.9 | *Prorocentrum mariae-lebouriae* | static bioassay 25C salinity 7.5 (reduced yield) | EC50 | Sanders & Abbe |
| 3.0 | *Mysidopsis bahia* mysid | flow through 23C, pH 8.0 | NOEL | Lussier *et al.* |
| 3.6 | *Chroomonas* | static bioassay 25C salinity 15 (yield) | $EC_{50}$ | Sanders & Abbe |
| 3.6 | *Chroomonas* | static bioassay 25C salinity 15 (growth) | $EC_{50}$ | Sanders & Abbe |
| 3.6 | *Chroomonas* | static bioassay 25C salinity 22.5 (growth) | $EC_{50}$ | Sanders & Abbe |
| 3.6 | *Prorocentrum mariae-lebouriae* | static bioassay 25C salinity 15 | $EC_{50}$ | Sanders & Abbe |

| | | (reduced yield) | | |
|---|---|---|---|---|
| 3.8 | *Skeletonema costatum* | static bioassay 25C salinity 7.5 (reduced yield) | $EC_{50}$ | Sanders & Abbe 1989 |
| $Ag^+$ (μg/L) | species | conditions | effects | |
| 4.1 | *Chroomonas* | static bioassay 25C salinity 22.5 (yield) | $EC_{50}$ | Sanders & Abbe |
| 4.2 | *Prorocentrum mariae-lebouriae* | static bioassay 25C salinity 22.5 (reduced yield) | $EC_{50}$ | Sanders & Abbe |
| 4.8 | *Thalassiosira pseudonana* | static bioassay 25C salinity 15 (reduced yield) | $EC_{50}$ | Sanders & Abbe |
| 5.2 | *Chroomonas* | static bioassay 25C salinity 22.5 (yield) | $EC_{50}$ | Sanders & Abbe |
| 5.3 | *Prorocentrum mariae-lebouriae* | static bioassay 25C salinity 30 (reduced yield) | $EC_{50}$ | Sanders & Abbe |

| | | | | |
|---|---|---|---|---|
| 5.8 | *Crassostrea virginica*<br>american oyster | static<br>bioassay<br>lab medium<br>26C<br>salinity 25<br>(development) | 48-h $EC_{50}$ | Calabrese<br>*et al.* |
| 5.9 | *Skeletonema costatum* | static<br>bioassay<br>25C<br>salinity 7.5<br>(growth) | $EC_{50}$ | Sanders<br>& Abbe |
| 6.7 | *Chroomonas* | static<br>bioassay<br>25C<br>salinity 30<br>(growth) | EC50 | Sander<br>& Abbe |
| 6.7 | *Prorocentrum mariae-lebouriae* | static<br>bioassay<br>25C<br>salinity 15<br>(growth) | $EC_{50}$ | Sanders<br>& Abbe<br>1989 |
| $Ag^{+}$<br>(μg/L) | species | conditions | effects | |
| 6.7 | *Prorocentrum mariae-lebouriae* | static<br>bioassay<br>25C<br>salinity 22.5<br>(growth) | $EC_{50}$ | Sanders<br>& Abbe |
| 8.1 | *Thalassiosira pseudonana* | static<br>bioassay<br>25C<br>salinity 30<br>(reduced<br>yield) | $EC_{50}$ | Sanders<br>& Abbe |
| 8.2 | *Prorocentrum mariae-lebouriae* | static<br>bioassay<br>25C<br>salinity 30<br>(growth) | $EC_{50}$ | Sanders<br>& Abbe |

| | | | | |
|---|---|---|---|---|
| 9.5 | *Spisula solidissima* surf clam (embryo) | static bioassay 20C, pH 8 salinity 30 (development) | 48-h NOEL | Eyster & Morse |
| 9.5 | *Spisula solidissima* surf clam (embryo) | static bioassay 20C, pH 8 salinity 30 (development) | 48-h $EC_{50}$ | Eyster & Morse |
| 10 | *Thalassiosira pseudonana* | static bioassay 25C salinity 22.5 (reduced yield) | $EC_{50}$ | Sanders & Abbe |
| 10 | *Thalassiosira pseudonana* | static bioassay 25C salinity 7.5 (growth) | $EC_{50}$ | Sanders & Abbe |
| 11 | *Mysidopsis bahia* mysid | flow through 23C, pH 8.0 (offspring) | 38-day effect level | Lussier *et al*. |
| 11 | *Mysidopsis bahia* mysid | flow through 23C, pH 8.0 (offspring) | 38-day NOEL | Lussier *et al*. 1985 |
| $Ag^+$ (µg/L) | species | conditions | effects | |
| 12 | *Skeletonema costatum* | static bioassay 25C salinity 30 (reduced yield) | $EC_{50}$ | Sanders & Abbe |

| 13.2 | *Skeletonema costatum* | static bioassay<br>25C<br>salinity 22.5<br>(reduced yield) | $EC_{50}$ | Sanders & Abbe |
|---|---|---|---|---|
| 14.5 | *Thalassiosira pseudonana* | static bioassay<br>25C<br>salinity 15<br>(growth | $EC_{50}$ | Sanders & Abbe |
| 15.4 | *Skeletonema costatum* | static bioassay<br>25C<br>salinity 15<br>(growth) | $EC_{50}$ | Sanders & Abbe |
| 14±2 | *Mytilus edulis* mussel embryos | Static bioassay<br>salinity 33.8<br>pH 8.1<br>(development) | 48-h $EC_{50}$ | Martin *et al.* |
| 16 | *Spisula solidissima* surf clam (embryo) | static bioassay<br>20C, pH 8<br>salinity 30<br>(development) | 48-h $EC_{50}$ | Eyster & Morse |
| 16 | *Spisula solidissima* surf clam (embryo) | static bioassay<br>20C, pH 8<br>salinity 30<br>(development) | 48-h $LC_{100}$ | Eyster & Morse |
| 16-18 | *Crassostrea gigas* oyster embryo | static bioassay<br>seawater<br>salinity 33<br>20C, pH 8.2<br>(development) | 48-h $EC_{50}$ | Coglianese & Martin |
| 19.8 | *Thalassiosira pseudonana* | static bioassay | $EC_{50}$ | |

| | | 25C<br>salinity 22.5<br>(growth) | | Sanders & Abbe 1989 |
|---|---|---|---|---|
| $Ag^+$ (µg/L) | species | conditions | effects | |
| 20 | *Chlamys varia* scallop | (detachment) | 7-d $EC_{50}$ | Metayer *et al.* |
| >20 | *Skeletonema costatum* | static bioassay<br>25C<br>salinity 22.5<br>(growth) | $EC_{50}$ | Sanders & Abbe |
| >20 | *Skeletonema costatum* | static bioassay<br>25C<br>salinity 30<br>(growth) | $EC_{50}$ | Sanders & Abbe |
| >20 | *Thalassiosira pseudonana* | static bioassay<br>25C<br>salinity 30<br>(growth) | $EC_{50}$ | Sanders & Abbe |
| 21.0 | *Mercenaria mercenaria* hard clam | static bioassay<br>lab medium<br>26C<br>salinity 25<br>(development) | 48-h $EC_{50}$ | Calabrese *et al.* |
| 22.50 | *Crepidula fornicata* slipper shell | | 12-d $EC_{50}$ | Nelson *et al.* |
| 24.2 | *Crassostrea virginica* american oyster | static bioassay<br>20C | 48-h $EC_{50}$ | MacInnes |

| | | salinity 26<br>seawater | | & Calabrese |
|---|---|---|---|---|
| 25.0 | *Crassostrea virginica*<br>oyster larvae | static bioassay<br>seawater<br>24C<br>salinity 25<br>(growth) | 12-d $EC_{50}$ | Calabrese<br>*et al.* |
| 32 | *Mysidopsis bahia*<br>mysid | flow through<br>23C, pH 8.0<br>(survival and # of offspring ) | 38-day effect level | Lussier<br>*et al.*<br>1985 |
| $Ag^+$ (µg/L) | species | conditions | effects | |
| 32.2 | *Crassostrea virginica*<br>american oyster | static bioassay<br>30C<br>salinity 26<br>seawater | 48-h $EC_{50}$ | MacInnes<br>& Calabrese |
| 35.3 | *Crassostrea virginica*<br>american oyster | static bioassay<br>25C<br>salinity 26<br>seawater | 48-h $EC_{50}$ | MacInnes<br>& Calabrese |
| 70.5 | *Pseudopleuronectes americanus*<br>winter flounder | flow through<br>3-5C<br>salinity 27-32<br>(chronic effect) | 18-d | Klein-Macphee<br>*et al.* |
| 108 | *Mysidopsis bahia*<br>mysid | flow through<br>23C, pH 8.0<br>(survival and no. of offspring ) | 38-day effect level | Lussier<br>*et al.* |
| 250 | *Nassarius obsoletus*<br>mud snail | static bioassay<br>lab seawater | 72-h LOEL | MacInnes |

|  |  |  |  |  |
| --- | --- | --- | --- | --- |
|  |  | 20C<br>salinity 25<br>($O_2$ uptake) |  | & Thurberg |
| 500 | *Nassarius obsoletus*<br>mud snail | static<br>bioassay<br>lab seawater<br>20C<br>salinity 25<br>($O_2$ uptake) | 72-h $EC_{50}$ | MacInnes<br>& Thurberg<br>1973 |

table 7.4

acute effects of silver on freshwater organisms.

| µg /L $Ag^+$ | species | conditions | effects | sources |
| --- | --- | --- | --- | --- |
| 0.39-2.9 | *Daphnia magna* | flow through<br>hardness 40-49 | 96-h $LC_{50}$ | Lemke<br>1981 |
| 0.6 | *Daphnia magna* | static/renewal<br>hardness 40<br>alkalinity 31<br>pH 7.2, no food | 48-h $EC_{50}$ | Nebeker<br>*et al*.<br>1983 |
| 0.6 | *Daphnia magna* | hardness 38<br>20C, no food | 48-h $EC_{50}$ | Nebeker<br>*et al*.<br>1983 |
| 0.64 | *Daphnia magna*<br>cladoceran | static<br>bioassay | 48-h $EC_{50}$ | Lemke<br>1981 |
| 0.7 | *Daphnia magna* | static<br>bioassay<br>pH 7.7<br>alkalinity 40<br>hardness 46 | 48-h $EC_{50}$ | Nebeker<br>1982 |
| 0.9 * | *Daphnia magna* | flow through<br>hardness 45 | 48-h $EC_{50}$ | Holcombe<br>*et al*. 1987 |
| 0.9 | *Daphnia magna* | flow through | 48-h | Khangarot |

| | | 17C, pH 7.39<br>hardness 44.7<br>alkalinity 43 | $EC_{50}$ | & Ray<br>1987 |
|---|---|---|---|---|
| 0.9 | *Daphnia magna* | static<br>bioassay<br>pH 7.2<br>alkalinity 49<br>hardness 60 | 48-h<br>$EC_{50}$ | Nebeker<br>1982 |
| 1.0 | *Daphnia magna* | static<br>bioassay<br>pH 7.4<br>alkalinity 50<br>hardness 46 | 48-h<br>$EC_{50}$ | Nebeker<br>1982 |
| 1.1 | *Daphnia magna*<br>cladoceran | static<br>bioassay | 48-h<br>$EC_{50}$ | Lemke<br>1981 |
| 1.1 | *Daphnia magna* | static/renewal<br>hardness 38<br>alkalinity 31<br>pH 7.5, no<br>food | 48-h<br>$EC_{50}$ | Nebeker<br>*et al*.<br>1983 |
| 1.1 | *Daphnia magna* | static/renewal<br>hardness 33<br>alkalinity 27<br>pH 7.5, no<br>food | 48-h<br>$EC_{50}$ | Nebeker<br>*et al*.1983 |
| 1.1 | *Daphnia magna*<br>cladoceran | hardness 40<br>20C, no food | 48-h<br>$EC_{50}$ | Nebeker<br>1983 |
| 1.1 | *Daphnia magna*<br>cladoceran | hardness 40<br>20C, with food | 48-h<br>$EC_{50}$ | Nebeker<br>1983 |
| 1.4 | *CerioDaphnia*<br>*reticulata*<br>cladoceran | static<br>bioassay<br>hardness 240 | 48-h<br>$EC_{50}$ | Einabarawy<br>*et al*.<br>1986 |
| 1.9 | *Daphnia pulex*<br>cladoceran | static<br>bioassay<br>hardness 45 | 48-h<br>$EC_{50}$ | Einabarawy<br>*et al*. 1986 |
| 2.2 | *Daphnia magna*<br>cladoceran | static<br>bioassay<br>hardness 54 | 48-h<br>$EC_{50}$ | Lemke<br>1981 |

| | | | | |
|---|---|---|---|---|
| 2.2 | *Leptophlebia* mayfly nymph | static bioassay hardness 47 | 96-h $EC_{50}$ | Brooke *et al.* 1986 |
| 3.9 | *Pimephales promelas* fathead minnow | flow through hardness 33 | 96-h $LC_{50}$ | Goettl & Davis 1978 |
| 3.9-12 | *Pimephales promelas* fathead minnow | flow through hardness 40-49 | 96-h $LC_{50}$ | Lemke 1981 |
| 4.1 | *Bufo melanostictus* Indian toad | static bioassay 31C, pH 7.4 hardness 185 | 96-h $LC_{50}$ | Khangarot & Ray 1987b |
| 4.2 | *Lymnaea luteola* snail | static bioassay 32C, pH 7.4 hardness 195 | 96-h $LC_{50}$ | Khangarot & Ray 1988a |
| 4.5 | *Gammarus pseudolimnaeus* scud | flow through 19.9C, pH 7.4 hardness 44 | 72-h $LC_{50}$ | Lima *et al.* 1982 |
| 4.5 | *Gammarus pseudolimnaeus* scud | flow through 19.9C, pH 7.4 hardness 44 | 96-h $LC_{50}$ | Lima *et al.* 1982 |
| 4.5 | *Gamaurus pseudolimnaeus* amphipod | flow through hardness 48 | 96 h $EC_{50}$ | Lima *et al.* 1982 |
| 4.7 | *Gammarus pseudolimnaeus* scud | flow through 19.9C, pH 7.4 hardness 44 | 24-h $LC_{50}$ | Lima *et al.* 1982 |
| 4.7 | *Gammarus pseudolimnaeus* scud | flow through 19.9C, pH 7.4 hardness 44 | 48-h $LC_{50}$ | Lima *et al.* 1982 |
| 4.8 | *Pimephales promelas* fathead minnow | flow through hardness 274 | 96-h $LC_{50}$ | Goettl & Davies 1978 |
| 4.9 | *Rhinichthys osculus* | flow through hardness 250 | 96-h $LC_{50}$ | Goettl & Davies |

|  |  |  |  |  |
|---|---|---|---|---|
|  | speckled dace |  |  | 1978 |
| 5.0 | *Crangonyx pseudogracilis* amphipoda | static bioassay 13C, pH 6.7-6.8 hardness 45-55 | 96-h $LC_{50}$ | Martin & Holdich 1986 |
| 5.3 | *Cottus bairdi* mottled sculpin | flow through hardness 350 | 96-h $LC_{50}$ | Goettl & Davies 1978 |
| 5.3 * | *Oncorhynchus mykiss* rainbow trout | flow through hardness 31 | 96-h $LC_{50}$ | Davies & Goettl 1978 |
| 5.6 | *Pimephales promelas* fathead minnow | flow through 22C, hardness 40 | 96-h $LC_{50}$ | Nebeker *et al.* 1983 |
| 6.0 | *Crangonyx pseudogracilis* amphipoda | static bioassay 13C, pH 6.7-6.8 hardness 45-55 | 48-h $LC_{50}$ | Martin & Holdich 1986 |
| 6 | *Oncorhynchus mykiss* rainbow trout | flow through 17C, pH 7.39 hardness 44.7 alkalinity 43 | 96-h $LC_{50}$ | Holcombe *et al.* 1987 |
| 6.2 * | *Oncorhynchus mykiss* rainbow trout | flow through hardness 20 | 96-h $LC_{50}$ | Davies & Goettle 1978 |
| 6.2 | *Bufo melanostictus* Indian toad | static bioassay 31C, pH 7.4 hardness 185 | 48-h $LC_{50}$ | Khangarot & Ray 1987 |
| 6.4 | *Lymnaea luteola* snail | static bioassay 32C, pH 7.4 hardness 195 | 72-h $LC_{50}$ | Khangarot & Ray 1988a |
| 6.4 | *Puntius sophore* | static | 96-h | Khangarot |

| | | | | |
|---|---|---|---|---|
| | | bioassay<br>30C, pH 7.3<br>hardness 250 | $LC_{50}$ | & Ray<br>1988b |
| 6.5 | *Oncorhynchus mykiss*<br>rainbow trout | flow through<br>hardness 26 | 96-h<br>$LC_{50}$ | Davies & Goettl<br>1978 |
| 6.7 | *Thymallus arcticus*<br>arctic grayling<br>alevin | static<br>bioassay<br>hardness 41<br>conductivity<br>156<br>pH 7.1 to 8.0<br>not fed,<br>DO>35% | 96-h<br>$LC_{50}$ | Buhl &<br>Hamilton<br>1991 |
| 6.7 | *Pimephales promelas*<br>fathead minnow | flow through<br>hardness 44 | 96-h<br>$LC_{50}$ | Holcombe<br>*et al*. 1983 |
| 7.3 | *Bufo melanostictus*<br>Indian toad | static<br>bioassay<br>31C, pH 7.4<br>hardness 185 | 24-h<br>$LC_{50}$ | Khangarot<br>& Ray<br>1987b |
| 7.4 | *Pimephales promelas*<br>fathead minnow | flow through<br>22C,<br>hardness 36 | 96-h<br>$LC_{50}$ | Nebeker<br>*et al*.<br>1983 |
| 7.6 | *Channa punctatus* | static<br>bioassay<br>30C, pH 7.3<br>hardness 250 | 96-h<br>$LC_{50}$ | Lemke<br>1981 |
| 8.1* | *Oncorhynchus mykiss*<br>rainbow trout | flow through<br>hardness 26 | 96-h<br>$LC_{50}$ | Davies &<br>Goettl<br>1978 |
| 8.5 | *Oncorhynchus mykiss*<br>rainbow trout | static<br>bioassay<br>12C,<br>hardness 35 | 96-h<br>$LC_{50}$ | Nebeker<br>*et al*.<br>1983 |
| 8.6 | *Oncorhynchus mykiss*<br>rainbow trout | flow through<br>12C,<br>hardness 29 | 96-h<br>$LC_{50}$ | Nebeker<br>*et al*.<br>1983 |

| 9.0 | *Daphnia magna*<br>cladoceran | flow through<br>17C, pH 7.4<br>hardness 45 | 48-h<br>$EC_{50}$ | Holcombe<br>*et al*.<br>1987 |
|---|---|---|---|---|
| µg /L<br>$Ag^+$ | species | conditions | effects | sources |
| 9.0 * | *Pimephales promelas*<br>fathead minnow | flow through<br>17C, pH 7.4<br>hardness 45 | 96-h<br>$LC_{50}$ | Holcombe<br>*et al*.<br>1987 |
| 9.2 | *Jordanella floridae*<br>flagfish | flow through<br>19.9C, pH 7.4<br>hardness 44 | 96-h<br>$LC_{50}$ | Lima<br>*et al*.<br>1982 |
| 9.2 | *Lymnaea luteola*<br>snail | static bioassay<br>32C, pH 7.4<br>hardness 195 | 48-h<br>$LC_{50}$ | Khangarot<br>& Ray<br>1988a |
| 9.2 | *Oncorhynchus mykiss*<br>steelhead trout | flow through<br>12C,<br>hardness 36 | 96-h<br>$LC_{50}$ | Nebeker<br>*et al*.<br>1983 |
| 9.4 | *Pimephales promelas*<br>fathead minnow | static bioassay<br>20C,<br>hardness 38 | 96-h<br>$LC_{50}$ | Nebeker<br>*et al.*<br>1983 |
| 9.7 | *Oncorhynchus mykiss*<br>rainbow trout | flow through<br>12C,<br>hardness 42 | 96-h<br>$LC_{50}$ | Nebeker<br>*et al*.<br>1983 |
| 9.7 | *Pimephales promelas*<br>fathead minnow | static bioassay<br>21C,<br>hardness 39 | 96-h<br>$LC_{50}$ | Nebeker<br>*et al*.<br>1983 |
| 9.7 | *Channa punctatus* | static bioassay<br>30C, pH 7.3<br>hardness 250 | 72-h<br>$LC_{50}$ | Lemke<br>1981 |
| 10 | *Daphnia magna* | static bioassay<br>13C, pH 7.6<br>hardness 240 | 48-h<br>$EC_{50}$ | Khangarot<br>& Ray<br>1987a |

| | | | | |
|---|---|---|---|---|
| 11 | *CerioDaphnia reticulata*<br>cladoceran | static<br>bioassay<br>hardness 45 | 48-h<br>$EC_{50}$ | Mount &<br>Norberg<br>1984 |
| 11 | *Pimephales promelas*<br>fathead minnow | flow through<br>24.7C, pH 7.4<br>hardness 44 | 72-h<br>$LC_{50}$ | Lima<br>*et al*.<br>1982 |
| 11 | *Pimephales promelas*<br>fathead minnow | flow through<br>24.7C, pH 7.4<br>hardness 46 | 96-h<br>$LC_{50}$ | Lima<br>*et al*.<br>1982 |
| 11 | *Oncorhynchus mykiss*<br>rainbow trout | static<br>bioassay<br>12C,<br>hardness 26 | 96-h<br>$LC_{50}$ | Nebeker<br>*et al.*<br>1983 |
| 11.1 | *Thymallus arcticus*<br>arctic grayling<br>juvenile | static<br>bioassay<br>hardness 41<br>conductivity<br>156<br>pH 7.1 to 8.0<br>not fed,<br>DO>35% | 96-h<br>$LC_{50}$ | Buhl &<br>Hamilton<br>1991 |
| 11.1 | *Oncorhynchus kisutch*<br>coho salmon<br>alevin | static<br>bioassay<br>hardness 41<br>conductivity<br>156<br>pH 7.1 to 8.0<br>not fed,<br>DO>35% | 96-h<br>$LC_{50}$ | Buhl &<br>Hamilton<br>1991 |
| 12 | *Pimephales promelas*<br>fathead minnow | flow through<br>24.7C, pH 7.4<br>hardness 44 | 48-h<br>$LC_{50}$ | Lima<br>*et al*.<br>1982 |
| 12 | *Daphnia magna* | static<br>bioassay<br>pH 8.<br>alkalinity 84<br>hardness 73 | 48-h<br>$EC_{50}$ | Nebeker<br>1982 |
| 12.5 | *Oncorhynchus* | static | 96-h | Buhl & |

| | | | | |
|---|---|---|---|---|
| | *kisutch* coho salmon juvenile | bioassay hardness 41 conductivity 156 pH 7.1 to 8.0 not fed, DO>35% | $LC_{50}$ | Hamilton 1991 |
| 12.5 | *Daphnia magna* | static/renewal H=33, a=27, pH 7.2, with food | 48-h $EC_{50}$ | Nebeker *et al.* 1983 |
| 12.7 | *Bufo melanostictus* Indian toad | static bioassay 31C, pH 7.4 hardness 185 | 12-h $LC_{50}$ | Khangarot & Ray 1987b |
| 13 | *Lepomis macrochirus* | flow through 17C, pH 7.39 hardness 44.7 alkalinity 43 | 96-h $LC_{50}$ | Holcombe *et al.* 1987 |
| 13 * | *Oncorhynchus mykiss* rainbow | flow through hardness 250 | 96-h $LC_{50}$ | Davies & Goettl 1978 |
| 14 | *Rhinichthys osculus* speckled dace | flow through hardness 250 | 96-h $LC_{50}$ | Goettl & Davies 1978 |
| 14 | *Cottus bairdi* mottled sculpin | flow through hardness 350 | 96-h $LC_{50}$ | Goettl & Davies 1978 |
| 14 | *Channa punctatus* | static bioassay 30C, pH 7.3 hardness 250 | 48-h $LC_{50}$ | Lemke 1981 |
| 14 | *Daphnia pulex* cladoceran | static bioassay hardness 45 | 48-h $EC_{50}$ | Mount & Norberg 1984 |
| 14 | *Pimephales promelas* fathead minnow | static bioassay hardness 44.7 | 96-h $LC_{50}$ | Holcombe *et al.* 1987 |

| | | | | |
|---|---|---|---|---|
| 15 | *Simocephalus vetulus*<br>cladoceran | static bioassay<br>hardness 45 | 48-h $EC_{50}$ | Mount & Norberg 1984 |
| 15 | *Pimephales promelas*<br>fathead minnow | flow through<br>24.7C, pH 7.4<br>hardness 44 | 24-h $LC_{50}$ | Lima *et al*. 1982 |
| 15.8 | *Jordanella floridae*<br>flagfish | flow through<br>24.7C, pH 7.4<br>hardness 44 | 72-h $LC_{50}$ | Lima *et al.* 1982 |
| 16.0 | *Pimephales promelas*<br>fathead minnow | hardness 29 to 38 | 48-h $LC_{50}$ | Bionomics 1979 |
| 16.0 | *Pimephales promelas*<br>fathead minnow | hardness 29 to 38 | 72-h $LC_{50}$ | Bionomics 1979 |
| 16.0 | *Pimephales promelas*<br>fathead minnow | flow through<br>hardness 38 | 96-h $LC_{50}$ | Le Blanc *et al*. 1984 |
| 16.1 | *Oncorhynchus mykiss*<br>rainbow trout alevin | static bioassay<br>hardness 41<br>conductivity 156<br>pH 7.1 to 8.0<br>not fed, DO>35% | 96-h $LC_{50}$ | Buhl & Hamilton 1991 |
| 17.3 | *Ictalurus punctatus*<br>channel catfish | flow through<br>hardness 44 | 96-h $LC_{50}$ | Holcombe *et al*. 1983 |
| 18.8 | *Channa punctatus* | static bioassay<br>30C, pH 7.3<br>hardness 250 | 24-h $LC_{50}$ | Lemke 1981 |
| 19 | *Lebistes reticulatus*<br>guppy | static bioassay<br>30C, pH 7.3<br>hardness 250 | 96-h $LC_{50}$ | Khangarot & Ray 1988b |
| 19.2 | *Oncorhynchus* | static | 96-h | Buhl & |

| | | | | |
|---|---|---|---|---|
| | *mykiss*<br>rainbow trout<br>juvenile | bioassay<br>hardness 41<br>conductivity 156<br>pH 7.1 to 8.0<br>not fed,<br>DO>35% | $LC_{50}$ | Hamilton<br>1991 |
| 21 | *Lebistes reticulatus*<br>guppy | static bioassay<br>30C, pH 7.3<br>hardness 250 | 72-h<br>$LC_{50}$ | Khangarot & Ray<br>1988b |
| 21.0 | *Pimephales promelas*<br>fathead minnow | hardness 29 to 38 | 24-h<br>$LC_{50}$ | Bionomics<br>1979 |
| 23.0 | *Daphnia magna* | static bioassay<br>13C, pH 7.6<br>hardness 240 | 24-h<br>$EC_{50}$ | Khangarot & Ray<br>1987a |
| 26.0 | *Hydra* | static bioassay<br>hardness 47 | 96-h<br>$EC_{50}$ | Brooke<br>*et al.* 1986 |
| 29 | *Oncorhynchus mykiss*<br>rainbow trout | flow through<br>pH 6.4-8.3 | 96-h<br>$TL_{50}$ | Hale<br>1977 |
| 29 * | *Nephelopsis obscura*<br>leech | flow through<br>17C, pH 7.39<br>hardness 44.7<br>alkalinity 43 | 96-h<br>$LC_{50}$ | Holcombe<br>*et al.*<br>1987 |
| 30 | *Lebistes reticulatus*<br>guppy | static bioassay<br>30C, pH 7.3<br>hardness 250 | 48-h<br>$LC_{50}$ | Khangarot & Ray<br>1988b |
| 31 | *Tubifex tubifex* | static bioassay<br>30C, pH 7.6<br>hardness 245 | 96-h<br>$LC_{50}$ | Khangarot<br>1991 |
| 39 | *Lebistes reticulatus* | static bioassay | 24-h<br>$LC_{50}$ | Khangarot & Ray |

| | guppy | 30C, pH 7.3<br>hardness 250 | | 1988b |
|---|---|---|---|---|
| 39 | *Tubifex tubifex* | static<br>bioassay<br>30C, pH 7.6<br>hardness 245 | 48-h<br>$EC_{50}$ | Khangarot<br>1991 |
| 41 | *Tubifex tubifex* | static<br>bioassay<br>30C, pH 7.6<br>hardness 245 | 24-h<br>$EC_{50}$ | Khangarot<br>1991 |
| 44 | *Channa punctatus* | static<br>bioassay<br>30C, pH 7.3<br>hardness 250 | 12-h<br>$LC_{50}$ | Lemke<br>1981 |
| 44 | *Jordanella floridae*<br>(flagfish) | flow through<br>24.7C, pH 7.4<br>hardness 44 | 24-h<br>$LC_{50}$ | Lima<br>*et al*.<br>1982 |
| 52 | *Daphnia magna* | static<br>bioassay<br>pH 7.8<br>alkalinity 368<br>hardness 255 | 48-h<br>$EC_{50}$ | Nebeker<br>1982 |
| 53 | *Nephelopsis obscura*<br>leech | static<br>bioassay<br>hardness 47 | 96-h<br>$EC_{50}$ | Brooke<br>*et al*.<br>1986 |
| 57 | *Puntius sophore* | static<br>bioassay<br>30C, pH 7.3<br>hardness 250 | 12-h<br>$LC_{50}$ | Khangarot<br>& Ray<br>1988b |
| 62 | *Lebistes reticulatus*<br>guppy | static<br>bioassay<br>30C, pH 7.3<br>hardness 250 | 12-h<br>$LC_{50}$ | Khangarot<br>& Ray<br>1988b |

| μg /L $Ag^+$ | species | conditions | effects | sources |
|---|---|---|---|---|
| 73 | *Oncorhynchus mykiss*<br>rainbow trout | static<br>bioassay<br>10C, | 96-h<br>$LC_{50}$ | Nebeker<br>*et al* .<br>1983 |

| | | | | |
|---|---|---|---|---|
| | | hardness 40 | | |
| 83 * | *Aplexa hypnorum*<br>snail | flow through<br>hardness 45 | 96-h<br>$EC_{50}$ | Holcombe<br>*et al.*<br>1987 |
| 84 | *Oncorhynchus mykiss*<br>rainbow trout | static bioassay<br>9C, hardness 37 | 96-h<br>$LC_{50}$ | Nebeker<br>*et al.*<br>1983 |
| 110-270 | *Pimephales promelas*<br>fathead minnow | flow through<br>hard water | 96-h<br>$LC_{50}$ | Bryan &<br>Langston<br>1992. |
| 241 | *Aplexa hypnorum*<br>snail | static renewal<br>hardness 50 | 96-h<br>$EC_{50}$ | Holcombe<br>*et al.*<br>1983 |
| 420 * | *Tanytarsus dissimilis*<br>midge | flow through<br>17C, pH 7.39<br>hardness 44.7<br>alkalinity 43 | 48-h<br>$LC_{50}$ | Holcombe<br>*et al.*<br>1987 |
| 560 * | *Orconectes immunis*<br>crayfish | flow through<br>17C, pH 7.39<br>hardness 44.7<br>alkalinity 43 | 96-h<br>$LC_{50}$ | Holcombe<br>*et al.*<br>1987 |
| 3160 | *Tanytarsus dissimilis*<br>midge | flow through<br>20C, pH 7.4<br>hardness 44 | 48-h<br>$LC_{50}$ | Lima<br>*et al.*<br>1982 |
| 5030 | *Tanytarsus dissimilis*<br>midge | flow through<br>20C, pH 7.4<br>hardness 44 | 24-h<br>$LC_{50}$ | Lima<br>*et al.*<br>1982 |
| 6300±<br>500 | *Saccharomyces cerevisiae*<br>baker's yeast | static bioassay<br>deionized water<br>30C<br>(respiration) | 60 min<br>$EC_{50}$ | Bitton<br>*et al.*<br>1984 |

Unless otherwise specified the silver salt used is $AgNO_3$. * salt not specified.

table 7.5

chronic effects of silver on freshwater organisms.

| µg/L $Ag^+$ | species | conditions | effects | sources |
|---|---|---|---|---|
| 0.03 to 0.06 as AgI | *Oncorhynchus mykiss* eyed-eggs | flow through 15.9C, pH 6.5-6.8 hardness 12.2 | 13 mo NOEL | Davies & Goettl 1978 |
| <0.1 as $AgNO_3$ | *Onycorhynchus mykiss* steelhead trout | | 60 day MATC | Davies & Goettl 1978 |
| 0.1 as $AgNO_3$ | *Onycorhynchus mykiss* steelhead trout | reduction | 60 day growth effects | Davies & Goettl 1978 |
| 0.5 as $AgNO_3$ | *Onycorhynchus mykiss* steelhead trout | survival | 60 day fewer living | Davies & Goettl 1978 |
| 0.18 to 0.40 as AgI | *Oncorhynchus mykiss* green eggs | flow through 15.9C, pH 6.5-6.8 hardness 12.2 | 10 mo NOEL | Davies & Goettl 1978 |
| 0.88 as AgI | *Oncorhynchus mykiss* rainbow trout fry | flow through 15.9C, pH 6.5-6.8 hardness 12.2 94% mortality, reduced growth and development | 6 week effect level | Davies & Goettl 1978 |
| <1.0 as $AgNO_3$ | *Ephemerella grandis* mayfly | flow through 3-9C, pH 7-7.2 hardness 30-70 | 14 day $TL_{50}$ | Nehring 1976 |
| 1.3 as $AgNO_3$ | *Onycorhynchus mykiss* steelhead trout | survival | $LC_{100}$ | Davies & Goettl 1978 |
| 1.6 | *Daphnia magna* | static renewal 20C, pH 7.2 alkalinity 49 | 21-day NOEL young | Nebeker 1982 |

| | | hardness 60 | per female per day | |
|---|---|---|---|---|
| 1.6 as $AgNO_3$ | *Daphnia magna* | hardness 60 alkalinity 49 pH 7.2 | 21 day MATC | Nebeker *et al.* 1983 |
| μg/L $Ag^+$ | species | conditions | effects | sources |
| 2.9 as $AgNO_3$ | *Daphnia magna* | hardness 60 alkalinity 49 pH 7.2 | 21 day $EC_{50}$ | Nebeker *et al.* 1983 |
| 3.4 as $AgNO_3$ | *Daphnia magna* | hardness 180 alkalinity 49 pH 7.2 | 21 day MATC | Nebeker *et al.* 1983 |
| 3.6 | *Daphnia magna* | static renewal 20C, pH 7.2 alkalinity 49, hardness 60 | 21-day $EC_{50}$ | Nebeker 1982 |
| 3.6 as $AgNO_3$ | *Daphnia magna* | hardness 75 alkalinity 49 pH 7.2 | 21 day $EC_{50}$ | Nebeker *et al.* 1983 |
| 3.9 | *Daphnia magna* | static renewal 20C, pH 7.2 alkalinity 49 hardness 60 | 21-day $EC_{50}$ | Nebeker 1982 |
| 3.9 as $AgNO_3$ | *Daphnia magna* | hardness 180 alkalinity 49 pH 7.2 | 21 day $EC_{50}$ | Nebeker *et al.* 1983 |
| 4.0 to 9.0 as $AgNO_3$ | *Pteronarcys californica* stonefly | flow through 3-9C, pH 7-7.2 hardness 30-70 | 14 day $TL_{50}$ | Nehring 1976 |
| 4.1 as $AgNO_3$ | *Daphnia magna* | hardness 60 alkalinity 49 pH 7.2 | young per female per day | Nebeker *et al.* 1983 |
| 8.0 as $AgNO_3$ | *Daphnia magna* | hardness 180 alkalinity 49 | percent living | Nebeker *et al.* |

| | | | | |
|---|---|---|---|---|
| | | pH 7.2 | and young per female per day | 1983 |
| μg/L $Ag^+$ | species | conditions | effects | sources |
| 8.8 | *Daphnia magna* | static renewal 20C, pH 7.2 alkalinity 49 hardness 60 | 21-day NOEL young per female per day | Nebeker 1982 |
| 8.8 as $AgNO_3$ | *Daphnia magna* | hardness 75 alkalinity 49 pH 7.2 | 21 day MATC | Nebeker *et al.* 1983 |
| 10.5 | *Daphnia magna* | static renewal 20C, pH 8.6 alkalinity 84 hardness 73 | 21-day NOEL young per female per day | Nebeker 1982 |
| 19.4 as $AgNO_3$ | *Daphnia magna* | hardness 75 alkalinity 49 pH 7.2 | percent living and young per female per day | Nebeker *et al.* 1983 |
| 20.0 | *Daphnia magna* | static renewal 20C, pH 7.2 alkalinity 49 hardness 60 | 21-day NOEL young per female per day | Nebeker 1982 |
| 270.0 as $AgNO_3$ | *Lemna minor* duckweed | static bioassay growing plant | 28 day $EC_{50}$ percent damage | Brown & Rattigan 1979 |

| $\mu g/L$ $Ag^+$ | species | conditions | effects | sources |
|---|---|---|---|---|
| 1000 | *Lemna paucicostata* duckweed | static bioassay 25±1C | 12 day $EC_{50}$ for frond growth | Nasu & Kugimoto 1981 |
| 7500 as $AgNO_3$ | *Elodea canadensis* | static bioassay growing plant | 28 day $EC_{50}$ for percent damage | Brown & Rattigan 1979 |

table 7.6

effects of acclimatization on silver toxicity in water of hardness 141 to *Pimephales promelas*, the fathead minnow.

| acclimation method | $Ic_{50}$ (µg Ag/L) |
|---|---|
| non | 30.0 |
| non | 36.6 |
| non | 37.2 |
| to 1.5 µg/L Ag for 7 days | 42.6 |
| to 1.5 µg/L Ag for 14 days | 41.3 |
| to 1.5 µg/L Ag for 14 days then de-acclimated for 7 days | 24.5 |
| to 15 µg/L Ag for 7 days | 45.6 |
| to 15 µg/L Ag for 14 days | 46.4 |
| to 15 µg/L Ag for 14 days then de-acclimated for 7 days | 29.1 |
| to 15 µg/L Ag for 14 days then de-acclinated for 14 days | 31.0 |

Birge *et al.* 1984.

table 7.7

comparison of the acute and chronic toxicity (to embryo/larvae) of different silver salts to the fathead minnow, *Pimephales promelas*.

| salt | result | remarks |
|---|---|---|
| acute effects (96-hour $LC_{50}$) | | |
| silver nitrate | 16 ± 4.0 | represents effect of free silver ion; toxic effects occured mainly during the first 24 hours. |
| silver thiosulfate | 280, 000 to 360,000 | estimate, based on the results both of the flowing test (lower value) and a range finding static test |
| silver sulfide dispersion | >240,000 | |
| silver sulfide slurry | no effect | due to settling, measured exposure concentration was <10% of nominal concentration |
| silver chloride | >4,600 | in the presence of 2,000 mg/L $Cl^-$ |
| chronic effects (30-day exposure) | | |
| silver sulfide | >11,000 | MATC; no observed effects on average wet weight or total length after 30 days of continuous exposure |
| silver thiosulphate | 16,000 to 35,000 | MATC; concentrations >35,000 resulted in significant decrease in % survival, length and weight |

LeBlanc *et al.* 1984. All values are expressed as total silver in µg/L

.table 7.8

effects of water hardness on the 96-h $lc_{50}$ of silver nitrate to larval rainbow trout, *Oncorhynchus mykiss*, and juvenile fathead minnows, *Pimephales promelas* in static and flow through bioassays.

| *Oncorhynchus mykiss* | | *Pimephales promelas* | | |
|---|---|---|---|---|
| larval rainbow trout | | juvenile fathead minnows | | |
| static ASSAY | flow through | static ASSAY | flow-through | water hardness |
| - | - | - | 7.4 | 36 |
| - | - | 9.4 | - | 38 |
| - | - | 9.7 | - | 39 |
| - | - | - | 5.6 | 40 |
| 10.9 | 8.4 | 6.7 | 5.3 | 46 |
| 11.8 | 6.9 | 12.3 | 3.9 | 46 |
| 19.9 | 17.9 | 30.4 | 10.9 | 48 |
| 31.8 | - | 22.7 | 11.8 | 48 |
| 48.0 | 14.0 | 13.8 | 11.1 | 54 |
| 54.0 | 16.4 | 19.6 | - | 54 |
| 22.5 | 11.5 | 8.7 | 5.0 | 75 |
| 24.6 | 9.7 | 10.3 | 6.3 | 75 |
| 240.0 | 240.0 | 230 | 110 | 255 |
| 280.0 | 170.0 | 270 | 150 | 255 |

Data from Lemke, 1981. Table entries are in µg/L of silver.

table 7.9

effects of hardness on the 48-h $ec_{50}$ of silver nitrate to *Daphnia magna* in static bioassays.

| silver: µg/L | hardness: mg/L | references |
|---|---|---|
| 0.6 | 39 | Nebeker *et al.* 1983 |
| 1.03 | 46 | Nebeker 1982 |
| 0.9 | 46 | Nebeker 1982 |
| 0.66 | 46 | Nebeker 1982 |
| 0.63 | 46 | Nebeker 1982 |

| | | |
|---|---|---|
| 0.24 | 47 | Chapman 1980 |
| 2.9 | 54 | Nebeker 1982 |
| 1.1 | 60 | Nebeker *et al*. 1983 |
| 0.6 | 60 | Nebeker 1982 |
| 1.1 | 60 | Nebeker 1982 |
| 1.5 | 72 | LeBlanc 1980 |
| 15 | 73 | Nebeker 1982 |
| 8.4 | 73 | Nebeker 1982 |
| 1.5 | 240 | Einabarawy *et al*. 1986 |
| 10 | 240 | Khangarot and Ray 1987a |
| 55 | 255 | Nebeker 1982 |
| 48 | 255 | Nebeker 1982 |

Table entries are for µg/L of silver, not for silver nitrate.

table 7.10

mortality of fathead minnows exposed to measured concentrations of silver.

| silver | total | free | percent mortality | | | |
|---|---|---|---|---|---|---|
| species | silver | silver ion | 24 hr | 48 hr | 72 hr | 96 hr |
| nitrate | 65 µg/L | 65 µg/L | 100 | 100 | 100 | 100 |
| | 29 µg/L | 29 µg/L | 80 | 100 | 100 | 100 |
| | 13 µg/L | 13 µg/L | 5 | 5 | 5 | 10 |
| | 5.8 µg/L | 5.8 µg/L | 5 | 5 | 5 | 5 |
| thiosulphate | 280 mg/L | 800 pg/L | 5 | 5 | 5 | 10 |
| | 140 mg/L | 330 pg/L | 0 | 0 | 0 | 0 |
| | 70 mg/L | 120 pg/L | 0 | 0 | 0 | 0 |
| sulphide | 240 mg/L | <1 fg/L | 0 | 0 | 0 | 0 |
| | 37 mg/L | <1 fg/L | 0 | 0 | 0 | 0 |
| chloride | 4.6 mg/L | 103 ng/L | 40 | 40 | 40 | 40 |

| (@ 2 g/L Cl) | 2.0 mg/L | 101 ng/L | 5 | 10 | 10 | 10 |
|---|---|---|---|---|---|---|
| | 380 µg/L | 101 ng/L | 0 | 0 | 0 | 0 |

Data from Dufficy *et al.* 1993.

table 7.11

aquatic life criteria derivation calculations.

| water hardness >100 | | | water hardness <100 | | |
|---|---|---|---|---|---|
| marine | | fresh | fresh water | | |
| acute | chronic | chronic | acute | chronic | noel |
| | | | | | |
| 12.2 | 2.9 | 3.9 | 0.39 | 0.10 | 0.06 |
| factor 0.1 | factor 0.5 | factor 0.5 | factor 0.1 | factor 0.5 | factor 1 |
| 1.22 | 1.45 | 1.9 | 0.039 | 0.05 | 0.06 |
| Sanders & Abbe, 1989 | Saunders & Abbe, 1989 | Nebeker *et al.*, 1983 | Lemke, 1981 | Davies & Goettl, 1978 | Davies & Goettl, 1978 |

Starting values are the lowest values considered to be reliable data, in µg/L, that were found in the literature. Maximum criteria are twice the 30-day average, providing the resulting value is less than the estimated acute $LC_0$ and does not exceed the chronic $EC_{50}$.