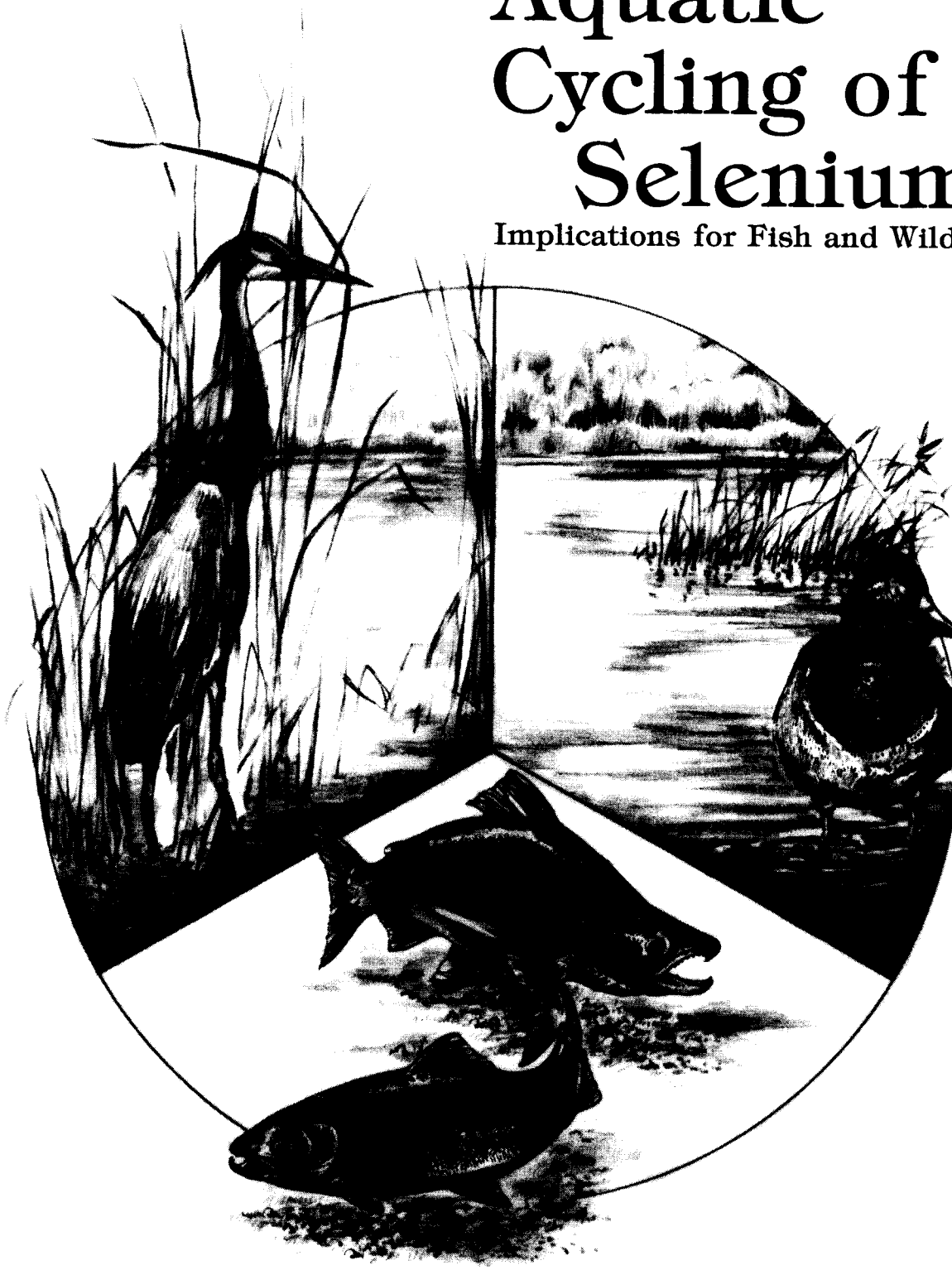
Aquatic Cycling of Selenium:

Implications for Fish and Wildlife



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Aquatic Cycling of Selenium: Implications for Fish and Wildlife

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Introduction

The processes by which selenium, a nonmetallic element, moves through the components of an aquatic habitat may affect fish and wildlife populations. Selenium occurs naturally in the environment in trace amounts; soil concentrations rarely exceed 2 μg/g (ppm) dry weight, except in soils produced by weathering of sedimentary rock. Such soils are widespread in the western United States. Although selenium is an essential micronutrient for normal animal nutrition, concentrations not greatly exceeding those required may produce toxic effects. These effects may range from physical malformations during embryonic development to sterility and death. Industrial and agricultural practices produce selenium-laden wastewater that may be discharged directly or indirectly into lakes, rivers, and wetlands. Two major sources are agricultural irrigation return flows that originate from high-selenium soils and drainage water from areas used for storage and disposal of ash produced by coal-fired power plants.

Because selenium in aquatic systems is readily taken up by organisms, concentrations sometimes reach levels toxic to fish and wildlife. The degree of mobility or cycling rate of selenium in the system determines, to a large extent, whether toxicity occurs and how long the environmental hazard remains. Cycling is important both when the selenium-laden wastewater is being discharged and after the discharge is stopped.

The Selenium Cycle

Basically, three things can happen to dissolved selenium when it enters an ecosystem: (1) it can be absorbed or ingested by organisms, (2) it can bind or complex with particulate matter, or (3) it can remain free in solution. Over time, most of the selenium is either taken up by organisms or bound to particulate matter. Through deposition of biologically incorporated selenium and settling of particulate matter (sedimentation), most of it usually accumulates in the top layer of sediment and detritus. However, because biological, chemical, and physical processes move selenium out of, as well as into, the sediments (Fig. 1), the sediments are only a temporary repository for selenium. Aquatic systems are dynamic, and selenium can be cycled back into the biota and remain at elevated levels for years after waterborne inputs of selenium are stopped.

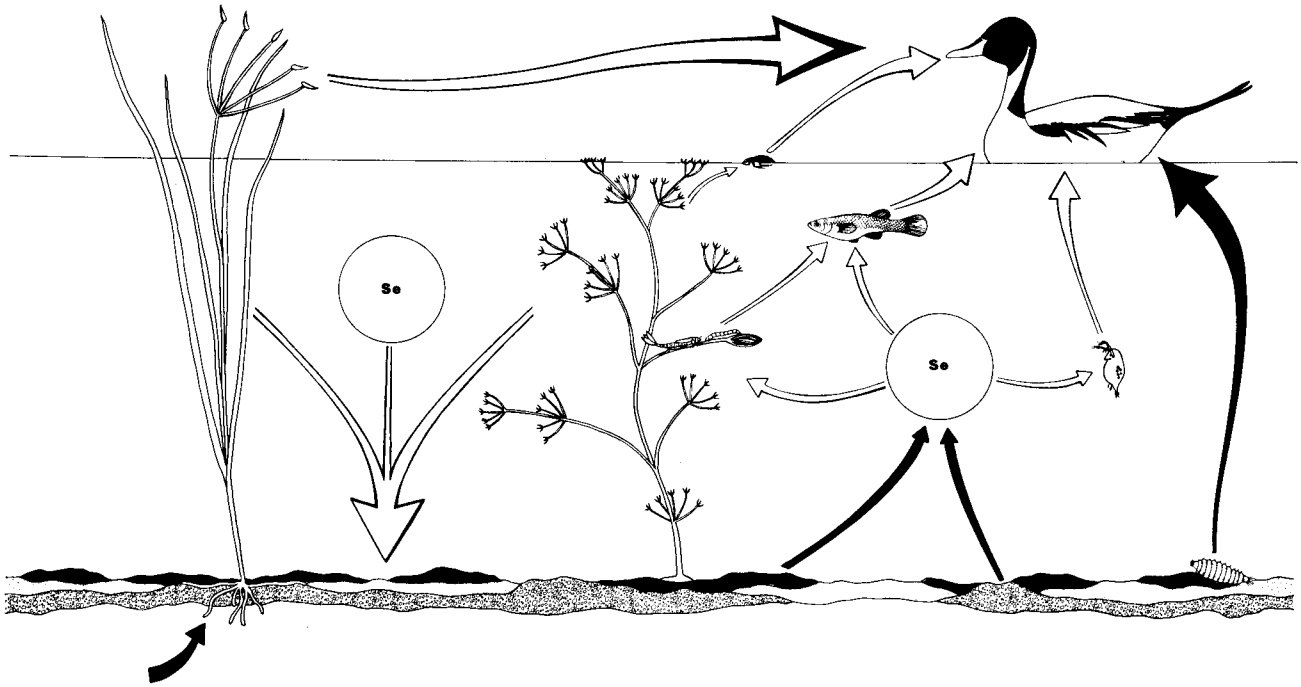

Fig. 1. A highly dynamic system: Biological, chemical, and physical processes cycle selenium into and out of the water, sediments, and biota. (In this and later figures, solid arrows indicate pathways by which selenium is remobilized from sediments into the food chain.)

Immobilization Processes

Selenium can be removed from solution and sequestered in sediments through the natural processes of chemical and microbial reduction of the selenate form (Se VI) to the selenite form (Se IV), followed by adsorption (binding and complexation) onto clay and the organic carbon phase of particulates, reaction with iron species, and coprecipitation or settling (Fig. 2). Regardless of the route, once selenium is in the sediments, further chemical and microbial reduction may occur, resulting in insoluble organic, mineral, elemental, or adsorbed selenium. Most selenium in animal and plant tissues is eventually deposited as detritus and, over time, isolated through the process of sedimentation. Some selenium, particularly certain organic forms, may be released into the atmosphere through volatilization by chemical or microbial activity in the water and sediments, or through direct release by plants.

In total, immobilization processes effectively remove selenium from the soluble pool, especially in slow-moving or still-water habitats and wetlands. Ninety percent of the total selenium in an aquatic system may be in the upper few centimeters of sediment and overlying detritus.

Mobilization Processes

Selenium in sediments is particularly important to long-term habitat quality because mechanisms present in most aquatic systems effectively mobilize such selenium into food chains and thereby cause long-term dietary exposure of fish and wildlife.

Selenium is made available for biological uptake by four oxidation and methylation processes (Figs. 2–4). The first is the oxidation and methylation of inorganic and organic selenium by plant roots and microorganisms. (Oxidation refers to the conversion of inorganic or organic selenium in the reduced organic, elemental, or selenite forms to the selenite or selenate forms; methylation is the conversion of inorganic or organic selenium to an organic form containing one or more methyl groups, which usually results in a volatile form.) The second process is the biological mixing and associated oxidation of sediments that results from the burrowing of benthic invertebrates and feeding activities of fish and wildlife. The third process is represented by physical perturbation and chemical oxidation associated with water circulation and mixing (current, wind, stratification, precipitation, and upwelling). Finally, sediments may be oxidized by plant photosynthesis.

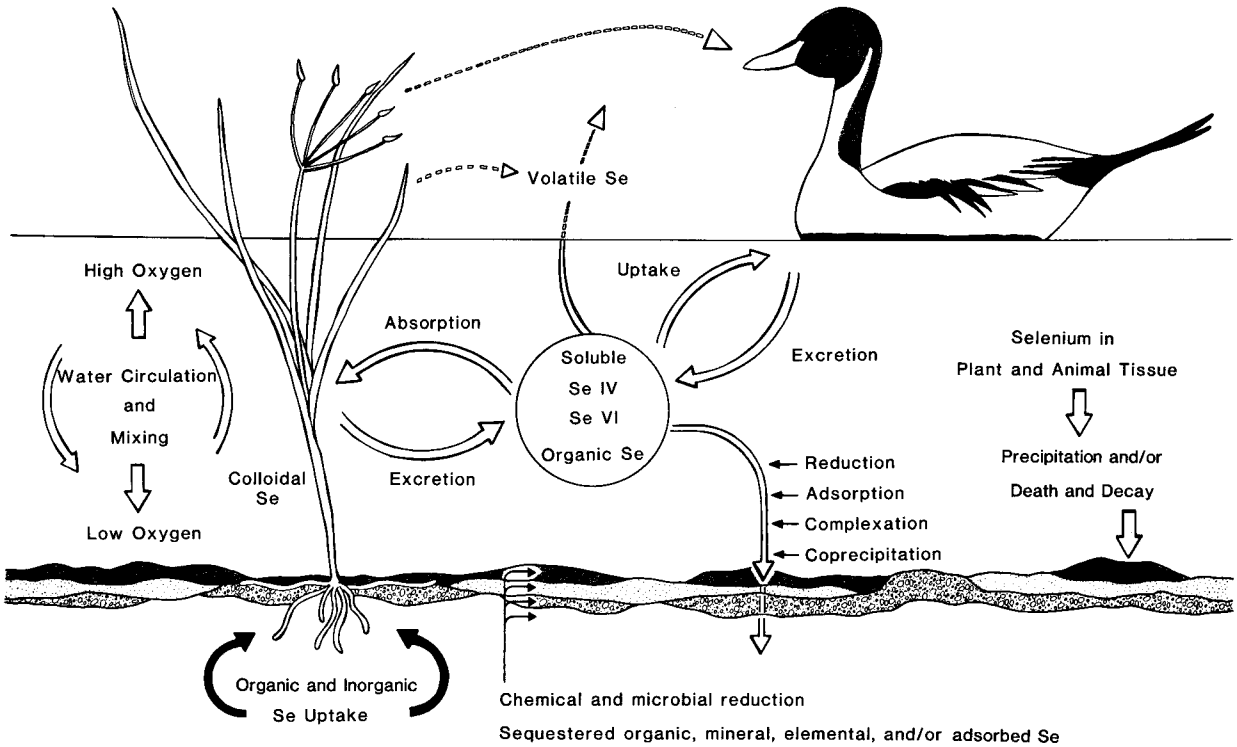

Fig. 2. Processes for the immobilization of selenium include chemical and microbial reduction, adsorption, coprecipitation, and deposition of plant and animal tissue; mobilization processes include uptake of selenium by rooted plants and sediment oxidation due to water circulation and mixing.

Two additional pathways provide for direct movement of selenium from sediments into food chains, even when the surface water does not contain the element. These pathways are uptake of selenium by rooted plants and uptake by bottom-dwelling invertebrates and detrital-feeding fish and wildlife. These two pathways may be the most important in the long-term cycling of potentially toxic concentrations of selenium. Thus, rooted plants and the detrital food pathway can continue to be highly contaminated and expose fish and wildlife through dietary routes even though concentrations of selenium in water are low.

Role of Habitat Variability

The processes regulating selenium cycling are similar in all aquatic habitats, but the relative contribution of each process may vary from habitat to habitat. In fast-flowing waters, fine organic sediments such as those produced by the deposition and decay of particulate matter and plant and animal tissue may be rare because they are continually flushed from the system. In such waters, there is little opportunity for a contaminated surface layer of sediment to develop and rooted plants are often scarce. The benthic–detrital components of the system and the associated food pathways thus play a smaller role in the selenium cycle in flowing waters than in standing water habitats such as wetlands or reservoirs.

Perhaps the aquatic systems that accumulate selenium most efficiently are shallow, standing or slow-moving waters that have low flushing rates. In these systems, biological productivity is often high and selenium may be trapped through immobilization processes or through direct uptake by organisms. Sediments build up a selenium load that can be remobilized gradually, yet continually, through detrital and planktonic food. These habitats are also some of the most important feeding and breeding habitats for fish and wildlife, especially waterfowl and shorebirds.

Several habitat types often occur together in one aquatic system. For example, rivers may have fast-flowing waters, slow-moving pools, and standing-backwater areas, all within a few hundred meters. The degree of fish and wildlife exposure to selenium varies

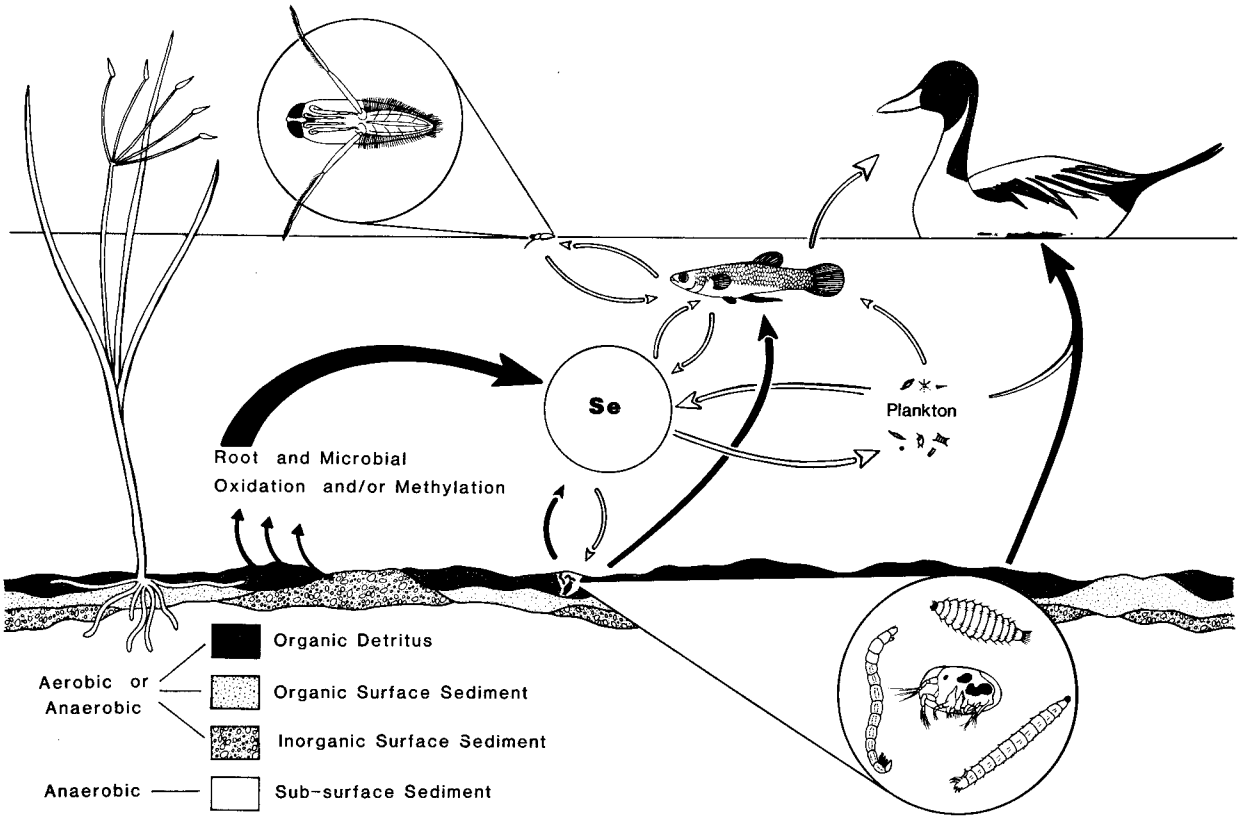

Fig. 3. Additional mobilization processes include direct uptake of selenium by benthic invertebrates and oxidation of sediments resulting from plant roots, microorganisms, and the burrowing activity of benthos.

among habitats according to intensity of use, type of use, and the relative contributions of the various processes that regulate selenium cycling. In any assessment of selenium contamination, variation among habitat types must be considered. And in assessment of toxicological risk, how individual species use different habitats, as well as the role of the physical environment in the selenium cycle, must be considered.

Toxicological Implications

Bioaccumulation

Perhaps the most important factor in selenium cycling and toxicity in aquatic systems is the ability of aquatic organisms to accumulate this element to concentrations one or more orders of magnitude greater than the concentrations in water or food. This extensive bioaccumulation may result because selenium is chemically similar to sulphur and because it is an essential micronutrient. Where fish have experienced chronic toxicity, selenium in the water has been concentrated from 100 to more than 30,000 times, depending on the species and tissue sampled. Selenium accumulation by organisms eaten by fish and wildlife is usually the major pathway leading to toxicity.

Biomagnification of selenium (the accumulation of progressively higher concentrations by successive trophic levels of a food chain) usually ranges from 2 to 6 times between the producers (algae and plants) and the lower consumers (invertebrates and forage fish). For example, fish that eat contaminated plankton or benthic invertebrates may contain 4 times the selenium concentration in their diet, which in turn could contain 500 times the selenium concentration in the water. The food chain biomagnification factor would then be 4 and the total bioconcentration factor for the fish would be 2,000 (i.e., 4 × 500). These relations are important in natural systems because they can cause top-level consumers, such as predatory fish, birds, and mammals, to receive toxic selenium levels in the diet even though

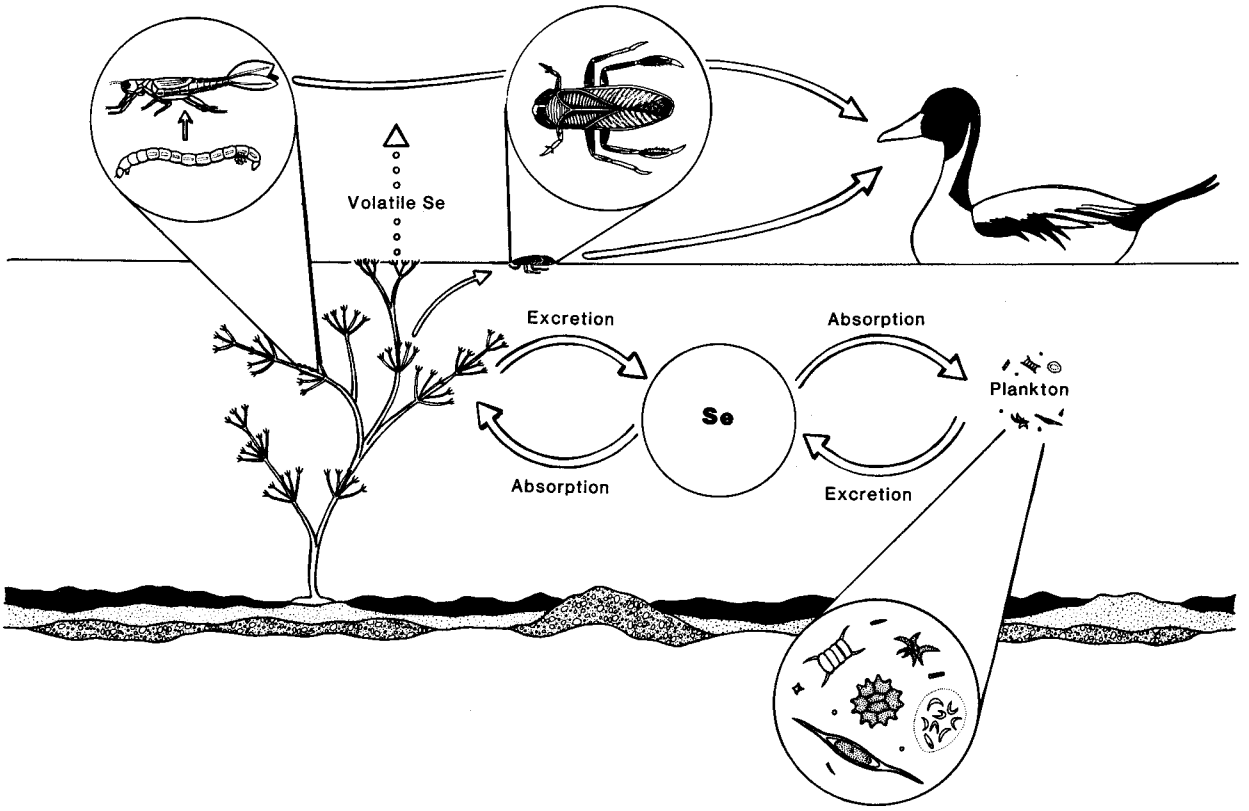

Fig. 4. Dissolved selenium, whether introduced from wastewater discharge or mobilized from sediments, is readily taken up by aquatic organisms and concentrated in food chains. These pathways converge on top consumer species of fish and wildlife. The effects may be severe even when the concentration of waterborne selenium is low.

the concentration in water is low. Moreover, the risk of toxicity through the detrital food pathway will continue despite a loss of selenium from the water column, as long as contaminated sediments are present.

Residues and Toxicity

Toxic effects of selenium fall into two categories: (1) mortality of juveniles and adults, and (2) reproductive effects. Reproductive failure, whether through effects on the adult ovary or on the embryonic development and survival of young, may be the first obvious biological symptom of a selenium contamination problem. Complete reproductive failure can occur with little or no tissue pathology or mortality in the adult population. Selenium concentrates in the eggs of birds and fish and is passed to the developing embryo, where it may cause death or developmental malformations. Reproductive failure accompanied by deformities in the embryos and young signals possible selenium toxicity.

Field and laboratory data suggest that selenium at concentrations greater than 2 to 5 μg/L (ppb) in water can be bioconcentrated in food chains and cause toxicity and reproductive failure in fish. About 10 mg of selenium (as selenomethionine) per kilogram of diet (parts per million; dry weight) caused adverse reproductive effects when fed to mallards. In food items, concentrations of 3 to 8 mg/kg represent contamination that could cause toxic effects in fish and wildlife. Further guidelines for assessing potential selenium toxicity are given in Tables 1 and 2. These guidelines should be applied with caution because the actual concentrations of selenium that are toxic may be increased or diminished by several other factors or other contaminants in the system. Selenium may interact with several metals that can alter the expression of biological effects. Moreover, several factors such as temperature, nutrition, disease, differences in species sensitivity, differences in the relative toxicity of the various chemical forms of selenium, and other environmental stress may affect the actual concentration of selenium that produces toxicosis.

Table 1. *Concentrations of selenium known to be hazardous to fish and wildlife.*

Source	Concentration µg/L (water) or µ/g dry weight (diet). Mean shown in parentheses	Exposure setting, duration, and test conditions	Species and life stage	Toxic effect	Tissue residue (µg/g or ppm)	Supplemental reading
Water[a]	1,100	Laboratory, 48 days, flow-through. Hardness = 330 mg/L	Fathead minnow, *Pimephales promelas*, larvae	50% mortality	—	Adams 1976
Water[a]	400	Laboratory, 48 days, flow-through. Hardness = 330 mg/L	Bluegill, *Lepomis macrochirus*, larvae	50% mortality	—	Adams 1976
Water[a]	500	Laboratory, 48 days, flow-through. Hardness = 330 mg/L	Rainbow trout, *Salmo gairdneri*, larvae	50% mortality	—	Adams 1976
Water[a]	160	Laboratory, 48 days, flow-through. Hardness = 330 mg/L	Coho salmon, *Oncorhynchus kisutch*, larvae	50% mortality	—	Adams 1976
Water[a]	30–170 (80)	Laboratory, 60 days, flow-through. Hardness = 28 mg/L, temperature = 11°C	Rainbow trout, eggs	Significant number of deformities	—	Goettl and Davies 1976
Water[a]	30–170 (80)	Laboratory, 12 months, flow-through. Hardness = 28 mg/L, temperature = 11°C	Rainbow trout, eggs	Significant mortality	—	Goettl and Davies 1976
Water[a]	47	Laboratory, 90 days, flow-through. Hardness = 272 mg/L, temperature = 12°C	Rainbow trout, sac fry	Significant mortality	Whole body = 1.07 wet weight (survivors)	Hunn et al. 1987
Water[a]	28	Laboratory, post-fertilization through hatching, flow-through. Hardness = 135 mg/L, temperature = 0°C	Rainbow trout, eyed eggs	Significantly reduced hatching	—	Hodson et al. 1980
Water[a]	17	Laboratory, 30 days, flow-through. Hardness = 371 mg/L, temperature = 12°C, sulfate = 200 mg/L	Chinook salmon, *Oncorhynchus tshawytscha*, fry	Significant mortality	—	Hamilton et al. 1986
Water[b]	90	Laboratory, post-fertilization through 60 days posthatch, flow-through. Temperature = 20–26°C, salinity = 3.5–5.5 ‰	Striped bass, *Morone saxatilis*, eggs 24 h postfertilization	Significant number of deformities	—	Klauda 1986
Diet[a]	8.9	Laboratory, 42 weeks, flow-through. Hardness = 28 mg/L, temperature = 11°C	Rainbow trout, juveniles	Significant mortality	—	Goettl and Davies 1977*b*

Table 1. *Continued.*

Source	Concentration μg/L (water) or μ/g dry weight (diet). Mean shown in parentheses	Exposure setting, duration, and test conditions	Species and life stage	Toxic effect	Tissue residue (μg/g or ppm)	Supplemental reading
Diet[c]	13	Laboratory, 6 weeks, flow-through, 3% body weight per day feeding. Hardness = 74 mg/L, temperature = 10°C	Chinook salmon, parr	Reduced smolting success	Whole body = 2.9 wet weight, 13.4 dry weight (survivors)	Hamilton et al. 1986
Diet[c]	54[g]	Laboratory, 44 days, flow-through, satiation feeding. Temperature = 21°C	Bluegill, juveniles	75% mortality	Skeletal muscle = 5-7 wet weight; liver = 8-86 wet weight	Finley 1985
Diet[c]	45	Laboratory, 7 days, flow-through, satiation feeding. Hardness = 18 mg/L, sulfate = 5.7 mg/L, temperature = 25°C	Bluegill, juveniles	100% mortality	Whole body = 21-32 dry weight	Carolina Power and Light Company 1984
Diet[c]	25-70	Laboratory, 61 days, flow-through, satiation feeding. Hardness = 19 mg/L, sulfate = 5.4 mg/L, temperature = 25°C	Bluegill, juveniles	100% mortality	Whole body = 44-53 dry weight	Carolina Power and Light Company 1985
Water[d] and diet[c]	8.9-12 (10) 21-73	Field (reservoir), 14 days. Alkalinity = 26 mg/L, temperature = 26°C	Bluegill, juveniles	100% mortality	Muscle = 13.1-17.5 dry weight; viscera = 27.5-37.5 dry weight	Duke Power Company 1980
Water[d] and diet[c]	5-22 (10) 15-70	Field (reservoir), 2 years. Alkalinity = 20-38 mg/L, sulfate = 5.5-17.1 mg/L	All life stages of centrarchids, percichthyids, ictalurids, cyprinids, percids, clupeids, catostomids	Mortality and deformity of fry, juveniles, and adults; total reproductive failure	Skeletal muscle = 3.2-22.3 wet weight; viscera (minus gonad) = 13-52.4 wet weight; ovary = 5.2-41.7 wet weight; testis = 15-22.8 wet weight (survivors)	Cumbie and Van Horn 1978; Lemly 1985*a*
Water[e] and diet[c]	8-12 (10) 25-45	Field (reservoir), 2 years. Alkalinity = 20 mg/L avg, sulfate = 27 mg/L avg	Bluegill, adults exposed in the field and spawned in the laboratory	Mortality and deformity of larvae; total reproductive failure	Carcass (minus gonad) = 5.9-7.8 wet weight; ovary = 6.9-7.2 wet weight (38-54 dry weight); testis = 4.3 wet weight	Gillespie and Baumann 1986

Table 1. *Continued.*

Source	Concentration μg/L (water) or μ/g dry weight (diet). Mean shown in parentheses	Exposure setting, duration, and test conditions	Species and life stage	Toxic effect	Tissue residue (μg/g or ppm)	Supplemental reading
Diet[f]	10[h]	Reproductive study	Mallard, *Anas platyrhynchos*, adults received treated diets	Productivity and duckling survival reduced	Concentrations in eggs ranged from 2.9 to 5.6 (wet weight) and wet weight concentration ranges in adult male and female livers were 6.1 to 12.0 and 2.6 to 6.2, respectively (use 71% moisture for conversion to dry weight)	Heinz et al. 1987

[a]In the form of selenite.
[b]In the form of selenate.
[c]Selenium source was food organisms from selenium-contaminated habitats.
[d]Measured as total recoverable selenium in filtered (0.45 μm) samples.
[e]In the form of selenite (57%), selenate (34%), and selenide (9%).
[f]In the form of selenomethionine.
[g]Converted from 13.6 μg/g wet weight, assuming 75% moisture. Formula for converting wet weight to dry weight:

$$\text{dry weight concentration} = \frac{\text{wet weight concentration}}{1 - \%\ \text{moisture of sample}}$$

[h]Fresh weight, diet contained about 10% moisture.

Ecosystem Recovery

Immobilization and mobilization processes occur simultaneously in aquatic systems, and the physical and biological characteristics of the environment such as season, climate, and habitat determine which process dominates at any given time. Continuous inputs of selenium at relatively constant levels generally result in an equilibrium in which the two groups of processes become more or less offsetting and selenium continues to cycle. When inputs stop, four processes operate to permanently remove selenium from the system and gradually reduce the residual contamination: (1) movement of selenium-contaminated organisms out of the system, as when fish and wildlife emigrate or aquatic insects emerge, (2) release of volatile selenium from plants and the water directly into the atmosphere, (3) removal by sediment and water transport (flushing) and groundwater seepage, and (4) burial by the process of sedimentation.

The rate at which an ecosystem recovers from selenium contamination depends on such factors as the degree of contamination, habitat type, annual productivity, flushing rates, and climate. The time required for these natural processes to lower selenium concentrations to nontoxic levels in specific habitats cannot now be estimated accurately. However, field evidence suggests that such time intervals may range from several years to several decades. In general, recovery is much slower in shallow impoundments and wetlands than in fast-flowing rivers and streams.

Because of its propensity to bioaccumulate, to move from sediments to biota, and to cause severe adverse reproductive effects as well as mortality, selenium should be recognized as a contaminant with the potential to markedly affect fish and wildlife populations.

Table 2. *Selenium levels of concern for fish and wildlife.*

Source or tissue residue	Concentration μg/L (water) or μg/g dry weight	Affected group	Suspected toxic effect	Supplemental reading
Water	>2-5	Fish and waterfowl	Reproductive failure or mortality due to food-chain bioconcentration	Heinz et al. 1987; Lemly 1985*a*; Lemly 1985*b*
Sediment	⩾4	Fish and waterfowl	As above	Duke Power Company 1980; Finley 1985; Garrett and Inman 1984
Food	⩾5	Fish	As above	Cumbie and Van Horn 1978; Lemly 1987
	⩾3	Waterfowl	As above	Heintz et al. 1987
Whole body residue	⩾12	Fish	Reproductive failure	Gillespie and Baumann 1986; Lemly 1985*a*
Visceral residue[a]	⩾16	Fish	Reproductive failure	Gillespie and Baumann 1986; Lemly 1985*a*
Skeletal muscle residue[a]	⩾8	Fish	Reproductive failure	Gillespie and Baumann 1986; Lemly 1985*a*
Egg	⩾15-20[b]	Waterfowl (mallard)	Teratogenesis or embryo mortality	Heinz et al. 1987

[a]Approximate conversion factors for fish:
Whole body to muscle = whole body × 0.6
Viscera (liver or female gonad) to muscle = viscera × 0.25
Viscera to whole body = viscera × 0.33
[b]Converted from a mean wet-weight concentration of 4.6 μg/g based on a 71% moisture content.
Note: The 85th percentile whole-body concentration of selenium in fish tissues measured in the National Contaminant Biomonitoring Program was 0.82, 0.70, and 0.71 μg/g wet weight for 1976-77, 1978-79, and 1980-81, respectively. Table 1, footnote g shows formula for converting wet weight to dry weight.

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Lemly, A. Dennis, and Gregory J. Smith. 1987. **Aquatic cycling of selenium: implications for fish and wildlife.** U.S. Fish Wildl. Serv., Fish Wildl. Leafl. 12. 10 pp.

Selenium, a non-metallic trace element, is readily accumulated by aquatic organisms. The degree of mobility or cycling rate of selenium in an aquatic ecosystem will largely determine whether toxicity occurs and how long the environmental hazard remains. The selenium cycle is made up of immobilization and mobilization processes that move the element between organisms, water, and sediment, with kinetics determined by chemical and physical factors related to the type of habitat present and its spatial variability. Because of its ability to bioaccumulate and cause adverse reproductive effects at low environmental concentrations, selenium should be recognized as a contaminant with the potential to severely impact fish and wildlife populations.

Key words: Selenium, fish, wildlife, aquatic ecosystems, bioaccumulation, toxicity, residues, fate, effects.

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