UNITED STATES DEPARTMENT OF THE INTERIOR

NATIONAL IRRIGATION WATER QUALITY PROGRAM
INFORMATION REPORT NO. 3

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

Selenium

Participating Agencies:

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

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Selenium

Description

Selenium (Se) is a semi-metallic trace element which has biochemical properties similar to those of sulfur. The pure element most often appears as lustrous trigonal crystals of gray selenium. Other common forms include a dark-red powder; the glassy, dark-brown vitreous selenium; and dense monoclinic crystals of red selenium, but these are all less stable than the gray variety and tend to convert to it over time. The most common selenium compounds in natural waters are selenious acid (H_2SeO_3) and selenic acid (H_2SeO_4), which correspond, respectively, to the salts selenite (Se^{+4}) and selenate (Se^{+6}). Certain metal and organic selenides (Se^{-2}) are also common in some environments, such as bottom sediments.

Occurrence

Selenium is widely distributed in rocks, soils, water, and living organisms. In the Western United States, it is most common in Upper Cretaceous and Tertiary marine sedimentary rocks (Seiler 1997). Figure 2 shows the distribution of these formations in the Western United States. Many geologic formations are seleniferous and capable of contributing to the mobile forms of selenium in soils. Selenium is highly mobile and biologically available in arid regions having alkaline soils—conditions typical of the Western United States. A number of plants, such as *Astragalus* (loco weed and milkvetch), can concentrate selenium extracted from the soil into a biologically available form, which is toxic to livestock when eaten (Hedlund 1993).

The concentration of selenium in rivers, streams, lakes, and wetland areas is greatly increased by irrigation drainage return flow in certain areas of the West. Upstream from irrigated areas in the Colorado River basin, waters generally have selenium concentra-tions of less than 1 µg/L, but downstream from irrigated areas, the concentration exceeds 30 µg/L in places (mainly backwater areas). Drainage from the Westlands Irrigation Project in California averaged 300 µg Se/L and ranged from 160 to 1,400 µg Se/L. Selenium was further concentrated in the collector drains by evaporation and bio-accumulation. Levels in plants and animals were high enough to kill some aquatic birds and fish and impair reproduction of others. (Hedlund 1993.)

Selenium can also be mobilized or released from the soil by a crop-fallow management system. Saline seeps developed in wheat-fallow areas of the plains from Texas into Canada may have high concentrations of selenium and may contaminate both ground water and surface runoff (Hedlund 1993).

Abnormally high mass-loading of selenium into aquatic environments most typically results from the disposal of coal fly ash, irrigation wastewater, or oil refinery waste-water. Mining of sulfide ores is also a common source of artificially mobilized selenium. In particular, selenium is a common waste product from uranium, bentonite, and coal mining. Soils, surface waters, and ground waters around these mining operations can become contaminated. Concentrations as high as 4,500 mg/kg have been reported in the overburden from the Powder River district in Wyoming. Bentonite mines in Wyoming, Montana, and SouthDakota are additional selenium sources, and it also may be concen-trated in coal deposits and carbonaceous

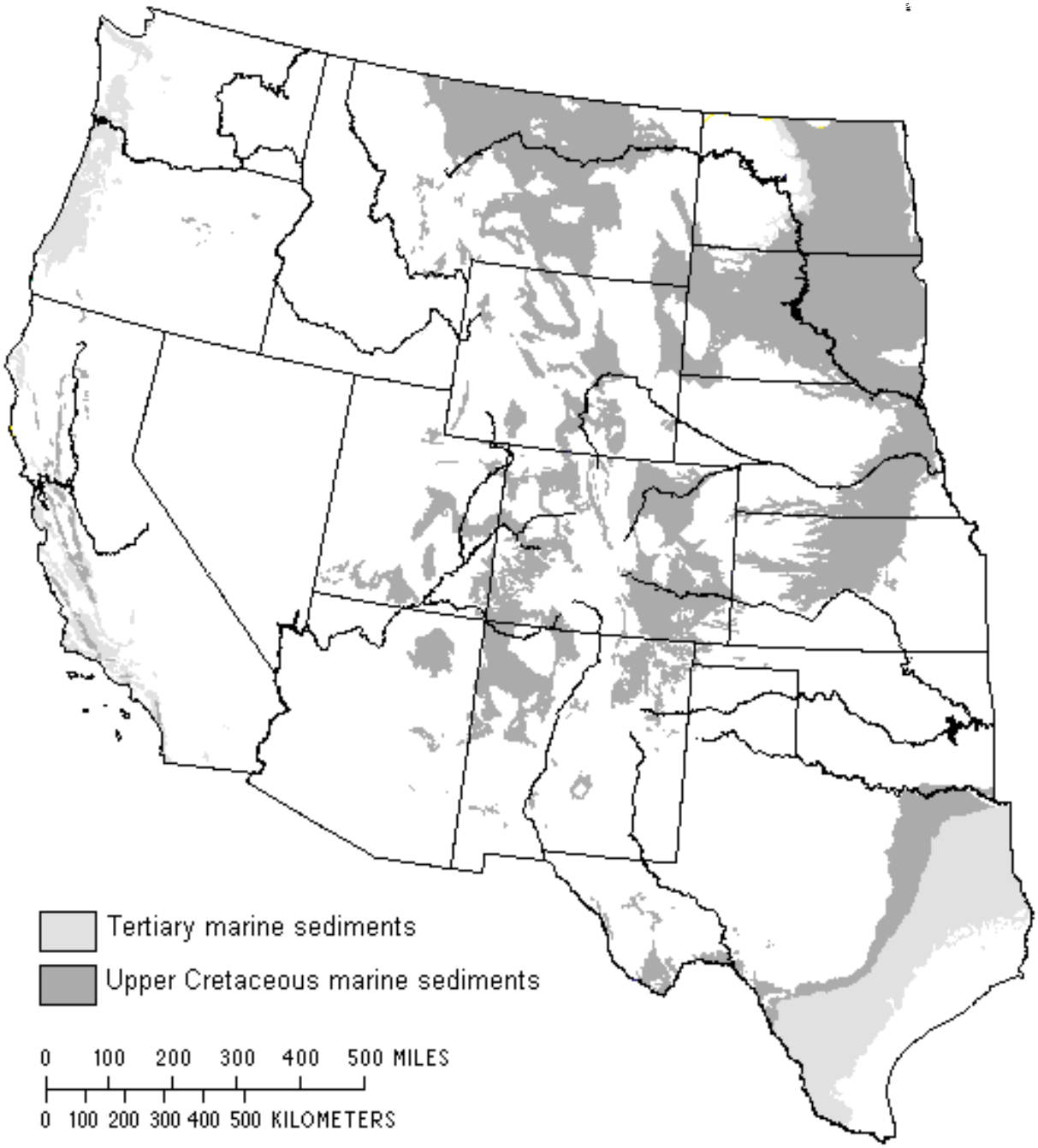

Figure 2. - Distribution of potentially seleniferous bedrock in the Western United States (adapted from Seiler 1997)

shales. Mining operations commonly increase the element's mobility and solubility. (Hedlund 1993.)

Background Concentrations.—Selenium has an average crustal abundance of 0.05 mg/kg and the following approximate background levels in various media:

Medium	Background (mg/kg dw, except as noted)
Freshwater	0.1–0.4 µg/L
Freshwater sediments	0.2–2.0
Plants:	
Freshwater algae	0.1–1.5
Freshwater macrophytes	0.1–2.0
Terrestrial plants	0.01–0.6
Invertebrates:	
Aquatic	0.4–4.5
Terrestrial	0.1–2.5
Fish:	
Liver	2–8
Other tissues	1–4
Reptiles/Amphibians:	
Liver	2.9–3.6
Other tissues	1–3
Birds: Whole body	<2
Muscle	1–3
Eggs	<5
Liver	<10
Feathers	1–4
Whole blood	0.1–0.4 mg/L
Mammals: Whole body	<1–4
Muscle	<1
Liver	1–10
Hair	<1–3
Milk	<0.05 mg/L
Whole blood	0.1–0.5 mg/L

See the separate sections for each of these media, below, for further discussion of these background levels.

Summary of Effects

Based on the known margins of safety between normal and toxic dietary exposures, selenium is more poisonous than either arsenic or mercury (Sorensen 1991). However, selenium is also an essential trace nutrient for animals, and it serves beneficial metabolic functions (Arthur and Beckett 1994). Thus, selenium deficiency as well as toxicity can cause adverse effects for fish and wildlife (Oldfield 1990; CAST 1994). Interestingly, both deficiency and toxicity cause similar effects: e.g., reproductive depression, anemia, weight loss, and immune dysfunction (Koller and Exon 1986). The known effects of selenium exposure to various classes of organisms are summarized in table 32.

One of the most important features of selenium ecotoxicology is the very narrow margin between nutritionally optimal and potentially toxic dietary exposures for vertebrate animals (Wilber 1980; NRC 1989). Nutritionally optimal dietary selenium expo-sure is generally reported as 0.1–0.3 mg/kg. Thresholds for dietary toxicity in animals are generally reported as 2–5 mg Se/kg—only 7 to 50 times the nutritionally optimal levels (Girling 1984; NRC 1989; Sorensen 1991; Eisenberg 1993). Thus, relatively small perturbations in the dietary exposure of vertebrate animals are potentially harmful. According to Spallholz (1994), extensive studies with rats have found the toxicity threshold for dietary selenite to be only 2.5 times above the nutritionally optimal dose. However, only the studies of rats have included controlled lifetime exposures and evaluated such sensitive response variables as longevity.

Table 32.—Summary of comprehensive biotic effects of selenium

Medium	No effect[1]	Level of concern[2]	Toxicity threshold[3]	Comments/Explanation
Water (µg/L, total recoverable Se)[4]	<1	1–2	>2	Peterson and Nebeker (1992)
Sediment (mg/kg dw)	<1	1–4	>4	Van Derveer and Canton (1997), SJVDP (1990), Lemly and Smith (1987)
Diet (mg/kg, dw)	<2	2–3	>3	Lemly (1996a)
Waterbird eggs (mg/kg dw)	<3	3–6	>6	No-effect level from Skorupa and Ohlendorf (1991). Toxicity threshold from Skorupa (1998a)
Fish, whole-body (mg/kg dw):				
Warm-water species	<3	3–4	>4	Lemly (1996a)
Cold-water species	<2	2–4	>4	

[1]Concentrations lower than this value produce no discernible adverse effects on fish or wildlife and are typical of background concentrations in uncontaminated environments.

[2]Concentrations in this range rarely produce discernible adverse effects but are elevated above typical background concentrations.

[3]Concentrations above this value appear to produce adverse effects on some fish and wildlife species.

[4]See the "Water" section of this chapter for a discussion of the difference between total recoverable Se and dissolved Se.

Selenium is much less toxic to most plants and invertebrate animals than to vertebrate animals. Among vertebrates, reproductive toxicity is one of the most sensitive endpoints; however, egg-laying vertebrates such as birds and fish seem to have substantially lower thresholds for reproductive toxicity than placental vertebrates (mammals).

A general ecotoxicological rule of thumb for selenium is that thresholds for adverse effects in vertebrate animals begin at concentrations less than one order of magnitude above normal (Lemly 1985b; Eisenberg 1993; Ohlendorf et al. 1993; Salyi et al. 1993). When environmental, dietary, or tissue concen-trations of selenium are 10 times normal background levels or higher, toxic effects are likely. Immunotoxic effects have been conclusively documented for birds and mammals at tissue concentrations of selenium less than five times normal background (Whiteley 1989; Schamber et al. 1995). How-ever, there are no well-documented case studies of selenium-mediated immunotoxicity and associated consequences among animals in nature.

The threshold of ecotoxicity for selenium is remarkably similar for fish and birds (Lemly 1995, 1996b), the two classes of animals most likely to be adversely affected by contamina-tion from agriculture or industry. With the exception of hepatic tissues, all fish and wildlife tissues normally average about 2 mg Se/kg or less. A concentration of 10–20 mg Se/kg in wildlife tissues or diets is above the threshold of toxicity for sensitive and moderately sensitive taxa, and at the level of 50–100 mg Se/kg, catastrophic impacts are highly likely.

Metabolic stress caused by winter weather can increase the susceptibility of birds (Heinz and

Fitzgerald 1993b), mammals (Ghosh et al. 1993), and fish (Sorensen 1991; Lemly 1993c, 1996b) to selenium poisoning. Toxicity data collected under benign climatic conditions may therefore underestimate sensitivity to selenium poisoning.

Selenium accumulates in and disperses from animal tissues fairly rapidly. Significant changes in tissue selenium status can occur within days, weeks, or months depending on the response criterion of interest and the target tissue being monitored (Wilber 1980; Bennett et al. 1986; USFWS 1990a; Heinz et al. 1990; Heinz and Fitzgerald 1993a; Heinz 1993). Furthermore, the overt symptoms of even near-fatal selenium poisoning in adult birds and mammals can be reversed quickly if the source of selenium exposure is eliminated (Ruta and Haider 1989; Heinz and Fitzgerald 1993b). By contrast, embryonic deformities caused by selenium poisoning are not reversible (Lemly 1993b), nor are some types of tissue damage in adult animals (Sorensen 1991).

Study Approaches

Selenium toxicology has been the subject of an extensive body of scientific literature. More than 5,500 titles were screened for this guidance document. Titles that were most directly relevant to evaluating *in situ* eco-toxicology of selenium were retrieved and reviewed. Finally, the retrieved literature was further screened to produce a "short list" of particularly useful reports for evaluating NIWQP measurements of environmental selenium.

Publications reporting field measures of exposure and response were afforded the greatest weight in formulating the guidelines presented in this chapter. Macrocosm and mesocosm studies were given the next highest priority. Among the remaining experimental studies of captive biota, those that utilized selenomethionine (a good surrogate for natural food-chain selenium) and that tested the effects of dietary exposure were given highest priority. Experimental studies that exposed vertebrate biota (other than eggs or larvae) to contaminated water, but provided no food or only clean food (i.e., standard aquatic toxicity tests), are not transferable to field conditions and therefore were not reviewed for this document (see EPA 1987; Maier et al. 1987; and SJVDP 1990 for reviews of studies of this type). Also, toxicity studies of marine organisms were not reviewed for this document.

Field Cases

To the extent possible, interpretive guidelines should be based either on field data or on experimental data that have been field validated. At least 11 incidents of fish or wildlife poisoning by selenium, studied in the field, have been documented in the technical literature. These incidents occurred in North Carolina, Texas, Colorado, California, Wyoming, Utah, and Sweden. Brief sum-maries of each incident are presented below.

(1) Belews Lake, North Carolina—This power-plant cooling reservoir received return flow from a fly-ash settling basin from 1974 to 1985. The ash-basin effluent contained about 150–200 μg Se/L. For the main reservoir, water-borne selenium was elevated to about 10 μg/L on average. Sediments averaged 14 mg/kg, benthic food-chain fauna averaged 20–50 mg/kg, plankton averaged 30 mg/kg, fish muscle averaged 20–40 mg/kg, and whole-body selenium concentrations averaged 40–125 mg/kg (approximate dry weights). Gonads of female fish contained about 20–170 mg Se/kg. Thus, the selenium concentrations were about 25–100× background in the water, 10–15× background in food-chain flora and fauna, as much as 130× normal in fish whole bodies, and about 5–85× normal in female fish gonads. Of 20 species of fish exposed to these contaminant conditions, 16 were extirpated, 2 had ceased reproducing, 1 was temporarily extirpated,

and 1 was unaffected. Only carp, black bullheads, and mosquitofish were present throughout the study. For most species, teratogenic effects and other overt abnormalities were observed in about 10–70 percent of the sampled fish.

Follow-up sampling of fish in 1992, 7 years after selenium loading to Belews Lake was substantially reduced, found whole-body selenium concentrations averaging 10–20 mg/kg (≈5–15× normal) associated with slightly elevated frequencies of abnormalities (5–10 percent versus a reference range of 1–3 percent). For centrarchids, a strong exposure-response relationship was evident between whole-body selenium and frequency of overt abnormalities. The EC25 was roughly 45 mg/kg whole-body Se (Lemly 1985a,b; 1993b). In an arm of Belews Lake that is semi-isolated from the main reservoir, known as the Highway 158 arm, selenium content averaged 3–4 µg/L in the water (10–20× normal), 0.7–3.0 mg/kg in sediments (1–10× normal), 4–8 mg/kg in benthic invertebrates (2–5× normal), 25–30 mg/kg in fish liver (4–6× normal), and 7–9 mg/kg in fish muscle (2–5× normal) (Sorensen et al. 1984; Cumbie pers. comm. cited in GLSAB 1981). Some fish in the Highway 158 arm showed sublethal toxic effects such as generalized edema and abnormal ovarian tissue damage (Sorensen et al. 1984). Nonetheless, an overtly normal fish fauna persisted (Lemly 1985b).

(2) Hyco Reservoir, North Carolina—This was another powerplant cooling reservoir, which received effluent from two fly-ash ponds that contained about 50–200 µg Se/L in their water. As a result, the waterborne concentra-tion of selenium in Hyco Reservoir was elevated to about 10 µg/L on average. In other media, selenium averaged approxi-mately 3–5 mg/kg (dw) in sediments, 10–30 mg/kg in benthic food-chain fauna, 2–20 mg/kg in plankton, 35–50 mg/kg in fish muscle, and 30–50 mg/kg in gonads of female fish. Compared to local reference populations, fish muscle contained about 40× normal selenium concentrations, and female gonads contained about 15–25× normal. As a consequence of this exposure, adult fish densities declined by 38–75 percent, and larval fish densities declined by 98.6 percent (Woock and Summers 1984; Gillespie and Baumann 1986).

(3) Martin Lake, Texas—This reservoir received discharges during 1978–79 from fly-ash ponds that contained selenium concentrations in excess of 2,000 µg/L. The water in Martin Lake was elevated to about 2.6 µg Se/L on average (≈5 µg/L in the impact area?). Other selenium concentrations (dw) averaged about 5 mg/kg in sediments, 30 mg/kg in fish muscle, and 17 mg/kg in gonads of female sunfish (≈8× the "normal" level of Hamilton and Waddell 1994). This level of exposure was implicated in a series of fish kills soon after Martin Lake received the ash-pond effluent (Garrett and Inman 1984; Lemly 1985a; Sorensen 1988; Texas Parks and Wildlife Department 1990). By 1986, fish muscle tissue was down to 5–10 mg Se/kg, but selenium in red-winged blackbird eggs was still elevated to 11 mg/kg (4× that of a local reference population). Two blackbird foods—grass-hoppers and mayflies—averaged 1.1 and 15 mg Se/kg, respectively. The hatching success of Martin Lake blackbird eggs was less than 50 percent that of a local reference population. Barn swallow eggs at Martin Lake averaged 3.3 mg Se/kg and showed normal hatchability com-pared to a local reference population (King 1988; King et al. 1994).

(4) Sweitzer Lake, Colorado—This lake, also known as Garnet Mesa Reservoir, was built in 1954 for recreational purposes. Sweitzer Lake is situated in an area of naturally seleniferous geological formations. It is unclear, however, how much of the cumulative selenium loading into Sweitzer Lake came from natural inputs and how much was artificial. Initial (1950's) water sampling revealed more than 100 µg Se/L. Biotic selenium contamination reached

as high as about 20 mg/kg (dw) in benthic food-chain fauna and 40 mg/kg in fish livers. This level of exposure caused progressive mortality of stocked game fishes. The Colorado Division of Wildlife decided to stop stocking the lake in 1974 but later did restock it with catfish in 1984. In the late 1980's, water samples from the lake contained about 10–25 µg Se/L. Fish muscle (channel catfish) averaged about 30 mg/kg, and bird eggs averaged about 9 mg Se/kg. At this level of exposure, there was no evidence of successful reproduction among the channel catfish, and the reproductive performance of birds at the lake was unknown; however, large popula-tions of green sunfish and carp, including various age classes, were present (Lemly 1985a; Butler et al. 1991).

(5) Kesterson Reservoir, California—This evaporation and seepage basin for irrigation drainage water in California's Central Valley received water containing about 330 µg Se/L over several years. Kesterson was operated as a series of 12 separate cells that contained 15–350 µg Se/L and averaged about 150 µg/L. Other average selenium contents were about 12 mg/kg in sediments, 20–110 mg/kg in benthic and water-column food-chain fauna, 170 mg/kg in mosquitofish (whole body), and about 10–70 mg/kg in bird eggs. These con-centrations were about 12–130× local reference values in food-chain fauna and 5–35× local reference values in bird eggs. At this level of exposure, it is suspected that several species of fish were extirpated before any systematic field studies began (USBR 1986: 4G-2). The brood size of mosquitofish in the San Luis Drain, the source of Kesterson's drainwater, was only 12.4 fry/brood, versus a local reference value of 25.7 fry/brood. The incidence of stillborn fry was about 20–30 per-cent compared to a local reference value of about 1–3 percent. Mosquitofish are extremely tolerant of selenium exposure.

The mosquitofish from the San Luis Drain contained whole-body concentrations of about 120 mg Se/kg or about 80× local reference levels. About 40 percent of 578 nests of ducks and other waterbirds at Kesterson contained one or more dead or deformed embryos. About 20 percent of all nests contained one or more overtly deformed embryos. Four species of waterbirds (American avocet, black-necked stilt, eared grebe, and American coot) experienced complete reproductive failure. Exposure-response data for black-necked stilt eggs revealed an eggwise threshold for embryo-toxicity in the vicinity of 10 mg/kg. Some adult birds also died, and many of these showed alopecia (loss of feathers), a classic symptom of acute selenium poisoning (Ohlendorf et al. 1986; Zahm 1986; Presser and Ohlendorf 1987; Ohlendorf et al. 1988a; Ohlendorf 1989; SJVDP 1990; Saiki and Ogle 1995).

(6) Tulare Lake Basin, California—About 25 evaporation and seepage ponds for irrigation drainage water in this basin received water containing from <1 to >1,000 µg Se/L. The "ponds" vary in size from 10 to 1,800 acres. Impounded water in these ponds averaged 0.5–1,014 µg Se/L. Sediments averaged 0.1–16 mg Se/kg, benthic and water-column food-chain fauna averaged 1–250 mg/kg, and bird eggs contained about 1–150 mg Se/kg. Except for intermittent introductions of mosquitofish, the Tulare evaporation ponds were never inhabited by fish. Impairment of avian reproduction was documented at about half of the evaporation ponds. Highly elevated rates of embryo teratogenesis (20 percent vs. 0.1 percent background) were documented for ponds with as little as 15 µg/L waterborne Se and 0.9 mg/kg sediment Se. At an individual level of analysis, embryos of black-necked stilts that were exposed *in ovo* to about 55–65 mg Se/kg (≈25× normal) and survived at least 8 days into incubation had about a 50 percent probability of overt teratogenesis. Data at the population level of analysis suggested that the threshold for hatchability depression in stilt eggs (only a portion of which is caused by overt terato-genesis) corresponds to a geometric mean value of >8 mg Se/kg (3–4× normal). Predictive regression equations for the ponds revealed that ponds averaging >2.7 µg Se/L in

water or >2.9 mg Se/kg in the food chain would be sufficiently contaminated for average bioaccumulation of >8 mg Se/kg in eared grebe eggs (the most proficient avian bioaccumulators of selenium). A statistical risk analysis of 354 nests revealed that black-necked stilt hens that had laid a sample egg containing as little as 4.2–9.7 mg Se/kg (the first quartile above background) had nearly a fourfold greater risk than normal of producing an inviable sibling egg. Based on a water-to-egg regression equation for stilts at the Tulare ponds (r=0.901), only 2.6–18 µg Se/L in water would result in 4.2–9.7 mg Se/kg in eggs. More recent analyses of the stilt data revealed that the embryotoxicity threshold occurs at 6 mg Se/kg in eggs and that 3-4 µg Se/L in water is sufficient to create such a concentration in eggs (Skorupa 1998a). Exposure-response data for avian terato-genesis in the Tulare Basin suggest that stilts are not the most sensitive avian species for Se-caused reproductive impairment. Ducks are nearly twice as sensitive as stilts to embryonic selenium exposure (≈50 percent probability of overt teratogenesis at 30 mg Se/kg in duck eggs, compared to 58 mg Se/kg in stilt eggs; Skorupa 1998b), but small sample sizes of duck nests in the Tulare Basin precluded any statistical risk analysis of egg viability (i.e., overall embryotoxicity). Among adult stilts, exposure-dependent loss of body weight was documented; however, no fatal poisoning of adults or alopecia was documented for any species of bird (Fujii 1988; SJVDP 1990; Skorupa and Ohlendorf 1991; CH2M Hill et al. 1993; Ohlendorf et al. 1993; CH2M Hill 1994).

(7) Chevron Oil Company Refinery Near Richmond, California—The refinery discharges process wastewater to a flow-through marsh for pretreatment prior to ultimate discharge into San Francisco Bay. The wastewater effluent contained about 10–30 µg Se/L, and water in the marsh averaged 7.5–17.5 µg Se/L. Sediment data are not available for the marsh. Food-chain organisms contained about 10–45 mg Se/kg, or about 10× normal levels. Randomly sampled eggs of black-necked stilts nesting at the marsh averaged 20–30 mg Se/kg (8–12× normal). About 18 percent of stilt nests contained at least one inviable egg, versus 9 percent in San Joaquin Valley nests confirmed to have had normal background exposure to selenium. That difference was statistically significant. Nonrandomly sampled inviable eggs of stilts, avocets, mallards, and coots contained about 15–60 mg Se/kg. Embryo teratogenesis was docu-mented for mallards, coots, and possibly stilts (CH2M Hill 1994, 1995; Medlin 1994).

(8) Rasmus Lee Lake and Goose Lake, Wyoming— These lakes, within the Kendrick Reclamation Project near Casper, received seleniferous irrigation drainage water. The median dis-solved selenium concentrations were 38 µg/ L in Rasmus Lee Lake and 54 µg/L in Goose Lake. At Rasmus Lee Lake, average selenium contents were about 4–9 mg/kg in sediments, 38 mg/kg in pondweed, 95–160 mg/kg in benthic and water-column food-chain fauna, and 5–85 mg Se/kg in bird eggs. At Goose Lake, the averages were 20–40 mg/kg in sediments, 14 mg/kg in pondweed, 45 mg/kg in benthic and water-column food-chain fauna, and 50–120 mg/kg in bird eggs. Selenium in the food chain at these lakes was elevated to about 3–16× local reference samples and about 2–50× in bird eggs. At these levels of exposure, rates of embryo teratogenesis in geese, avocets, and grebes were 4–23 percent. Nearly 40 percent of 126 monitored nests contained at least one inviable egg, and 7 percent of 120 nests contained at least one deformed embryo. Egg viability (hatchability) ranged from 45.1 to 86.2 percent in all species monitored over all years of study (compared to normal hatchability for these species of >90 percent). Poor post-hatch survivorship of avian hatchlings was also suspected (See et al. 1992).

(9) Ouray National Wildlife Refuge (NWR), Utah—Waterfowl ponds at the refuge received seleniferous irrigation drainage water via seepage of shallow groundwater from upgradient

agricultural fields and from flooding on natural drainages. The shallow ground-water near Ouray NWR showed much spatial variation in selenium content but generally averaged 10–700 µg/L. Water in each of the affected ponds, North and South Roadside Ponds, averaged about 40 µg Se/L. These two ponds are hydrologically connected. Sedi-ment from North Roadside Pond averaged 17 mg Se/kg. Sediment was not sampled from South Roadside Pond. Based on pooled data for both ponds, selenium averaged 10–30 mg/kg in aquatic plants, about 25 mg/kg in benthic and water-column food-chain fauna, about 40–80 mg/kg in fish (whole body), and about 8–90 mg/kg in bird eggs. These concentrations were about 20–60× normal in plants, 10× normal in food-chain fauna, 15–30× normal in fish, and 3–35× normal in bird eggs. At these exposure levels, more than 85 percent of all coot eggs were inviable, and deformed embryos were found in about 10 percent of the nests. Reproductive performance was very poor in small samples of grebe and duck nests, and deformed embryos were found in nests of mallards and redhead ducks. A randomly collected egg from a redhead nest that contained all dead or deformed embryos contained about 20 mg Se/kg (12× normal).

Forty-four wing-clipped game-farm mallards were released to the roadside ponds to monitor the dynamics of tissue selenium and adult survivorship. At the time of release, these birds averaged 2.8 mg Se/kg in their livers. After 1 week, liver selenium averaged 27 mg/kg (10× normal); after 4 weeks the last surviving mallard died, and its liver was found to contain 106 mg Se/kg. Breast muscle averaged about 1 mg Se/kg at release and increased to 37 mg/kg in the last surviving bird. Most of the released mallards had died by the end of the second week, at which time their livers averaged about 40–50 mg Se/kg (≈15× normal) and breast muscle had 4–8 mg Se/kg (3–10× normal). Within 2 weeks, the released mallards had lost about 20 percent of their body mass (Stephens et al. 1992), a finding consistent with selenium-induced cachexia (Albers et al. 1996; Green and Albers 1997).

(10) Imperial Valley of Southern California—Colorado River water averaging 1–2 µg Se/L is diverted into the valley for irrigation, and irrigation wastewater averaging 2–10 µg Se/L (evaporative concentration) is discharged to the Salton Sea (a 230,000-acre terminal sink). Impounded water at the Salton Sea averages about 1.5 µg Se/L (3× normal for saline sinks), and sediments contain 0.2–3.3 mg Se/kg. Algae averaged 0.9 mg Se/kg (3× normal), other food-chain organisms averaged 2–13 mg Se/kg (1–7× normal), and avian eggs averaged 4–7 mg Se/kg (2–4× normal). A monitored population of black-necked stilts, which averaged 6 mg Se/kg in their eggs, exhibited a slight depression (5.6 percent) in reproductive performance. That degree of impairment was consistent with known exposure-response curves for stilts in nature and has tentatively been attributed to selenium toxicity. At such a small effect level, however, larger sample sizes of monitored nests will ultimately be required to conclusively evaluate preliminary findings (Setmire et al. 1990; Westcot et al. 1990a; Setmire et al. 1993; Bennett 1997).

Because stilt eggs collected during Bennett's (1997) study came from numerous locations, only a few of which constituted Salton Sea "shoreline" sites, it is not precisely known what concentration of selenium in water can be associated with this case study. It is, however, highly probable that the birds in this study were predominantly using wetlands with selenium concentrations in water in the range of 2-10 µg/L.

(11) Selenite-Treated Lakes in Sweden—Eleven lakes were treated with selenite in an attempt to mitigate high levels of mercury in edible fish (Lindqvist et al. 1991; Paulsson and Lundbergh 1991). In fact, the selenite was highly success-ful in lowering the bioaccu-mulation of mercury in the fish, but it also had some detrimental effects of its own (Lindqvist et al. 1991; Meili 1996). Treatments consisted of a leachable

rubber matrix containing sodium selenite suspended in a sack 1–2 m below the lake surface for 2 years. The selenium-free rubber skeletons, remaining after continuous leaching of sodium selenite, were removed (and replaced?) at intervals of several months. During the first year of treatments (beginning in September 1987), the doses were calibrated for a target lake concentration of 3–5 μg Se/L (lakes initially contained about 0.1 μg Se/L). On average, the target concentration was achieved at most of the lakes, although selenium levels as high as 25–35 μg/L were measured within 100 m of the leach sacks. Four lakes never exceeded about 2.6 μg/L average waterborne Se. Because mitigation of mercury residues was as pronounced in the four lower selenium lakes as in the target concentration (3–5 μg/L) lakes, the dosing was adjusted in the second year of treatment for a target lake concen-tration of 1–2 μg Se/L.

Prior to treatment, pike muscle concentrations of selenium averaged 0.7–2.4 mg/kg (1.3 mg/kg grand mean) in the 11 lakes. After the first year of treatment, muscle concentrations averaged 0.9–2.3 mg Se/kg (1.6 mg/kg grand mean), and after 2 years treatment, they averaged 2.8–7.4 mg Se/kg (4.6 mg/kg grand mean). There was no evidence of catastrophic declines of pike populations in any of the lakes. Muscle concentrations of selenium in perch fry averaged 0.8–2.0 mg/kg prior to treatment and 6–36 mg/kg after 1 year. By the end of the second year of treatment, researchers were unable to find any perch fry in four of the lakes and had a substantially reduced catch from a fifth lake. Among these five lakes, concentrations in perch muscle had averaged 6.9–36 mg Se/kg (23 mg/kg grand mean) at the 1-year sampling point; by comparison, among the other six lakes, concentrations in perch muscle had averaged only 6–18 mg Se/kg (12 mg/kg grand mean). At the end of 2 years, perch muscle con-centrations in the six lakes that still had reproductively viable perch populations averaged 6.9–26 mg Se/kg (15 mg/kg grand mean).

Paulsson and Lundbergh (1991, p. 837) concluded that, "There seems to be a dependence between the selenium concentration in fish tissue and a maximum concentration in lake water being less tank [sic] 2 μg/L." Although the language of the report was confusing, the authors seemed to be suggesting that 2 μg/L waterborne Se was a threshold point for avoidance of excessive tissue selenium in perch. Lindqvist et al. (1991, p. 214) more clearly stated, "It is important not to dose so that Se concentrations in water rise above about 1 to 2 μg Se/L." They also concluded that the observed reproductive failures in perch were due to ". . . the selenium treatment and not to any of the other factors . . .." More recently, Meili (1996), as well as Nuutinen and Kukkonen (1998), concluded that the selenium treatments caused adverse effects on fish populations. For example, Meili (1996) noted the correlation between selenium dosing, elevated tissue selenium, and disappearance of perch fry and concluded, ". . . high Se levels can be linked to high doses, and furthermore to fatal ecological consequences. The results suggest that a selenium concentration of only 3 μg/L can seriously damage fish populations."

Table 33 represents a collection of the *minimum* estimates for real-world (*in situ) toxic exposures* that have been documented for natural populations of fishes and birds. These values do not necessarily correspond with true threshold points (e.g., EC10's) for toxicity because, in some cases, exposure levels are clearly above threshold regions. They do, however, provide field-validated ceilings for the exposure intervals within which true threshold points occur. Over time, the pro-gressive accumulation of data from field cases has lowered these ceilings so that most of the values are now only 5–10× normal back-ground concentrations for selenium. Data from field cases also reveal that selenium exposures in the range of 30–50× normal levels are almost certain to cause widespread *severe* adverse biological effects.

Table 33.—Summary from field cases of minimum selenium contamination having adverse effects on natural fish and wildlife populations

Matrix	Minimum toxic concentrations	Response variable	Study site
Water, µg/L	2–10	Avian reproduction	Salton Sea, CA
	2.0	Fish reproduction	Sweden (five different lakes)
	2.6–5	Fish population collapse	Martin Lake, TX
	2.6–18	Avian reproduction	Tulare Basin, CA
	3–4	Fish sublethal effects	Belews Lake, NC, Hwy 158 Arm
	7.5–17.5	Avian reproduction	Chevron Marsh, CA
Sediment, mg/kg dw	0.9	Avian reproduction	Tulare Basin, CA
	1–3	Avian reproduction	Salton Sea, CA
	<3	Fish sublethal effects	Belews Lake, NC, Hwy 158 Arm
	3–5	Fish survival and reproduction	Hyco Res., NC
Food chain fauna, mg/kg dw	2.9	Avian reproduction	Tulare Basin, CA
	4–8	Fish sublethal effects	Belews Lake, NC, Hwy 158 Arm
	3.1 (pileworms)	Avian reproduction	Salton Sea, CA
Fish muscle, mg/kg dw	7–9	Sublethal effects	Belews Lake, NC, Hwy 158 Arm
	<(10–20)	Teratogenesis	Belews Lake, NC
	6.9–36 (means), 23 grand mean	Reproductive failure	Sweden (five different lakes)
Fish whole body, mg/kg dw	10–20	Teratogenesis	Belews Lake, NC
Fish gonads, mg/kg dw	17	Population collapse	Martin Lake, TX
Fish liver, mg/kg dw	25–30	Sublethal effects	Belews Lake, NC, Hwy 158 Arm
Bird liver, mg/kg dw	40–50	Adult mortality	Ouray NWR, UT
Bird muscle, mg/kg dw	4–8	Adult mortality	Ouray NWR, UT
Bird egg, mg/kg dw	4.2–9.7	Hatchability	Tulare Basin, CA
	6 (mean)	Hatchability	Salton Sea, CA
	10	Hatchability	Kesterson, CA
	11 (mean)	Hatchability	Martin Lake, TX

Abiotic Factors Affecting Bioavailability

Water

Selenium commonly occurs as a mixture of several chemical species in natural waters, although two inorganic chemical species, selenite and selenate, are usually the predominant forms (Masscheleyn and Patrick 1993).

Normal background concentrations of selenium in uncontaminated freshwater ecosystems have been estimated (in µg/L) as 0.25 (Wilber 1980), 0.1–0.3 (Lemly 1985b), 0.2 (Lillebo et al. 1988), and 0.1–0.4 (average <0.2, Maier and Knight 1994). Even in California, a State heavily influenced by agricultural mobili-zation of selenium, the median concentration in 226 streams was 0.4 µg/L (Westcot et al. 1990b). A survey of inland saline lakes in Oregon, California, Nevada, and Utah yielded

a geometric mean concentration for selenium of 0.6 µg/L (Westcot et al. 1990a). Inland saline lakes of the western United States are subject to pronounced evaporative concen-tration of dissolved constituents and thus are likely to have the highest natural concentra-tions of selenium. Behra et al. (1993) considered 1 µg/L selenium in running waters in Switzerland to be "higher than concentrations in moderately polluted waters."

An important factor confounding interpre-tation of field data for waterborne selenium is the differential partitioning of selenium mass loads between the water column and other compartments of an aquatic ecosystem. Partitioning ratios can be strongly influenced by the overall biotic productivity of a water body. In highly productive waters, less dissolved selenium is left in the water column even though food-chain exposure of fish and wildlife may be substantial. Therefore, low waterborne selenium concentrations can indicate either low mass loading (low risk) or high biotic uptake (high risk). This inter-pretive problem can be partially ameliorated by measuring total recoverable selenium (i.e., unfiltered samples) rather than dissolved selenium (filtered samples) (Skorupa and Ohlendorf 1991). Total recoverable selenium includes suspended detrital particulate matter, a function of biotic uptake, and thus more accurately reflects the total mass load of selenium fluxing through a water column.

Estimates of normal *background* concentrations for waterborne selenium are unlikely to differ significantly regardless of whether one measures dissolved or total recoverable selenium. For contaminated waters, however, the differences between these two measures increase with the degree of eutrophication. For eutrophic, shallow waters, such as evaporation ponds, the differences can be pronounced. For one evaporation pond in California, a split sample yielded 7 µg/L dissolved Se but 25 µg/L total recoverable Se (Fujii 1988). The higher value was much more consistent with the >20 mg Se/kg found in bird eggs at the pond (Skorupa and Ohlendorf 1991). Threshold values presented below based on toxicity tests or drinking-water exposure refer to dissolved selenium, whereas those based on field sampling for bioaccumu-lative risk refer to total recoverable selenium; these two types of values should not be equated to each other. Many of the total recoverable selenium values would be lower if expressed on a dissolved basis (e.g., Peterson and Nebeker 1992). To assess biotic risk for selenium toxicity, unfiltered samples of water should be analyzed for both particulate and dissolved selenium (ERG 1998); this is referred to as "total recoverable" selenium in this document and is roughly equivalent to EPA's "acid soluble" selenium.

Waterborne selenium, ***per se,*** is not very toxic to fish and wildlife. When water is the only exposure route (e.g., standard aquatic toxicity test), toxic thresholds for selenium are generally >1,000 µg/L (≈10,000× normal) for adult fish. SJVDP (1990), Maier et al. (1987), and EPA (1987) provide good reviews of aquatic toxicity test results for fish and wildlife populations. Chronic toxicity in experimental animals and livestock has been observed when drinking water exceeds 2,000 µg Se/L (NRC 1980). Drinking water containing 2,200 µg/L selenomethionine appeared to suppress certain aspects of the mallard immune response (Fairbrother and Fowles 1990). A family of Ute Indians in Colorado suffered hair loss, nausea, and fatigue after drinking well water that contained 9,000 µg Se/L (Anonymous 1962). Eggs and larvae of both fish and amphibians may be the most sensitive vertebrate life stages to waterborne selenium *per se*. Toxicity testing revealed that the LC50 for eggs of the narrow-mouthed toad was only 90 µg Se/L. Rainbow trout sac fry are adversely affected by about 50–100 µg/L waterborne Se (Birge et al. 1979). Hamilton and Wiedmeyer (1990) reported that 70 µg/L waterborne Se (inorganic mixture)

was sufficient to reduce the 90-day survival of larval chinook salmon (exposed as eyed eggs and as larvae).

Much lower concentrations of selenium in water can be bioaccumulated to toxic levels in fish and wildlife via dietary exposure to the aquatic food chain. Field cases of selenium poisoning in fish and birds have been docu-mented for waters averaging as little as 1–10 µg Se/L (see "Field Cases" section). NIWQP data from 23 study areas in 13 western States illustrate the effect of bioaccumulation on bird eggs. Out of 10 study areas where the 75th percentile value for selenium in surface waters was 2 µg/L or less, none (0 percent) had a 75th percentile value for selenium in bird eggs that exceeded the embryotoxic threshold. Out of nine study areas where the 75th percentile for surface waters was 3–10 µg Se/L, three (33 percent) had 75th percentile values for bird eggs above the embryotoxic threshold. Of the four study areas where the 75th percentile for surface waters was >10 µg Se/L, all (100 percent) had 75th percentile values for bird eggs above the embryotoxic threshold (Seiler and Skorupa 1995).

Bluegill fish residing for 319 days in outdoor experimental streams supplemented with 2.5 µg Se/L produced larvae with elevated frequencies of edema, lordosis, and hemorrhaging (Hermanutz et al. 1990). With reference to larval edema, the 2.5-µg/L concentration was about the EC10, and a 10-µg/L treatment was about the EC95. Seven other recent reports that estimate waterborne thresholds for food-chain-mediated toxicity to fish and wildlife are summarized in Maier and Knight (1994). All the reports conclude that the threshold is about 3 µg/L or less, even though no two of them had used the same combination of field data, clinical data, and predictive modeling.

In California, human health advisories have been issued for localities where edible tissues in fish or wildlife contained >2.0 mg Se/kg on a wet weight basis (Fan et al. 1988). Those advisories recommended zero consumption of such tissues by pregnant woman and children (<15 years old). In 1975, Kaiser et al. (1979) collected trout that had been stocked 2 years earlier in a Wyoming lake containing about 2 µg/L waterborne Se. The fillets (with skin) that Kaiser et al. (1979) harvested contained about 2 mg Se/kg (wet weight).

The speciation of waterborne selenium can substantially affect the potential for bioaccumulation in fish and wildlife tissues, at least at relatively high waterborne concentrations. Waterborne selenite (coal fly-ash effluent and

Summary: Effects of selenium in water

Interpretive guidance	Waterborne selenium concentration (µg/L)
True background, freshwater environments	0.1–0.4 (typically <0.2)
Approximate background, California streams (maximum probably not natural)	<0.2–73 (median = 0.4)
Approximate background, Western inland saline lakes (maximum probably not natural)	<0.2–490 (geometric mean = 0.6)
Validated LOAEL's for fish and wildlife via bioaccumulation	1–3
10× normal background averages	<2–6
Toxicity-test LOAEL for fish and amphibian eggs/larvae (waterborne exposure only)	50–100
Experimental LOAEL for drinking water (mallards)	2,000 (seleno-methionine)
Validated LOAEL for drinking water (humans)	9,000 (inorganic)

oil refinery wastewater) is more readily bio-accumulated than waterborne selenate (irrigation wastewater) (e.g., Besser et al. 1993). For example, in California, oil refinery wastewater containing 10–30 µg Se/L produced the same tissue concentrations of selenium as irrigation wastewater containing 330 µg Se/L. Both sources of water produced water boatmen (a type of insect) averaging about 20 mg Se/kg (10× normal) and black-necked stilt eggs averaging about 25 mg Se/kg (10× normal) (SJVDP 1990; CH2M Hill 1994, 1995). Thresholds for toxicity, however, have been observed in the 1–3 µg/L range for both selenate- and selenite-dominated waters. Possibly, at these lower concentrations, selenate reduces readily to the more rapidly metabolized selenite, but higher concentra-tions of selenate may overwhelm the reduction pathways.

Bottom Sediment

Currently, there is little empirical basis for assessing fish and wildlife risk as a function of sediment concentrations of selenium. Only one study has matched sediment concentra-tions of selenium with concentrations in a benthic invertebrate from the same sediment— an obvious first step for developing empirically based risk thresholds (ERG 1998). One post hoc comparison of means for unmatched samples showed a poor correlation between selenium concentrations in sediments and benthic invertebrates
(Van Derveer 1995).

Sediments present formidable methodological and statistical obstacles for data interpretation. Methodologically, the depth to which a sediment sample is collected will strongly influence the results. Even when sampling depth is standardized between studies, results will vary depending on whether the analysis is performed on whole-bed samples or some size-fraction subsample (particle fractions <0.062 mm and <2.0 mm are frequently used). Precisely which sampling method a particular study employed is not always clear in the literature (SJVDP 1990). Furthermore, there is an immense amount of spatial variability in measures of sediment selenium. Samples collected only a few meters apart can yield substantially different results (e.g., Chilcott et al. 1990; Setmire et al. 1993; Wu et al. 1995). Thus, in comparison to water or biotic tissues, many more samples of sediment are required to adequately characterize selenium content. This statistical obstacle requires careful consideration when designing a plan for sediment sampling. Rarely will a simple random sampling design be adequate.

Because of the high variance and generally inadequate sampling designs of many existing studies, the maximum sediment values reported for selenium may have more inter-pretive value than the means. As a general rule, anytime the maximum selenium con-centration in sediments exceeds 5 mg/kg, further investigation is strongly warranted (GLSAB 1981). To the extent possible, the guidelines provided here are based on samples no deeper than the upper 3 inches of sediment and on chemical analyses of whole-bed samples.

In a selenium-deficient landscape (Finland), sediments of lakes usually contained <0.1 mg Se/kg, typically 0.04–0.08 mg/kg (Makela et al. 1995). The average concentrations of selenium for sediments in selenium-normal environ-ments are usually <1.0 mg/kg (Maier and Knight 1994). The 90th percentile value for freshwater sediments in Texas was 1.9 mg/kg (Davis 1987), but the proportion of contami-nated sites included in this statewide survey is unknown. SJVDP (1990) estimated normal background values to average as much as about 0.5 mg Se/kg in the sediments of a naturally seleniferous region of California. Martin and Hartman (1984) reported mean sediment concentrations for selenium of
0.89 and 0.52 mg/kg in pothole and riverine wetlands located primarily in North and South Dakota (two relatively seleniferous States). At 25 U.S. Department of the Interior study sites in the Western United States, median selenium

concentrations were 0.5 and 0.3 mg/kg, respectively, in fine and coarse sediment fractions (Presser 1995).

Based on a review of 27 eclectic studies, Van Derveer and Canton (1997) concluded that sedimentary selenium is a reliable predictor of adverse biological effects and that a preliminary toxic threshold existed at about 2.5 mg Se/kg (the 10th percentile for effects). They also noted that, in the literature they reviewed, adverse effects were always observed at selenium concentrations greater than 4.0 mg/ kg in sediments. For a set of 13 independent evaporation ponds in the Tulare Lake Basin of California, Skorupa et al. (unpub. data) found that mean concentrations of selenium in pond sediments correlated reasonably well with mean concentrations of selenium in the eggs of black-necked stilts nesting at the ponds (r =0.777, N=25, p<0.01). All ponds that averaged ≥1 mg Se/kg in sediments yielded stilt eggs averaging >6 mg Se/kg (the embryo-toxicity threshold). All ponds that averaged <0.4 mg Se/kg in sediments yielded stilt eggs averaging <6 mg Se/kg. Based on the relationship between sediments and stilt eggs, eggs would be expected to exceed an average of 20 mg Se/kg (≈85 percent risk level for avian populations, i.e., high risk) when sediments exceeded 2.7 mg Se/kg. A survey of fish in California's San Joaquin River system found elevated concentrations of whole-body selenium (>4 mg/kg) in most mosquitofish at sites containing 0.2–1.9 mg Se/kg in sediments. At sites having <0.13 mg Se/kg in sediments, selenium concentrations in mosquitofish were generally at background levels (<2 mg/kg) (USFWS 1990b).

Summary: Effects of selenium in sediment

Interpretive guidance	Sediment Se concentration (mg/kg)
Selenium-deficient environments	<0.1
Approximate background, Se-normal freshwater environments	0.2–2.0 (typically <1.0)
Approximate background, Texas freshwater environments	<1.9 (90th percentile)
Maximum zero-response (NOAEL) boundary for birds nesting at shallow terminal ponds (population basis)	0.4
Minimum total-response (EC100 boundary for birds nesting at shallow terminal ponds (population basis)	1.0
EC10 for fish and birds in a variety of freshwater aquatic systems (population basis)	2.5
EC100 for fish and birds in a variety of freshwater aquatic systems (population basis)	>4.0
10× normal background averages	3–5
Various regulatory clean-up criteria for soils	3–10

Soil

The presence of selenium in geologic formations does not mean it is present in toxic amounts in the soils derived from these strata. The solubility of selenium in soils depends on pH, moisture, oxidation-reduction conditions, and the degree of aeration. In areas where annual precipitation exceeds 20 inches and there is deep percolation, selenium is slowly leached and does not become concentrated in the soil. However, ground water in such areas may be contaminated from leached selenium. Where rainfall is less than 20 inches and where the soil is alkaline and generally well aerated (as in much of the Western United States), selenium in the soil may be mobilized in its oxidized, readily soluble, selenate form and may be concentrated near the surface (Hedlund 1993).

The amount of selenium in the soil is not a reliable indicator of how much is available to plants or how much can be leached by deep percolation. In moist, acidic, and reducing soils, selenium is present as insoluble selenides, elemental selenium, or insoluble pyritic selenides and not available to plants. Under dry, alkaline conditions in seleniferous soils, microorganisms produce selenates and organic selenium complexes which are very soluble. Excess precipitation or irrigation water draining these soils would leach soluble selenites and selenates and also carry dissolved and suspended organic forms of selenium (Hedlund 1993).

Various regulatory clean-up criteria for selenium in soils typically range from about 3 to 10 mg/kg (Beyer 1990).

Biotic Effects

Interpretation of field data for biota can be confounded by a sampling bias that favors "survivors." Most biological sampling techniques are designed to sample live biota. In contaminated environments, live biota represent "survivors" and are potentially biased subsets of the study populations with regard to selenium exposure and/or sensitivity.

Plants

Selenium-Concentrating Vegetation.—Different plant species have widely varying abilities to take selenium from the soil, accumulate it, and tolerate it. Common types of selenium-concentrating vegetation include *Astragalus* (loco weed and milkvetch—24 species), *Machaeranthera* (thistle), *Haplopappus* (goldenweed), and *Stanleya* (mustard). These plant species have an extraordinary ability to accumulate selenium and can achieve selenium concentrations of hundreds or even thousands of milligrams per kilogram, dry weight.

Primary producers are the foundation for most food chains supporting fish and wildlife populations. In aquatic ecosystems, algae serve as the primary source of energy assimilation and as the base of most aquatic food chains (Ogle et al. 1988). Aquatic macrophytes are important in chemical cycling and as a major input source for detrital food chains.

Filamentous algae in California's San Joaquin River system contained 0.1–0.4 mg Se/kg at sites with <1.0 µg/L waterborne Se (USFWS 1990b; Saiki et al. 1993). Algae in an uncon-taminated San Joaquin Valley freshwater marsh averaged <0.5 mg Se/kg (Saiki and Lowe 1987; Schuler et al. 1990). Background concentrations of selenium in aquatic macro-phytes usually average <1.5 mg/kg (Maier and Knight 1994). Aquatic macrophytes in an uncontaminated San Joaquin Valley freshwater marsh averaged <1.0 mg Se/kg (Saiki and Lowe 1987; Hothem and Ohlendorf 1989; Schuler et al. 1990). Terrestrial plants on nonseleniferous soils usually average <0.25 mg Se/kg (Girling 1984). The two dominant herbaceous plants collected from a normal-selenium (0.25 mg Se/kg soil) reference site in the San Joaquin Valley averaged 0.3 and 1.4 mg Se/kg (Wu et al. 1995). On seleniferous soils, non-accumulator plants may contain 1–200 mg Se/kg, and selenium-accumulator plants contain even higher concentrations (Girling 1984).

No studies in the literature report selenium toxicity thresholds for plants based on exposure in nature (i.e., based on field data). In standard toxicity tests, sublethal effects of selenium exposure are initially observed for green algae at waterborne concentrations of 10 µg/L (selenate) and 75 µg/L (selenite) (Vocke et al. 1980; Foe and Knight 1986). Growth of the green alga *Selenastrum capricornutum* was significantly reduced at tissue concentrations of 4 mg Se/kg (Williams et al. 1994). Bioaccumulation of selenate (but not selenite or selenomethionine) by algae is strongly influenced by waterborne sulfate concentrations (e.g., Kiffney and Knight 1990; Williams et al. 1994). The 10-µg/L threshold for sublethal effects of waterborne selenate occurred in both high-

sulfate and low-sulfate waters**:** algal tissue accumulated 4 mg Se/kg in high-sulfate waters and 17 mg Se/kg in low-sulfate waters (Williams et al. 1994). Blue-green algae are generally less sensitive than green algae to selenium exposure (Maier et al. 1987; Kiffney and Knight 1990), and even among green algae there are extreme species differences in sensitivity. Sublethal thresholds in green algae have ranged from 10 to 300 µg/L waterborne Se (Vocke et al. 1980; EPA 1987). Toxicity profiles for aquatic macrophytes are not known, but the sublethal toxicity thresholds for aqua-cultured lettuce were 200 µg/L waterborne selenate and 3,000 µg/L waterborne selenite. For both selenite and selenate exposure, the threshold tissue concentration for lethal effects was 800 mg Se/kg (Berry and Savage 1986). Apparently, irrigation water with ≤50 µg/L total Se is considered by agronomists to be protective of all crop plants (e.g., Eisler 1985).

The quality of habitat for fish and wildlife is closely linked to particular plant communities. Therefore, selenium contamination could impact fish and wildlife populations indirectly if plant communities are altered by its toxic effects. However, there are no documented field cases of this type of indirect effect on fish and wildlife populations. Additionally, because of the absence of threshold data for ecologically important endpoints in nature, no regulatory values have been established for aquatic toxicity of selenium to plants (EPA 1987). For all practical purposes regarding protection of fish and wildlife populations, the direct toxic effects of consuming selenium-contaminated plants are apparently more important than indirect ecological effects from changes in plant communities. Fish and wildlife risk thresholds for dietary exposure to selenium are addressed in the summaries for vertebrate animals presented below.

Summary: Effects of selenium on plants

Interpretive guidance	Plant Se concentration (mg/kg)
Background, freshwater algae	0.1–1.5 (typically <0.5)
Background, freshwater macrophytes	0.1–2.0 (typically <1.5)
Background, terrestrial plants on nonseleniferous soil	<0.01–0.6 (typically <0.25)
Experimental LOAEL for sublethal effects (growth), algal tissue	4.0
Experimental LOAEL for sublethal effects (growth), macrophyte (lettuce) tissue	250
Experimental LOAEL for lethal effects, macrophyte (lettuce) tissue	800
	Waterborne Se exposure (µg/L)
Toxicity-test LOAEL for sub-lethal effects on green algae	10–300 selenate 75 selenite
Toxicity-test LOAEL for sub-lethal effects on blue-green algae	100 seleno-methionine 3,000 selenate 3,000 selenite
Toxicity-test LOAEL for sublethal effects on a macrophyte (lettuce)	200 selenate 3,000 selenite
Irrigation water standard to protect crop plants	≤50 total

Invertebrates

Invertebrate populations are important sources of food for most species of fish and wildlife. Many species of fish and wildlife require high-protein diets for optimal reproduction, and the invertebrate component of the diet is often the

principal source of protein. Consequently, selenium-induced alterations of invertebrate density or community structure could have indirect ecological impacts on fish and wildlife populations.

For aquatic invertebrates, Maier and Knight (1994) report a range of 0.5–2.0 mg Se/kg as the national background concentrations. In an Se-normal aquatic ecosystem in Colorado, Birkner (1978) reported average invertebrate concentrations of 2.3–4.2 mg Se/kg. At an uncontaminated San Joaquin Valley wetland, invertebrates averaged 0.9–3.0 mg/kg (Saiki and Lowe 1987; Hothem and Ohlendorf 1989; Schuler et al. 1990). Small experimental freshwater ponds serving as control macro-cosms (waterborne Se <1.0 µg/L) contained invertebrate taxa with selenium concentra-tions averaging 1.4–3.8 mg/kg (Crane et al. 1992). In the San Joaquin River system, midges and amphipods averaged 0.4–1.5 mg Se/kg, and crayfish averaged 0.5–0.9 mg Se/kg at sites with ≤1.0 µg/L waterborne Se (Saiki et al. 1993). At Se-normal sites of the lower Colorado River system, crayfish contained 0.6–2.5 mg Se/kg and usually averaged ≤1.5 mg/kg (Welsh and Maughan 1994).

For terrestrial invertebrates (grasshoppers and mantids) at two reference sites near the former Kesterson Reservoir, Wu et al. (1995) reported average concentrations of <1.0 mg Se/kg. Grasshoppers and beetles collected at ref-erence agroforestry plantations in the San Joaquin Valley contained 1.3–2.5 mg Se/kg (SJVDP 1990).

No studies were found in the literature that report selenium toxicity thresholds for invertebrates based on exposure in nature (i.e., based on field data). It has been established that tissue concentrations of selenium in field-collected aquatic invertebrates are strongly related to waterborne concentrations of selenium (Birkner 1978; Wilber 1980; Lillebo et al. 1988). Crayfish caged in the ash-pit drain of a Wisconsin power plant bioaccumulated 30 mg Se/kg in the hepato-pancreas and had significantly altered respiration rates; the ash pit effluent also had high concentrations of chromium, iron, and zinc (Magnuson et al. 1980). No major effects on benthic macroinvertebrate communities were detected in experimental freshwater ponds treated with 2, 10, and 25 µg/L inorganic Se (60:40 ratio selenate to selenite) (Crane et al. 1992). Abundance data for midge larvae, however, are suggestive enough to warrant further study at doses above 2 µg/L waterborne Se. In standard laboratory toxicity tests, 4 µg/L waterborne selenomethionine was acutely toxic to an amphipod. Many factors influence toxicity-test results, including water chemistry, species tested, and life-stage tested, but the lowest waterborne thresholds for acute toxicity are approximately 200 µg/L selenite and 500 µg/L selenate (EPA 1987; Maier et al. 1987; Ingersoll et al. 1990). Lowest thresholds for chronic toxicity occur at approximately 25–100 µg/L for selenite or selenate and perhaps at <0.5 µg/L waterborne selenomethionine (Johnston 1987; EPA 1987; Boyum and Brooks 1988; Ingersoll et al. 1990).

Experimental studies of dietary toxicity are rare. Amphipods showed no adverse effects from dietary exposures to algae containing as much as 300 mg Se/kg (Foe and Knight 1986). Dietary exposure of larval midges to algae containing ≥2.1 mg Se/kg significantly inhibited growth. The inhibited larvae contained ≥2.5 mg Se/kg in their tissues (Malchow et al. 1995). Alaimo et al. (1994) exposed midge larvae to diets based on naturally contaminated widgeon grass (*Ruppia*) detrital substrates from evaporation ponds in the Tulare Basin, California. Alaimo et al.'s 14-day egg-to-prepupation feeding study produced results very similar to those of Malchow et al. (1995). Detrital substrates containing as little as about 2 mg Se/kg significantly inhibited the growth of midge larvae even though the associated selenium concentrations in larval tissue were in some cases <4 mg/kg (Alaimo et al. 1994). Tissue concentrations of 15 and 32 mg Se/kg in

amphipods were associated with reduced growth and reproduction, respectively (Ingersoll et al. 1990).

There is almost no selenium toxicity data for terrestrial invertebrates. Aphids are reportedly sensitive to selenium, and spiders and mites have been controlled by commercial selenium insecticides (Trelease 1945). Simmons et al. (1988) reported that selenite in the drinking water of house flies (4,000 µg/L) caused 28 percent mortality after 12 days.

Among microinvertebrates, such as protozoans, an extremely wide range of toxicity-test results has been reported (Sanders and Gilmour 1994). Toxic thresholds in some taxa are as high as 10,000 µg/L selenite and in other taxa are as low as 3 µg/L selenite (inhibited growth of a heterotrophic flagellate). In microcosm studies, protozoan diversity (but not total biomass) was affected by 21-day exposures to 20–160 µg/L selenite.

The field applicability of guidelines generated by toxicity tests or controlled feeding trials is highly uncertain. Populations of brine shrimp in nature exhibit substantial variability in resistance to selenium toxicity (Freeman et al. 1987). Such findings suggest a potential in nature for rapid selection of populations that are more resistant than those used in tests. On the other hand, tests that replicate drainwater ionic chemistry and the presence of multiple trace elements suggest that toxicity thresholds in nature may be lower than in standard freshwater single-element toxicity tests (Dwyer et al. 1992; Naddy et al. 1995).

There are no documented field cases of fish and wildlife populations being affected adversely by selenium-induced alterations of invertebrate density or community structure. As noted above for plants, the direct toxic effects of consuming selenium-contaminated invertebrates are apparently more important than any indirect ecological effects. Fish and wildlife risk thresholds for dietary exposure to selenium are addressed in the summaries for vertebrate animals presented below.

Summary: Effects of selenium on invertebrates

Interpretive guidance	Invertebrate Se concentration (mg/kg)
Background, aquatic invertebrates	0.4–4.5 (typically <2.0)
Background, terrestrial invertebrates	<0.1–2.5 (typically <1.5)
Experimental LOAEL for sublethal effects (growth), midge larvae and amphipod tissue concentrations	2.5–15
Experimental LOAEL for sublethal effects (respiration rate) in crayfish	30 (hepato-pancreas)
Experimental LOAEL for reproductive effects, amphipod tissue concentration	32
	Dietary Se exposure (mg/kg)
Experimental LOAEL for sublethal effects (growth) in midge larvae	2.1
Experimental NOAEL for acute toxicity in amphipods	300
	Waterborne Se exposure (µg/L)
No clear community-level effects on benthic macroinvertebrates, outdoor macrocosm studies	25 (inorganic mixture)
Altered protozoan species diversity	20–160 (selenite)
Toxicity-test LOAEL's for acute toxicity, in midge larvae and amphipods	4.0 (seleno-methionine) 200 (selenite) 500 (selenate)
Toxicity-test LOAEL for sublethal (growth) effects on protozoans	3.0 (selenite)
Toxicity-test LOAEL's for chronic toxicity in midge larvae and amphipods	<0.5(?) (seleno-methionine) 25–100 (selenite) 25–100 (selenate)
Experimental LOAEL for drinking water toxicity in house flies	4,000 (selenite)

Fish

National and global monitoring programs have revealed that most species of fish average less than 4 mg Se/kg on a whole-body basis (e.g., Walsh et al. 1977; Schmitt and Brumbaugh 1990; Jenkins 1980). These surveys include data from both contaminated and uncontaminated sites and therefore include maximum values that do not repre-sent true "background." For comparison, Lemly (1993b) recently reported whole-body concentrations of selenium for more than
20 species of fish sampled at two confirmed Se-normal (waterborne Se ≤1 µg/L; Lemly 1985b) lakes in North Carolina. With few exceptions, the species averages were below
2 mg Se/kg. At several verified Se-normal sites in the San Joaquin River system of California, Saiki (1989) and Saiki et al. (1993) also found that mosquitofish, carp, bluegill, and largemouth bass averaged <2 mg/kg whole-body Se. Sunfish sampled at confirmed Se-normal sites in the lower Colorado River system averaged 1.6–2.4 mg/kg whole-body Se (Welsh and Maughan 1994). Controlled dietary exposures as high as 2 mg Se/kg in experimental studies consistently yield whole-body selenium concentrations <2.0 mg/kg (e.g., Ogle and Knight 1989; Hamilton et al. 1990; USFWS 1990b; Cleveland et al. 1993; Coyle et al. 1993; Lorentzen et al. 1994). Dietary exposures in this range would be typical of Se-normal environments (see summary tables for plants and invertebrates above). Background concentrations of selenium in skeletal muscle, gonads, and eggs also tend to average 2–4 mg/kg or less (e.g., Baumann and Gillespie 1986; Coughlan and Velte 1989; Crane et al. 1992; Hermanutz et al. 1992; Hamilton and Waddell 1994). Back-ground concentrations for hepatic selenium have been reported to range from 2 to 8 mg/ kg but are usually <5 mg/kg (Sorensen 1988; USFWS 1990b; Hermanutz et al. 1992; Lorentzen et al. 1994).

Lemly (1993a, 1996a) provided excellent reviews of selenium toxicity thresholds for fish. Based on that review, Lemly concluded that the most precise way to assess risks associated with exposure of fish to selenium is to measure selenium levels in gravid ovaries. More recently, Hamilton and Waddell (1994) reviewed the "gonad/egg" literature and concluded that fish gonads and eggs normally average 2–3 mg Se/kg and that 16–18 mg/kg was the current LOAEL for warm-water fish. This LOAEL, however, does not indicate the *true* threshold for adverse effects because it was associated with at least 50–80 percent reproductive impairment (Hermanutz et al. 1992). The lowest concentration of selenium in gonads and eggs reported for a case of *total* reproductive failure in fish (IC100) is 25–
30 mg/kg (Crane et al. 1992). Considering selenium concentrations in bird eggs, Heinz et al. (1989) found that the ratio between the threshold of total reproductive failure (IC100) and the threshold for reproductive impairment (≈IC10) was about 3–3.5 to 1. (See terato-genesis response table for black-necked stilts presented below.) Since background levels of selenium in eggs are similar for birds and fish (Hamilton and Waddell 1994; Skorupa and Ohlendorf 1991), and exposure-response curves for embryo teratogenesis are broadly similar (Lemly 1993b, p. 201: "It therefore appears that the teratogenic effects of selenium in natural populations of fish and aquatic birds are essentially the same."), it seems likely that the IC100 to IC10 ratio (3–3.5×) is also similar and that the threshold region for reproductive failure in *sensitive* species of fish extends as low as 7–10 mg Se/kg in gonads and eggs. Crane et al.'s (1992) study is consistent with this con-clusion because perch averaging about 8 mg Se/kg in gonads had slightly lower repro-ductive output than controls. As is often the case, though, Crane et al.'s study did not have the statistical power to conclusively test for the presence of a true threshold effect.

For bluegill, a sensitive species, Coyle et al. (1993) didn't find statistically significant reproductive impairment until the gonads and eggs had accumulated about 40 mg Se/kg. Because controls in Coyle et al.'s study exhibited

only 20 percent reproductive success, the study did not have the statistical power to detect anything but catastrophic reproductive impairment. If it is assumed that Coyle et al.'s treatment group of bluegill that had 40 mg Se/kg in gonads and eggs was relatively close to the IC100 threshold point, then the projected IC10 (based on a 3–3.5 ratio) would be 11–13 mg Se/kg.

At least two studies have related levels of 30–35 mg/kg dietary selenium (organic) to total reproductive failure (Woock et al. 1987; Coyle et al. 1993). Since selenium concentra-tions in gonad and egg tissues are propor-tional to dietary exposure, this suggests that the threshold dietary exposure to organic selenium (predominantly selenomethionine and selenocysteine) for reproductive impair-ment (assuming only parental exposure) is no higher than about 10 mg/kg (30–35 divided by 3–3.5). Results from Woock et al. (1987) are consistent with this estimate. In Woock et al.'s study, a diet of 13 mg Se/kg (organic) resulted in an "apparent" 8 percent reproductive depression. Thus, when adult female fish of sensitive species are exposed to dietary Se ≥ approximately 10 mg/kg, they are likely to produce eggs that contain enough selenium to impair survival of at least some offspring, even if the offspring themselves are never exposed to elevated dietary selenium. Conversely, studies of offspring from uncontaminated eggs suggest that larval fish are very sensitive to direct dietary selenium. Dietary exposures as low as 3–8 mg Se/kg (organic) have been demonstrated to impair normal juvenile survival and/or development in salmonids (Hamilton et al. 1990) and centrarchids (Cleveland et al. 1993; Lemly 1993c). In a more recent study, larval razorback suckers fed field-collected seleniferous invertebrates from the Green River system in Utah accumulated whole-body selenium concentrations >8 mg/kg (i.e., >2× the toxicity threshold) in two out of four trials, although the invertebrate food supply contained total Se of only 2.3–3.5 mg/kg (Hamilton et al. 1996).

In the study by Coyle et al. (1993), whole-body selenium values of about 15–20 mg/kg were associated with complete reproductive failure in bluegill. Crane et al. (1992) did not report whole-body residues but did report muscle concentrations associated with total repro-ductive failure in perch. Based on the muscle-to-whole-body conversion regression for bluegill provided by Saiki et al. (1991), whole-body selenium in the perch presumably was also about 15–20 mg/kg. Again, using a ratio of about 3–3.5 to back-calculate an estimate of the threshold region, the reproductive effects threshold for sensitive species would be expected at about 4–6 mg/kg whole-body Se. That is roughly the same as the threshold range of whole-body values associated with impaired survival and/or development of larval fish (Hamilton and Wiedmeyer 1990; Hamilton et al. 1990; Cleveland et al. 1993; Hamilton 1996).

Data on hepatic concentrations associated with exposures of fish to organic forms of selenium are insufficient for estimating a sensitive-species threshold. Lemly (1993a) suggested a risk threshold of 12 mg Se/kg based on experimental results that linked selenite exposure to perturbations of blood chemistry. However, the fish component of a hazard assessment protocol for selenium presented by Lemly (1995, 1996b) relies on data for fish eggs or whole-body residues (as a surrogate for eggs) but not on data for hepatic tissues. As recognized by Lemly, only a weak basis exists for assessing ecological risk in nature based on hepatic selenium concentrations and, therefore, they have little interpretive value.

No sublethal effects have been reported for adult or juvenile fish at levels lower than the thresholds estimated above for reproductive effects (e.g., Lemly 1993a, 1995, 1996a, b). Mortality of adult fish, even in sensitive species, occurs at exposure and tissue thresholds much higher than those that produce reproductive impairment. For example, even among fingerling bluegill, some

specimens survived a 44-day dietary exposure of 130 mg Se/kg (organic) (Finley 1985; Coyle et al. 1993).

Consumption advisories to protect human health were issued in California when edible fish tissue was known to exceed 2 mg Se/kg on a wet weight basis (Fan et al. 1988; Saiki et al. 1991). When edible tissues exceed 5 mg Se/kg on a wet weight basis, health professionals advise against any human consumption (A. Fan, pers. comm., cited in Texas Parks and Wildlife Department 1990).

Summary: Effects of selenium on fish

Interpretive guidance	Fish Se concentration (mg/kg)
Background, whole body	<1–4 (typically <2)
Background, skeletal muscle	<1–4 (typically <2)
Background, gonads/eggs	<1–4 (typically 2–3)
Background, hepatic	2–8 (typically <5)
Lowest validated concentration in edible tissue (trout fillet) warranting human health advisory	2.0
Outdoor macrocosm LOAEL for reproductive impairment (bluegill)	16–18 (gonad/ egg tissue)
Estimated true threshold range (≈IC10) for reproductive impairment in sensitive species (perch, bluegill)	7–13 (gonad/egg tissue)
Experimental LOAEL for total reproductive failure (bluegill)	15–20 (whole body, parental)
Estimated true threshold range (≈IC10) for reproductive impairment in sensitive species (perch, bluegill, salmon)	4–6 (whole body, parental or offspring)
Complete reproductive failure (IC100) in sensitive species (bluegill)	30–35 (food-chain Se or selenomethionine)
Estimated true threshold range (≈IC10) for reproductive failure in sensitive species (bluegill), parental exposure only	10 (food-chain Se or selenomethionine)
Experimental LOAEL's for reproductive impairment via lethal larval dietary exposure (salmon, bluegill, razorback suckers)	3–8 (food-chain Se or selenomethionine)
	Edible tissue Se (mg/kg)
Health advisories recommend limited fish consumption by healthy adults and no consumption by children and pregnant women	2 (wet weight basis)
Complete ban on human consumption of fish recommended	5 (wet weight basis)

Amphibians and Reptiles

Bullfrogs collected from reference agroforestry plantations in the San Joaquin Valley con-tained 1.0–1.9 mg/kg whole-body Se (CDFG 1993). Frog and toad livers from reference sites in the same area contained 2.9–3.6 mg Se/kg (Bryne et al. 1975; Ohlendorf et al. 1988b). In lizards and snakes from these areas, whole-body selenium averaged 0.7–2.0 mg/kg, and these values probably repre-sent normal background concentrations even though some of the snakes were collected at sites where selenium-contaminated water is used to irrigate trees (CDFG 1993). Water snakes from Florida also contained about 1–2 mg/kg whole-body Se (Winger et al. 1984). Livers of gopher snakes from reference sites near Kesterson Reservoir contained about 1–4 mg Se/kg (Ohlendorf et al. 1988b). Pine snake hatchlings from the New Jersey pine barrens

region averaged 2.6 mg Se/kg in skinless whole-body samples (skins averaged 1.6 mg/kg) (Burger 1992). These hatchlings are probably good indicators of normal selenium levels in snake eggs. American alligator eggs from Florida contained about 1.0–2.1 mg Se/kg (Heinz et al. 1991). Both the pine snake hatchlings and the alligator eggs suggest that normal background concentra-tions of Se in amphibian and reptile eggs are the same as in fish and bird eggs (i.e., typically averaging 1–3 mg/kg).

Apparently, no field data are available for assessing toxic thresholds in amphibians and reptiles. Experimental data are limited to toxicity tests that found *Xenopus* (frog) larvae sensitive to >1,000 µg/L waterborne selenite (Browne and Dumont 1979) and found an LC50 of 90 µg/L waterborne selenite for eggs and larvae of the narrow-mouthed toad (Birge et al. 1979).

Based on how similar the toxic threshold values are for fish and bird eggs (e.g., Lemly 1995, 1996b), two other classes of egg-laying vertebrates, it is probably safe to assume for amphibians and reptiles that (1) reproductive impairment is among the most sensitive response variables and (2) populations producing eggs with ≥10 mg Se/kg are reproductively impaired. Another provisional interpretive guideline that seems justified, based on existing knowledge for all other taxa of vertebrates, is that whole-body concentra-tions at or above 10× normal background (or ≥20 mg/kg) are probably toxic to populations of sensitive species.

Birds

Birds are rarely analyzed for contaminant concentrations on a whole-body or carcass basis. One national monitoring program, however, analyzed selenium concentrations in starling carcasses (feathers, legs/feet, beaks removed). Starling carcasses usually averaged less than 2 mg Se/kg (White et al., 1988).

Summary: Effects of selenium on amphibians and reptiles

Interpretive guidance	Biomass Se concentration (mg/kg)
Background, whole body	0.7–3 (typically <2)
Background, eggs	1–3
Background, hepatic	2.9–3.6
Presumptive reproductive impairment threshold	≥10 (eggs)
Presumptive adverse effects threshold on a whole body basis (10× normal)	≥20 (whole body)
	Waterborne Se concentration (µg/L)
Lowest toxicity-test LC50 for amphibian eggs/larvae	90

Selenium exposure in birds is more typically measured in specific tissues: muscle, liver/ kidney, eggs, feathers, or blood/plasma. Normal concentrations of selenium in muscle are about 1–3 mg/kg (e.g., White et al., 1987; Ohlendorf et al. 1990; Barnum 1994). Liver and kidney tissue usually contain comparable concentrations of selenium (Ohlendorf et al. 1988a; Heinz 1996) and will be referred to in aggregate as hepatic tissue. Reference inter-quartile ranges for selenium in avian hep-
atic tissue (in mg/kg) vary from 2.0–4.3 (3.3 median) in rallids (mostly American coots), to 5.2–9.5 (7.5 median) in anatids (dabbling ducks), to 6.0–9.9 (8.2 median) in recurviro-strids (stilts and avocets) (Skorupa et al., 1992). These three taxa represent herbivorous, omnivorous, and carnivorous (invertebrate prey) groups of birds and therefore should represent the full range of normal background concentrations. Bird eggs collected from Se-normal study areas usually average ≤3 mg Se/kg (grand median across all taxa of 1.9 mg/ kg), and the maximums are usually <5 mg Se/kg (Skorupa and Ohlendorf 1991; Ohlendorf et al. 1993). The selenium content
of feathers is usually 1–2 mg/kg (Parrish et al.

1983; Burger et al. 1992a; Burger and Gochfeld 1992a,b; Burger and Gochfeld 1993; Burger et al. 1994) but may be <1 mg/kg in areas of selenium-poor soil (Burger et al. 1993). However, the presence of mercury has the effect of directing more selenium content into the feathers, and so feathers may reach concentrations of 2–4 mg Se/kg in environments containing elevated mercury levels (GLSAB 1981; Burger et al. 1992b). Avian whole blood normally contains about 0.1–0.4 mg Se/kg on a wet weight basis (e.g., Ihnat 1989; Heinz et al. 1990; USBR 1995).

Heinz (1996) provides an excellent recent review of selenium toxicity thresholds for birds. He found that reproductive impairment is one of the most sensitive response variables and eggs are the most reliable tissues for interpretive purposes. Unlike most sampling techniques, egg studies easily avoid a sampling bias that favors survivors. Bird eggs are sampled without regard for the status of the embryo inside the egg. Live and dead embryos have equal probabilities of being sampled. Therefore, accurate population-level exposure assessments and unbiased exposure-response curves can be obtained from field samples of eggs. Reproductive impairment is generally a more sensitive response variable than adult mortality, *in ovo* exposure to selenium is discrete and easily measured, and birds' sensitivity to selenium is equal to or greater than that of other taxa. Consequently, bird eggs constitute one of the best biotic matrices for risk/impact interpretation (Ohlendorf et al. 1986; Heinz et al. 1987, 1989; Skorupa and Ohlendorf 1991; CH2M Hill et al. 1993; Ohlendorf et al. 1993; Skorupa 1994; Seiler and Skorupa 1995; Heinz 1996; Skorupa 1998a).

Based on a review of experimental and field data, Heinz (1996) estimated that the embryotoxic threshold for selenium in bird eggs is about 10 mg/kg. Several taxa-specific exposure-response profiles for avian embryos have been derived from field sampling during the last decade (Skorupa et al., unpub. data; Skorupa et al. 1993; Skorupa 1998a). Based on those profiles (presented below), bird species differ substantially in embryo sensitivity to selenium exposure. The variation seems to have more to do with salinity tolerance than with phylogeny. Species of birds that prefer athalassohaline (nonmarine saline) wetlands produce embryos that are more tolerant of selenium than closely related species that favor freshwater wetlands. For example, embryos of American avocets tolerate selenium better than do those of black-necked stilts, and snowy plover embryos are more tolerant than those of killdeer. A possibility that deserves some attention is that differences in embryonic selenium tolerance may be a consequence of natural selection for tolerance to sulfate salinity. Whatever is causing the interspecific variation, once identified, it would have tremendous interpretive implications.

With the possible exception of cinnamon teal, dabbling ducks appear to be among the most sensitive species of waterbirds. A recent statistical analysis of the teratogenesis data for ducks revealed that the IC10 was 23 mg Se/kg egg (Skorupa 1998b). Field-collected teratogenesis response data for duck embryos are distributed as follows:

Ducks

Egg Se range: ducks (mg/kg)	Observed probability of overt embryo teratogenesis (%)
00–10	0.0
11–20	3.2
21–30	8.7
31–40	40.0
41–50	Insufficient data
51–60	Insufficient data
61–70	Insufficient data
71–80	100.0

Notes: The field-measured background rate of overt embryo teratogenesis for Se-normal duck populations in Montana (i.e., representative eggs contained <5 mg Se/kg) was approximately 0.3 percent based on a monitored sample of >3,000 eggs (calculated from data in Dubois 1988). Only eggs that were both randomly sampled in the field and randomly selected for chemical analysis are included above. Observed probabilities are for true teratogenesis only, not for all types of abnormalities. Total *N*=138 eggs, mostly from the San Joaquin Valley of California.

For individual duck eggs containing as little as 11–20 mg/kg, the observed probability of teratogenesis is low in absolute terms but is nevertheless 10× background. The above numbers are individual-level data, but they support the population-level analysis of Skorupa and Ohlendorf (1991). For inter-pretive purposes, however, it is very impor-tant to distinguish between individual-level and population-level guidelines. Population-level thresholds are usually lower than individual-level thresholds because they are based on population averages even though it is actually the maximum values that deter-mine when a population crosses the toxicity threshold (i.e., the point at which just a few hens in the population show a toxic response). Quite often, only population levels of selenium exposure (means and standard errors) are reported in the literature, and that is why Skorupa and Ohlendorf (1991) were limited to a population-level analysis of published field data. The response profiles provided here are guides for individual-level risk interpretation (the level that is most useful but requires far more effort to con-struct field-validated profiles). Teratogenesis is not as sensitive a response variable as egg hatchability, but due to high rates of nest parasitism among the duck populations sampled, it was not possible to construct a response profile based on hatchability.

Black-necked stilts are not as sensitive to selenium poisoning as ducks but are still moderately sensitive. Field-collected teratogenesis response data for stilt embryos are distributed as follows:

Stilts—Teratogenesis

Egg Se range: stilts (mg/kg)	Observed probability of overt embryo teratogenesis (%)
00–10	0.4
11–20	1.3
21–40	5.0
41–60	24.4
61–80	71.4
81–100	100.0
101–120	100.0

Notes: The field-measured background rate of overt embryo teratogenesis for Se-normal stilt and avocet populations (recurvirostrids) in the San Joaquin Valley of California was approximately 0.15 percent based on a monitored sample of >3,000 eggs (Skorupa et al., unpub. data). Only eggs that were both randomly sampled in the field and randomly selected for chemical analysis are included above. Observed probabilities are for true teratogenesis only, not for all types of abnormalities. Total *N*=547 eggs, mostly from the San Joaquin Valley of California.

The response profile above is not directly comparable to the embryotoxicity curve for stilts presented by Ohlendorf et al. (1986, figure 2) because their curve was not restricted to true teratogenesis (irreversible structural deformities) but included all forms of embryo impairment (including reversible pathologies). More importantly, Ohlendorf et al.'s data set included some samples that were non- randomly selected for chemical analysis specifically because the samples contained abnormal embryos (H.M. Ohlendorf and R.L. Hothem, pers. comm.). Inclusion of some nonrandom data points statistically biases the response curve upward. Within each exposure category, eggs with abnormal embryos had a higher probability of being sampled (i.e., selected for chemical analysis) than unim-paired eggs and, therefore, the frequency of response at each exposure interval is overestimated. Comparing the duck data to that for stilts, notice that the incidence of teratogenesis in ducks shows a substantive increase at a distinctly lower threshold (≈35 mg/kg versus ≈50 mg/kg).

Field-collected clutch viability data for stilts are distributed as follows:

Stilts—clutch viability

Egg Se range: stilts (mg/kg)	Observed probability of impaired clutch (%)
0–5	8.7 (background)
6-15	18.9
16-30	26.9
31-50	33.7
51-70	65.4
71-90	100.0
91-110	100.0

Notes: These data represent the percentage fo hens within each exposure interval that were reproductively impaired; a henwise response rate based on whole-clutch viability (4-egg clutches). The background rate is a function of normal infertility. Total *N*-410 full-term, monitored, and chemically characterized clutches, mostly from the San Joaquin Valley of California.

Avocets—teratogenesis

Egg Se range: avocets (mg/kg)	Observed probability of overt embryo teratogenesis (%)
00–40	0.0
41–60	3.8
61–80	7.1
81–100	9.1
101–120	50.0

Notes: The field-measured background rate of overt embryo teratogenesis for Se-normal stilt and avocet populations (recurvirostrids) in the San Joaquin Valley of California was approximately 0.15 percent based a monitored sample of >3,000 eggs (Skorupa et al., unpub. data). Only eggs that were both randomly sampled in the field and randomly selected for chemical analysis are included above. Observed probabilities are for true teratogenesis only, not for all types of abnormalities. Total *N*=542 eggs, mostly from the San Joaquin Valley of California.

The henwise (=clutchwise) response profile presented above provides individual-level interpretive guidance that incorporates all forms of embryo impairment, not just terato-genesis. Ohlendorf et al. (1986, figure 3) also presented a clutchwise response profile for stilts, but it is biased upward due to the inclusion of some samples nonrandomly selected for chemical analysis. For example, Ohlendorf et al.'s clutchwise response curve suggests that 60 percent of stilt hens in the 31–50 mg Se/kg egg exposure category will be reproductively impaired as opposed to an estimate of 33 percent (which is below Ohlendorf et al.'s lower 95 percent confidence boundary) based on strictly random samples. Skorupa (1998a) has recently provided a detailed statistical analysis of the Tulare Basin stilt data and shown that the IC10 for teratogenesis is 37 mg Se/kg egg and that the threshold point for hatchability effects is 6–7 mg Se/kg egg.

As a final species-specific example, American avocets are very tolerant to selenium poisoning, even compared to the closely related stilts. Field teratogenesis response data for avocet embryos break down as follows:

The IC50 for embryo teratogenesis in avocets is 105 mg Se/kg egg, or about 40–45× normal background (Skorupa 1998a). That is roughly four times the value for ducks and about twice the value for stilts. Even the IC10 for avocets, 74 mg Se/kg egg, is very high (Skorupa 1998a). Interestingly, Goodsell (1990), studying Australian varieties of avocets and stilts, found a similar degree of differences in these birds' tolerance for salinity. Red-necked avocet chicks were far more salinity tolerant than black-winged stilt chicks in the same areas. As noted in Skorupa (1996), selenium tolerance and salinity tolerance are closely related. The predominant salt at many saline-sink wetlands is sodium sulfate, and the biochemistry of selenium is very similar to that of sulfur. Thus, any mechanism that has evolved to cope with the effects of sulfate salinity is likely to be equally effective against selenium. The Australian avocets and stilts are close ecological equivalents to the North American species. (Johnsgard [1981] considers black-winged and black-necked stilts to be the same species.)

Field-collected clutch viability data for avocets are distributed as follows:

Avocets—clutch viability

Egg Se range: avocets (mg/kg)	Observed probability of impaired clutch (%)
00–20	13.5 (background)
21–40	14.6
41–60	11.8
61–80	33.3
81–100	50.0

Notes: These data represent the percentage of hens within each exposure interval that were reproductively impaired; a henwise response rate based on whole-clutch viability (4-egg clutches). The background rate is a function of normal infertility. Total *N*=230 full-term, monitored, and chemically characterized clutches, mostly from the San Joaquin Valley of California.

Whereas 30 percent of stilt hens are reproductively impaired when eggs contain 40 mg Se/kg, avocet eggs have to contain 70 mg/kg for 30 percent of the hens to be reproductively impaired.

At Heinz's (1996) recommended threshold concentration of 10 mg Se/kg in avian eggs, threshold proportions (≈10 percent) of duck and stilt hens would indeed exhibit reproductive selenosis. Probably, though, no avocet hens would show any such effect. More limited sets of field response data for snowy plovers and killdeer (Skorupa et al., unpub. data) suggest profiles that are very similar to those for avocets and stilts, respec-tively. In addition to the differences between species in their responses to equal *in ovo* exposures, other differences are related to dietary habits. Three experimental studies of flesh-eating birds have found that less selenium was transferred from the hen's diet to the egg than is typical of plant- and invertebrate-eating species of birds (Smith et al. 1988, black-crowned night-herons; Wiemeyer and Hoffman 1996, screech owls; and USBR 1995, American kestrels). Based on Wiemeyer and Hoffman's screech-owl study, the only one with straightforward repro-ductive performance data, general interpretive guidelines based on *in ovo* exposure are probably applicable to embryos of flesh-eating birds, but greater environmental exposure of the hens is probably required for them to produce eggs that attain threshold concentra-tions. Based on experimental studies with chickens and quail (see Heinz 1996), embryos of upland gallinaceous game birds may be more sensitive than duck embryos. Studying white-faced ibis at Carson Lake, Nevada, Henny and Herron (1989) reported that nests in which the eggs contained >6 mg Se/kg were 16 percent less productive than nests that had eggs containing <4 mg Se/kg. That's about the magnitude of effect that would be expected at that exposure level (6–9 mg/kg) for a moderately sensitive species (like stilts), but the small sample of >6 mg/kg eggs and the confounding presence of high DDE and/or mercury levels in some eggs made Henny and Herron's (1989) study inconclusive with regard to selenium.

In birds, reproductive impairment can result from diets containing as little as about 3–8 mg Se/kg (Wilber 1980; Martin 1988; Heinz 1996). Nonbreeding adult birds can tolerate higher levels of selenium, but still it is recommended that their dietary exposure not exceed 10–15 mg Se/kg (Heinz 1989; Heinz and Fitzgerald 1993b). These dietary thresholds have been estimated primarily from feeding trials with selenomethionine. When selenium is added to artificial diets as purified seleno-methionine, its toxicity and bioavailability are virtually identical to those of naturally incor-porated selenium in high-selenium wheat (Heinz et al. 1996) and in contaminated small mammals that were collected on the site of the former Kesterson Reservoir and fed to captive American kestrels (USBR 1995). Skorupa and Ohlendorf (1991), however, presented data suggesting that perhaps naturally incorporated selenium in highly chitinous species or life stages of invertebrates might be less bio-available than purified selenomethionine added to artificial diets. As was found in the results for

fish, the lowest levels of dietary selenium that were fatal to either juvenile or adult birds occurred in treatments that coincided with winter metabolic stress (Tully and Franke 1935; Heinz 1996).

Determinations of selenium concentrations in avian hepatic tissues provide a very limited basis for interpretation. Although hepatic concentrations are always elevated in popu-lations of birds exposed to toxic levels of selenium (e.g., USFWS 1990a; Heinz 1996), they are sometimes very elevated in popu-lations inhabiting Se-normal environments (e.g., Rowe et al. 1991; Rinella and Schuler 1992). In the latter cases, avian eggs contain back-ground concentrations of selenium (consistent with environmental conditions) even though the hepatic concentrations are elevated. The converse situation, background hepatic concentrations combined with elevated egg concentrations, has never been recorded. Heinz and Hoffman (1998) reported that simultaneous dietary exposure to organic mercury and organic selenium increased the hepatic concentrations of selenium in captive mallards by >10× the level from selenium-only diets. Perhaps the confusing field cases in which low environmental (and egg) levels of selenium are accompanied by elevated hepatic levels would be explained by elevated exposures to mercury. In normal-mercury environments, it appears that hepatic con-centrations ≥30 mg Se/kg are highly likely to be associated with reproductive impairment (USFWS 1990a; Skorupa et al. 1992). The 30-mg Se/kg level may mark the approximate threshold for toxicity in young and adult birds (Heinz 1996). In any environment, hepatic concentrations ≤10 mg Se/kg indicate normal (safe) selenium exposure. In summary, hepatic concentrations of selenium are more reliable for delineating populations that are *not* suffering from selenium toxicity than they are for identifying poisoned populations. Elevated levels of hepatic selenium should not be interpreted as anything more than an indication that further study is warranted.

Field data have not been collected that reliably relate concentrations of selenium in avian muscle (usually breast muscle) to toxic effects. An important factor that might confound such attempts is the fact that muscle tissue accumu-lates and disposes of contaminants more slowly than other tissues do (Heinz 1996), and so samples are less likely than other tissues to show the effects of recent dietary exposure. In experimental studies, concentrations ≥20 mg Se/kg in breast muscle of adult mallards were sufficient to cause death (USFWS 1990a). Breast muscle is most frequently sampled in the field to provide interpretive data for assessing human health hazards (e.g., Barnum 1994). Concentrations that warrant health advisories and consumptive bans are the same as summarized above for edible fish tissues.

Feathers and blood are important sample tissues because they are the only ones that can be collected without sacrificing the animals being sampled. Therefore, these are the primary avian analytical matrices available for assessing the selenium status of endangered species. Despite this important role, no substantive interpretive basis has been developed for either tissue. For example, no study has yet established threshold concen-trations of selenium in feathers or blood that can be reliably associated with reproductive impairment. Experimental studies found that selenium-poisoned captive mallards had 5–14 mg Se/kg wet weight basis in their blood (or in the blood of treatment group survivors exposed to the same diet) (Heinz 1996). In American kestrels, blood containing ≥1 mg Se/kg (wet weight) resulted from a diet of ≥5 mg Se/kg (USBR 1995)—a level that could cause reproductive impairment in sensitive species of birds (e.g., Lemly 1995, 1996b). A follow-up study of captive kestrels suggested an adverse effects threshold between 1.2 and 2.1 mg Se/kg ww in blood, based on fertility and body condition as the response variables (USBR 1997; Gary Santolo, CH2M Hill, pers. comm. to J. Skorupa, USFWS). As a general interpretive guide, any selenium concentration

that exceeds about 5 mg Se/kg in feathers or 1 mg Se/kg in blood (wet weight) will mean that further study is warranted.

Summary: Effects of selenium on birds

Interpretive guidance	Bird Se concentration (mg/kg)
Background, whole body	Typically < 2
Background, muscle	1–3
Background, eggs	Mean <3 (typically 1.5–2.5) Maximum <5
Background, hepatic (median values)[1]	3.3 (herbivore) 7.5 (omnivore) 8.2 (carnivore)
Background, feathers	1–4 (typically 1–2)
Background, blood	0.1–0.4 (wet weight basis)
Embryo teratogenesis threshold (≈IC10), wild ducks (sensitive taxon)	23 (in ovo)
Embryo viability (=egg hatchability) threshold, captive mallards	10 (in ovo)
Embryo teratogenesis threshold (≈IC10), American avocets (tolerant taxon)	74 (in ovo)
Embryo viability (=egg hatchability) threshold, American avocets	61–80 (in ovo)
Hepatic threshold for juvenile and adult toxicity	30 (liver)
Muscle threshold for juvenile and adult toxicity	≈20 (breast muscle)
Provisional feather threshold warranting further study	5 (breast feathers)
Provisional blood threshold warranting further study	1 (whole blood) (wet weight basis)

Interpretive guidance	Dietary Se exposure (mg/kg)
Reproductive impairment threshold	3–8
Toxicity threshold for nonbreeding birds exposed to winter-stress	10–15
	Edible tissue Se (mg/kg)
Health advisories recommend limited consumption by healthy adults and no consumption by children and pregnant women	2 (wet weight basis)
Complete ban on human consumption recommended	5 (wet weight basis)

[1] Background hepatic concentration is typically <10 but can be much higher in Hg-contaminated environments with normal Se.

Mammals

Based on the lowest whole-body selenium values reported by Clark (1987) for Kesterson Reservoir, and the diet-to-whole-body transfer values reported by Ohlendorf and Santolo (1994), normal whole-body selenium concentrations in small mammals are probably <2 mg/kg. For example, kangaroo rats, little brown myotis bats, and Brazilian freetail bats from agroforestry plantations in the San Joaquin Valley had a median whole-body selenium concentration of about 1.2 mg/kg (CDFG 1993).

Normal concentrations of selenium in muscle are typically <1 mg/kg in bats (Schroeder et al. 1970), pronghorn antelope (Raisbeck et al. 1996), gophers, voles, deer mice, house mice, rabbits, hares (CDFG 1993), and macaque monkeys (Hawkes et al. 1994). The data for wild species of mammals agree with data from many species of domestic mammals (Jenkins 1980).

Normal concentrations of selenium in livers of mammals characteristic of aquatic habitats, such as muskrats and raccoons, range from about 1 to

10 mg/kg and typically average ≤5 mg/kg (Schroeder et al. 1970; Clark 1987; Clark et al. 1989). Similar results have been reported for terrestrial predators that may seasonally focus their hunting in aquatic habitats, such as foxes and coyotes (e.g., Paveglio and Clifton 1988), and for an assortment of small terrestrial mammals trapped in the vicinity of an Se-normal
San Joaquin Valley wetland (Clark 1987). Another group of small mammals collected near agroforestry plantations averaged <3 mg Se/kg in their livers, except at plantations using high-selenium water for irrigation (CDFG 1993).

Selenium in whole blood normally averages <0.5 mg/L in domestic mammals (Jenkins 1980) and in raccoons, pronghorn antelope, and coyotes (Paveglio and Clifton 1988; Clark et al. 1989; Schamber et al. 1995). Less than 0.1 mg/L in whole blood is considered deficient, and deficiency seems to be more common than excessive exposure among wild ungulates such as deer (e.g., Oliver et al. 1990; Hein et al. 1994). Human whole blood normally contains 0.1–0.3 mg/L (USPHS 1989).

Data for a wide range of mammalian species suggest that the normal concentration of selenium in individual samples of hair is less than 3 mg/kg. Population averages normally range from about 0.5 to 1.5 mg Se/kg, including those for human populations (Huckabee et al. 1972; Paveglio and Clifton 1988; Clark et al. 1989; USPHS 1989).

One study reported an average concentration of 0.015 mg Se/L in the milk of a control group of nursing macaque monkeys (Hawkes et al. 1994). Most herds of dairy cows also average <0.05 mg Se/L in milk (Jenkins 1980).

Clark et al. (1989) reported that the feces of raccoons averaged about 1 mg Se/kg at an Se-normal wetland in the San Joaquin Valley of California.

There have been no well-documented cases of widespread selenosis among wild mammals comparable to the multiple examples available for fish and birds (Skorupa 1998a). Poisoning in nature has been reported for mammals but has been largely restricted to free-range domestic livestock, primarily horses, cows, and sheep (e.g., Rosenfeld and Beath 1946; Olson 1986; Raisbeck et al. 1993). As is the case for fish and birds, young animals are generally more sensitive (Thompson et al. 1991).

The lowest dietary threshold for mammalian toxicity reported in the literature is 1.4 mg Se/kg (natural selenium, dry feed basis); sublethal effects were noted in rats following lifetime exposure at that level (Eisler 1985). At 3 mg/kg in the lifetime diet, longevity was reduced (Eisler 1985). Also at 3 mg/kg (high-Se wheat), Olson (1986) reported reproductive selenosis in rats. Sublethal effects were observed in dogs exposed to about 7 mg/kg (high-selenium corn) (Rhian and Moxon 1943). Most domestic animals exhibit signs of toxicity on diets containing ≥3–5 mg/kg (natural selenium) (NRC 1980; Eisler 1985; Olson 1986). Pronghorn antelope showed some symptoms of immunotoxicity on a diet of 13–16 mg Se/kg (high-selenium hay), but were overtly healthy and more tolerant to dietary selenium than would be expected based on veterinary standards developed for domestic ungulates (Schamber et al. 1995; Raisbeck et al. 1996). Macaque monkeys given intravenous doses equivalent to about 16 mg/ kg (dry feed basis) dietary selenomethionine showed obvious toxic effects (Hawkes et al. 1994). The minimum long-term dietary exposure found to produce sublethal toxic effects in humans is 1.9 mg/kg (natural selenium, *wet weight*) (USPHS 1989).

Several studies summarized by Hawkes et al. (1994) suggest that mammalian teratogenic

effects occur only when maternal dietary exposure is high enough to adversely affect the mother. Under such circumstances it is not clear whether the teratogenesis is a direct effect of fetal selenium exposure or a consequence of maternal poisoning (Ferm et al. 1990).

Others have reported nonteratogenic reproductive depression as the principal effect of chronic selenosis in mammals, even when maternal selenosis is not apparent (e.g., James et al. 1981). Clark et al. (1989) reported that, for raccoons, hair selenium was one of the best indicators of the extent of exposure to selenium, and James et al. (1981) presented guidelines for interpreting risk of selenium-induced reproductive depression based on hair selenium. Less than 5 mg/kg was considered nontoxic, 5–10 mg/kg borderline toxic, and >10 mg/kg hair selenium was considered a toxic exposure. Samples of hair with more than about 10 mg Se/kg from natural popu-lations of pronghorn antelope, coyotes, meadow voles, mule deer, shrews, and raccoons have been reported from California, Idaho, North Carolina, and Wyoming (Huckabee et al. 1972; Paveglio and Clifton 1988; Clark et al. 1989). A chronically poisoned human population averaged 32 mg Se/kg in hair (USPHS 1989).

Based on whole blood, toxicologically significant thresholds for land mammals are generally cited as about 1–5 mg Se/L for chronic selenosis (e.g., Rosenfeld and Beath 1946; Edwards et al. 1989). Raisbeck et al. (1993) documented four recent cases of overt equine selenosis in Wyoming and reported blood values of 0.86, 0.96, 1.1, and 1.3 mg Se/L. Pronghorn antelope fed high-selenium hay exhibited immune system disorders and had blood selenium values of 1.0–1.3 mg/L (≈4–5× normal) (Schamber et al. 1995). Raccoons at Kesterson Reservoir had as much as 9.4 mg Se/L in their blood (average 2.6 mg/L) without overt signs of selenosis (among "survivors"), although four of eight specimens had deformed liver cells. A blood level of 5 mg Se/L was found in captive sea lions that had died after eating high-selenium fish (Edwards et al. 1989). A chronically poisoned human population had blood selenium averaging 3.2 mg/L (USPHS 1989).

General guidelines for interpreting concentrations of liver selenium have been developed primarily through veterinary studies. For example, Edwards et al. (1989) cite a toxicological threshold range of about 45–60 mg/ kg for liver selenium in domestic livestock, based on a veterinary handbook (Osweiler et al. 1985). Raisbeck et al. (1996) cited a more conservative toxic threshold of about 20 mg Se/kg in the liver but noted that feeding trials with pronghorn antelope show that the criterion may not be reliable. Buechner (1950), however, had already reported that prong-horn antelope are notably tolerant of toxic forage. This raises the question of whether the diagnostic criterion is broadly inapplicable or whether pronghorn antelope are a particularly insensitive taxon. More recently, O'Toole and Raisbeck (1998) reported for cattle and horses that hepatic selenium concentrations exceeding 2 mg/kg wet weight (~6-8 mg/kg dry weight), combined with other typical clinical symptoms, was a sufficient basis for a firm diagnosis of selenium poisoning. Interpretive criteria for hepatic tissues are not well supported, and as suggested above for birds, the primary interpretive value of hepatic measures may be to identify Se-normal populations of mammals rather than to identify poisoned populations.

Presumably the guidelines used for human health advisories regarding consumption of fish and birds (see above) should also apply to edible tissues of mammals. These call for targeted restrictions at 2 mg/kg (ww) and a complete consumptive ban at 5 mg/kg (ww).

Summary: Effects of selenium on mammals

Interpretive guidance	Mammal Se concentration (mg/kg dw, except as noted)
Background, whole body	<1–4 (typically <2, mean)
Background, muscle	<1
Background, liver (aquatic habitat mammals)	1–10 (typically <5, mean)
Background, blood	0.1–0.5 mg/L (typically 0.2–0.3, mean)
Deficient, blood	<0.1 mg/L
Background, hair	<1–3 (typically 0.5–1.5, mean)
Background, milk	<0.05 mg/L
Background, feces	<2
Reproductive depression threshold, hair	>10
Overt equine selenosis threshold, blood	1 mg/L
Human chronic selenosis threshold, blood	3 mg/L
Acute lethal toxicity LOAEL, sea lions, blood	5 mg/L
Veterinary toxicological handbook threshold, domestic livestock, liver	45–60
	Dietary Se exposure (mg/kg dw, except as noted)
Sublethal effects threshold, lifetime exposure of rats	1.4
Chronic selenosis threshold, humans	1.9 (ww)
Reduced longevity threshold, lifetime exposure, rats	3
LOAEL for reproductive selenosis, in rats	3
Overt toxicity thresholds, domestic livestock	3–5
Sublethal effects LOAEL, dogs	7
Health advisories recommend limited consumption by healthy adults and no consumption by children or pregnant women	≥2
Complete ban on human consumption recommended	≥5

Bioaccumulation

Toxicity varies for different forms of selenium, animal species, duration of exposure, method of uptake, and other factors. In wetland areas, bioaccumulation increases the levels of selenium in the food chain. Selenium is taken up by aquatic biota, including phytoplankton, zooplankton, and insects, which contribute to the diet of higher forms of wildlife. In particular, selenium accumulation in the food chain has caused the deaths of many fish and aquatic birds, young and old, and has led to reproductive failure and deformed offspring (Skorupa 1998a).

Selenite and selenate, the most common aqueous forms of selenium, are biotrans-formed into organic chemical species after uptake by primary producers such as algae (Ogle et al. 1988). Speciation of dissolved selenium in water strongly influences how much aquatic loading is required to bio-accumulate dangerous concentrations of selenium in the food chain, but waterborne speciation does not appear to influence the unit toxicity of food chain incorporated selenium (USFWS 1990b; Besser et al. 1993). After selenium becomes incorporated in the food chain, apparently the issue of chemical speciation is not an important interpretive factor. Toxicologically, food chain selenium in nature seems to be fairly uniform, with a toxicity profile very similar to that of selenomethionine (e.g., Woock et al. 1984; Hamilton et al. 1990; Heinz 1996). This is a particularly useful interpretive consideration since dietary exposure is the primary exposure pathway for fish and wildlife populations.

Dietary plant selenium is readily absorbed by animals. Most of the selenium (70–80 percent) is quickly metabolized and eliminated, but the remaining selenium becomes bound or incorporated into blood and tissue and is only slowly eliminated (Olson, 1978). Selenium easily enters metabolic pathways and there-fore is highly bioaccumulative. The high pro-pensity for biotic uptake of selenium is at least partially explained by its biochemical similarity to sulfur.

Interactions

Interactions between selenium and mercury have been extensively documented (Cuvin-Aralar and Furness 1991; Sorensen 1991), although many conflicting results have been reported. Depending on the exact chemical speciation of the two elements, and other factors, the toxicity of selenium can be increased, reduced, or unaffected by the presence of mercury. Few Se-Hg interaction studies have examined combined dietary exposure to methyl mercury and seleno-methionine in fish or wildlife, even though these forms of mercury and selenium are the ones most likely to be combined in a natural diet. A recent study using captive mallards found conflicting antagonistic and synergistic Se-Hg interactions with reference to adult toxicity and reproductive impairment: i.e., selenium and mercury together are less likely to poison adult birds than either element separately but more likely to impair repro-duction (Heinz and Hoffman 1998).

Dietary protein, boron, arsenic, and methionine concentrations are other factors clinically demonstrated to alter the toxicity of selenium to wildlife (Hoffman et al. 1991, 1992a,b; Stanley et al. 1994). However, the dose combinations of these factors that altered selenium toxicity in the laboratory would rarely be found in nature. Stanley et al. (1996) recently reported a lack of interaction effects between selenium and boron added to the diets of captive mallards.

The strong exposure-response relationships documented for selenium in field-collected bird eggs and fish suggest that interactions with other elements rarely affect selenium toxicity in the field (Ohlendorf et al. 1986; Skorupa and Ohlendorf 1991; Lemly 1993b; Ohlendorf et al. 1993; Skorupa 1998a). A multivariate statistical analysis of chemical data for bird eggs from California's
San Joaquin Valley (Skorupa 1998b) demonstrated a strong correlation between embryo deformity and *in ovo* selenium concentration, which was unaffected by other potentially interactive chemical constituents present in the eggs. There is, however, sub-stantial individual and taxonomic variability in sensitivity to selenium poisoning (EPA 1987; SJVDP 1990; USFWS 1990a, b; Sorensen 1991; Lemly 1993b).

In some cases, levels of exposure to selenium that wouldn't be directly toxic may increase susceptibility to otherwise benign pathogens due to a selenium-induced immune dys-function (Fairbrother and Fowles 1990; Whiteley and Yuill 1991; Schamber et al. 1995).

Regulatory Standards

U.S. Environmental Protection Agency Standards and Criteria
[See Appendix II for explanation of terms. Sources: EPA 1995; Federal Register 57(246):60911]

Status	EPA priority pollutant
Drinking water MCL	50 µg/L
Freshwater criteria	20 µg/L for acute exposure 5 µg/L for chronic exposure
1/1,000,000 cancer risk	10 µg/L (water and organisms)

For standards and criteria set by State agencies, contact those agencies directly. See Appendix I for a listing of water-quality officials in the 17 Western States.

Existing Literature Reviews

Over the last two decades, many publications have surveyed the literature pertaining to the effects of selenium exposure on fish and wildlife populations. Some of these reviews are listed here:

Adams and Johnson 1981
GLSAB 1981
Brooks 1984
Eisler 1985
Lemly 1985a
Lemly and Smith 1987
EPA 1987
Lillebo et al. 1988
UC Committee 1988
DuBowy 1989
Ohlendorf 1989
Beyer 1990
Hodson 1990
SJVDP 1990
USFWS 1990a,b
Skorupa and Ohlendorf 1991
Sorensen 1991
Peterson and Nebeker 1992
CH2M Hill et al. 1993
Emans et al. 1993
Lemly 1993a
CAST 1994
Maier and Knight 1994
Lemly 1995
Albers et al. 1996
Heinz 1996
Lemly 1996a,b,c
Green and Albers 1997
O'Toole and Raisbeck 1997
Van Derveer and Canton 1997
Adams et al. 1998
Hamilton 1998
Lemly 1998a,b
O'Toole and Raisbeck 1998
Skorupa 1998a

In addition, some useful interpretive guidance is found in the more general reviews of selenium chemistry and toxicology by Wilber (1980), Maier et al. (1987), Ogle et al. (1988), USPHS (1989), and Oldfield (1990). The core scientific basis for interpretive guidance is contained in these references. Where other sources are not cited to support statements in this chapter, the papers listed above are the source(s) of information.

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