

# ENVIRONMENTAL CHEMISTRY of SELENIUM

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## **Selenium Poisoning of Fish and Wildlife in Nature: Lessons from Twelve Real-World Examples**

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### **I. INTRODUCTION**

Ecotoxicologists ultimately endeavor to understand the attributes of toxicants in the real world. Although controlled studies can contribute to that understanding, they are often plagued by an inability to translate laboratory results to real ecosystems: the so-called lab-to-field dilemma (Landis and Yu, 1995). The lab-to-field dilemma is of particular concern for bioaccumulative toxicants because standardized aquatic bioassay testing rarely includes a dietary pathway, and dietary exposure of fish and wildlife to bioaccumulative toxicants is usually the primary risk factor. That critical flaw was appropriately recognized by the U.S. Environmental Protection Agency (EPA) when the freshwater chronic criterion for selenium was established at 5 micrograms per liter ( $\mu\text{g/L}$ ) in 1987. EPA established the 5  $\mu\text{g/L}$  criterion based largely on a single well-documented episode of selenium poisoning in nature rather than on a larger accumulation of data from bioassay toxicity testing (USEPA, 1987). Although that choice was clearly prudent, a national water quality criterion based largely on one real-world case study is an easy target for criticism. A single study does not provide sufficient basis for assessing a criterion's applicability across a variety of aquatic ecosystems and site-specific environmental conditions.

During the decade since EPA's publication of the 5  $\mu\text{g/L}$  freshwater chronic criterion for selenium (USEPA, 1987), there has been much research on the toxicity of selenium to fish and wildlife populations, as well as numerous reviews

of available information (Lillebo et al., 1988; UC Committee, 1988; DuBow, 1989; Ohlendorf, 1989; Beyer, 1990; Moore et al., 1990; USFWS, 1990a, 1990b; Skorupa and Ohlendorf, 1991; Sorensen, 1991; Peterson and Nebeker, 1992; CH2M HILL et al., 1993; Lemly, 1993a, 1995; Council for Agricultural Science and Technology, 1994; Gober, 1994; Maier and Knight, 1994; Heinz, 1996; O'Toole et al., 1996). None of these recent reviews, however, systematically presents an updated inventory of real-world case studies and the associated comparative results. In this chapter I will endeavor to provide such an inventory and, in some cases, provide the first detailed documentation to be found outside government reports and regulatory environmental assessment documents. This is not trivial: there are at least a dozen real-world case studies of clearly confirmed or highly probable selenium poisoning in nature (Fig. 1). Finally, I will examine this new abundance of real-world information for practical insights and the applicability of EPA's 5  $\mu\text{g/L}$  criterion.

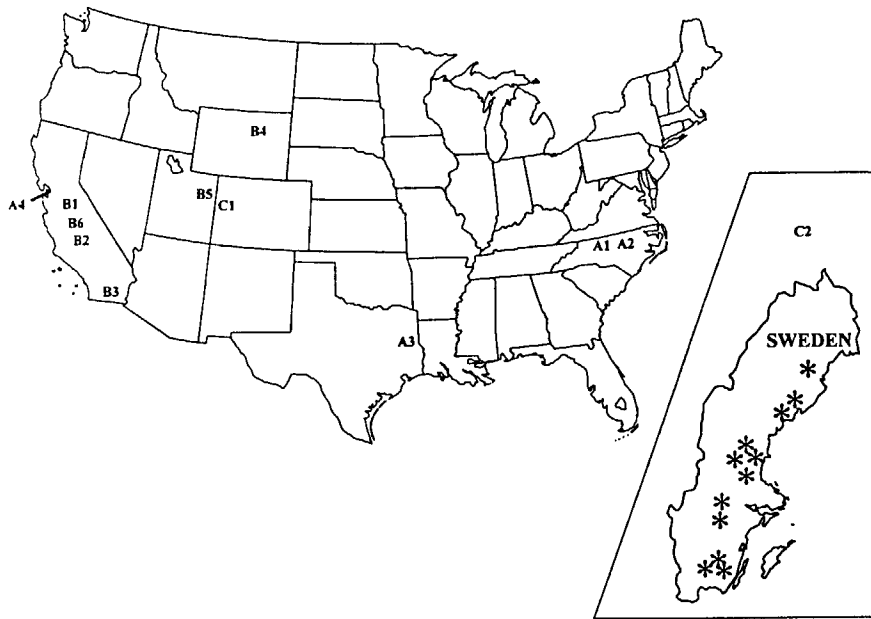


FIGURE 1 Geographic distribution of case studies documenting toxic episodes of fish and wildlife exposure to selenium. Site numbering follows text section divisions of Section II. Thus site A1 = Belews Lake (subsection A.1), site B1 = Kesterson Reservoir (subsection B.1), and so on. Asterisks indicate the distribution of individual lakes included in the Swedish lakes study.

## II. REAL-WORLD TOXIC EPISODES

### A. Case Studies Associated with Coal and Petroleum

#### 1. Belews Lake, North Carolina: Power Plant Cooling Basin

Belews Lake (A1 in Fig. 1), a man-made reservoir, was constructed to provide condenser cooling water for a large coal-fired electric power plant. The reservoir began filling in 1970 and biological monitoring also commenced immediately. The first unit of the power station began operating in 1974. Within a year (1975) juvenile recruitment among most species of fish was very low, and by 1977 the fish community had been drastically altered. Water entering Belews Lake from a fly ash settling basin contained 150 to 200  $\mu\text{g/L}$  selenium, and of 16 trace elements studied, only selenium was highly elevated in water and biota. By 1978, it was clear that a severe episode of selenium poisoning had occurred at Belews Lake (Cumbie and Van Horn, 1978; Lemly, 1985a, 1985b; 1993b).

Although the selenium concentration in the main portion of Belews Lake had been elevated only to values on the order of 8 to 22  $\mu\text{g/L}$ , and an average value of 10  $\mu\text{g/L}$ , highly elevated rates of teratogenic fish (10–70% vs. normal baseline of 1–3%) were documented, and populations of 16 species of fish completely collapsed. Only 4 species of fish remained by 1978. As summarized in Table 1, this level of waterborne selenium resulted in biotic tissues that sometimes exceeded 100 mg/kg selenium. (All tissue concentrations in this chapter are expressed on a dry weight basis unless otherwise noted.) Compared to normal reference values, which are uniformly in single digits, the tissue data were extremely elevated and were consistent with the severe adverse effects data. Both the exposure and the response data from the main reservoir at Belews Lake indicated that 10  $\mu\text{g/L}$  waterborne selenium was well above the toxicity threshold (Lemly, 1985a, 1985b, 1993b).

In contrast to the main reservoir, a semi-isolated reach known as the Highway 158 arm contained less than 5  $\mu\text{g/L}$  waterborne selenium, and an overtly normal fish community persisted there (Cumbie and Van Horn, 1978). A subsequent histopathological and hematological study of green sunfish (*Lepomis cyanellus*) measured 3 to 4  $\mu\text{g/L}$  waterborne selenium in the Highway 158 arm and detected in some of the fish sublethal toxic effects such as generalized edema and abnormal ovarian tissue damage (Sorensen et al., 1984). Thus, the threshold region for toxicity at Belews Lake appeared to be in the neighborhood of 2 to 5  $\mu\text{g/L}$  waterborne selenium. By the mid-1980s the Belews Lake episode had been closely studied and documented for more than a decade. Based largely on that body of documentation, EPA revised the national chronic criterion for selenium downward from 35  $\mu\text{g/L}$  to 5  $\mu\text{g/L}$  (USEPA, 1987).

A dry ash system was implemented in late 1985 at Belews Lake, curtailing the input of selenium to the reservoir. Fish sampling 7 years postremediation,

TABLE I Comparative Selenium Concentrations in Abiotic and Biotic Environmental Sampling Matrices of Selenium-Normal and Selenium-Poisoned Nonmarine Aquatic Ecosystems

Case study	Primary speciation of source Se	Environmental sample matrix <sup>a</sup>										Effects on Fish and Wildlife Resources	Sources <sup>d</sup>	
		Water source ( $\mu\text{g/L}$ )	Sediment (mg/kg dw)	Food chain fauna (mg/kg dw)	Fish			Bird						
					Whole body	Eggs	Muscle	Hepatic <sup>c</sup>	Eggs	Muscle	Hepatic <sup>c</sup>			
Selenium—normal background	Selenate	Range Typical	0.1–1 <0.5	0.2–2 <1	0.4–4.5 <2	<1–4 <2	<1–4 <3	<1–4 <2	2–8 <5	0.5–4.5 <3	1–3 <2	2–15 <10	None	1–40
1. Belews Lake, NC Main reservoir	Selenite		150–200	4–20	20–50	40–125	20–170	25–200	ND	ND	ND	ND	16 Fish spp extirpated, 10–70% rates of terata	2, 21, 41, 42, 47, 48
Semi-Isolated Hwy 158 Arm	Selenite		N/A	0.7–3	4–8	ND	ND	7–9	25–30	ND	ND	ND	Sublethal effects: ovarian damage, generalized edema	2, 43, 44, 47, 48
Main Res., 7 yr Postremedial	Selenite		N/A	ND	ND	10–20	ND	ND	ND	ND	ND	ND	5–10% fingering rates of terata (1–3% normal)	21, 47
Main Res., 10 yr Postremedial	Selenite		N/A	1–4	2–5	ND	3–20	ND	ND	ND	ND	6–15	3–6% fry rates of terata (0% fingering terata)	84
2. Hyco Reservoir, NC	Selenite		50–200	3–40	10–30	ND	30–50	35–50	ND	ND	ND	ND	Fish densities reduced 38–75%, reproductive failure	41, 45–47
3. Martin Reservoir, TX Initial sampling 1979–80	Selenite		2200–2700	5	0.8–68	ND	ND	10–40	25–100	ND	ND	ND	Collapse (>90%) of planktivorous fish biomass	41, 47, 49, 50
7.5 yr Post release 1986–1987	Selenite		N/A	ND	1–15	5–10	17	ND	20–50	<0.5–12+	ND	10–35	Blackbird hatch decline (54%), fish ovary damage	32, 47, 51, 52
9.5 yr Postrelease 1988–89	Selenite		N/A	ND	ND	5–10	ND	5–10	15–25	ND	ND	ND	Not monitored	47, 53
F. Chevron Richmond Refinery, CA	Selenite		10–60	3	10–45	ND	ND	ND	ND	1.9–57	ND	ND	10–30% teratogenic waterfowl nests (vs. <1% normal)	54–57
J. Keasterson Reservoir, CA	Selenite		230–420	0.3–67	5.9–290	69–430	ND	ND	ND	2.3–180	3.5–40	3.1–360	4–49% unviable waterbird eggs, adult & juvenile toxicosis	6, 12–14, 36, 58–61
1. Tulare Basin, CA	Selenate		460–943	0.8–15	4.7–230	ND	ND	ND	ND	15–148	ND	ND	33–50% teratogenic shorebird eggs (vs. <0.5% normal)	62–65, 67
Summer-Peck Ranch	Selenate		83–671	29–603	0.2–4.1	9.4–140	ND	ND	ND	2.1–164	5.5–36	6.7–120	2–16% teratogenic shorebird eggs (vs. <0.5% normal)	62–65, 67
Lost Hills Water District	Selenate		19–30	8.7–23	<0.2–2.4	8.0–27	4.7–18	ND	ND	1.9–80	1.5–22	4.4–57	14–15% teratogenic duck eggs (vs. <0.5% normal)	62–65, 67
Tulare Lake Drainage District-S	Selenate		158–212	1–10	7.5–57	ND	ND	ND	ND	2.5–115	ND	ND	4–10% teratogenic shorebird eggs (vs. <0.5% normal)	62–67
Rainbow Ranch	Selenate													

<sup>a</sup>Table continues

TABLE I Continued

Case study	Primary speciation of source Sc	Environmental sample matrix <sup>a</sup>										Effects on Fish and Wildlife Resources <sup>b</sup>	Sources <sup>c</sup>	
		Water source ( $\mu\text{g/L}$ )	Water system ( $\mu\text{g/L}$ )	Sediment (mg/kg dw)	Food chain fauna (mg/kg dw)		Fish		Bird		Hepatic <sup>d</sup>			
					Eggs	Whole body	Eggs	Muscle	Eggs	Muscle				Hepatic <sup>d</sup>
1. Salton Sea, CA	Selenate	2-10	1.5	3.3	0.8-12.1	6.1-16	ND	7.9-14	ND	1.6-35	2.7-7.2	2.7-42	5% reduction of black-necked stilt nesting proficiency	68-72
2. Kendrick Reclamation Project, WY	Selenate	ND	7-1300	4-17	87-166	ND	ND	ND	ND	2.4-135	17-35	2.6-170	38-52% inviable avocet and Canada goose eggs	73-75
3. Goose Lake	Selenate	ND	44-70	20-43	36-65	ND	ND	ND	ND	39-160	23-26	22-134	8-26% inviable avocet and eared grebe eggs	73-75
4. Ouray NWR, UT	Selenate	<1-830	9-93	8-26	10-71	22-104	ND	ND	ND	3.8-120	6-84	19-213	10% teratogenic coot nests, >85% inviable coot eggs	76, 77
5. Red Rock Ranch, CA	Selenate	1151-2114	1600-11,300	ND	ND	ND	ND	ND	ND	7.2-81	ND	ND	14-57% teratogenic shorebird eggs (vs. <0.5% normal)	67, 78
6. Sweitzer Lake, CO	Selenate	ND	96-160	ND	14-20	ND	ND	ND	<2-40	ND	ND	ND	Progressive mortality of stocked game birds	41, 79, 83
7. 1970s Sampling	Selenate	ND	<5-80	305	ND	ND	<48-243	<74-255	ND	ND	ND	ND	Human health warnings posted	79
8. 1980s Sampling	Selenate	ND	10-170	8.6-11	27-30	15-50	22-31	40-78	5.6-18	ND	9.0-84	ND	Possible reproductive failure among carfish	79
9. 1990s Sampling	Selenate	ND	24	31	12-26	29-62	ND	ND	2.3-32	ND	ND	ND	Not monitored	85
10. Swedish Lakes Project, Sweden	Selenite	N/A	3-5	ND	ND	ND	1.4-52	53-135	ND	ND	ND	ND	No catastrophic effects evident	80, 82
11. Lake Olerjarn 1984-87	Selenite	N/A	1-5	ND	ND	ND	0.9-36	ND	ND	ND	ND	ND	Collapse of perch populations in five lakes	81, 82

<sup>a</sup>Sampling methods for water and sediment are less standardized between studies than are sampling methods for biotic tissues.

<sup>b</sup>ID, no data; N/A, not applicable; dw, dry weight.

<sup>c</sup>kidney and/or liver.

<sup>d</sup>sources: 1, Wilber, 1980; 2, Lemly, 1985b; 3, Lillebo et al., 1988; 4, Maier and Knight, 1994; 5, Irwin, 1996; 6, Moore et al., 1990; 7, Martin and Hartman, 1984; 8, Presser, 1995; 9, USFWS, 1990a; 10, Beyer, 1990; 11, Birkner, 1978; 12, Saiki and Lowe, 1987; 13, Hothorn and Ohlendorf, 1989; 14, Schuler et al., 1990; 15, Crane et al., 1992; 16, Saiki et al., 1993; 17, Welsh and Maughan, 1994; 18, Walsh et al., 1977; 19, Schmit and Brumbaugh, 1990; 20, Jenkins, 1980; 21, Lemly, 1993b; 22, Saiki, 1989; 23, Ogle and Knight, 1989; 24, Hamilton et al., 1990; 25, USFWS, 1990b; 26, Cleveland et al., 1993; 27, Coyle et al., 1993; 28, Lorentzen et al., 1994; 29, Coughlan and Velte, 1989; 30, Hermanutz et al., 1992; 31, Hamilton and Waddell, 1994; 32, Sorensen, 1988; 33, White et al., 1977; 34, Wells et al., 1988; 35, White et al., 1987; 36, Ohlendorf et al., 1990; 37, Barmum, 1994; 38, Skorupa et al., unpublished data—bird, hepatic; 39, skorupa and Ohlendorf, 1991; 40, Ohlendorf et al., 1993; 41, Lemly, 1985a; 42, Lemly, 1993c; 43, Sorensen et al., 1984; 44, Great Lakes Science Advisory board, 1981; 45, Wock and Summers, 1984; 46, Gillespie and Baumann, 1986; 47, Cutter, 1986; 48, Cumbie and Van Horn, 1978; 49, Garrett and Inman, 1984; 50, Sorensen, 1986; 51, King, 1988; 52, King et al., 1994; 53, Texas Parks and Wildlife, 1990; 54, S. R. Hansen and Associates, 1994; 55, CH2M HILL, 1994; 56, CH2M HILL, 1995; 57, Chevron USA, unpublished data—sediments; 58, Presser and Ohlendorf, 1987; 59, USBR, 1986; 60, Ohlendorf, 1989; 61, Ohlendorf and Hothorn, 1994; 62, Westcot et al., 1988; 63, Chilcott et al., 1990a; 64, Chilcott et al., 1990b; 65, Moore et al., 1989; 66, Central Valley [California] regional Water Quality Control Board, unpublished data—water; 67, Skorupa et al., unpublished data—bird eggs; 68, Saiki, 1990; 69, Setmire et al., 1990; 70, Setmire et al., 1993; 71, Bennett, 1997; 72, Westcot et al., 1990; 73, Peterson et al., 1988; 74, See et al., 1992a; 75, See et al., 1992b; 76, Stephens et al., 1988; 77, Stephens et al., 1992; 78, Westside Resource Conservation District, 1996; 79, Butler et al., 1991; 80, Paulsson and Lundbergh, 1989; 81, Paulsson and Lundbergh, 1991; 82, Paulsson and Lundbergh, 1994; 83, Barnhart, 1957; 84, Lemly, 1997; 85, Krueger and Osmundson, unpublished data.

a small (36 ha) constructed wetland prior to discharge into the San Francisco Bay estuary. Initial (1989–91) water monitoring revealed that outflow from the constructed wetland typically contained substantively less selenium (ca. 10  $\mu\text{g/L}$ ) than the inflow had contained (ca. 20  $\mu\text{g/L}$ ). By 1994 the marsh was attracting substantive use by waterbirds, prompting officials at the San Francisco Bay Regional Water Quality Control Board to request that Chevron USA conduct a study of selenium exposure and reproductive performance among birds nesting at the marsh. The board requested an additional study of food chain bioaccumulation of selenium for 1995.

During 1995, wastewater inflow averaged about 20  $\mu\text{g/L}$  selenium and after flowing through the three segments of the marsh averaged about 5  $\mu\text{g/L}$  in the outflow. Aquatic invertebrates and birds using this flow-through system accumulated highly elevated tissue concentrations of selenium (Table 1). Black-necked stilts (*Himantopus mexicanus*) were chosen as a focal bird species for random sampling of eggs in 1994 and 1995. Nests of other species of waterbirds were also monitored in 1994, and some fail-to-hatch eggs were nonrandomly collected. In both 1994 and 1995, random stilt eggs averaged about 20 to 30 mg/kg selenium and concentrations exceeding 50 mg/kg were observed among fail-to-hatch eggs of stilts and other bird species. Deformed embryos were recovered from about 30% of mallard (*Anas platyrhynchos*) nests and about 10% of American coot (*Fulica americana*) nests that yielded one or more assessable embryos. Normally, fewer than 1% of assessable nests should yield a deformed embryo. Selenium exposure at Chevron Marsh was sufficient to expect a 6.7% deformity rate in stilt eggs (based on the exposure–response data for stilts exposed to seleniferous agricultural drainage water; see Tulare Basin data presented in Section II.B.2), but none of 16 assessable stilt embryos were deformed. At an expected 6.7% deformity rate, a sample size of 16 has a power of only 69% for detecting a deformed embryo. Doubling the sample size would have provided a more acceptable power of 90%.

Although selenium poisoning of mallards and coots was confirmed, and poisoning of stilts seems likely, net effects on the local breeding populations of these species is unknown. Chevron USA operates a predator control program at the marsh, and the benefits of that protection could counterbalance losses caused by selenium poisoning. To date, neither the predator control benefits nor the selenium poisoning losses (especially posthatch) have been quantified to a level of scientific certainty that would permit a reliable cost–benefit evaluation.

Stilt eggs averaged about the same selenium exposure at Chevron Marsh (20–30 mg/kg) as that observed at Kesterson Reservoir, California (25–37 mg/kg), but the source water at Chevron Marsh averaged less than 10% as much selenium as the source water at Kesterson (20 vs. 300  $\mu\text{g/L}$ ). This unexpected result prompted the 1995 follow-up bioaccumulation study at Chevron Marsh, which revealed that transfer of selenium from water to aquatic invertebrates was

greatly enhanced compared to Kesterson and other agricultural drainage water sites, while transfer of selenium from aquatic invertebrates to black-necked stilt eggs was comparable. Thus, the unexpectedly high level of selenium in stilt eggs at Chevron Marsh appears to be a function of selenium chemistry in the water (primarily selenite at Chevron Marsh vs. selenate at Kesterson), not in the food chain. A 5-year remedial management and monitoring plan is being implemented in 1997 by Chevron USA and the San Francisco Bay Regional Water Quality Control Board, but the plan will not include any further monitoring of avian reproductive performance beyond the collection of small numbers of eggs in 1997, 1999, and 2001 (CH2M HILL, 1994, 1995; Chevron USA, 1996; San Francisco Bay Regional Water Quality Control Board file data).

## B. Agricultural Drainage Water Associated Case Studies

### 1. Kesterson Reservoir, California: Drainage Water Evaporation Impoundment

Kesterson Reservoir (B1 in Fig. 1), a 500 ha shallow impoundment (1–1.5 m deep) subdivided into 12 interconnected cells, was constructed in the northern San Joaquin Valley as part of a federal irrigation project. Located at the terminus of the San Luis Drain, Kesterson Reservoir served dual roles as an evaporation basin for agricultural drainage water and a managed wetland intended to benefit fish and wildlife populations. Following an initial period (1972–78) of receiving high quality agricultural spill water, during which time robust marsh vegetation and animal populations were established, Kesterson Reservoir began receiving highly saline subsurface drainage water. By 1981, virtually all inflow to the reservoir was saline drainage water. By the spring of 1982, federal biologists and resource managers noted an apparent deterioration of the aquatic ecosystem. Detailed ecotoxicological research was conducted during 1983–85 (Zahm, 1986; Ohlendorf, 1989).

Saline drainage water discharged from the San Luis Drain to Kesterson Reservoir averaged about 300  $\mu\text{g/L}$  selenium. The cells at Kesterson Reservoir were operated in series, such that concentrations of total dissolved solids, boron, and some other elements increased as water moved down series. In contrast, selenium concentrations in impounded water decreased as water moved down series from the receiving cells. Even in down-series cells, however, selenium concentrations still usually exceeded 50  $\mu\text{g/L}$ . This resulted in a severely contaminated aquatic habitat, with selenium concentrations in some food chain fauna, fish, and wildlife samples as high as 100 to 400 mg/kg (Table 1).

Although there are no hard data on the species composition of the fish fauna at Kesterson Reservoir prior to inflows of saline drainage water, as late as September, 1983, a multispecies warmwater fish assemblage was sampled in the San Luis Drain near its discharge point to Kesterson Reservoir (M. K. Saiki,

U.S. Geological Survey, personal communication). Therefore, there is at least a circumstantial basis to suspect that Kesterson Reservoir initially also contained a similar multispecies assemblage of warm-water fish. Shortly thereafter, however, only the pollution-tolerant mosquitofish (*Gambusia affinis*) persisted in the San Luis Drain and Kesterson Reservoir. The role of selenium in the disappearance of all other fish species, if selenium played any role, is unknown. During 1984 and 1985, samples of mosquitofish from the San Luis Drain averaged whole-body concentrations of about 120 mg/kg selenium and were documented to have a 20 to 30% incidence of stillborn fry compared to 1 to 3% in reference samples. The high incidence of stillborn fry was believed to be attributable to the high exposures of these fish to selenium (as opposed to a salinity effect) because many of the stillborn fry exhibited superficial signs of teratogenesis, and the salinity of San Luis Drain water, although elevated, was well below the upper limits known to be tolerated by mosquitofish (Saiki and Ogle, 1995; M. K. Saiki, personal communication).

The geometric mean selenium content of waterbird eggs at Kesterson Reservoir during 1983–85 varied by species and ranged from means of about 4 to 70 mg/kg (Ohlendorf and Hothem, 1994). Kesterson was surrounded by a rich landscape mosaic of cleaner wetlands, and wide-ranging species of birds, such as ducks, exhibited lower average selenium exposures than the more sedentary species did. Depressed egg viability (hatchability) and elevated incidence of embryo deformities were documented for several species of waterbirds. Upon pooling data for all species, it was found that at least 39% of 578 nests contained one or more inviable eggs and 26% of 2281 fully incubated eggs were inviable (vs. 1.2% for pooled reference eggs). When the reproductive data were segregated by species, findings were as follows: 4 to 49% of the fully incubated Kesterson eggs failed to hatch, and 0 to 15% contained deformed embryos. Embryo deformities were often multiple and typically involved the eyes, beak, and limbs (Ohlendorf et al., 1988). This distinctive pattern of multiple embryo deformities associated with highly elevated egg selenium is hereafter referred to as the “Kesterson syndrome.” Complete posthatch juvenile mortality was reported for several species. Signs of acute poisoning of adults, such as carcasses that exhibited alopecia, also were evident (Ohlendorf et al., 1986a; Ohlendorf, 1989; Ohlendorf and Skorupa, 1989; Skorupa and Ohlendorf, 1991).

The avian reproductive data collected at Kesterson Reservoir and nearby comparison sites were sufficient to permit the construction of several exposure–response curves. The curves clearly indicated that reproductive toxicity was exposure responsive and, combined with data from experimental feeding studies using captive mallards, confirmed the causative link between selenium exposure and embryotoxicity. However, because most data came from the highly contaminated Kesterson Reservoir, the lower end of the exposure axis (x axis) was poorly

represented and threshold points for adverse effects could not be identified very precisely (Ohlendorf et al., 1986b; Heinz, 1996).

All agricultural drainage discharges to Kesterson Reservoir were halted in 1986. By the end of 1988, Kesterson Reservoir had been dried up and the low-lying areas within the ponds filled with soil to at least 15 cm above the expected average seasonal rise of groundwater. Although ephemeral pooling of rainwater still occurs, Kesterson Reservoir has been transformed to a mosaic of primarily terrestrial habitats that are much less contaminated than the aquatic habitats they replaced. Postclosure biological monitoring has been conducted annually since 1987. Selenium concentrations in animal tissues have stabilized at slightly elevated levels, and no toxic effects are apparent (Ohlendorf and Santolo, 1994).

## 2. Tulare Basin, California: Drainage Water Evaporation Impoundments

The Tulare Basin (B2 in Fig. 1), located in the southern San Joaquin Valley about 160 km south of Kesterson Reservoir, has no natural drainage to the ocean. To meet the demand for agricultural drainage generated by extensive irrigation within the basin, more than 20 shallow impoundments were constructed during 1972–85 to provide for evaporative disposal of saline drainage water. These facilities varied from large multiple-celled systems (similar in design to Kesterson Reservoir) to small single-celled “ponds.” None of these facilities were intended to provide fish or wildlife benefits and, unlike Kesterson, they are devoid of emergent marsh vegetation. Nonetheless, these impoundments proved very attractive to waterbirds, including populations of nesting birds. Although all the facilities received saline drainage water, there was a wide span of ionic and trace element composition of impounded water. The selenium content of water discharged to these impoundments varied from less than 1  $\mu\text{g/L}$  to more than 1000  $\mu\text{g/L}$ . Detailed studies of avian exposure to contaminants and reproductive performance, methodologically matched to the Kesterson studies, were conducted during 1987–89. Additional wildlife monitoring and research at these facilities has occurred from 1982 to present (Moore et al., 1990; CH2M HILL et al., 1993; Robinson et al., 1997).

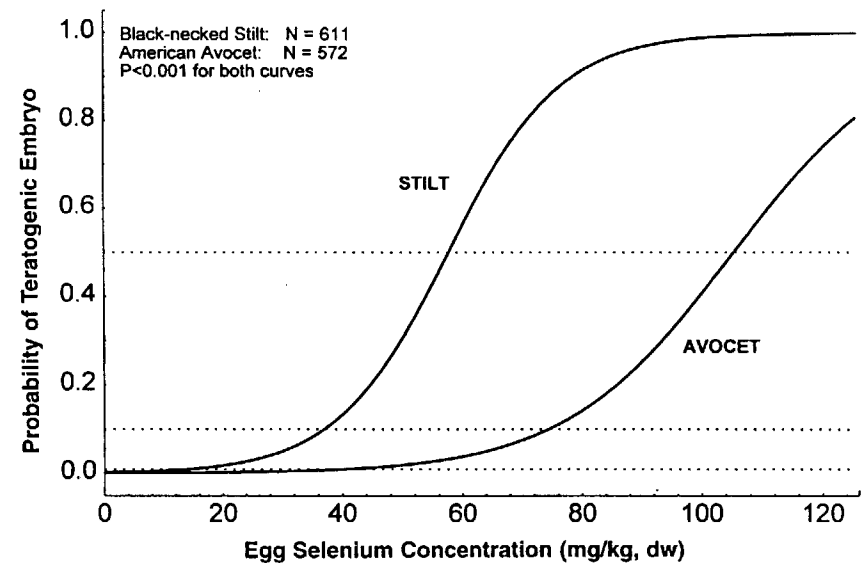
During 1987–89, four sites were found to exhibit highly elevated rates of embryo teratogenesis (10–50%) in one or more species of waterbirds (Table 1). The embryo deformity types at these sites matched the Kesterson syndrome, and maximum selenium concentrations in bird eggs at all four sites were highly elevated (80–164 mg/kg). The least contaminated of the four sites, the Tulare Lake Drainage District South facility, averaged about 15  $\mu\text{g/L}$  selenium in impounded water and about 20 mg/kg in duck eggs (Skorupa and Ohlendorf, 1991). This confirmed that reproductive toxicity could occur at agricultural drainage water sites with far less contamination than at Kesterson Reservoir. The true research value of the Tulare Basin, however, was the wide range of selenium

exposures associated with different evaporation facilities and the ubiquitous abundance of two species of birds, black-necked stilts and American avocets (*Recurvirostra americana*). Stilt and avocet eggs could be sampled across an exposure risk of four orders of magnitude (i.e., <1 to >100 mg/kg selenium in eggs). Thus, the Tulare Basin presented ideal circumstances for examining exposure-response relationships and threshold points for avian reproductive toxicity.

It is possible to determine unambiguously the overt teratogenic status (e.g., presence or absence of eyes) of any fresh stilt or avocet embryo incubated to at least 6 to 8 days of age. Consequently, teratogenic response is a very precise variable for examining species' relative sensitivity to selenium exposure. Sufficient field data have now been collected for stilts and avocets to allow delineation of species-specific teratogenic response curves (Fig. 2). Surprisingly, even though stilts and avocets are each other's closest phylogenetic relatives (Sibley et al., 1988), their response coefficients for selenium (coefficient  $b_1$  in Fig. 2) are significantly different ( $Z = 2.70$ ,  $P < 0.01$ ; Afifi and Clark, 1996). Based on predicted 50% effect concentrations ( $EC_{50}$  values in Fig. 2), stilt embryos are about twice as sensitive as avocets to in ovo selenium exposure. Stilts, therefore, are the more sensitive model species for further examination of threshold points.

Although embryo teratogenesis is a precise response variable, it is also relatively insensitive because teratogenesis is a severe response. Subtle disruptions of embryonic physiology and development are likely to cause inviability of stilt eggs at exposure levels less severe than those required for selenium-induced overt teratogenesis. Analysis of egg viability (hatchability) should therefore provide a sensitive estimate of the exposure threshold for reproductive toxicity. By examining the clutchwise incidence of inviable stilt eggs as a function of selenium concentrations measured in sibling sample eggs, it was determined that the threshold exposure for impaired hatchability occurred between 4 and 9 mg/kg selenium in the egg (Ohlendorf et al., 1993). Enough additional data have accumulated since 1993 to warrant a reexamination of that threshold region by single milligram-per kilogram increments. These additional data produce an estimate of the stilt embryotoxicity threshold that is narrowed to the region between 6 and 7 mg/kg selenium in eggs (Fig. 3). Thus, the upper boundary of safe exposure levels for stilt eggs (embryos) is about 6 mg/kg, or roughly three times the normal background exposure of about 2 mg/kg.

A strong relationship between the mean concentration of selenium in impounded water and in stilt eggs at the Tulare Basin sites ( $r^2 = 0.81$ ) is described by the equation shown in Figure 4, where egg and water selenium are expressed in milligrams per kilogram and micrograms per liter, respectively. Based on that regression equation, the average concentration of selenium in a population sample of stilt eggs would be expected to exceed 6 mg/kg when the mean selenium content of impounded drainage water exceeds 6  $\mu\text{g/L}$ . If the average exposure of embryos is 6 mg/kg selenium when water contains 6  $\mu\text{g/L}$ , then roughly 50%



GENERAL LOGISTIC MODEL:  $Y = \text{EXP}(b_0 + b_1 X) / (1 + \text{EXP}(b_0 + b_1 X))$

MODEL COEFFICIENTS:

MODEL	$b_0$	(S.E.)	$b_1$	(S.E.)
STILT	-6.125	(0.575)	0.1061	(0.0115)
AVOCET	-7.479	(1.179)	0.0710	(0.0144)

PREDICTED EFFECT CONCENTRATIONS (mg/kg, dw):

	STILT	AVOCET
$EC_{01}$	14	41
$EC_{10}$	37	74
$EC_{50}$	58	105

FIGURE 2 Logistic response curves for selenium-induced embryo teratogenesis among black-necked stilt and American avocet populations exposed to agricultural drainage water.

of individual eggs still exceed the toxic risk threshold (see Salton Sea data presented in Section II.B.3). Thus, the zero-exceedance threshold point must be associated with water containing less than 6  $\mu\text{g/L}$  total recoverable selenium. During the 1987-89 studies, eggs of stilts were collected at two sites with

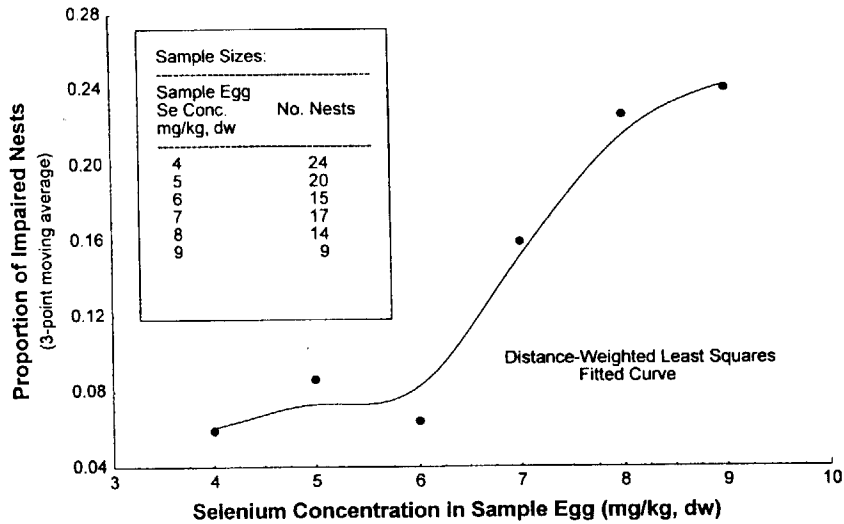


FIGURE 3 Response threshold for selenium-induced inviability of black-necked stilt eggs among populations exposed to agricultural drainage water.

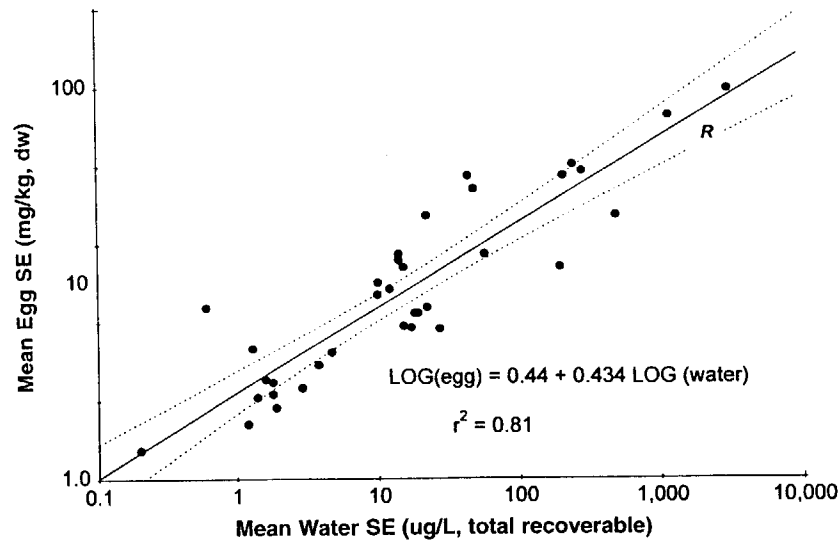


FIGURE 4 Regression relationship for mean selenium concentrations in black-necked stilt eggs as a function of mean selenium concentrations in impounded drainage water at study sites in the Tulare Basin, California. A data point for the Red Rock Ranch Agroforestry Demonstration Site is plotted as the letter R.

waterborne selenium in the 3 to 6  $\mu\text{g/L}$  range (Skorupa et al., unpublished data). At a site that averaged 3.8  $\mu\text{g/L}$  selenium in impounded water, the selenium content of five randomly sampled stilt eggs ranged from 2.2 to 5.6 mg/kg (zero exceedance, and a maximum value close to the 6 ppm safe limit). At a site that averaged 4.7  $\mu\text{g/L}$  selenium in impounded water, two of eight randomly sampled stilt eggs contained more than 6 mg/kg selenium (25% exceedance rate). Consequently, the reproductive effects threshold for black-necked stilts nesting at drainage evaporation facilities in the Tulare Basin appears to be about 4  $\mu\text{g/L}$  selenium in impounded water (about 10 times normal background for saline sinks).

This estimate of a toxicity threshold point at 4  $\mu\text{g/L}$  selenium in impounded drainage water is species-specific. As illustrated in Figure 2, even closely related species can substantially vary in their sensitivity to equivalent selenium exposures. The reproductive toxicity threshold point for stilts would be overly protective for avocets. Conversely, it might not be fully protective for other waterbird species. For example, based on a sample of 126 eggs from several species of dabbling ducks, the  $\text{EC}_{50}$  for overt teratogenesis is 31 mg/kg egg selenium (Skorupa et al., unpublished data) compared to 58 and 105 mg/kg for stilts and avocets (Fig. 2). These comparative results suggest that ducks may be roughly twice as sensitive as stilts to selenium-induced embryotoxicity and that depressed egg viability might be possible at sites with less than 4  $\mu\text{g/L}$  selenium in impounded drainage water.

About half the drainage water evaporation facilities in the Tulare Basin are no longer in operation. Those still running are regulated by means of Waste Discharge Requirements (WDR permits, as provided for pursuant to the California Water Code) that require creation of clean wetlands to mitigate unavoidable toxic impacts to breeding waterbirds. The mitigation wetlands are not allowed to average more than 2.7  $\mu\text{g/L}$  total recoverable selenium in impounded water (e.g., Central Valley Regional Water Quality Control Board, 1993).

### 3. Salton Sea, California: Regional Drainage Water Terminal Sink

The Salton Sea (B3 in Fig. 1), with a surface area of about 93,000 ha, is California's largest inland body of water. The present sea was created in 1905–07 when the entire flow of the Colorado River was accidentally diverted for about 16 months into the Salton Trough. Consistent with a desert climate, where evaporation greatly exceeds precipitation, the elevation of the sea fell by 18 m, and the salinity increased to 40,000 mg/L during 1907–25. Since the early 1920s the sea has served as a terminal sink for irrigation drainage water. By 1925 the elevation of the sea stopped declining because of counterbalancing inflows of drainage water. As agricultural development proceeded, and drainage water inflows increased, the sea has regained 10 m of lost elevation. Currently, more than 125,000 ha  $\cdot$  m/yr (1 million acre  $\cdot$  ft/yr) of agricultural drainage water is diverted to three

major river channels for conveyance to the Salton Sea (Saiki, 1990; Setmire et al., 1990; 1993).

Irrigation in the Coachella, Imperial, and Mexicali Valleys occurs with water diverted from the Colorado River. This source water contains selenium concentrations of about 2  $\mu\text{g/L}$ . Evaporative concentration of selenium in the shallow groundwater results in subsurface drainage containing up to 360  $\mu\text{g/L}$  selenium. The final mixture of surface and subsurface drainage conveyed to the Salton Sea contains 2 to 10  $\mu\text{g/L}$  selenium. Mass loading to the Salton Sea amounts to more than 8000 kg (>9 tons) of selenium per year. Although the water column of the sea contains only 1.5  $\mu\text{g/L}$  total recoverable selenium (Westcot et al., 1990), food chain fauna and tissues of fish and birds exhibit substantively elevated concentrations of selenium (Table 1). The levels of avian exposure to selenium revealed by the systematic surveys of Setmire et al. (1990, 1993) were sufficiently elevated to prompt follow-up studies of reproductive performance among black-necked stilts during 1992–93 (Bennett, 1997).

In 1992, 38 black-necked stilt eggs were sampled and averaged 6.6 mg/kg selenium. Twenty-two, or 58%, of the eggs exceeded the toxic risk threshold of 6 mg/kg associated with impounded drainage water in the Tulare Basin. The only reproductive performance variable measured in 1992 was the incidence of embryo teratogenesis. No selenium-induced terata were found among the 20 eggs that contained assessable embryos. However, because almost all the eggs contained selenium concentrations well below the 1% effect concentration ( $\text{EC}_{01}$ ) of 14 mg/kg for embryo teratogenesis in stilts (Fig. 2), a failure to find selenium-induced terata would be the predicted outcome. In 1993, 44 stilt eggs were sampled and averaged 5.8 mg/kg selenium. Twenty-one, or 48%, of the eggs exceeded the 6 mg/kg toxic risk threshold. Once again, as expected, no selenium-induced terata were found among 28 eggs that contained assessable embryos.

During 1993, however, the nests from which eggs were sampled also were monitored and the viability of sibling eggs was recorded. Among the 23 nests that survived to full term, 13% contained one or more inviable sibling eggs. Normally 8.9% of stilt nests contain one or more inviable eggs due to infertility and other natural causes. If the slightly elevated incidence of "affected" nests were due to selenium-induced inviability of eggs, it would represent about a 5% depression in nesting proficiency, where proficiency is defined as the proportion of nests in which all sibling eggs are viable  $[(0.911 - 0.87)/0.911 = 0.045 = \text{ca. } 5\%]$ . This putative magnitude of reproductive depression very closely matches predictions based on 410 sample eggs and sibling fates from full-term nests of stilts that were monitored at Kesterson Reservoir and in the Tulare Basin. Based on the Kesterson–Tulare logistic regression curve for nesting proficiency of stilts, the exposure-specific ( $N = 23$ ) predicted probability of affected nests for the Salton Sea sample is 11.9% (Skorupa et al., unpublished data). The close match between the 11.9% prediction and the 13% observed value supports the view

that the apparent 5% reproductive depression is biologically real. For such a small putative effect, about 225 full-term nests would have to be monitored to statistically distinguish between a true effect and random sampling error. Additional nest monitoring has been recommended for the Salton Sea (Bennett, 1997).

Because stilt eggs collected during the 1993 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 highly probable that the birds in this study were predominantly using wetlands with selenium concentrations in water of 10  $\mu\text{g/L}$  or less.

#### 4. Kendrick Reclamation Project, Wyoming: Seepage Wetlands

The Kendrick Reclamation Project (B4 in Fig. 1), near Casper, Wyoming, was constructed to store and divert water from the North Platte River for use within the Casper–Alcova Irrigation District. The project has been in operation since 1946, and by the mid-1960s it had expanded from an initial 425 ha of irrigated land to the present service area of 8000 to 10,000 ha. Soil in the service area is derived principally from seleniferous Cretaceous age geologic formations. Surveys of shallow groundwater revealed localized selenium concentrations as high as 23,000  $\mu\text{g/L}$ . Although the high bioavailability of selenium to plants within the Kendrick Project area had been reported much earlier (Rosenfeld and Beath, 1964), the potential for fish and wildlife exposure to selenium was not extensively investigated until 1986–90 (Peterson et al., 1988; See et al., 1992b).

Highly elevated selenium concentrations in bird eggs ( $\leq 135$ –160 mg/kg), along with elevated frequencies of overt embryonic terata consistent with the Kesterson syndrome, were documented at two wetlands within the project area: Rasmus Lee Lake and Goose Lake (Table 1). Both lakes functioned as terminal sinks during 1986–90. Both sites are considered seepage wetlands that are at least partially dependent on seepage from Casper Canal, the primary conveyance ditch for delivery of irrigation water (Peterson et al., 1988). Rasmus Lee and Goose Lakes also receive direct and indirect irrigation drainage and natural runoff, including snowmelt. The selenium content of those diverse inflows is mostly uncharacterized. Selenium concentrations in impounded water averaged 38  $\mu\text{g/L}$  in Rasmus Lee Lake and 54  $\mu\text{g/L}$  in Goose Lake. Based on ratios of selenium and chloride, it was concluded that evaporative concentration of Casper Canal source water (averaging 1  $\mu\text{g/L}$  selenium) was the only mechanism necessary to explain the highly elevated concentrations of selenium in water impounded at Rasmus Lee and Goose Lakes (See et al., 1992a). Consistent with highly elevated selenium in impounded water, sediments and food chain fauna at Rasmus Lee and Goose Lakes also contained very high concentrations of selenium (Table 1).

The most unambiguous selenium poisoning occurred among American avocets nesting at Rasmus Lee Lake in 1989. At least 6% of all embryos were

teratogenic (ca. 0.15% is normal for avocets), including some embryos exhibiting the suite of multiple malformations (eyes, limbs, bill) that is particularly characteristic of avian selenosis. More than 50% of the full-term avocet nests ( $N = 47$ ) contained one or more inviable eggs (ca. 12% is normal for avocets). These high rates of embryo deformity and inviability indicate a system that is far beyond the threshold for toxic effects, a conclusion consistent with the exposure data. A sample of 86 eggs had a median selenium concentration of 79 mg/kg (See et al., 1992a). The true median selenium content of eggs was probably somewhat lower because many nonrandomly selected "effect" eggs were included in the sample (See et al., 1992b).

The American avocet is relatively insensitive to selenium poisoning (Fig. 2), yet avocets experienced severe reproductive failure at Rasmus Lee Lake in 1989. Thus, the level of selenium contamination prevalent at Rasmus Lee Lake (Table 1) may be an order of magnitude or more above toxic thresholds for sensitive species. However, among randomly sampled avocet embryos at Kendrick ( $N = 28$ ), the incidence of teratogenesis was statistically consistent with the response curve for Tulare Basin avocets ( $\chi^2 = 1.29$ ,  $P > 0.05$ ; Skorupa and Ramirez, unpublished data) suggesting that Tulare Basin threshold estimates may also apply at Kendrick.

Poor reproductive performance also was documented for eared grebes (*Podiceps nigricollis*) and Canada geese (*Branta canadensis*). Grebe eggs exhibited selenium concentrations comparable to the avocet eggs, but interpretation of exposure-response data for grebes is complicated because grebes build floating nests. Water often seeps into grebe nests, and salts in the water can pass through the eggshells. Thus, potential embryotoxic effects from the ionic composition of the water (e.g., sulfates) would be difficult to partition from the effects of maternally deposited selenium (Skorupa et al., unpublished data). Severe deformities, such as anophthalmia, exhibited by eared grebe embryos at Goose Lake (See et al., 1992b), were very likely attributable to selenium poisoning, but the overall rates of embryonic inviability in eared grebe eggs could have been due to more than just selenium. The data for Canada geese do not present a strong case for selenium poisoning because embryo exposure to selenium was not consistent with the severity of observed effects. Unless Canada geese are extremely sensitive to selenium compared to known response curves (Fig. 2), or are exposed to a different form of selenium in their herbivorous diet (known response curves are from species with predominantly animal diets), it would be unreasonable to conclude that the severe reproductive depression documented for geese at Rasmus Lee Lake was solely the result of selenium poisoning.

The level of selenium in impounded water at Rasmus Lee and Goose Lakes (roughly 35–60  $\mu\text{g/L}$ ) was far above threshold levels for wildlife poisoning. Other than that conclusion, the data from Kendrick do not provide much insight on toxic threshold points. The U.S. Bureau of Reclamation and the Casper-Alcova

Irrigation District are planning a remediation strategy for reducing selenium levels in Rasmus Lee and Goose Lakes. Continued monitoring of these sites as remediation actions are implemented and concentrations of selenium are reduced could have great potential for precisely delineating toxic thresholds.

##### 5. Ouray National Wildlife Refuge, Utah: Seepage Wetlands

Ouray National Wildlife Refuge (Ouray NWR: B5 in Fig. 1) lies adjacent to the Green River near the town of Ouray, Utah. The refuge was established in 1960 as part of the mitigation for Flaming Gorge Reservoir and is managed primarily as waterfowl habitat. Agricultural land just northwest of the refuge is irrigated with water delivered from Pelican Lake by the Ouray Park Irrigation Company. The natural flow of groundwater toward the Green River results in subsurface seepage of irrigation drainage into wetlands near the western boundary of Ouray National Wildlife Refuge. The seepage contains elevated concentrations of selenium, probably from a combination of evaporative concentration of the source water (Pelican Lake water contains  $\leq 1 \mu\text{g/L}$  selenium) and leaching of selenium-enriched geologic formations (Stephens et al., 1992).

A pair of hydrologically connected ponds, the North and South Roadside Ponds, are maintained by seepage inflows and also periodically receive surface inflow of irrigation drainage water. The overall average selenium content of seepage and surface inflows to the Roadside Ponds was not determined, but upgradient shallow groundwater contained  $< 1\text{--}830 \mu\text{g/L}$  selenium. Impounded water in these flow-through ponds averaged about 40  $\mu\text{g/L}$  selenium during 1988–89. This level of contamination was sufficient to cause highly elevated selenium concentrations in food chain fauna, fish tissues, and bird tissues compared to normal reference values (Table 1). The most abundant species of waterbird nesting at the Roadside Ponds was the American coot (Stephens et al., 1992).

A sample of 21 coot eggs collected at random from the Roadside Ponds contained a geometric mean concentration of 50 mg/kg selenium (ca. 25–50 times normal). Embryo teratogenesis was documented for about 10% of the nests that survived to full term, but many inviable eggs were not assessable for embryo condition and therefore the percentage of teratogenic nests is probably underestimated. More than 85% of all full-term eggs failed to hatch, a severe level of reproductive depression comparable to the observations for American coots at Kesterson Reservoir in California. Although fewer than 10 duck nests were monitored, embryo teratogenesis also was documented for two species of ducks in eggs that contained about 20 to 45 mg/kg selenium. This case study provides another example of environmental contamination and avian exposure that is well beyond toxic thresholds. At another Ouray NWR impoundment (Sheppard Bottom-5), however, two eggs of black-necked stilts sampled in 1989 contained 5.3 and 5.4 mg/kg selenium (Peltz and Waddell, 1991), which is just below the

embryotoxicity threshold of greater than 6 mg/kg for stilt eggs (see Tulare Basin, Section II.B.2). Water at Sheppard Bottom-5 sampled in 1987 and 1988 contained 2 to 4  $\mu\text{g/L}$  selenium ( $N = 4$ , mean = 3.25  $\mu\text{g/L}$ ; Stephens et al., 1988; 1992), which appears very consistent with the toxic threshold estimate of about 4  $\mu\text{g/L}$  waterborne selenium determined for stilts in the Tulare Basin, California (see above).

#### 6. Red Rock Ranch, California: Agroforestry Demonstration Site

The California Department of Food and Agriculture and several cooperating agencies devised an integrated system of irrigation, drainage, and salt management that employs a series of increasingly salt-tolerant crops (including trees) and reuse of drainage water to greatly reduce the flow of saline wastewater from irrigated croplands (Cervinka, 1990). This "agroforestry" system was developed primarily to meet the need for managing irrigation salt loads in the San Joaquin Valley. A reduced flow of more highly concentrated saline drainage water facilitates salt recovery and maintenance of a systemwide salt balance. An additional anticipated benefit of the proposed system was the opportunity to replace traditional evaporation basins that create unavoidable selenium hazards for wildlife (see Kesterson Reservoir and Tulare Basin case studies above) with much smaller, plastic-lined solar evaporators that could be managed to prevent wildlife exposure to selenium. With the saline drainage water discharged to the solar evaporator at a rate equivalent to daily evaporative loss, no more than a thin film of water would ever cover the solar evaporator's plastic liner (or salt crust) and no aquatic habitat suitable for waterbirds would be created.

Red Rock Ranch (B6 in Fig. 1), near Five Points, California, includes one section of land (259 ha) set aside as an agroforestry demonstration site. The demonstration site includes 251 ha of traditional cropland, 5 ha of tree plantation, 1.8 ha for halophyte crops (such as *Salicornia*), and a small (0.86 ha) solar evaporator basin (Westside Resource Conservation District, 1995). Subsurface drainage systems were installed and trees planted in 1994. The solar evaporator basin was completed and halophytes planted in 1995. The full system of traditional irrigation integrated with sequential recovery and blending of drainage water to irrigate increasingly salt-tolerant crops, trees, and halophytes prior to terminal drainage discharge to the solar evaporator was operational in July 1995.

In May 1996 staff from the Central Valley Regional Water Board discovered that nonuniform distribution of drainage water inflow to the solar evaporator was great enough to cause patches of ponding (puddles) sufficient to attract breeding shorebirds. A sample of the water impounded in the solar evaporator contained more than 11,000  $\mu\text{g/L}$  selenium (Table 1). The irrigation furrows of the halophyte plot were also flooded with standing water, and a survey of nesting shorebirds conducted by U.S. Fish and Wildlife Service and Regional Water Board

staff in early June 1996 revealed that 12 of 13 nests at the demonstration site were located in the halophyte plot. Water entering the halophyte plot during the spring of 1996 averaged about 1600  $\mu\text{g/L}$  selenium (Westside Resource Conservation District, 1996). Representative sample eggs from 7 stilt nests contained a geometric mean selenium concentration of 58 mg/kg. Assuming that these stilts fed mostly in the halophyte plot, a mean egg selenium of 58 mg/kg is consistent with the water-to-egg regression for Tulare Basin stilt eggs (see point R plotted on Fig. 4). Overall, the status of 30 stilt embryos was determined, of which 17 (56.7%) were teratogenic (Skorupa et al., unpublished data). That is the highest incidence of selenium-induced avian teratogenesis reported by any field study to date, and it is consistent with the predicted rate of 50.4% based on the selenium content of the 7 sample eggs and the response curve for stilts presented in Figure 2. The status of 7 killdeer (*Charadrius vociferus*) embryos was also determined of which 1 (14%) contained a teratogenic embryo. Three representative sample eggs for killdeer contained a geometric mean selenium concentration of 19 mg/kg, but the teratogenic egg contained 57 mg/kg. Killdeer feed in upland as well as aquatic habitats and typically exhibit lower mean exposure to selenium than stilts when the two species co-occur at the same site (Skorupa et al., unpublished data).

During the summer of 1996, the method of water delivery to the solar evaporator was modified to provide a more even distribution of water, and this measure is expected to eliminate the ponding of water. Additionally, a wildlife monitoring and management program was devised in an attempt to prevent future avian nesting in the halophyte plot or elsewhere at the demonstration site (Westside Resource Conservation District, 1996).

#### C. Other Case Studies

##### I. Sweitzer Lake, Colorado: Mining Drainage//Ambient Seleniferous Geology//Irrigation Drainage

Sweitzer Lake (aka Garnet Mesa Reservoir; C1 in Fig. 1) near Delta, Colorado, was built in 1954 for recreational purposes. Sweitzer Lake occurs in an area with naturally seleniferous geological formations, but there is also a great deal of mining activity and irrigation drainage in the regions surrounding Sweitzer Lake (Barnhart, 1957). It is unestablished, however, how much of the cumulative selenium loading into Sweitzer Lake is of natural versus anthropogenic origins. Initial (1950s) water sampling revealed concentrations of selenium exceeding 100  $\mu\text{g/L}$ . Biotic selenium concentrations as high as about 20 mg/kg in benthic food chain fauna, and 40 mg/kg in fish hepatic tissue, were reported (Table 1). This level of exposure was associated with progressive mortality of stocked game fishes (7 species) attributed to excessive dietary intake of selenium (Barnhart, 1957; Lemly, 1985a; Butler et al., 1991).

In 1974 and 1977, the Colorado Division of Wildlife measured more than 100 mg/kg selenium in muscle tissue of fish and decided to stop stocking the lake, although catfish (*Ictalurus* spp.) were restocked in 1984. In 1978 water with selenium concentrations as high as 25  $\mu\text{g/L}$  was being discharged to Sweitzer Lake via agricultural drainage ditches, and impounded water contained up to 45  $\mu\text{g/L}$  selenium. Samples of impounded water in the late 1980s contained about 10 to 25  $\mu\text{g/L}$  selenium except for a deep-water sample containing 170  $\mu\text{g/L}$ ; muscle and eggs of catfish averaged about 30 mg/kg selenium, and a multispecies collection of six waterbird eggs contained 5.6 to 18 mg/kg selenium (Butler et al., 1991).

At that level of exposure, there was no evidence of successful reproduction among catfish. It was not determined, however, whether habitat conditions were suitable for catfish reproduction. It is not unusual for man-made reservoirs to be devoid of suitable spawning substrate for catfish (M. K. Saiki, personal communication). Large populations of green sunfish and carp (*Cyprinus carpio*) that included various age classes were reported by Butler et al. (1991), but selenium concentrations in eggs from those species were not measured. About 85% of waterbird eggs exceeded the 6 mg/kg toxic threshold point for black-necked stilts; however, stilts were not one of the species whose eggs were sampled at Sweitzer Lake. Insufficient nesting activity occurred at Sweitzer Lake to permit a systematic examination of avian reproductive proficiency. Based on the catfish and avian egg residue data, 10 to 25  $\mu\text{g/L}$  waterborne selenium at Sweitzer Lake is distinctly above toxic threshold points.

The most recent environmental monitoring conducted at Sweitzer Lake occurred in 1995 (R. Krueger and B. Osmundson, U.S. Fish and Wildlife Service, unpublished data). A water sample collected in April contained 24  $\mu\text{g/L}$  selenium. Sediment, aquatic invertebrates, fish, and avian eggs all contained highly elevated concentrations of selenium (Table 1). The eight American avocet eggs collected in 1995 are of particular significance because of the interpretive data available for avocets (Fig. 2). Additionally, a "stilt response standard" can be inferred directly from avocet exposure data because stilt and avocet eggs usually contain similar concentrations of selenium when these species co-occur at a study site (e.g., Ohlendorf and Skorupa, 1989; Skorupa and Ohlendorf, 1991; Ohlendorf and Hothem, 1994). The avocet eggs contained 23 to 32 mg/kg selenium with a geometric mean of 27 mg/kg. For a selenium-tolerant species like the avocet, that level of exposure would not be expected to have teratogenic impacts (Fig. 2). Based on a more sensitive standard, such as a "stilt response standard," that level of exposure would be expected to cause about a 4% incidence of embryo teratogenesis and an overall 21% depression in nesting proficiency (Fig. 2; Skorupa et al., unpublished data). Therefore, at Sweitzer Lake, a clear potential for moderate reproductive impacts among sensitive species of waterbirds is associated with impounded water containing less than 25  $\mu\text{g/L}$  selenium.

## 2. Swedish Lakes Project, Sweden: Mercury Remediation Treatments

Following initial experimentation at Lake Oltertjärn in 1985–86 (Paulsson and Lundbergh, 1989), 11 additional lakes widely distributed across Sweden (C2 in Fig. 1) were treated with selenite in an attempt to mitigate high levels of mercury in edible fish (Paulsson and Lundbergh, 1991, 1994). Treatments consisted of a leachable rubber matrix containing sodium selenite suspended in a sack 1 to 2 m below the lake surface for 2 years. The selenium-depleted rubber skeletons, remaining after continuous leaching of sodium selenite, were removed 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 to 5  $\mu\text{g/L}$  selenium (lakes initially contained about 0.1  $\mu\text{g/L}$  selenium). On average, the target concentration was achieved at most of the lakes, although up to 25 to 35  $\mu\text{g/L}$  was measured within 100 m of the leach sacks. Four lakes, however, never exceeded about 2.6  $\mu\text{g/L}$  average waterborne selenium. Because mitigation of mercury residues was equally successful in the four low-selenium lakes just cited and in the target concentration (3–5  $\mu\text{g/L}$ ) lakes, the dosing was adjusted in the second year of treatment for a target lake concentration of 1 to 2  $\mu\text{g/L}$  selenium.

Prior to treatment, concentrations of selenium in pike (*Esox lucius*) muscle tissue averaged 0.7 to 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 to 2.3 mg/kg selenium (1.6 mg/kg grand mean), and after 2 years of treatment they averaged 2.8 to 7.4 mg/kg (4.6 mg/kg grand mean). There was no evidence of catastrophic declines of pike populations in any of the lakes.

Prior to treatment, concentrations of selenium in perch (*Perca fluviatilis*) muscle tissue averaged 0.8 to 2.0 mg/kg. After the first year of treatment these tissues averaged 6 to 36 mg/kg selenium. By the end of the second year of treatment, researchers were unable to find any perch in four lakes and had a severely reduced catch from a fifth lake. Among the five lakes with an apparent collapse of perch populations, muscle tissues had averaged 6.9 to 36 mg/kg selenium (23 mg/kg grand mean) at the 1-year sampling point; by comparison, among the other six lakes muscle tissue of perch had averaged only 6 to 18 mg/kg selenium (12 mg/kg grand mean) at the 1-year sampling point, and in some cases had declined by the 2-year sampling point (consistent with lower dosing of lakes in the second year). At the end of 2 years, muscle concentrations of selenium in the six lakes where perch populations persisted averaged 6.9 to 26 mg/kg (15 mg/kg grand mean). The authors could not clearly establish the cause of the collapse of some perch populations, but they concluded that the possibility of selenium poisoning could not be excluded (Paulsson and Lundbergh, 1994). They also concluded that one of their "most important findings" was the need to keep waterborne selenium levels below 2  $\mu\text{g/L}$  to avoid undesirable levels

of selenium bioaccumulation in fish and unintentional side effects. Considering the differential persistence of pike (which coincided with substantively lower selenium exposure than perch), the absolute magnitude of the perch tissue data (well into the toxic range for other species of fish), and the stability of perch populations after selenium treatments of lakes were reduced to 2  $\mu\text{g/L}$  or less, the circumstantial case for selenium poisoning of perch in this field study seems very strong.

### III. MAJOR CASE STUDY GAPS

#### A. Mining Associated Case Studies

The mining of sulfide ores is commonly identified as a major source of anthropogenically mobilized selenium (see, e.g., Eisler, 1985). Despite the recognition that deep pit mining is producing numerous "pit lakes" with elevated levels of waterborne selenium (Miller et al., 1996), no case studies of wildlife at pit lakes nor biotic exposure assessments for selenium have yet been reported. A recent risk assessment for wetlands affected by copper smelting and refining on the south shore of the Great Salt Lake, Utah, reported selenium concentrations as high as 69 mg/kg in eggs of black-necked stilts (Fairbrother et al., 1997). In the San Joaquin Valley, stilt eggs containing that much selenium would have about a 75% probability of embryo teratogenesis (Fig. 2). Unfortunately, the few high-exposure stilt eggs in the Great Salt Lake sample were cracked and did not contain embryos that were assessable for terata. Future case studies of wildlife exposure to seleniferous mining drainage should have substantial potential to yield insights regarding toxic threshold points and the generality of currently delineated thresholds.

#### B. Feedlot and Feedbarn Associated Case Studies

As a result of the supplementation of livestock diets with selenium, it is not uncommon for the liquid manure in pits beneath feedlots or feedbarns to contain highly elevated concentrations of selenium (50–150  $\mu\text{g/L}$ ; see, e.g., Oldfield, 1994). Some livestock production schemes route this liquid manure to outdoor holding ponds where the potential for wildlife exposure exists (personal observation). To date, no environmental hazards associated with selenium supplementation of livestock feeds are known, but no systematic examination of wildlife use or selenium exposure at outdoor manure ponds has been reported either. The biochemistry of selenium in liquid manure could be quite unique compared to other sources of environmental selenium (Council for Agricultural Science and Technology, 1994), suggesting that future case studies in this arena could provide important new insights.

### IV. LESSONS FROM NATURE

#### A. Uniformity of Toxic Thresholds

Research at Belews Lake identified a toxicity threshold for fish of 2 to 5  $\mu\text{g/L}$  waterborne selenium (predominantly as selenite). It is now clear that there is nothing unique about the results from Belews Lake. The Martin Reservoir (fish and selenite), Tulare Basin (birds and selenate), and Swedish lakes (fish and selenite) case studies also provide strong documentation of a toxicity threshold at 5  $\mu\text{g/L}$  waterborne selenium or less. The Salton Sea (birds and selenate) and Ouray NWR (Sheppard Bottoms; birds and selenate) case studies provide circumstantial evidence for a threshold of 5  $\mu\text{g/L}$  or less. Thus, all six of the real-world toxic episodes that included biotic exposures near the Belews Lake threshold region uniformly support a criterion of  $\leq 5 \mu\text{g/L}$  for selenium in water. Equally noteworthy, no case studies of selenium exposure and response among biota in nature have *affirmatively* documented discordant estimates of toxicity thresholds.

The most extensive and detailed set of real-world toxic threshold data has been produced by long-term studies of birds exposed to irrigation drainage water in the San Joaquin Valley of California. For a focal species of intermediate sensitivity, the black-necked stilt, the toxic threshold for selenium in eggs ( $>6 \text{ mg/kg}$ ) was associated with 3 to 4  $\mu\text{g/L}$  selenium (predominantly as selenate) in impounded water. More importantly, the exposure–response relationships and bioaccumulation curves established for the Tulare Basin have proven reliable when applied to data from the other five irrigation drainage water case studies. Typical of this between-site convergence of data are the nearly identical teratogenesis response curves for stilt eggs from the Kesterson and Tulare Basin case studies (Fig. 5). Although insufficient data have been accumulated elsewhere to provide comparable site-specific response curves, additional examples of the interpretive consistency of data between sites were presented above for every irrigation drainage water case study. Thus, even the irrigation drainage water case studies with levels of biotic exposure and response too severe to yield direct estimates of threshold points (e.g., Kesterson, Kendrick, Ouray NWR—Roadside Ponds) indirectly support the threshold estimates derived from the Tulare Basin research.

#### B. Selenite Versus Selenate

In nature, selenite-dominated waters appear to have much steeper environmental response curves than selenate-dominated waters. Although response thresholds below 5  $\mu\text{g/L}$  have been identified for both types of water, the biotic consequences of threshold exceedance appear much more severe for selenite-dominated waters. For example, although a toxicity threshold point of about 3 to 4  $\mu\text{g/L}$  waterborne selenium was identified earlier in this chapter for black-necked stilts exposed to selenate-dominated irrigation drainage water in the Tulare Basin, exposures of

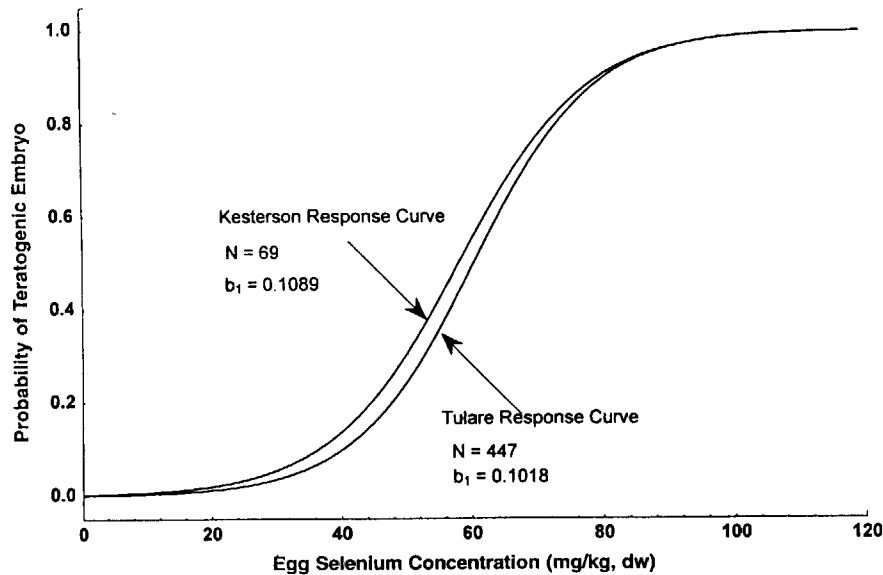


FIGURE 5 Comparison of teratogenesis response curves generated from black-necked stilt data at Kesterson Reservoir versus Tulare Basin evaporation basins. The selenium response logistic regression coefficients,  $b_1$ , are not significantly different.

stilts to irrigation drainage containing up to  $10 \mu\text{g/L}$  selenium result in reproductive depression of only a few percentage points (e.g., Salton Sea case study and Tulare Basin regression relationships). It was only because very large data sets (with the statistical power to detect small effects) were accumulated for stilts exposed to irrigation drainage that a precise threshold point below  $10 \mu\text{g/L}$  was ultimately identified.

In clear contrast to the situation for selenate, by the time selenite-dominated waters reach concentrations of  $10 \mu\text{g/L}$  total selenium, fish populations are completely collapsing (e.g., Belevs Lake, Hyco Reservoir, Martin Reservoir, and Swedish lakes), and avian populations are exhibiting the severe reproductive impacts more typical of selenate-dominated waters with total selenium exceeding  $100 \mu\text{g/L}$  (e.g., Martin Reservoir, Chevron Marsh). The bioaccumulation study at Chevron Marsh clarified the basis for this trend by showing that bioaccumulation of selenium into the food chain is much more proficient when the source water is selenite-dominated than when it is selenate-dominated. Consequently, selenite-dominated aquatic systems have “supercharged” food chains compared to selenate-dominated systems.

### C. Offstream Versus Instream Aquatic Systems

All 12 real-world toxic episodes of selenium exposure presented in this chapter are from off-stream aquatic environments. This pattern in nature probably explains why advocates for strengthening or relaxing regulatory criteria for aquatic selenium generally are polarized along a boundary that separates scientists whose primary experience is with off-stream systems from those whose primary experience is with in-stream systems. This axis of contention persists, at least in part, because it has not been demonstrated to any degree of scientific certainty whether the lack of well-documented in-stream toxic episodes is the result of categorical differences in toxic risk or the result of categorical bias against documenting in-stream risks (due to methodological constraints associated with studies of in-stream biota).

Two primary factors have greatly facilitated documentation of off-stream toxic episodes. First, off-stream case studies of fish have dealt with demographically closed populations. For demographically closed populations, even moderate selenium-induced reproductive deficiency leads to population collapse, an easily detected secondary response. Second, off-stream case studies of demographically open bird populations have relied on sampling eggs (embryo viability). Selenium-impaired embryo viability is a primary response not dependent on demographically closed populations for valid assessment of population response. Equally important, samples of bird eggs provide unbiased measures of biotic response to selenium exposure because the health of the embryo inside the egg does not influence a scientist's probability of sampling the egg, whereas samples of free-living birds or fish are “self-selected” to be insensitive (biased) measures of biotic response because only survivors (live specimens) are sampled. Clearly, only eggs provide unbiased exposure–response data in field studies.

Unfortunately, most in-stream studies of biotic response to selenium exposure are based on samples of free-living fish (biased survivors) from demographically open populations (i.e., populations that are regularly replenished by immigration of individuals from outside the segment of water being studied). Thus, it is common for in-stream studies to report the counterintuitive combination of abnormally elevated levels of selenium in fish tissue associated with what is viewed as a normally abundant and diverse fish fauna. It is then usually concluded that *conventional wisdom* regarding toxic thresholds for selenium, as derived from clinical data and off-stream field data, does not apply to the in-stream case at hand. Such studies meet neither the closed-population nor the unbiased exposure–response sampling criterion of a typical off-stream study and therefore are categorically very low power approaches for detecting toxic episodes.

To further illustrate this point, consider the six irrigation drainwater case studies summarized in this chapter. In all cases, normally abundant and diverse

bird faunas, or even superabundant bird populations, were present at the study sites despite selenium concentrations in avian liver or muscle tissues that were abnormally elevated. Without the egg data, demonstrating embryotoxic effects, researchers could easily have concluded in at least five of the six cases that the high levels of selenium in tissues of free-ranging birds were not associated with any evidence of harmful effects (Kesterson being the exception because adult mortality was widespread and led to the sampling of dead as well as live specimens of free-ranging birds). Of course in all five cases we know that the researchers would have been committing type II error (acceptance of a false negative). The crucial flaw of the dominant paradigm for in-stream studies is the interpretation of what are effectively only exposure surveys, because of their low power to detect a biotic response, as exposure–response studies. Such interpretations are highly vulnerable to type II error.

The preceding observations do not imply an absence of scientifically valid categorical reasons to question the applicability of off-stream toxicity results to in-stream risk management. For example, the lower primary productivity categorically associated with in-stream aquatic environments compared to off-stream systems should have major implications for the proportional within-system fluxes of selenium partitioned into biotic versus abiotic compartments. Similarly the categorically higher input of allochthonous energy into in-stream systems as opposed to off-stream systems could cause in-stream biota to be less tightly linked to waterborne levels of selenium (M. K. Saiki, personal communication). If it can be demonstrated that such differences result in categorically different bioaccumulation dynamics in-stream as opposed to off-stream, or that species-specific exposure–response curves for selenium in fish eggs are unequivocally different for off-stream and in-stream data sets, then a strong basis for context-dependent regulatory discrimination would indeed be established.

Since, however, all in-stream water can eventually become off-stream water, the off-stream–in-stream issue is moot. Even if valid site-specific arguments could be made for setting in-stream selenium criteria above off-stream toxic threshold values, from a systems level perspective the cumulative effects of maintaining in-stream water above off-stream toxic thresholds could be severe for fish and wildlife populations. For example, it is very conceivable that the millions of fish and birds that utilize California's Salton Sea would be in serious jeopardy if the cumulative effects of implementing site-specific selenium criteria within the Colorado River and its major tributaries caused even a 1 to 2  $\mu\text{g/L}$  increase downstream in the water diverted for irrigating the Coachella, Imperial, and Mexicali Valleys. That seemingly trivial increase would double the selenium load delivered to a Salton Sea system already exhibiting threshold toxicity. One of the lessons nature has provided at the Salton Sea is that the off-stream ecotoxicology of selenium should be considered as a limiting constraint on in-stream regulatory largesse.

#### D. Sulfate Dependency

It has been demonstrated clinically that environmentally relevant concentrations of sulfate inhibit the uptake of selenate by algae (e.g., Williams et al., 1994). Sulfate has also been clinically demonstrated to inhibit the bioconcentration of selenate by aquatic invertebrates (Hansen et al., 1993). Consequently, it has been suggested that aquatic ecosystems with relatively low sulfate levels may be particularly susceptible to selenium toxicity (i.e., may have lower toxic threshold points). Further, it has been asserted that EPA's chronic criterion of 5  $\mu\text{g/L}$  for selenium is inapplicable to high-sulfate waters because the criterion was derived from research at Belews Lake, which is a low-sulfate aquatic ecosystem (Mongan and Miller, 1995).

Real-world toxic episodes of fish and wildlife exposure to aquatic selenium, however, have not been restricted to low-sulfate systems. At least 7 of the 12 case studies reviewed for this chapter were high-sulfate systems. Moreover, the toxic threshold point (3–4  $\mu\text{g/L}$  selenium) for a waterbird utilizing high-sulfate irrigation drainage water is consistent with the toxic threshold point (2–5  $\mu\text{g/L}$  selenium) originally delineated by researchers at Belews Lake for fish in a low-sulfate system. The apparent absence of sulfate dependency in real-world episodes of selenium poisoning is consistent with Birkner's (1978) survey of 30 field sites in Colorado and Wyoming, which led him to conclude that levels of dissolved sulfate (5–9611 mg/L) did not influence the level to which selenium is accumulated by aquatic organisms. In the Tulare Basin (Fig. 4), sulfate concentrations from 2,000 to 100,000 mg/L did not significantly confound rates of selenium accumulation in avian eggs (via maternal dietary exposure to aquatic organisms).

Thus, the sulfate issue appears to be a good example of the lab-to-field dilemma (Landis and Yu, 1995). Both Hansen et al. (1993) and Williams et al. (1994) explicitly discussed the possibility that their lab results (based on only 48–96 h exposures in grossly simplified systems) might not be relevant to the field. The lesson from nature is that toxic threshold points for selenium are not sulfate-dependent.

#### V. CONCLUSION: RISK MANAGEMENT

Conceptually, a *national* water-based criterion for selenium is a safety net set at the lowest height necessary to be reasonably certain that across a broad (national) array of environmental permutations, no potentially injurious falls will occur below the level of the safety net. Consider the safety net analogy further: even though the trapeze artist may work at a height of 35 m above the ground, the safety net is set at 2 m above the ground, not 30 m, because the artist *potentially* could fall at any point along the climb up to 35 m or along the climb down

from it, and from 2 m upward that fall could *potentially* be injurious. Risk management must focus on the *constraining potentials for risk*. The fact that in many individual cases trapeze artists routinely climb up to and down from a height of 35 m without falling does not justify raising the net from 2 m to 30 m (even though the greatest risk of falling is from 35 m, the height at which the trapeze artist performs). However, even one injurious fall from 2 m would be sufficient to justify lowering the safety net to below 2 m.

If we consider short-term catastrophic demographic impacts to fish or wildlife as the “fall” we want our selenium criterion “safety net” to protect against (a minimal level of protection), where “catastrophic” might be defined as a population collapse of 50% or more among sensitive species, then the real-world data from nature support the viewpoint that EPA’s current chronic criterion for selenium of 5  $\mu\text{g/L}$  is set too high. Short-term catastrophic demographic impacts on birds at Martin Reservoir and on fish in Swedish Lakes were evident at below 5  $\mu\text{g/L}$ . If we want our safety net to be just under known toxic thresholds (a reasonable level of protection), even if those thresholds are associated with modest demographic impacts, then nature’s lessons at Belews Lake (second-generation studies, i.e., Lemly, 1993b; 1997), Hyco Reservoir, Chevron Marsh, Tulare Basin, Salton Sea, and Ouray NWR (Sheppard Bottom) also dictate a national water-based criterion for selenium of <5  $\mu\text{g/L}$ .

Managing the potential for risk across a broad (national) array of environmental permutations, where the riskiest permutations are not fully predictable solely from concentrations of selenium in a water column, means that there will be site-specific cases where *potential* risk is unlikely ever to be *realized*. Thus arises the question of how to identify and appropriately manage site-specific risk, including establishment of site-specific criteria. To delve into that topic in detail is beyond the scope of this chapter, but nature’s relevant lesson is that avian and fish eggs are the appropriate metric for precise and reliable site-specific risk management. So far, all the real-world data suggest that selenium concentrations in bird and fish eggs incorporate and boil down all the confounding between-site variability in environmental permutations to a universal currency that is very reliable for risk assessment and management.

Consequently, nature’s two bottom lines are that it is not unusual for toxic risk to fish and wildlife populations to be associated with less than 5  $\mu\text{g/L}$  selenium in impounded water (thus the current national chronic criterion of 5  $\mu\text{g/L}$  is not an adequate safety net), and that only bird and fish eggs provide a risk metric reliable enough to justify fiddling with the safety net on a site-specific basis (and even then, downstream effects must be given full consideration).

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# 19

## Magic Numbers, Elusive Lesions: Comparative Pathology and Toxicology of Selenosis in Waterfowl and Mammalian Species

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I gave careful consideration to the possibility that anatomical dissection might be used to check speculation.

Andreas Vesalius (1539), *Epistola, docens venam axillarem dextri cubiti in dolore laterali secundem: Et melancholicum succum ex venae portae remis dedem pertinaentibus, purgari.* (O'Malley, 1965)

Characterization of disease syndromes caused by excess dietary selenium (selenosis) represents one of the historic research achievements of the U.S. agricultural experiment station system. In the late 1920s and 1930s this success was exemplified by the interdisciplinary work of Kurt Franke and his colleagues at South Dakota State University (Franke et al., 1934; Moxon, 1937; Moxon and Rhian, 1943).

Research into selenosis also had its failures, particularly with regard to disease syndromes incorrectly attributed to the element. For some years, based on experimental studies using rats fed seleniferous grain or inorganic selenium, selenium was considered a cause of hepatic tumors (Nelson et al., 1943). Since selenium is an essential micronutrient in animal rations, this led to continuing regulation of selenium supplements by the FDA under the Delaney clause, and recurrent debates about benefits and risks of such supplementation (Oldfield et al., 1994). Subsequently it was established that original studies were flawed by poor experimental design and the inability of investigators to distinguish age-related changes from hepatic carcinomata (Shamberger, 1985). Excess dietary