ASSESSING THE TOXIC THREAT OF SELENIUM TO FISH AND AQUATIC BIRDS

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Abstract. A procedure is given for evaluating the toxic threat of selenium to fish and wildlife. Toxic threat is expressed as hazard, and is based on the potential for food-chain bioaccumulation and reproductive impairment in fish and aquatic birds, which are the most sensitive biological responses for estimating ecosystem-level impacts of selenium contamination. Five degrees of hazard are possible depending on the expected environmental concentrations of selenium, exposure of fish and aquatic birds to toxic concentrations, and resultant potential for reproductive impairment. The degree of hazard is given a numerical score: 5 = high hazard, 4 = moderate hazard, 3 = low hazard, 2 = minimal hazard, and 1 = no identifiable hazard. A separate hazard score is given to each of five ecosystem components; water, sediments, benthic macroinvertebrates, fish eggs, and aquatic bird eggs. A final hazard characterization is determined by adding individual scores and comparing the total to the following evaluation criteria: 5 = no hazard, 6–8 = minimal hazard, 9–11 = low hazard, 12–15 = moderate hazard, 16–25 = high hazard. An example is given to illustrate how the procedure is applied to selenium data from a typical contaminant monitoring program.

Introduction

The importance of selenium as an environmental contaminant has gained widespread attention among scientists, natural resource managers, and water quality regulators in the U.S. during the past two decades. Selenium mobilized from the combustion of coal at electric generating stations has contaminated several major reservoirs, leading to reproductive failure and elimination of entire communities of fish (Cumbie and Van Horn, 1978; Garrett and Inman, 1984; Woock and Summers, 1984; Woock *et al.*, 1985; Lemly, 1985a, 1993a). Irrigation of seleniferous soils has produced subsurface drainage that contaminated wetlands and poisoned fish and migratory birds at several locations in the western U.S. (Lemly, 1993b, 1994; Presser, 1994; Presser *et al.*, 1994). The U.S. Environmental Protection Agency and some states have formulated and adopted increasingly restrictive water quality standards for selenium because of these incidents of environmental contamination (NCDEM, 1986; USEPA, 1987; CEPA, 1992).

Most natural resource management agencies are aware of the toxic threat posed by selenium and many have implemented contaminant monitoring programs to measure selenium concentrations in water, sediments, and aquatic biota. Once these data are collected it is important to conduct an overall evaluation and determine the degree of hazard present in order to identify appropriate management actions. However, few agencies address this information need by performing a

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comprehensive hazard assessment. There likely are two reasons for this. First, it has been difficult for those conducting the monitoring programs to determine the toxicological significance of selenium residues in aquatic organisms. Taking time to locate, obtain, and interpret the results of selenium toxicity tests for a variety of aquatic species is a difficult task. Fortunately, this type of information synthesis has been done. Guidelines are now available for evaluating selenium in food-chain organisms and fish and wildlife tissues based on current toxicological information (Skorupa and Ohlendorf, 1991; Lemly, 1993c; Beyer *et al.*, 1996; Skorupa *et al.*, in press). Interpreting selenium residues, for the most part, is no longer a primary concern.

The second reason, which still persists, is the lack of a broadly applicable procedure for conducting an aquatic hazard assessment of selenium. This has left investigators on their own, struggling with selenium monitoring data and trying to come up with a reasonable approach for characterizing hazard. Several approaches have been tried. Early attempts relied on comparisons between waterborne concentrations measured in the field and concentrations that were toxic to aquatic organisms in the laboratory (Cumbie and Van Horn, 1978; Adams and Johnson, 1981; Lemly, 1982). However, the laboratory data did not adequately explain the greater toxic effects observed for fish in the field. Subsequent research on selenium indicated that food-chain bioaccumulation, dietary intake, and reproductive effects should be given high priority in hazard assessment (Woock and Summers, 1984; Garrett and Inman, 1984; Sorensen *et al.*, 1984; Lemly, 1985a, 1985b; Baumann and Gillespie, 1986; Gillespie and Baumann, 1986; Heinz *et al.*, 1987). Utilizing this and other information, some investigators have developed mathematical models to describe and predict selenium cycling and toxicity (e.g., Porcella *et al.*, 1991; Peterson and Nebeker, 1992; Bowie and Grieb, 1991; Bowie, 1995). Models can be a useful tool for predicting toxicological risk. The major drawback is that these methods are relatively complex and, in order to provide reliable predictions, require considerably more information than would be available from a typical contaminant monitoring program. Thus, the potential for models to be routinely used in hazard assessment is limited.

Despite the large amount of new toxicity information generated during the past decade (e.g., USFWS, 1990a, 1990b; Figure 1), and the need to evaluate selenium hazards at many locations across the U.S. (Peterson and Nebeker, 1992; Presser *et al.*, 1994; Seiler and Skorupa, 1995), there is no common assessment method in use. This has resulted in confusion and frustration for those involved in hazard assessment of both site-specific and regional selenium contamination problems (Sylvester *et al.*, 1991; Presser *et al.*, 1994).

Field biologists and fish and wildlife managers would benefit by having an easily applied, scientifically credible procedure for conducting aquatic hazard evaluations of selenium. The procedure presented here was developed to address that information need. It includes the key parameters that are implicit in basic toxicological risk assessment such as concentration and exposure, but it also integrates biotic and

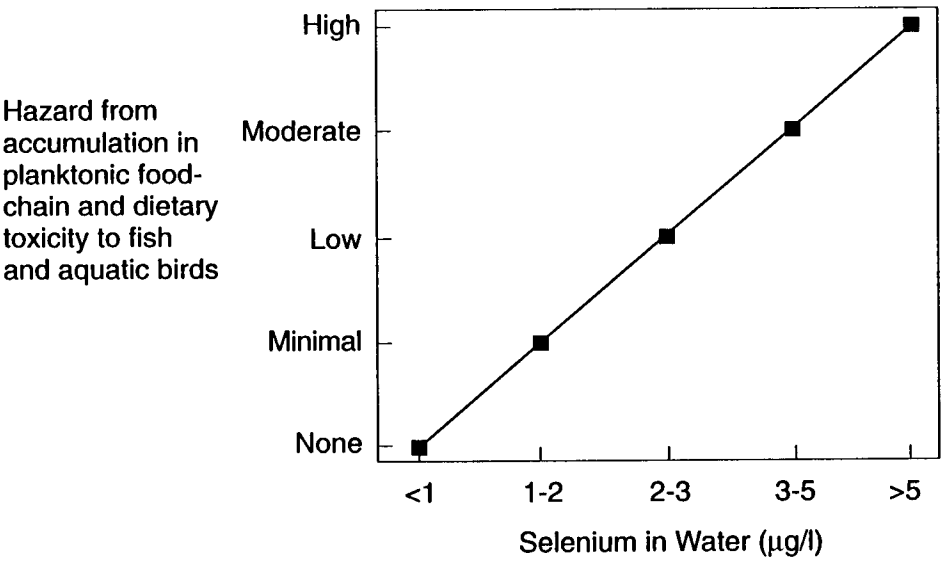

Figure 1. Hazard profile for selenium accumulation from water into the planktonic food chain, and resultant dietary toxicity to fish and aquatic birds. Waterborne concentrations are for 0.45 μm filtered samples. This and subsequent hazard profiles (Figures 2–5) are based on a synthesis of toxicity, bioaccumulation, and sediment data from Traversy *et al.*, 1975; Adams and Johnson, 1977; Birkner, 1978; Cumbie, 1978; Cumbie and Van Horn, 1978; Goettl and Davies, 1978; Cherry *et al.*, 1979; Furr *et al.*, 1979; Guthrie and Cherry, 1979; Holland, 1979; Duke Power Company, 1980; Hilton *et al.*, 1980; Nassos *et al.*, 1980; Speyer, 1980; Hilton and Hodson, 1983; Hodson and Hilton, 1983; Nriagu and Wong, 1983; Rudd and Turner, 1983; Sorensen and Bauer, 1983, 1984a, 1984b; Turner and Rudd, 1983; Bryson *et al.*, 1984; Garrett and Inman, 1984; Hicks *et al.*, 1984; Martin and Hartman, 1984; Sager and Cofield, 1984; Sorensen *et al.*, 1982a, 1982b, 1983a, 1983b, 1984; Wiener *et al.*, 1984; Woock, 1984; Woock and Summers, 1984; Finley, 1985; Lemly, 1985a, 1993a, 1993d; Winger and Andreason, 1985; Woock *et al.*, 1985, 1987; Baumann and Gillespie, 1986; Bennett *et al.*, 1986; Byrne and DeLeon, 1986; Foe and Knight, 1986; Gillespie and Baumann, 1986; Hamilton *et al.*, 1986, 1990; Kleinow and Brooks, 1986; Ohlendorf *et al.*, 1986a, 1986b, 1987, 1988, 1989, 1990; Saiki, 1986; TUGCO, 1986; White *et al.*, 1986, 1987; Heinz *et al.*, 1987, 1988, 1989, 1990; Presser and Ohlendorf, 1987; Saiki and Lowe, 1987; Thompson and Watling, 1987; Barnum and Gilmer, 1988; Boyum and Brooks, 1988; Gillespie *et al.*, 1988; Hoffman and Heinz, 1988; Hoffman *et al.*, 1988, 1991; Ogle *et al.*, 1988; Smith *et al.*, 1988; Sorensen, 1988; Stephens *et al.*, 1988, 1992; Besser *et al.*, 1989, 1993; Coughlan and Velte, 1989; Dubowy, 1989; Hothem and Ohlendorf, 1989; Ogle and Knight, 1989; Ohlendorf, 1989; Pyron and Beitinger, 1989; Williams *et al.*, 1989; Fairbrother and Fowles, 1990; Ingersoll *et al.*, 1990; Kiffney and Knight, 1990; Schuler *et al.*, 1990; Schultz and Hermanutz, 1990; USFWS, 1990a, 1990b; Allen, 1991; Cutter, 1991; Horne, 1991; Ornes *et al.*, 1991; Peltz and Waddell, 1991; Riedel *et al.*, 1991; Skorupa and Ohlendorf, 1991; Crane *et al.*, 1992; Hernmanutz, 1992; Hermanutz *et al.*, 1992; Waddell and Stanger, 1992; Cleveland *et al.*, 1993; Coyle *et al.* 1993; Goede, 1993; Hallock and Hallock, 1993; Heinz, 1993; Heinz and Fitzgerald, 1993a, 1993b; Maier and Knight, 1993; Olson and Welsh, 1993; Saiki *et al.*, 1993; Hamilton and Waddell, 1994; King *et al.*, 1994; Presser *et al.*, 1994; Sanders and Gilmour, 1994; Stanley *et al.*, 1994; Welsh and Maughan, 1994; and Skorupa *et al.*, in press.

abiotic cycling components that are essential for site-specific hazard evaluation of selenium. The method generates numerical scores that can be compared between years and across sites and locations, thereby providing a consistent approach for

evaluating hazard. The assessment focuses on food-chain bioaccumulation and reproductive impairment in fish and aquatic birds, which are the most sensitive biological endpoints for determining potential ecosystem-level impacts of selenium (Lemly, 1993a, 1993c).

Definition of Toxic Threat

In the context of this assessment procedure, toxic threat is expressed as the hazard to fish and aquatic birds that use a specific habitat known or suspected of being contaminated with selenium. Hazard is characterized from two types of information: (1) the degree of selenium contamination present, which is used as an estimate of the expected environmental concentration, and (2) the extent of fish and wildlife exposure to potentially toxic selenium concentrations. Exposure can be difficult to characterize precisely. However, the data set required for assessment helps with this problem because it specifies the use of egg selenium concentrations. This focuses the assessment on the reproductive cycle, which is the critical period of exposure necessary for toxic impacts. Hazard is maximized when the degree of contamination is high and exposure occurs during the breeding season.

Five categories of hazard are recognized in the procedure based on the potential for reproductive impairment in fish and aquatic birds. High hazard denotes an imminent, persistent toxic threat sufficient to cause complete reproductive failure in most species of fish and aquatic birds. Moderate hazard indicates a persistent toxic threat of sufficient magnitude to substantially impair, but not eliminate reproductive success; some species will be severely affected while others will be relatively unaffected. Low hazard denotes a periodic or ephemeral toxic threat that could marginally affect the reproductive success of some sensitive species, but most species will be unaffected. Minimal hazard indicates that no toxic threat is identified but concentrations of selenium are slightly elevated in one or more ecosystem components (water, sediment, invertebrates, fish, birds) as compared to uncontaminated reference sites; continued comprehensive environmental monitoring is recommended. No hazard denotes that no toxic threat is identified and selenium concentrations are not elevated in any ecosystem component; periodic baseline reconnaissance monitoring is recommended.

Data Requirements

The procedure requires a set of data for selenium concentrations measured in five ecosystem components – water (0.45 μm filtered samples), sediments, benthic macroinvertebrates, fish eggs, and aquatic bird eggs. Incomplete data sets, i.e., one or more ecosystem components missing, will weaken the predictive power of the assessment but it can still be performed. Depending on species and time of

year, bird eggs and gravid fish ovaries may be difficult or impossible to obtain. In this case, selenium can be measured in bird livers and whole-body samples of fish, and the results converted to approximate egg concentrations using the following conversion factors: bird egg selenium = bird liver selenium × 0.33; fish egg selenium = fish whole-body selenium × 3.3 (Lemly and Smith, 1987; Skorupa *et al.*, in press). When nesting birds are sampled it is desirable to collect eggs of species that use a localized feeding area which represents the study site and thus the local conditions of exposure to dietary selenium. Species such as American coots (*Fulica americana*), grebes (*Podilymbus* spp.), and dabbling ducks (*Anas* spp.) are good choices in this regard. With fish, species that are resident in the area will suffice. Depending on location, minnows (Cyprinidae), sunfish (Centrarchidae), suckers (Catostomidae), catfish (Ictaluridae), and trout (Salmonidae) should be readily available and easy to sample. For some species, particularly cyprinids in streams, it will probably be necessary to select large individuals in order to obtain sufficient egg mass for analysis. Migratory species of birds and fish that could have recently arrived (within 2 weeks) at the study site from distant locations should be avoided because they may not have had time to accumulate tissue residues of selenium that reflect local exposure conditions (Heinz *et al.*, 1990).

Data for the procedure can be from a one-time sampling effort or from an ongoing monitoring program of considerable duration. However, if the later case exists, it is recommended that the most recent data (1–2 years) be used since it would yield the best estimate of current hazard. Eggs are a key component in the assessment because of the fact that reproductive success is the most sensitive biological response for determining selenium toxicity to fish and aquatic birds (Lemly, 1993c). Selenium ingested in the diet is readily transmitted to developing eggs from the parent, where it can cause teratogenic deformities and embryomortality. Complete reproductive failure can occur with no observable toxic effects on the adults (Gillespie and Baumann, 1986; Woock *et al.*, 1987; Heinz *et al.*, 1987, 1989; Coyle *et al.*, 1993). The procedure thus incorporates a sensitive indicator that reflects the mode of toxicity responsible for the most serious community- and ecosystem-level effects.

In order for the assessment to be reliable, the data for selenium concentrations must also be reliable. Analytical methods used to determine selenium should be sensitive and under strict, documented quality control. Methods for water and tissues should have limits of detection in the sub-μg/l range and sub-μg/g range respectively. This is important because an error of only 1 μg/l or 1 μg/g in measured concentrations could cause the hazard rating for an environmental component to fall in the wrong category. Propogation of this error throughout the five environmental components used in the procedure could result in an inappropriate final hazard characterization. Acceptably sensitive analytical methods include neutron activation, hydride-generation atomic absorption spectrophotometry, and cathodic stripping voltammetry (McKown and Morris, 1978; Jarzabek and Kublik, 1982; Brooks *et al.*, 1983). Tissue and sediment preparation techniques appropriate for

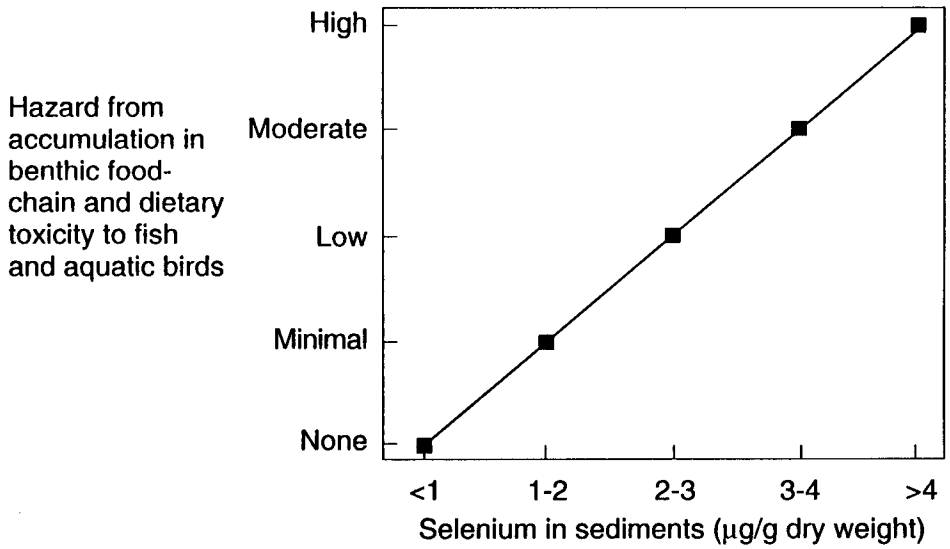

Figure 2. Hazard profile for selenium accumulation from sediments into the benthic food chain, and resultant dietary toxicity to fish and aquatic birds.

use with these methods are available (e.g., Grant, 1981; May, 1982; Krynitsky, 1987; Fio and Fujii, 1988; Brumbaugh and Walther, 1989). Analytical methods such as inductively-coupled argon plasma spectroscopy (ICAP) should be avoided if possible. Although ICAP scans are good for reconnaissance-level monitoring to identify field sites of concern, the detection limits are generally too high and the precision too low for determining concentrations to be used in the hazard assessment. Establishing a contract for selenium measurements with a reputable environmental laboratory is a desirable, cost effective alternative to developing in-house analytical chemistry capabilities.

Conducting an Assessment

Selenium concentrations measured in samples collected from a study site are compared to hazard profiles (Figures 1–5). These profiles are based on the results of extensive field and laboratory studies of selenium, and reflect the most current information on bioaccumulation and toxic thresholds (see references for Figure 1). Waterborne concentrations are compared to the profile for water, sediments to sediments, and so forth. A hazard rating is determined for each of the five ecosystem components based on where the highest concentrations of selenium in the samples fall on the hazard scale (high, moderate, low, minimal, none). Individual hazard ratings are then given a numerical score: high hazard = 5, moderate hazard = 4, low hazard = 3, minimal hazard = 2, and no identifiable hazard = 1. A final haz-

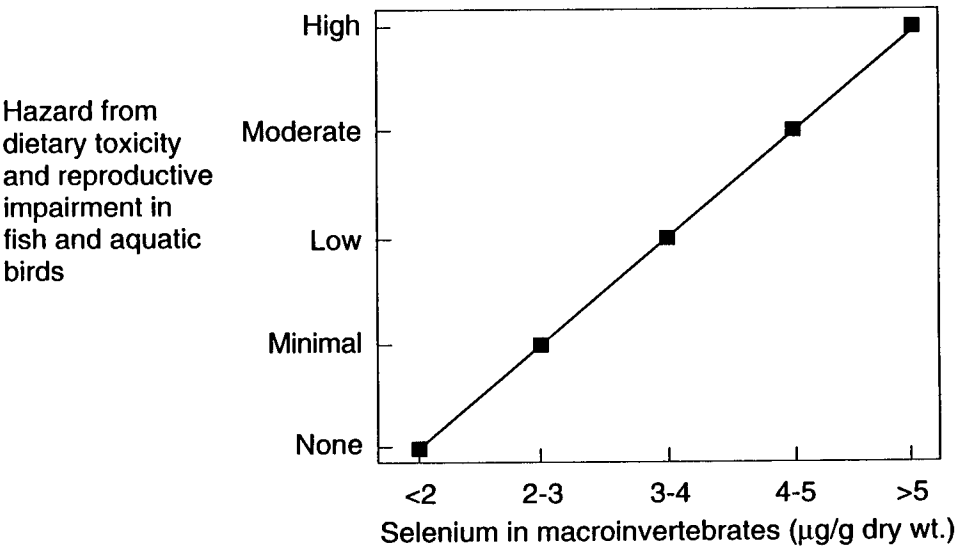

Figure 3. Hazard profile for dietary toxicity and reproductive failure in fish and aquatic birds from ingestion of selenium-contaminated macroinvertebrates.

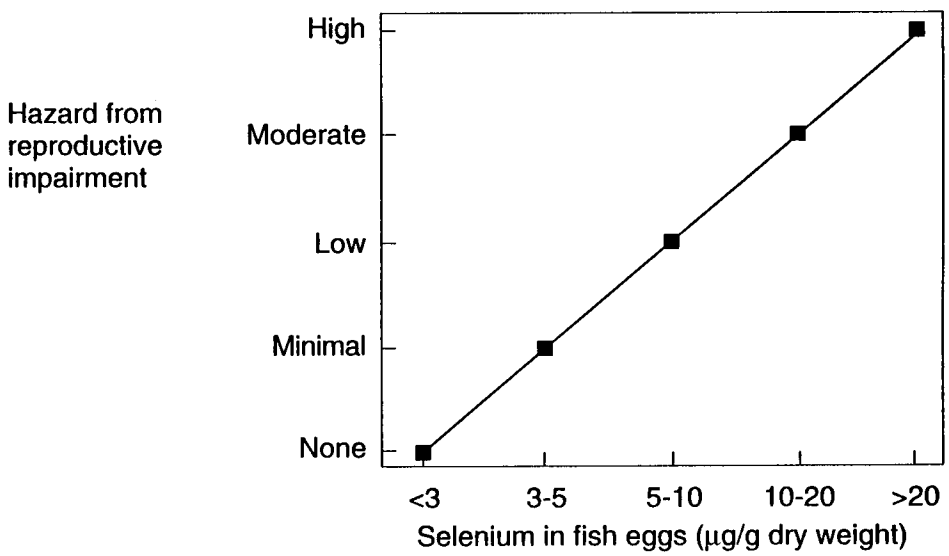

Figure 4. Hazard profile for selenium-induced reproductive impairement in fish.

ard characterization is determined by adding together the five individual scores and comparing the total to the following evaluation criteria: 5 = no hazard, 6–8 = minimal hazard, 9–11 = low hazard, 12–15 = moderate hazard, 16–25 = high

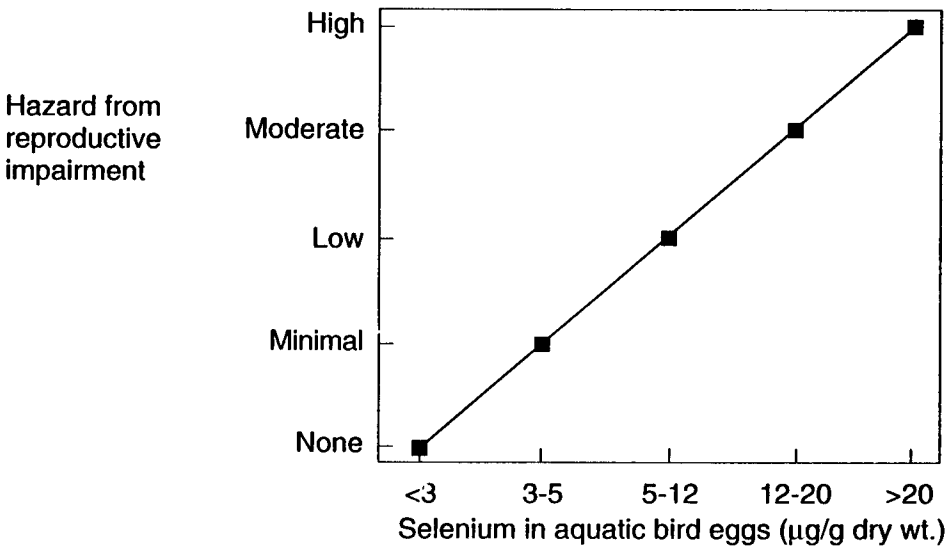

Figure 5. Hazard profile for selenium-induced reproductive impairment in aquatic birds.

hazard. The assessment thus integrates hazard component scores and provides an ecosystem-level evaluation.

The intervals for the final hazard characterization are based on the scores for individual components but they are not a simple average or a midpoint. For example, a final hazard rating of "low" can occur even though each of the individual components was rated "minimal". The rationale for this is that three distinct routes of exposure are possible for selenium (water, planktonic food-chain, detrital food-chain). Based on field evidence, the hazard from all three together should be greater than if each is considered separately. Thus, the aggregate hazard for the environment as a whole can be greater than the hazard for the individual components. This is analogous to synergism, i.e., the parts (hazard) are not simply additive.

Depending on the needs of the investigator, the procedure can be applied to data collected for a single sampling site or to pooled data from a monitoring effort covering a large area. The procedure can thus address several levels of landscape scale and provide site-specific as well as regional assessment of selenium hazard. Comparisons can be made between locations and years, and follow-up assessments can be made to evaluate the success of management actions and cleanup activities at selenium contaminated sites.

The procedure was recently used to evaluate the toxic threat of selenium to fish and migratory birds at Ouray National Wildlife Refuge, Utah (Lemly, 1995). The example given here was taken from that evaluation. Data for selenium concentrations were obtained from the records of a contaminant monitoring program (Stephens *et al.*, 1988, 1992; Peltz and Waddell, 1991; Waddell and Stanger, 1992).

Table I

Data set for aquatic hazard assessment of selenium at Ouray National Wildlife Refuge, Utah

Site and environmental component	Selenium concentration[a]	Evaluation by component		Totals for the site	
		Hazard[b]	Score	Score	Hazard
Leota Bottom					
Water	<1–3	Low	3		
Sediments	0.7–1	None	1		
Invertebrates	1–3	Minimal	2		
Fish eggs	2–4	Minimal	2		
Bird eggs	2–7	Low	3		
			11	11	Low
Roadside Ponds					
Water	9–93	High	5		
Sediments	7–41	High	5		
Invertebrates	12–72	High	5		
Fish eggs	75–120	High	5		
Bird eggs	12–120	High	5		
			25	25	High
Sheppard Bottom					
Water	3–4	Moderate	4		
Sediments	0.6–3	Low	3		
Invertebrates	3–33	High	5		
Fish eggs	8–27	High	5		
Bird eggs	1–17	Moderate	4		
			21	21	High

[a] Selenium concentrations in μg/l (parts per billion) for water; μg/g (parts per million) dry weight for sediments, invertebrates, and eggs.
[b] Hazard ratings were determined by comparing selenium concentrations to hazard profiles given in Figures 1–5.

Three managed wetlands on the Refuge were examined and separate hazard assessments were generated for each of these locations (Table I). The ratings given to the sites ranged from low hazard to high hazard, reflecting differences in waterborne selenium concentrations and associated influences on the sediment-detrital food pathway and dietary exposure of fish and migratory birds. Corrective actions have been prescribed and implemented at the high hazard sites and the procedure can soon be used to begin evaluating the success of these actions in reducing selenium hazards.

Conclusions

The assessment procedure provides a straightforward, consistent approach to evaluate the toxic threat of selenium using a set of data from routine contaminant monitoring. Toxic threat, expressed as hazard, is characterized from sensitive endpoints linked to important biological effects. Although the method is simple and easy to carry out, it is based on a large body of field and laboratory research data on selenium cycling and toxicity in aquatic ecosystems. This, combined with the integrative nature of the method, i.e., utilizing information from five ecosystem components, should make it a useful tool for assessing the toxic threat of selenium to fish and wildlife.

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