

US EPA ARCHIVE DOCUMENT

SUPPORTING INFORMATION

**Emerging opportunities in management of selenium
contamination**

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SI 1. Selenium Toxicity

Selenium is an essential element for all animals, but the margin of safety is narrow between Se concentrations that are essential and those that are toxic, especially for wildlife (1). The narrow window between essentiality and toxicity means there is little room for maneuverability in managing ecological risks from Se. Small changes in exposure hold great potential for ecological damage. The standardized toxicity tests traditionally used to establish the concentration at which Se elicits toxicity suggest the opposite, however. Dissolved toxicity tests with Se suggest thresholds for ecological risk should be in the 100's of $\mu\text{g/L}$ (2,3). Dietary toxicity tests (4) and field observations of adverse effects on birds (5) and fish (6) are documented in freshwater systems at dissolved concentrations as low as 2 – 5 $\mu\text{g/L}$. The disparity between dissolved toxicity tests and field observation is the single greatest cause of the differences among jurisdictions in managing Se.

Biogeochemical processes convert Se to concentrations at the base of the food web orders of magnitude greater than in water and passage of that Se through the food web is the source of Se exposure. Therefore, ecosystems will be badly damaged by dietary exposure before dissolved Se concentrations reach levels that themselves are dangerous to animals. Dissolved toxicity tests cannot predict the concentration at which that damage will occur. Similarly, the convention traditionally used to quantitatively link Se bioaccumulation by animals to Se in the environment is the Bioaccumulation Factor (BAF): the ratio of Se in animal tissue to dissolved Se concentrations. BAFs for Se vary by orders of magnitude with concentration, transformation, environment, and species (7), making BAFs of little value in either explaining or predicting Se bioaccumulation into food webs.

Dietary exposures show that, in birds, the first signs of toxicity include failure of the eggs to hatch and occurrence of deformities in young (5). In fish, failure of eggs to hatch, deformities or damage in embryos, and teratogenesis are also early signs of toxicity (6). These effects correlate with Se concentrations in the organism, with the strongest correlations occurring between concentrations in the egg and the onset of effects (5). However, the concentration at which occurs the effect of Se on the ability of a species to produce viable young cannot be determined by acute or dissolved toxicity

testing. Reproductive toxicity also means that species-specific demographics will be especially important in determining risks to populations.

Therefore, exposure via diet must be considered in order to evaluate risks from Se in nature. This is the single greatest deviation from the conventional risk assessment paradigm.

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SI 2 References for Figure 2.

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