ARCADIS
Infrastructure · Water · Environment · Buildings

MEMORANDUM

August 3, 2012

To: Michelle Cagley, Tennessee Valley Authority

Subject: Response to Comments from Industrial Economics, Incorporated (IEc), on behalf of Tennessee Valley Authority, on the *Toxicological Profiles* — Selenium

ARCADIS, on behalf of the Tennessee Valley Authority (TVA), has reviewed and respectfully submits the following responses to the comments made by IEc in the memorandum dated July 16, 2012. The memorandum provided comments, on behalf of Tennessee Valley Authority, on the *Toxicological Profiles* report prepared by ARCADIS, dated November 11, 2011.

IEc's comments and our responses are as follows:

GENERAL COMMENTS

Proposed Additions To The Selenium Toxicity Section

Although selenium is a micronutrient, and therefore provides some benefit to biota, it can also be toxic, causing sub-lethal and lethal effects (Hamilton 2004). In aquatic environments, uptake by biota can occur via gills, epidermis, and gut, with dietary exposure being the primary pathway. Once exposed to elevated levels of selenium, the effects on natural resources are dependent on interactions with the surrounding matrices, the life stage and sensitivity of the organism, the chemical form of selenium and the exposure pathway (Chapman *et al*. 2010, Fordyce 2005, Hamilton 2004, Eisler 1985). In addition, other contaminants affect selenium's toxicity. For example, when mercury and selenium occur together, they appear to have an antagonistic relationship, reducing observed toxicity (Bjerregaard *et al.* 2011), whereas the arsenic compound arsenite has a synergistic relationship with methylated selenium compounds, enhancing the toxicity of forms of selenium (Kraus and Gather 1989).

In general, selenium can adversely affect biochemical, neurological, growth, developmental, reproductive, and survival endpoints in a suite of organisms. The remainder of this section contains a brief, resource-specific review of the toxicological effects of selenium on plants, aquatic invertebrates, fish, birds, amphibians and reptiles, and mammals. Additional details are provided in the Attachments.

Selenium is an essential nutrient for growth in plants; however it can also be toxic at elevated concentrations. Uptake into plants is influenced by the form of selenium present, concentration, pH, and species (USEPA 2007). The toxic effects of selenium on plants generally include, but are not limited to, growth and survival endpoints (Eisler 1985). For example, growth was significantly reduced in green algae at tissue concentrations of 4 parts per million (ppm) (Williams et al. 1994 as cited in USDOI 1998, Foe and Knight 1986, Vocke et al.1980). Survival endpoints were investigated by Kumar and Prakash (1971), who examined blue-green algae exposed to selenium After 96 hours, they observed a median lethal concentration (LC50) at water concentrations of 15 mg/L.

Although invertebrates have been shown to be relatively tolerant to elevated selenium, they can experience selenium-related adverse effects, including reduced biochemical rates, reproduction, and growth (Chapman *et al*. 2010, USDOI 1998). For example, altered respiration rates were found in crayfish caged in the ash-pit drain of a Wisconsin power plant with concentrations of 30 ppm found in the hepato-pancreas (Magnuson *et al*. 1980). Reduced reproduction and growth resulted when daphnids were exposed for 30 days to 0.348 and 0.156 mg/L, respectively (Ingersoll et al. 1990). Further, a study

on potworms reported lethality to 50 percent of the population (LC_{50}) after 42 days of exposure at a soil concentration of 5.69 ppm dry weight (dw) (Somogyi *et al.,* 2007).

Field and laboratory studies on selenium toxicity in fish show effects that include, but may not be limited to, reproductive effects, teratogenic deformities, and mortality (Eisler 1985; Jacobs 1989). Lemly (1996a) recommended a toxicity threshold of 4 ppm dw for whole body, adult fish for mortality of juveniles and reproductive failure, and a 10 ppm dw threshold for ovary/eggs. In Belews Lake, North Carolina, Sorenson *et al.* (1984) observed reproductive failure, reduced immune and liver functions, and population decline in green sunfish with elevated selenium levels of 0.005 mg/L in water. Laboratory studies of selenium exposure from maternal transfer to embryo for bluegill showed lowest effect concentration (LOEC) at 16 ppm dw in the ovary/egg, resulting in larval edema, larval mortality, and larval deformities (Coyle *et al*. 1993 in DeForest and Adams 2011). In a long-term study with bluegill sunfish, dietary selenium caused adult and embryo mortality, and maternally-transferred selenium resulted in but larval mortality at even lower dietary concentrations (Chapman *et al*. 2010).

The literature on selenium toxicity to birds is robust, and studies identify a suite of effects that include, but are not limited to, reproductive effects and mortality. Reproductive impairment is considered the most sensitive indicator of selenium toxicity in birds and has been observed at concentrations as low as 3 ppm dw in the diet of mother birds (Chapman *et al*. 2010; US DOI 1998). Embryos suffer brain tissue, spinal cord and limb bud deformities at maternal dietary concentrations of 6-9 ppm dw. Additionally, reduced egg production and growth were observed at maternal dietary concentrations above 7 ppm dw (Ohlendorf and Heinz 2011, Jacobs 1989, WHO 1987). Wayland *et al.* (2007) used logistic regression analysis on experimental studies of six species (mallard, American kestrel, domestic chicken, black-crowned night-heron, eastern screech owl and ring-necked pheasant), and calculated an effects concentration for 10% of the population (EC_{10}) for reduced egg hatchability of 4 ppm dw. Further, at Kesterson Reservoir (California), where selenium in runoff from agriculture accumulated at toxic levels of 50 ppm in birds' diets, reproductive failure and adult mortality were observed (Ohlendorf et. al.1990; Ohlendorf and Heinz 2011).

Little is known regarding the toxicity of selenium to amphibians and reptiles, but studies have identified histological and growth effects. Larval deformities, deformities of the mouthparts, spinal and oral abnormalities that affected swimming and feeding, reduced growth, altered predator avoidance, reduced larval survival, and altered time and size to metamorphosis have all been reported in various studies in which amphibians were exposed to selenium and other contaminants (Chapman *et al*. 2010). Effects on reptiles include histopathological differences such as liver necrosis and increased respiratory rates, which indicate a high energetic cost associated with exposure (Chapman *et al*. 2010). Exposure of reptiles to selenium-laden prey did not affect growth, survival, overwinter survival, or metabolism (Chapman *et al*. 2010).

Selenium toxicity studies on mammals report immunological and reproductive effects. For example, Peterson and Nebeker (1992) modeled thresholds of waterborne selenium toxicity, and estimated immunological effects thresholds of 0.0009 mg /L for bats and shrews, 0.0011 mg/L for mink, and 0.0007 mg/L for river otters. Similar thresholds (i.e., between 0.001-0.002 mg/L were recommended by several studies cited by Lemly (1996b). Further, inhibition of reproductive success and failure to breed was observed in a 3-generation study of mice exposed to a single dose of selenium at 0.056 ppm in food and 3 mg/L in drinking water (Schroeder and Mitchener 1971).

Response: The highlighted text has been added to Section 18.4 of the Toxicological Profiles Report, as suggested.

SPECIFIC COMMENTS

Proposed Ammendments To Appendix C: Fish

1. Remove all information on fish species that are not relevant to the assessment area from the eco-toxicity and effects-based tissue concentration tables.

Response: While some fish species not specifically found in the assessment area were included in the database of eco-toxicity and effects-based tissue concentration tables, these data were not removed as they provide additional comparisons for sizes and feeding guilds.

2. Remove all references that had NR as endpoints from the eco-toxicity and effects-based tissue concentration tables.

Response: While the proposed change might improve the overall readability of the tables, it is unnecessary and impractical to make this revision given the required submission date of the BERA.

3. Remove the fields “response site description”, “class” “con 2”, and “con 2 type” from the eco-toxicity table.

Response: While the proposed changes might improve the overall readability of the tables, it is unnecessary and impractical to make these revisions given the required submission date of the BERA.

4. Remove the field “class” from the effects-based tissue concentration table.

Response: While the proposed change might improve the overall readability of the tables, it is unnecessary and impractical to make this revision given the required submission date of the BERA.

5. Remove duplicate entries.

Response: While removing the duplicate entries might improve the overall readability of the tables, it is unnecessary and impractical to make this revision given the required submission date of the BERA.

6. Add endpoints for the following references: Doroshov et al. (1992), Schultz and Hermanutz (1990), Jacobs et al. (1989), and Sorensen et al. (1984).

Response: The suggested references and corresponding effects-based tissue concentrations are currently under review.

7. Revise endpoints related to the following references to reflect maternal transfer studies: Hamilton et al. (2005), Saiki et al. (2004), CP&L (1997), Coyle et al (1993), Ogle and Knight (1989), Gillespie & Baumann (1986).

Response: The suggested revisions of endpoints are currently under review.

Proposed Ammendments To Appendix C: Birds

1. Simplify the eco-toxicity table, so that it is easier to understand. Remove the fields “Wet Weight Reported,” “Percent Moisture,” “Route of Exposure,” “Exposure Duration,” “Duration Units,” “Age,” “Age Units,” “Lifestage,” “Sex,” “Response Site,” “Study NOAEL,” “Study LOAEL,” “Body Weight Reported?, Body Weight in kg, Ingestion Rate Reported?, Ingestion Rate in kg/day or L/day, NOAEL Dose (mg/kg/day), and LOAEL Dose (mg/kg/day). Add the fields “Effect”, “Endpoint”, “Concentration”, “Units” and “Test Type”.

Response: While the proposed change might improve the overall readability of the tables, it is unnecessary and impractical to make this revision given the required submission date of the BERA.

2. Remove the fields class and water type from the effects-based tissue concentrations table.

Response: While the proposed changes might improve the overall readability of the tables, it is unnecessary and impractical to make these revisions given the required submission date of the BERA.

3. Remove duplicate entries.

Response: While removing the duplicate entries might improve the overall readability of the tables, it is unnecessary and impractical to make this revision given the required submission date of the BERA.

4. Add endpoints for the following references: Adams *et al*. (2003), Skorupa (1998), US DOI (1998).

Response: The suggested references and corresponding effects-based tissue concentrations are currently under review.

5. Expand information for endpoints related to the following references: Lam *et al*. (2005); Ohlendorf (2003); O’Toole and Raisbeck (1998); Ohlendorf et al. (1986); Ort and Latshaw (1978).

Response: The suggested revisions of endpoints are currently under review.