

SALTON SEA ECOSYSTEM RESTORATION PLAN

Final Selenium Summary

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FINAL SELENIUM SUMMARY

This report briefly summarizes current scientific information from readily available literature with respect to environmental and human health risks associated with ambient exposure to selenium. The main focus is on environments analogous to the Salton Sea, but other information that may be relevant for ecological and human health risk assessment (ERA and HHRA, respectively) is included. The report provides brief summaries on fate and transport, persistence and bioaccumulation, interactions with other chemicals, chronic and acute toxicity levels in wildlife and humans, action levels and levels of concern, impacts from selenium at other sites, previously performed HHRA and ERA results, and existing protocols to evaluate avian exposure.

Several reviews and assessments on selenium have been published recently, including those by Hamilton (2004), Ohlendorf (2003), the Agency for Toxic Substances and Disease Registry (ATSDR 2003), Luoma and Presser (2000), Eisler (2000), Frankenberger and Engberg (1998), U.S. Department of the Interior (USDI 1998), and Frankenberger and Benson (1994). These reviews along with other published reports have been used to compile general information on selenium that is most relevant to the selenium issues at the Salton Sea.

FATE AND TRANSPORT

Fate and transport processes are important because they control the cycling of selenium through the environment, and understanding those processes will be essential for the evaluation of restoration alternatives at the Salton Sea. These processes are complex because of the biogeochemistry of selenium in the environment.

Selenium is a naturally occurring element found in rocks and soils. Selenium occurs in several forms, including multiple oxidation states, which vary depending on ambient conditions (such as pH, Eh [oxidation/reduction potential], and microbial activity), as well as the environmental medium (such as water, sediment, or biological tissue). Biologically significant oxidation states include selenide (Se^{2-}), elemental selenium (Se^0), selenite (Se^{4+}), and selenate (Se^{6+}).

The behavior of selenium in the environment is largely influenced by its oxidation state as well as physical factors such as geology, climate, and hydrology. Selenium is often more abundant in environmental media in areas with Upper Cretaceous marine sedimentary rocks and other formations naturally high in selenium (USDI 1998). Climate also affects selenium distribution, because it behaves differently in arid climates than in humid or wet climates. In areas that have a local geologic source of selenium (as discussed above), concentrations generally increase as aridity increases. Hydrology can increase selenium contamination by acting as a transporting agent, and receiving waterbodies may become sinks for the mobilized selenium. Selenium can be transported via rivers, streams, creeks, and irrigation drainage water. Terminal waterbodies may become contaminated due to evaporative enrichment and concentration over several seasons of runoff. These physical factors influence the fate and transport of selenium in various environmental media. Fate and transport in sediment, water, soil, and air are briefly described below, and also in the Draft Report on Selenium at the Salton Sea and Summary of Data Gaps prepared under this Task Order.

Sediments

In an aquatic system, like the Salton Sea, selenium is generally associated with sediments (acting as a sink and reservoir) or plants and animals. In bottom sediments, metal and organic selenides are most common (Ohlendorf 2003). The sediment contamination process that occurred at Kesterson Reservoir during disposal of agricultural drainage water, as discussed later in this paper, was simulated in a laboratory study to identify the selenium transformations that were occurring (Tokunaga et al. 1996). Water

contaminated with selenate was ponded over uncontaminated soil. The study revealed that in water, selenate was reduced to selenite and both forms were removed from the aqueous phase into sediment. Once in sediment, the selenite was reduced to elemental selenium, which may have made up 99 percent of the selenium found in sediments. Within 2 days of sampling in some aerated ponds, 60 percent of the selenium was reoxidized to selenite and selenate in water. Ponds amended with organic matter retained the elemental selenium in sediments.

In a flow-through experimental wetland system receiving agricultural drainage water in the San Joaquin Valley, most of the waterborne selenium was removed as water passed through the system (Gao et al. 2003). Vegetation was considered to play an important role in the removal of selenium from the water. The inflow water was dominated by selenate (91 percent selenate, 7 percent selenite, and 2 percent organic selenium), which was reduced to selenite and organic selenium in the wetland (47 percent selenate, 32 percent selenite, and 21 percent organic selenium). Surficial sediment appeared to be a large sink for selenium, with the highest concentrations in fallen litter; however, the greatest mass of selenium was in the sediment itself.

Wetting and drying cycles, as normally found in wetlands, are important factors that contribute to selenium mobilization and potential toxicity. Selenium is often present in reduced forms (less available and therefore less toxic) when wetlands are submerged and have high organic matter. This condition favors volatilization (Masscheleyn and Patrick 1993). When the water level is lowered the selenium becomes more oxidized and bioavailable. As a result, the initial wetting period increases selenium bioavailability in sediments and organic matter.

Studies at Kesterson Reservoir found that while selenate was the predominant form in surface waters, sediments accumulated high concentrations (tens to hundreds of mg/kg) of elemental and organically associated forms of selenium (Byron et al. 2003). After the area was dried out and low-lying areas were filled in with soil, the concern shifted to the ephemeral pools that formed during the rainy season. The highest selenium concentrations (up to 1000 µg/L) found in the pools occurred immediately after the pools had formed. These concentrations decreased to more stable concentrations (1 to 200 µg/L) after a few weeks. This mobilization is caused by the dissolution of salt crusts and diffusion of dissolved selenium from pore waters into the overlying surface waters. Anoxic conditions may form in underlying sediment once the pool is formed. Then selective adsorption of selenium to organic carbon in the sediment or diffusion of the selenium from the pools back into the underlying soils depletes the concentrations in the surface water. Upon diffusion into the soils, the selenium is reduced back to immobile forms (Byron et al. 2003). According to Masscheleyn and Patrick (1993), repeated cycles of wetting and drying increased the amount of elemental selenium in sediments. Elemental selenium and selenium-containing sulfide minerals release soluble selenium species upon drying or prolonged oxidation of sediments. The volatilization of selenium was positively correlated with sediment temperatures (Masscheleyn and Patrick 1993).

The selenium in shallow, oxidized sediments is readily bioavailable primarily through uptake by benthic invertebrates and subsequent uptake by benthic-feeding fish and fish-eating birds (Setmire et al. 1993). However, most of the selenium found at the Salton Sea is located within deep, anoxic sediments where the selenium is relatively unavailable to biota because of low exposure rates (USGS 2003). Modifications to the Salton Sea that would decrease water depth and increase oxidation of the deep sediments would tend to increase overall selenium bioavailability. If exposed areas around the Salton Sea shoreline (as a result of falling Sea levels) undergo frequent wetting and drying cycles or if shallow water is ponded there (associated with dust control measures), selenium bioavailability in the food chain could be greatly increased in a manner similar to that associated with wetlands and ephemeral pools.

Water

In general, relatively small amounts of selenium are found dissolved in water (Furr et al. 1979, Nriagu and Wong 1983, and Lemly 1985a as cited in Ohlendorf 1989). The most common forms of selenium in water are selenic and selenious acids. Soluble selenate salts of selenic acid are expected to occur in alkaline waters. Sodium selenate is highly mobile due to its high solubility and inability to adsorb onto soil particles. Bender et al. (1991) found that bacteria and cyanobacteria have two mechanisms for the uptake and transformation of selenate (as cited in ATSDR 2003). The uptake method reduces selenate to elemental selenium, which is physically held within the algal mat. The microorganisms were found to transform soluble selenium into volatile alkyl selenium compounds. Selenious acid, a weak acid, and the diselenite ion predominate in waters between pH 3.5 and 9. In general, selenites are less soluble in water than the corresponding selenates. In most surface waters, sodium predominates as the counter ion of selenate and selenite. Microbial activity in deep aquifers is believed to retard the selenium transport in groundwater by causing chemical reduction and precipitation (White et al. 1991 as cited in ATSDR 2003).

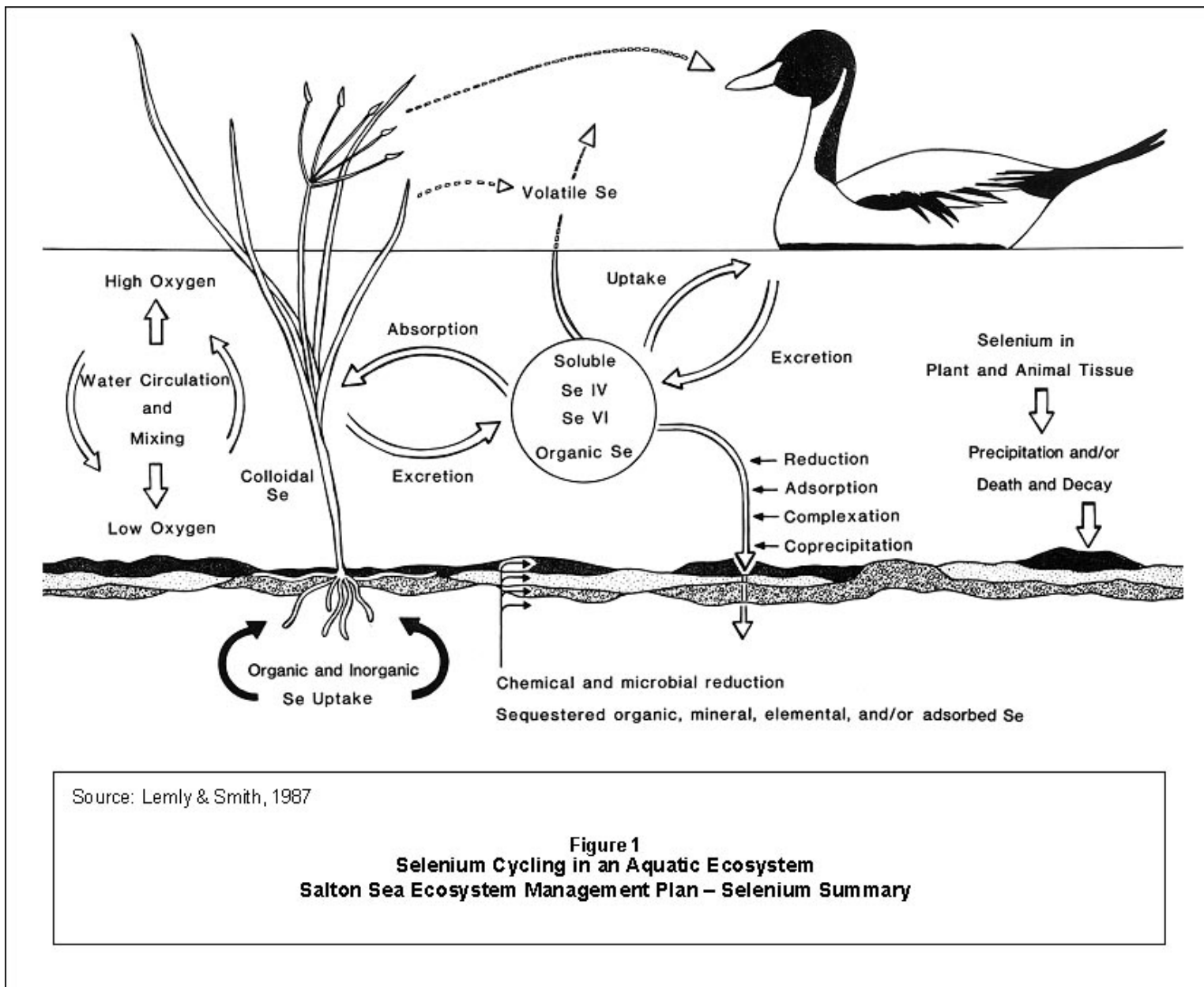
Figure 1 illustrates selenium cycling in an aquatic ecosystem, as presented in Lemly and Smith (1987). Inorganic forms of selenium (selenate and selenite) usually predominate in water, but inorganic as well as organic forms of selenium occur in water, sediment, and biological tissues.

Soil

Some soils have naturally high concentrations of selenium, and selenium is transported as a result of weathering rocks. Irrigation of seleniferous soils can also dissolve and mobilize selenium and then transport it to irrigation drains (Ohlendorf et al. 2003). Transport and partitioning of selenium in soils is highly influenced by pH and Eh (oxidation/reduction conditions). Elemental selenium is essentially insoluble and stable in soils when anaerobic conditions occur. Heavy metal selenides and selenium sulfides are insoluble and will remain in soils with low pH or high organic matter. The selenides of other metals, including copper and cadmium, have low solubility. In contrast, selenates are very mobile (Kabata-Pendias 2001) and easily taken up by biological systems (Klaassen 2001) or leached through the soil due to their high solubility and low adsorption potential (onto soil particles). Selenates dominate in alkaline, well-oxidized soil environments and some (e.g., sodium and potassium) dominate in neutral, well-drained, mineral soils. While soluble selenates are responsible for the naturally occurring accumulation of high levels of selenium by plants, much of the total selenium measured in soils may be present in other forms. Under alkaline and oxidizing conditions, the soluble forms of selenium can be accumulated by plants though selenate seems to be the preferred form for uptake. In acidic soils, with high moisture, selenite (the predominant form) is bound to colloids as iron hydroxide selenium complexes. These complexes are generally not bioavailable to plants (Galgan and Frank 1995 as cited in ATSDR 2003). Lime and plant ash are sometimes used as fertilizers, raising the pH of the soil and favoring the formation of selenate. These fertilizers have been associated with accumulation of selenium in crops grown in high-selenium soils (Yang et al. 1988 as cited in ATSDR 2003).

Air

Volatile selenium compounds, both inorganic (selenium dioxide and hydrogen selenide) and organic (dimethyl selenide and dimethyl diselenide), partition into the atmosphere. These compounds can persist in air, but typically hydrogen selenide is rapidly oxidized to elemental selenium and water. Selenium dioxide, released to the air from combustion of fossil fuels, reacts with atmospheric moisture to generate selenious acid aerosols (Oehm et al. 1991 as cited in ATSDR 2003). Selenium compounds in air can be removed via wet or dry deposition to soils or surface water (ATSDR 2003).



PERSISTENCE IN THE ENVIRONMENT AND BIOACCUMULATION

Selenium is persistent in the environment, changing chemical forms and moving from one medium to another, but it does not “degrade” in the sense of organic chemicals. Biogeochemical processes cause cycling from abiotic media to biotic media, and back to abiotic media, but it does not disappear. Thus, it is important to consider this persistence in relation to water, sediment, and biota within the Salton Sea. Although selenium (including organic and inorganic forms) is bioaccumulated readily from water by aquatic organisms, it does not “biomagnify” significantly in successively higher levels of the food web (Ohlendorf 1989, 2003).

Bioaccumulation is the combined net accumulation of a chemical from abiotic media and ingestion of chemical-containing biota. Selenium can bioaccumulate in both aquatic and terrestrial food chains including higher trophic-level animals that feed on those plants and animals. Ingestion is the primary route of uptake in both aquatic and terrestrial food chains, and toxic effects from food-borne selenium may be more significant than those from water-borne selenium (Sandholm et al. 1973, Birkner 1978, Brooks 1984, Girling 1984, Lemly 1985a,b as cited in Ohlendorf 1989). Waterborne selenium generally does not pose a high risk to the terrestrial food chain when it is the only route of exposure because, in general, only small amounts of selenium are dissolved in water (Furr et al. 1979, Nriagu and Wong 1983, and Lemly 1985a as cited in Ohlendorf 1989; Ohlendorf 2003).

Bioaccumulation and overall concentrations are usually higher in marine organisms than in freshwater organisms (Ohlendorf 2003, Eisler 2000). In freshwater biota, selenate represented about 36 percent of the total selenium (selenite and selenide made up the remainder) while in marine samples, only 24 percent of the total selenium was selenate (Cappon and Smith 1982 as cited in Eisler 2000). The significance of this is not well understood but may affect the ability of selenium to reduce toxicity of heavy metals as discussed later in this paper.

Selenium is bioaccumulated in the aquatic food chain. Selenite and selenate are the most common aqueous forms and are biotransformed into organic chemical species after uptake by primary producers (such as algae, phytoplankton, and rooted plants) (Ogle et al. 1988 as cited in USDI 1998, Ohlendorf 2003). Bioaccumulation is often a function of chemical species. Organic selenium is especially bioaccumulative, so that aquatic organisms exposed to organic selenium (such as selenomethionine) are likely to bioaccumulate much more selenium than those exposed to inorganic selenium in water (Ohlendorf 2003). For example, Besser et al. (1989) found that selenium accumulated from selenomethionine more readily than from selenite or selenate. As noted above, inorganic selenium is converted to organic selenium by organisms such as algae when it is taken up from the water. In an experimental treatment system using an algal-bacterial selenium reduction process, 80 percent of the total selenium was removed from the water, but aquatic organisms living in treated water had 2 to 4 times more selenium than those living in untreated water (Amweg et al. 2003). This illustrates the importance of understanding the cycling processes that convert selenium from one form to another, potentially increasing bioavailability and uptake (and therefore risk to consumers).

In the terrestrial food chain, leaves, roots, stems, and seeds of plants often have differing concentrations of selenium (Beath et al. 1937, NAS-NRC 1976 as cited in Ohlendorf 1989) with more elevated concentrations usually occurring in leaves. Loco weed, milkvetch, thistle, goldenweed, and mustard are known to concentrate selenium and have been found to accumulate selenium to hundreds or even thousands of milligrams per kilogram dry weight (dw) (USDI 1998). Plants can absorb both inorganic and organic forms of selenium (Adriano 1986 as cited in Ohlendorf 1989). Selenomethionine is the most common form of selenium ingested by wildlife while foraging on plants and 85 to 100 percent of selenium from plants is biologically available (Scott 1973, Glover et al. 1979). In contrast, only 20 to 50 percent of selenium present in meat and fish is absorbed by wildlife (Ohlendorf 1989). Overall,

selenium bioavailability is greater from plant selenium than from animal foods (Ohlendorf 2003). This has important implications for human exposures as well as for wildlife.

INTERACTIONS WITH OTHER CHEMICALS

Selenium interacts with various heavy metals (especially arsenic and mercury), vitamins A, C, and E, sulfur-containing amino acids, and paraquat herbicides (Ohlendorf 1989, 2003). These interactions can be antagonistic or synergistic when related to uptake and metabolic effects, thereby either increasing or decreasing the toxic effects of selenium or the other chemical. Other selenium interactions which may be of interest at the Salton Sea include boron, as described below.

At appropriate dietary levels, selenium generally, but not always, protects animals from toxic effects of arsenic and certain other metals. Overall, interactions between selenium and other chemicals are very complex, because they are influenced by many factors. The most relevant interactions potentially affecting birds at the Salton Sea are briefly described here.

Variations in the mercury-to-selenium ratio in fish reflect relative mercury bioavailability in the environment and potentially the bioavailability of selenium. In fish-eating birds, selenium was found to be correlated with inorganic mercury in the liver (Henny et al. 2002, Spalding et al. 2000). These studies suggest that selenium may contribute to the sequestration of mercury, thereby reducing its toxicity and agree with the results from a selenium-mercury interaction study with mallards by Heinz and Hoffman (1998). In that study, mercury toxicity in adult male ducks was reduced when dietary exposure included selenium. At higher selenium concentrations, however, reproductive effects were worse than for each element alone and the storage of selenium in duck tissues was enhanced. Simultaneous administration of selenium and mercury to animals in equimolar doses reduced toxicity of both elements in acute and chronic studies with both inorganic and organic mercury and selenium. The inorganic forms of selenium are apparently more effective at reducing adverse effects of mercury than the organic forms (Chang 1983, Rao et al. 1998, Skerfving 1978 as cited in ATSDR 2003).

The interactive effects of dietary selenium in combination with arsenic were studied to evaluate effects on reproduction in mallards (Stanley et al. 1994). Ducks fed selenium and arsenic in combination had an antagonistic effect whereby arsenic reduced the accumulation of selenium in duck livers and eggs and reduced the impact of selenium on hatching success and embryo deformities.

The interactive effects of selenium in combination with boron were also studied to evaluate effects on reproduction in mallards (Stanley et al. 1996). Mallards were fed diets containing either boron or selenium alone or boron in combination with selenium. Boron exposure alone resulted in significant adverse effects when mallards were fed high dietary levels. Boron and selenium in combination did not appear to have any significant interaction, so that effects were similar to when each chemical was fed separately.

Selenium has been found to reduce the nephrotoxic and hepatotoxic (kidney and liver, respectively) effects of cadmium in rats (Flora et al. 1982, Lindh et al. 1996, Nehru and Bansal 1996, Stajn et al. 1997 as cited in ATSDR 2003). Studies have also shown selenium to reduce effects in testes (Jones et al. 1997, Mason and young 1967, Ohta and Imamiya 1986, Wlodarczyk et al. 1995, Yiin et al. 1999 as cited in ATSDR 2003) and to reduce cardiovascular effects of cadmium (Jamall et al. 1989 as cited in ATSDR 2003). In addition, selenium has also been found to protect against toxic levels of cadmium in freshwater snails, marine crabs, and earthworms (Wilber 1983, Bjerragaard 1982, Helmke et al. 1979, Beyer et al. 1982 as cited in Eisler 2000).

Selenium was also found to reduce the toxic effects of the herbicide paraquat in mammals and cold-blooded animals (Wilber 1983 as cited in Eisler 2000, ATSDR 2003).

Little information was found directly relating salinity and selenium but some studies suggest that salinity tolerance may influence effects of selenium in wildlife. Salinity tolerance has been found to be a large factor in embryo sensitivity to selenium exposure. Birds that prefer nonmarine saline wetlands are more tolerant of selenium than those that prefer freshwater wetlands (USDI 1998). For example, embryos of American avocets tolerate selenium much better than do those of black-necked stilts, and snowy plover embryos are more tolerant than those of killdeer. Overall, dabbling ducks (such as the mallard) are considered more sensitive than black-necked stilts (a moderately sensitive species), and the stilt is more sensitive than the avocet. In a study using rainbow trout, hypersaline conditions were found to protect fish from dietary seleno-L-methionine toxicity (Schlenk 2003). These differences in sensitivity may be important in evaluating the potential effects of selenium in current and future habitats at the Sea.

CHRONIC AND ACUTE TOXICITY

Selenium is an essential trace element for animals, and is a component of glutathione peroxidase (GSH-PX), which aids in the protection of tissues against peroxidation by destroying hydrogen peroxide or organic hydroperoxides (Ohlendorf 2003). The presence of selenium at increased dietary levels results in the replacement of sulfur in some metabolic pathways disrupting some biological processes. Selenite and selenate are readily absorbed through the small intestine in animals. Selenides and elemental selenium are poorly absorbed. Selenomethionine has been found to be the best surrogate form of selenium in experimental studies with fish and birds to represent environmental exposures. Elemental selenium (and other insoluble forms) appears to be least toxic. About 70 to 80 percent of the inorganic selenium intake is quickly excreted in the urine, breath, perspiration, and bile. The remaining selenium is eliminated after becoming bound or incorporated into blood and tissue proteins (Ohlendorf 1989). Toxicity of selenium to wildlife, aquatic biota, and humans is briefly summarized in this section.

Concentrations in biota tissues can be expressed either on wet-weight or fresh-weight basis (which are considered to be synonymous), or on dry-weight basis. Conversion from one basis to the other is a function of the moisture content in the sample, as follows:

$$\text{Dry-weight conc.} = \text{Wet-weight conc.} \times \frac{100}{(100 - \text{Moisture percentage})}$$

For example, 10 µg/g on wet-weight (ww) basis in a sample having 80 percent moisture is equal to 50 µg/g on dry-weight (dw) basis. When selenium concentrations in tissue were originally reported in ww, the approximate dw concentration is presented in this summary.

Wildlife

The most important food-chain pathway for selenium in the Salton Sea begins with accumulation from sediment by benthic invertebrates, particularly pileworms, and includes subsequent uptake by benthic-feeding fish and fish-eating birds (Setmire et al. 1993). Of the benthic invertebrates sampled for the Setmire et al. study, only pileworms had selenium concentrations that were in excess of the critical dietary threshold selenium concentrations of 5 µg/g (on dry-weight basis) for food-chain organisms (Skorupa and Ohlendorf 1991). Selenium at the Salton Sea was transferred through successive trophic levels in the food-chain at increasing concentrations (Setmire et al. 1993). Selenium concentrations in food-chain organisms of tributary rivers and agricultural drains were similar to those for the Salton Sea food chains, but with lower levels at similar trophic levels. Setmire et al. (1993) noted that large birds feeding in rivers do not accumulate nearly as much selenium as those feeding in the Salton Sea. In general, selenium concentrations at the highest freshwater trophic levels were only one-half of those in the Salton Sea.

Subacute or chronic selenosis can occur when wildlife are gradually exposed to concentrations of selenium ranging from 1 to 44 mg/kg in their diets (Eisler 2000). Chronic selenium toxicosis can result in

reproductive problems including decreased conception and increased loss of fetuses in animals and embryocidal damage in birds (Mayland 1994). Domestic livestock and birds have been found to be sensitive to the effects of excess selenium, though early life stages of birds are the most sensitive wildlife. Selenium accumulates and depurates from tissues fairly quickly (i.e., uptake and loss are very responsive to the current level of exposure). As a result, symptoms of selenium poisoning in adult birds and mammals can be reversed rapidly if the sources of selenium is eliminated (Ruta and Haider 1989, Heinz and Fitzgerald 1993 as cited in USDI 1998). Embryonic deformities, however, are not reversible (Lemly 1993 as cited in USDI 1998).

Selenium exposure in mammals can result in congenital malformations and reproductive problems (Rhian and Moxon 1943; Harr 1978; NAS-NRC 1980, 1983 as cited in Ohlendorf 1989). The lowest chronic dietary threshold for mammals in literature is 1.4 mg/kg (natural selenium, dry feed basis) with sublethal effects occurring after a lifetime exposure in rats (NRC 1980, Olson 1986 as cited in USDI 1998, Eisler 2000). Dietary concentrations from 3 to 5 mg/kg (natural selenium) induce signs of toxicity in domestic animals. Teratogenic effects in mammals are not as pronounced as in avian receptors and seem to occur only when levels are high enough to adversely affect the mother (Hawkes et al. 1994 as cited in USDI 1998).

Avian embryos are highly sensitive to the toxic effects of selenium (Poley and Moxon 1938, Thapar et al. 1969, Arnold et al. 1973, NAS-NRC 1976, El-Begearmi et al. 1977, Ort and Latshwa 1978 as cited in Ohlendorf 1989, 2003). Hatchability of fertile eggs is considered the most sensitive endpoint. Dabbling ducks, such as mallards and cinnamon teal, are among the most sensitive species (USDI 1998). Dietary levels from 6 to 9 mg/kg are known to reduce the hatchability of chicken eggs (Ohlendorf 1989), but reproductive impairment can result from diets of only 3-8 mg/kg (Wilber 1980, Martin 1988, Heinz 1996 as cited in USDI 1998). Ohlendorf (2003) used the results of six studies with mallards to determine the selenium concentrations in diet and eggs that were associated with reduced egg hatchability. Dietary concentration of 4.87 mg/kg and egg concentrations of 12.5 mg/kg were associated with a 10 percent reduction in hatchability. Duck eggs containing 11 to 20 mg/kg selenium exhibit an observed probability of overt embryo teratogenesis (deformities) ten times greater than background. Heinz (1996) estimated that the embryotoxic threshold for selenium in bird eggs is about 10 mg/kg. Concentrations of 5 to 20 mg/kg in the diet may result in selenium accumulation in eggs above teratogenic thresholds.

Aquatic Biota

Waterborne selenium can be acutely toxic to some aquatic invertebrates when concentrations range from 70 to 760 $\mu\text{g/L}$ (Adams 1976, USEPA 1980, Halter et al. 1980, Nassos et al. 1980, and Murphy 1981 as cited in Ohlendorf 1989; USEPA 1998). Acute toxicity was observed in amphipods exposed to 4 $\mu\text{g/L}$ waterborne selenomethionine (ATSDR 2003) while no adverse effects were noted from a dietary concentration of 300 mg/kg selenium (Foe and Knight 1986, as cited in ATSDR 2003). Larval midges exposed to algae containing greater than or equal to 2.1 mg/kg selenium showed significantly inhibited growth (Malchow et al. 1995).

Eggs and larvae of fish and amphibians may be the most sensitive stages of vertebrate animals to direct exposure to waterborne selenium. Excess selenium in the diet of fish leads to substitution of selenium for sulfur during protein synthesis (Lemly 1998). This disrupts normal chemical bonds resulting in improperly formed or dysfunctional proteins and enzymes affecting sub-cellular, cellular, organ, and system functions. Effects include teratogenicity in developing embryos, reduced survival of fry, and reduced health and survival of adult fish (Sorensen 1986). Typical deformities include scoliosis, missing or deformed fins, missing or deformed gills and gill covers, abnormally shaped head, missing or deformed eyes, and deformed mouth (Lemly 1998). Parental transfer of selenium to eggs and larvae of fish can be lethal or teratogenic (Ohlendorf 2003).

In general, fish studies indicate that in elevated selenium conditions, sensitive fish species disappear due to direct mortality or reproductive failure while a few tolerant species persist (Garrett and Inman 1984, Sorensen 1988, Vencil 1986, NRC 1989 as cited in Hamilton 2004). Both field and lab studies indicate that selenium results in reduced fish populations in cooling reservoirs of coal-fired power plants (Ohlendorf 1989). However, when water is the only exposure route, toxic thresholds for selenium for adult fish are usually greater than 1000 µg/L (USDI 1998). For rainbow trout sac fry, adverse effects are seen at waterborne concentrations of 50 to 100 µg/L (Birge et al. 1979 as cited in USDI 1998).

Plants

Selenium is not generally considered essential for plant growth and under natural conditions is usually not toxic. However, in some nonaccumulator species, it has been found that soluble selenium compounds may interfere with seed germination and growth (Shrift 1973, NAS-NRC 1983 as cited in Ohlendorf 1989). In toxicity tests, sublethal effects in green algae are observed at waterborne concentrations of 10 µg/L selenate and 75 µg/L selenite (Vocke et al. 1980, Foe and Knight 1986 as cited in USDI 1998).

Almost all of the selenium contained within plants is bioavailable, so when plants are consumed by animals, the selenium is transferred directly into the food-chain. Selenium levels in aquatic and terrestrial vegetation that can result in toxic effects in wildlife range between 3 to 20 mg/kg for chronic exposure and 400 to 800 mg/kg for acute exposure (Girling 1984 as cited in Ohlendorf 1989).

ACTION LEVELS/LEVELS OF CONCERN

Optimal dietary levels of selenium range from 0.05 to 0.3 mg/kg (dw) in wildlife, and from 0.055 to 0.4 mg/day (ww) for adult humans. Dietary concentrations resulting in toxicity are usually an order of magnitude higher than those resulting in selenium deficiency. A chronic dietary concentration of 2 mg/kg (dw) has been suggested as a maximum tolerable level for domestic animals (NAS-NRC 1980). Levels of concern, threshold concentrations, other action levels, and water quality standards applicable to wildlife, aquatic organisms, and humans are presented in Table 1.

The National Ambient Water Quality Criterion for protection of aquatic life under chronic exposure is 5 µg/L in freshwater and 71 µg/L in saltwater (USEPA 2002). These criteria may be revised to a tissue-based concentration, as USEPA has prepared a draft proposal for such a change (USEPA 2004). Levels of concern for fish range from 2 to 3 mg/kg (dw) in the diet and from 1 to 2 µg/L in water, as noted in Table 1. Levels of concern in fish tissue (whole body) range from 2 to 4 mg/kg (dw). For semi-aquatic birds, levels of concern range from 3 to 6 mg/kg in bird eggs, 2 to 3 mg/kg in diet, and 1 to 2 µg/L in water.

IMPACTS OF SELENIUM EXPOSURES TO WILDLIFE AT OTHER SITES

Kesterson Reservoir

Subsurface agricultural drainage water was used for maintaining marsh habitats at Kesterson Reservoir (located in Merced County, California) during the late 1970s to the mid-1980s. Studies on toxicity and bioaccumulation of selenium were conducted at Kesterson from 1983 until 2001. Results of these studies have shown that selenium from the subsurface agricultural drainwater bioaccumulated in plants and animals to levels that were toxic to a wide variety of aquatic birds. The most notable effects observed included mortality and impaired reproduction of grebes, waterfowl, and shorebirds.

The average selenium concentration in water entering Kesterson Reservoir from 1983 to 1985 was about 300 µg/L (Ohlendorf 1989) with a general decrease as water moved through the ponds. Downstream ponds

Table 1
Selenium Action Levels and Levels of Concern in Fish, Wildlife, and Humans

Value	Unit	Type	Receptor/Media	Tissue	Comment	Reference
Fish and Wildlife						
2-4	mg/kg dw	level of concern	cold-water fish	whole body	Above background, but rarely appear to be related to adverse effects in fish or wildlife	USDI 1998
>4	mg/kg dw	toxicity threshold	cold-water fish	whole body	Appear to be related to adverse effects on some fish and wildlife species	USDI 1998
3-4	mg/kg dw	level of concern	warm-water fish	whole body	Above background, but rarely appear to be related to adverse effects in fish or wildlife	USDI 1998
>4	mg/kg dw	toxicity threshold	warm-water fish	whole body	Appear to be related to adverse effects on some fish and wildlife species	USDI 1998
3	mg/kg dw	toxicity threshold	fish diet	NA	Associated with adverse effects	Lemly 1993, 1996
2-3	mg/kg dw	level of concern	diet	NA	Above background, but rarely appear to be related to adverse effects in fish or wildlife	USDI 1998
>3	mg/kg dw	toxicity threshold	diet	NA	Appear to be related to adverse effects on some fish and wildlife species	USDI 1998
4.87	mg/kg dw	EC10	mallard diet	NA	Caused effect on egg hatchability in 10 percent of eggs	Ohlendorf 2003
5.86	mg/kg dw	EC20	mallard diet	NA	Caused effect on egg hatchability in 20 percent of eggs	Ohlendorf 2003
8.05	mg/kg dw	EC50	mallard diet	NA	Caused effect on egg hatchability in 50 percent of eggs	Ohlendorf 2003
3-6	mg/kg dw	level of concern	waterbird eggs	NA	Above background, but rarely appear to be related to adverse effects in fish or wildlife	USDI 1998
>6	mg/kg dw	toxicity threshold	waterbird eggs	NA	Appear to be related to adverse effects on some fish and wildlife species	USDI 1998
10	mg/kg dw	threshold level	avian eggs	NA	Threshold for effects on hatchability	Heinz 1996
12.5	mg/kg dw	EC10	mallard eggs	NA	Caused effect on egg hatchability in 10 percent of eggs	Ohlendorf 2003
16.3	mg/kg dw	EC20	mallard eggs	NA	Caused effect on egg hatchability in 20 percent of eggs	Ohlendorf 2003
25.7	mg/kg dw	EC50	mallard eggs	NA	Caused effect on egg hatchability in 50 percent of eggs	Ohlendorf 2003
1-4	mg/kg dw	level of concern	sediment	NA	Above background, but rarely appear to be related to adverse effects in fish or wildlife	USDI 1998
>4	mg/kg dw	toxicity threshold	sediment	NA	Appear to be related to adverse effects on some fish and wildlife species	USDI 1998
1-2	µg/L	level of concern	water	NA	Above background, but rarely appear to be related to adverse effects in fish or wildlife	USDI 1998

Table 1
Selenium Action Levels and Levels of Concern in Fish, Wildlife, and Humans

Value	Unit	Type	Receptor/Media	Tissue	Comment	Reference
>2	µg/L	toxicity threshold	water	NA	Appear to be related to adverse effects on some fish and wildlife species	USDI 1998
Human						
0.02	mg/day	RDA	children (1-3 yrs)	NA	Recommended Dietary Allowance	ATSDR 2003
0.03	mg/day	RDA	children (4-8 yrs)	NA	Recommended Dietary Allowance	ATSDR 2003
0.04	mg/day	RDA	children (9-13 yrs)	NA	Recommended Dietary Allowance	NAS 2000
0.015	mg/day	RDA	infant (0-6 months)	NA	Recommended Dietary Allowance	ATSDR 2003
0.02	mg/day	RDA	infant (7-12 months)	NA	Recommended Dietary Allowance	ATSDR 2003
0.07	mg/day	RDA	lactating female	NA	Recommended Dietary Allowance	ATSDR 2003
0.055	mg/day	RDA	men and women	NA	Recommended Dietary Allowance	ATSDR 2003
0.06	mg/day	RDA	pregnant woman	NA	Recommended Dietary Allowance	ATSDR 2003
0.005	mg/kg/day	chronic oral RfD	human	NA	chronic oral reference dose	ATSDR 2003
0.9	µg/kg	Drinking water intake	human	NA	recommended daily intake for adults	ATSDR 2003
0.4	mg/day	Tolerable Upper Intake Level	human	NA	maximum daily nutrient intake likely to pose no risk to individuals	ATSDR 2003
0.015	mg/kg/day	NOAEL	human	NA	disappearance of symptoms of selenosis	ATSDR 2003
Water Quality Standards						
5	µg/L	CCC	freshwater	NA	criterion continuous concentration	USEPA 2002
20	µg/L	CMC	freshwater	NA	criteria maximum concentration	USEPA 2002
71	µg/L	CCC	saltwater	NA	criterion continuous concentration	USEPA 2002
290	µg/L	CMC	saltwater	NA	criteria maximum concentration	USEPA 2002

Notes:

CCC – Criterion continuous concentration
 CMC – criterion maximum concentration
 dw – dry weight
 NA – not applicable
 NOAEL – No observed adverse effect level
 RDA – Recommended Dietary Allowance

had selenium levels ranging from 50 to 200 $\mu\text{g/L}$. Total selenium in the ponds was primarily selenite (20-30 percent) while the drainage water only contained about 2 percent selenite and 98 percent selenate.

Bioconcentration factors in algae, rooted plants, and organic detritus were typically below 1000, but exceeded 1000 in animals such as the mosquitofish (Ohlendorf 1989). In 1983, selenium concentrations in submerged rooted aquatic plants, algae, widgeongrass, and rhizomes of emergent aquatic plants generally ranged from low- to mid-teens to about 300 mg/kg. Concentrations in aquatic invertebrates ranged from about 6 to 180 mg/kg (average of 60.4 mg/kg) in 1984 with an average of about 100 mg/kg in 1983. Mosquitofish were the only fish found from 1983 to 1985. Composite mosquitofish samples collected in 1983 had a mean selenium concentration of 170 mg/kg. Fish collected from various ponds in 1984 had mean selenium concentrations of 380 and 339 mg/kg. Liver concentrations of selenium in semi-aquatic birds ranged from about 20 to 127 mg/kg in 1983. Selenium levels appeared to increase from the early to late segments of the nesting season. Selenium liver concentrations in mammals from Kesterson averaged 522 times higher than those from the nearby reference site, Volta. All measured selenium tissue concentrations at Kesterson exceeded those at Volta (Ohlendorf 1989).

Semi-aquatic nesting birds at Kesterson were found to have high rates of embryo deformities and mortalities in 1983. In 1984, high numbers of dead birds were identified. Effects were associated with high levels of selenium. Selenium-induced effects, including dead or deformed embryos or chicks, were found in 39 percent of the nests monitored from 1983 to 1985 (Ohlendorf 1989).

Grasslands

In western Merced County, California, 30,000 hectares of permanent or seasonal wetlands are managed for the benefit of waterfowl or for livestock grazing (Ohlendorf and Hothem 1995). Most of this land is managed by the Grassland Water District. Portions of this land were historically irrigated with fresh irrigation water and agricultural drainage water, with an average selenium concentration of about 50 $\mu\text{g/L}$ in 1984. Field studies in 1984 found elevated selenium concentrations in birds and fish in this area, but not clear indication of selenium-associated reproductive impairment. Modifications to the water conveyance system were made so that since autumn 1985 the selenium concentrations were reduced to less than 2 $\mu\text{g/L}$. A more comprehensive study of the reproductive success of ducks and shorebirds was conducted in the Grasslands in 1986 and 1987 (Hothem and Welsh 1994a, b). Although elevated levels of selenium were found in some eggs, no clear effects on reproduction were detected.

Tulare Basin Evaporation Ponds

Tulare Basin is located in the southern San Joaquin Valley, California, and had about 25 evaporation and seepage ponds for irrigation drainage water (USDI 1998). The ponds ranged from 10 to 1800 acres, and water concentrations of selenium in these ponds averaged 0.5 to 1014 $\mu\text{g/L}$ while sediments averaged 0.1 to 16 mg/kg selenium. These elevated selenium concentrations were found to have contributed to embryonic malformations in some birds similar to malformations found at Kesterson (Ohlendorf 1989). In a study conducted by the USDI, 26 sites, under the influence of irrigation drainages, were evaluated for impacts from selenium (USDI 1998). The study identified selenium concentrations in surface water at Tulare ranging from less than 1 to 390 $\mu\text{g/L}$ (seventy-fifth percentile of 265 $\mu\text{g/L}$). Selenium concentrations in some of the 132 sets of avian eggs sampled were greater than 8 $\mu\text{g/g}$, a level considered to be embryotoxic (Seiler et al. 1999) although other studies indicate this threshold level to be closer to 6 $\mu\text{g/g}$ selenium in eggs (Skorupa 1998). Minimum selenium concentrations having adverse effects on avian reproduction at the Tulare Basin ranged from 2.6 to 18 $\mu\text{g/L}$ in water, 0.9 mg/kg dw in sediment, and 2.9 mg/kg dw in food chain fauna (USDI 1998). The USDI study also investigated the relationship between selenium toxicity and evaporation index (EI) at 26 sites and found that a higher EI sometimes indicated greater selenium toxicity although there were other contributing factors. The EI at Tulare Lake

Bed was 11.1, surpassed only by the Lower Colorado River Valley (California-Arizona) (EI=18.9) and the Salton Sea [EI=24.5] (Seiler et al. 1999).

Green River Basin

The Green River Basin of Wyoming, Colorado and Utah has naturally elevated selenium concentrations in sedimentary formations of the basin resulting in an excess of selenium in river alluvium. From 1986 to 1995 selenium was measured in surface water, bottom sediment, aquatic plants, invertebrates, whole-body fish, bird eggs, and bird livers from nine areas within the Green River Basin. The highest concentration in water across all sites was 16,000 µg/L with a maximum sediment concentration of 720 mg/kg (dw). Maximum concentrations in biota were 91 µg/g (dw) in aquatic plants, 71.7 µg/g (dw) in invertebrates, 120 µg/g (dw) in bird eggs, and 125 µg/g (dw) in bird livers. Muscle tissue samples collected from adult razorback suckers in the Green River from 1991 to 1995 had a median concentration around 34 µg/g (dw). Several samples exceeded the 4 µg/g (dw) threshold limit for cold-water fish. Bioassays performed on waters from various areas in the Green River suggested that the water was directly lethal to larval razorback suckers during a 10-day exposure. In addition, bioassays conducted using fathead minnows and razorback sucker larvae indicated that selenium exhibited a statistically significant relation to reproductive failure or mortality. Overall, while direct exposure to dissolved selenium in the Green River did not appear to result in acute lethality to endangered fish, concentrations in water were sufficient to inhibit reproduction in razorback suckers (Ohlendorf 1989) and result in multiple overt deformities in bird embryos (Seiler et al. 1999).

PREVIOUS HUMAN HEALTH RISK ASSESSMENTS AND ECOLOGICAL RISK ASSESSMENTS

Human Health Risk Assessments

Human exposure to excess selenium can result in acute or chronic toxic effects. Short-term oral exposure to high levels of selenium can result in nausea, vomiting, and diarrhea. Short-term exposure to elemental selenium or selenium dioxide in air can result in respiratory tract irritation, bronchitis, breathing difficulty, and stomach pains. Chronic oral exposure can result in a disease called selenosis. Symptoms include hair loss, nail brittleness, and neurological abnormalities. Chronic exposure via air may cause respiratory irritation, bronchial spasms, and coughing. However, selenium also is an essential nutrient, and some researchers agree that selenium deficiency is a greater threat to human health than selenium poisoning (Frost and Ingvald 1975, Stadtman 1977). Selenate or selenite supplements of 0.020 mg/kg/day usually prevent or reverse dietary deficiencies (Eisler 2000). Minor increases in dietary exposure can exert toxic effects in some individuals but not others. For example, an oral dose of only 0.023 mg/kg/day organic selenium over a lifetime resulted in selenosis in a female while a dose of 0.015 mg/kg/day had no observed adverse effect (Yang et al. 1989, ATSDR 2003). Both the ATSDR and U.S. EPA divided the no observed adverse effect level (NOAEL) established by Yang et al. (1989) by an uncertainty factor of three to allow for sensitive individuals, giving a maximum safe level for chronic oral ingestion of 0.005 mg Se/kg/day or 0.35 mg/day for a 70-kg adult. In 1994, the Food and Agriculture Organization of the United Nations and the World Health Organization also accepted Yang's NOAEL, resulting in a maximum safe dietary intake of 0.007 mg Se/kg/day using a safety factor of two. The minimum long-term dietary exposure found to produce sublethal toxic effects in humans is 1.9 mg/kg (natural selenium, ww) (USPHS 1989 as cited in USDI 1998). This concentration was considered a chronic selenosis threshold.

A health advisory related to selenium exposure from the consumption of sportfish has been developed for the Salton Sea. The assessment provides guidance on the maximum selenium exposures from fish consumption that are considered protective of human health. The advisory determines a safe exposure or dose based on selenium concentrations in fish tissue, fish consumption rates, and a numerical hazard

value specific to total selenium (i.e., all forms combined), and is based on the following general relationship developed by USEPA (2000) for assessing chemical contaminant data for use in fish advisories:

$$CR_{lim} = \frac{RfD \times BW}{C_m}$$

where

CR_{lim} is the maximum safe daily consumption rate;
 RfD is the reference dose (mg/kg/day) determined by USEPA;
 BW is average human body weight (kg); and
 C_m is the average contaminant concentration in the edible portions of fish.

A recent study by Moreau et al. (in press) evaluated health risks from selenium concentrations in tilapia fillets from the Salton Sea. The study used a mean selenium concentration of 1.67 $\mu\text{g/g}$ ww (based on measured concentrations in 24 tilapia samples collected in 1998) and a reference dose of 0.005 mg Se/kg/day that was adjusted for a background selenium intake from other sources of 0.0016 mg/kg/day. The risk-based analysis indicated that a 70-kg adult could consume as much as 1,000 grams per week (or 19 8-oz meals per month), and a 30-kg child could consume as much as 430 grams per week (or 16 4-oz meals per month). If the daily selenium intake from other sources was considered zero, adults and children could safely consume 28 and 24 meals per month of tilapia fillets, respectively. These study results are consistent with a previous study by Costa-Pierce et al. (2000) that indicated selenium exposure through the consumption of Salton Sea fish should be limited to 130 to 190 g/day for a 70-kg adult (or 17 to 25 8-oz. meals per month). Results from both studies are less conservative than current USEPA (2000) guidelines for selenium exposure via fish consumption. USEPA allows 16 227-g (8-oz) meals per month for an average 70-kg adult consuming fish with an average selenium concentration of 1.5 to <2 $\mu\text{g/g}$. Regardless, these maximum safe consumption rates are approximately one order of magnitude higher than the current advisory limits issued by the State of California (discussed below). A similar effort to evaluate potential health risks for 3 other fish species (bairdiella, orangemouth corvina, and sargo) is in progress (Moreau et al. in review).

The Office of Environmental Health Hazard Assessment (OEHHA) web site (http://www.oehha.ca.gov/fish/so_cal/saltonsea.html) was updated on September 17, 2004, with the following advisory:

“Because of elevated selenium levels, no one should eat more than four ounces [114 g] of croaker, orangemouth corvina, sargo, or tilapia taken from the Salton Sea in any two-week period.”

This advisory was originally issued in 1986, and it was based on a tissue threshold of 2 $\mu\text{g/g}$ ww from a study conducted in Australia, which was not risk-based (Dalton and Bird 2003). It was also applied to Kesterson Reservoir (Fan et al. 1988), and it has been used as a screening level in a number of other California areas. Further details on the derivation of this number, however, are unclear. The most recent state-issued health advisory recommends that humans should not consume more than four ounces of croaker, orangemouth corvina, sargo, or tilapia taken from the Salton Sea in any two-week period due to high selenium levels (Cal/EPA 2004, CDFG 2004), which is also consistent with the OEHHA advisory. This is the current advisory; modification of this advisory would depend on new data as well as OEHHA evaluation. An additional warning for the New River has been published and posted by the Imperial County Health Department for people to avoid physical contact with the waters of the New River and to avoid eating fish of any variety taken from the river. This advisory may be due to exposures from

multiple contaminants, including elevated concentrations of organochlorine compounds, such as DDE and PCBs (Riedel et al. 2002; Sapozhnikova et al. 2004), and not just selenium.

Ecological Risk Assessments

Selenium was one of the chemicals of potential ecological concern (COPECs) in an ecological risk assessment that was completed for land-based solar evaporation ponds near the Salton Sea (Tetra Tech 2004). This ecological risk assessment indicated that selenium conditions in the Salton Sea and in 4 or 5 evaporation ponds (where invertebrates are present) would result in potential ecological risks to aquatic birds (American avocet, black-necked stilt, eared grebe, and snowy plover). In addition to the risks from direct selenium exposures, a 7 percent increase in black-necked stilt clutches containing at least one inviable egg, was estimated from selenium bioaccumulation in the solar evaporation ponds. It should be noted that the birds were not foraging within the evaporation ponds and that bioaccumulation from sediment was actually based on limited samples of detritus within the lined ponds.

Ecological risk assessments (ERAs) were conducted for Kesterson Reservoir three times, including to evaluate cleanup alternatives (CH2M HILL 1986) as well as the success of remediation and subsequent monitoring requirements after remediation was completed in 1988 (CH2M HILL 1993, Ohlendorf and Santolo 1994, CH2M HILL and LBNL 2000, Byron et al. 2003). The first ERA (CH2M HILL 1986) evaluated the potential ecological impacts of three cleanup alternatives that were identified in the Environmental Impact Statement for Kesterson Reservoir (USBR 1986). The ecosystem and selenium transfer through the ecosystem were modeled using data available from several research programs. Selenium exposure to organisms high in the food chain that would result from changes in soil and waterborne selenium levels for each cleanup alternative were predicted. Because none of those alternatives was considered environmentally protective, the reservoir was de-watered and filled in 1988 so that all areas had at least 15 cm of soil above the expected seasonal (winter) elevation of high-selenium groundwater. These actions effectively transformed the Reservoir into terrestrial habitats, as described by Ohlendorf and Santolo (1994).

The second ERA (CH2M HILL 1993, Ohlendorf and Santolo 1994) was completed to estimate the most likely levels of biologically available selenium in various biota (plants and animals) at the site within the next 20 years, assess the risks of adverse effects to animals that could be caused by the selenium contamination, assess the significance of the site's selenium toxicosis risks, assess needs for alternative management plans, and recommend research and monitoring that would provide information needed to improve management for the site.

By 1999, more extensive monitoring data were available for the site (including the results of sampling during the wettest year on record, 1997-1998) that were useful for evaluating the risks associated with rainwater-formed pools within the site. Thus, another ERA was conducted for the terrestrial and ephemeral aquatic habitats with the expanded data set (CH2M HILL and LBNL 2000, Byron et al. 2003). The primary goal of this ERA was to use the results of the ongoing monitoring activities to update the characterizations of exposure and risks to Kesterson wildlife. The secondary goal was to evaluate the results of the EcoRA with respect to management strategies and the environmental monitoring program, and to make recommendations to optimize future monitoring and management of the habitat.

Overall, ERA was a useful tool for evaluating available information about Kesterson Reservoir and in evaluating needs for monitoring or management of the site. Results of the most recent ERA indicated that risks of adverse effects to wildlife are in the acceptable range, the monitoring program could be scaled down, and only limited management actions were needed to further reduce risks of adverse wildlife effects.

EXISTING PROTOCOLS FOR EVALUATING AVIAN EXPOSURE TO SELENIUM

The Bay-Delta Selenium Model was designed to predict the effects of selenium on wildlife in the San Francisco Bay-Delta Estuary (Luoma and Presser 2000). The model considers loads, water-column concentrations, speciation, transformation to particulate forms, particulate concentrations, bioaccumulation, and trophic transfer to predators to predict ecological impacts. The model was developed because future regulations may call for the evaluation of proposals and discharge permits and may include discharge requirements for an extension of the San Luis Drain to the estuary to convey subsurface agricultural drainage from the western San Joaquin Valley. They may also call for a renewal of an agreement to allow the existing portion of the San Luis Drain to convey subsurface agricultural drainage to a tributary of the San Joaquin River, and refinements to promulgate selenium criteria for the protection of aquatic life for the estuary. Results of the Bay-Delta model and forecasts suggest that many of the most likely combinations of load, hydrology, climate, selenium reactivity and bioavailability pose significant ecological risk to the Bay-Delta. It is probable that this model could be adapted for use at the Salton Sea to predict impacts to wildlife based on selenium concentrations and various other modeled parameters (T.S. Presser, USGS, personal communication).

Protocols for evaluating avian exposure to selenium include ERA approaches that consider appropriate exposure factors for the representative receptor species (e.g., body weight, food ingestion rate, etc.) along with an exposure term (referred to as an exposure point concentration. This kind of approach can be used for the Salton Sea, as it has for a number of other sites where selenium was evaluated (such as at Kesterson Reservoir, discussed above).

An alternate approach to typical ERA (referred to as a Protocol) has been proposed by Lemly (2002) for assessing hazards from selenium to aquatic life, including birds as well as fish. The Protocol uses a set of selenium data from several media including sediment, water, macroinvertebrates, fish eggs, and aquatic bird eggs. Although there are erroneous assumptions and statements in part of Lemly's description for risk calculation (e.g., p.62 about derivation of a hazard quotient), the Protocol can be useful if used appropriately.

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