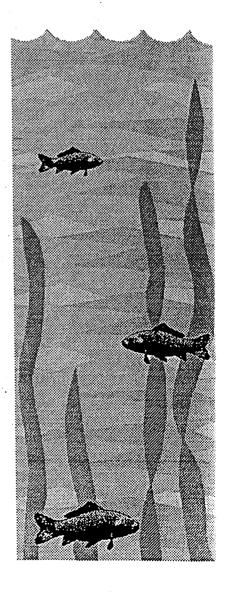
Salton Sea Symposium

January 13, 1994 Indian Wells, California

Skorupa, J.P. 1994. Impacts of selenium on the biological systems of the Salton Sea. In: <u>Proceedings of the Salton</u> <u>Sea Symposium</u>, January 13, 1994, Indian Wells, California. Salton Sea Authority, Imperial, CA. 15 pp, typed.



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Coachella Valley Water District Imperial County Imperial Irrigation District Riverside County Speaker: Dr. Joseph P. Skorupa, U.S. Fish & Wildlife Service

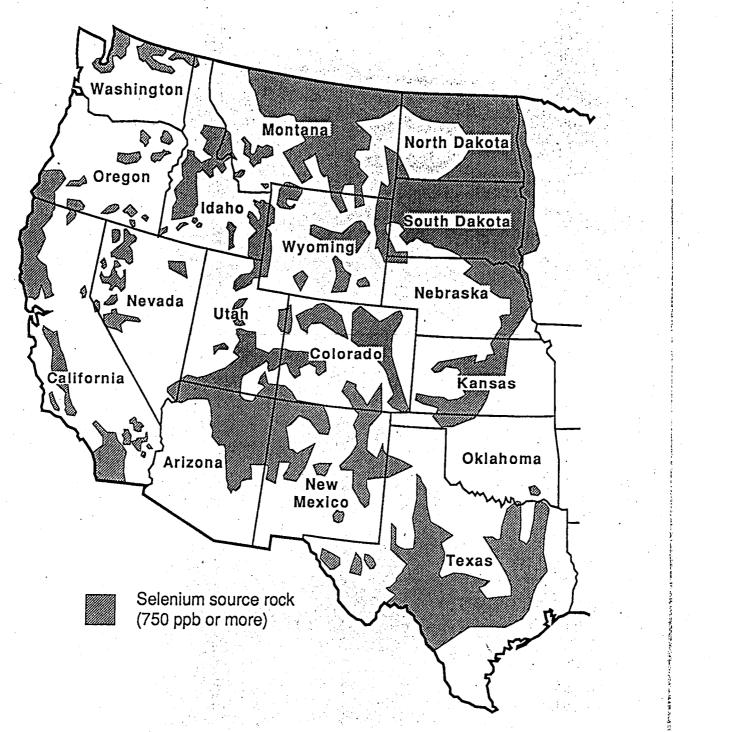
<u>Presentation Title</u>: "Impacts of Selenium on the Biological Systems of the Salton Sea"

Narrative Summary of Presentation:

Some Basics of Selenium Chemistry and Ecotoxicology

Selenium is a naturally occurring, semimetallic trace element. It typically occurs in the earth's crust at concentrations of less than one part-per-million (ppm). Much of the selenium found in arable soils of the western United States was originally deposited via prehistoric volcanic eruptions. The current distribution of seleniferous soils is well known (Figure 1).

From an ecotoxicology perspective, the chemistry of selenium is dominated by two central features. Chemically, selenium behaves very similarly to sulfur and therefore is biologically active, that is, can easily enter into the metabolic chemistry of plants and animals. Secondly, although selenium is a required nutrient for animals, it has one of the narrowest tolerance margins between beneficial and toxic doses of any nutrient. As a very general rule, environmental concentrations just 10-times or more above normal background concentrations are sufficient to cause biological hazards.



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FIGURE 1. DISTRIBUTION OF SELENIFEROUS SOILS IN THE WESTERN U.S. Plants take up selenium from water and concentrate it in their tissues to varying degrees depending on the particular species of plant. Fish and wildlife bioaccumulate selenium in their tissues prinicipally by eating contaminated plants, insects, and/or other animals. Even in highly contaminated environments, fish and wildlife are rarely poisoned directly by drinking or living in contaminated water.

Aquatic plants, such as algae, biotransform elemental forms of selenium into seleno-amino acids and other biologically active forms of selenium that are far more toxic to fish and wildlife than the elemental forms that predominate in the earth's crust and in water. Accumulation and biotranformation of selenium by aquatic plants and insects can result in foodchains for fish and wildlife that contain up to 360,000 times the concentration of selenium in the water where the fish and wildlife are feeding. Experimental systems with less than 1 part-per-billion (ppb) of added seleno-amino acids in the water have led to tissue concentrations of selenium in aquatic insects that would be toxic to fish and wildlife eating those insects.

What's Normal

Freshwater ecosystems that are not heavily influenced by agriculturally or industrially mobilized sources of selenium

("selenium-normal environments") usually contain less than 0.5 parts-per-billion (ppb) total recoverable selenium. Inland saline sinks surveyed in Oregon, California, Nevada, and Utah usually contained less than 1 part-per-billion (ppb) total recoverable selenium.

Aquatic plants and insects in selenium-normal aquatic environments usually average less than 2 parts-per-million (ppm) total recoverable selenium on a dry-weight basis.

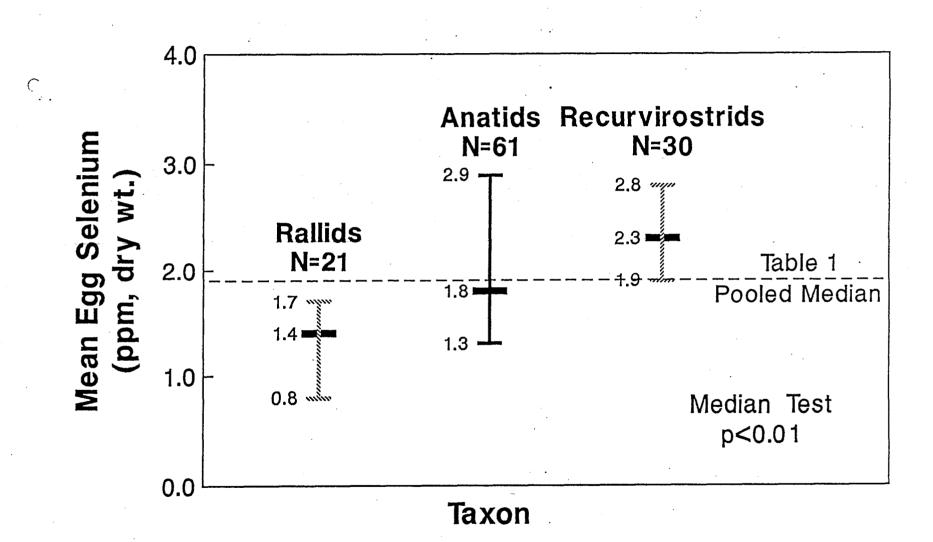
Bird's eggs, one of the best wildlife tissues for monitoring exposure to selenium in the environment, usually average less than 3 parts-per-million (ppm) selenium on a dry weight basis (Figure 2). Whole-body concentrations of selenium in freshwater fish are usually less than 2 parts-per-million (ppm) dry weight basis in selenium-normal environments.

Current Predictive Capability -- Bird's Eggs

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Field studies of avian exposure and response to selenium in the San Joaquin River Basin and near the Tulare Lakebed in California, and elsewhere throughout the western United States have provided an unprecedented base of data for classic doseresponse and epidemiological statistical analyses.

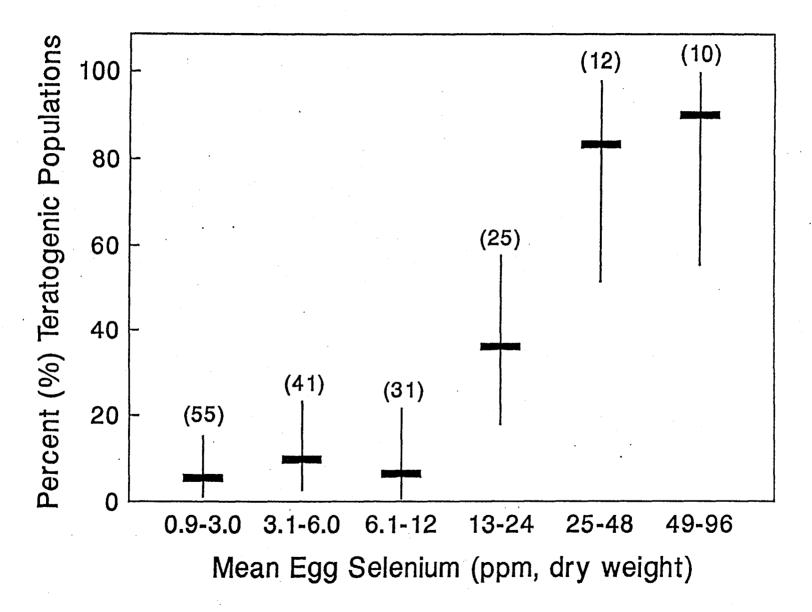
FIGURE 2. BACKGROUND CONCENTRATIONS OF SELENIUM IN AVIAN EGGS



The threshold exposure level associated with severe deformities of avian embryos occurs between 13 and 24 parts-per-million (ppm) total recoverable selenium on a dry-weight-basis (Figure 3). The overall viability of avian eggs, a more sensitive measure of toxic effects than deformities, can be examined by clutch-wise logistic odds ratios (the same statistical technique used to assess the human cancer risk associated with exposure to cigarette smoke). Results show that black-necked stilt eggs with as little as 4.2 to 9.7 parts-per-million (ppm) total recoverable selenium on a dry-weight-basis are about 4 times as likely to be associated with reproductive failure as eqqs with less than 4.2 parts-per-million total recoverable selenium (Table 1). In addition to direct embryotoxicity, selenium can adversely affect the survival rates of chicks after they hatch by slowing down their growth rates. A pilot study conducted by the Fish and Wildlife Service and the Environmental Protection Agency, showed significantly reduced growth rates in shorebird chicks hatched from eggs with more than 12 parts-per-million (ppm) total recoverable selenium on a dry weight basis.

Because there is a close correlation between waterborne selenium and egg selenium in sedentary species of waterbirds (Figure 4), and a close correlation between egg selenium and toxic effects, toxicity thresholds for avian eggs can be used to estimate toxicity thresholds in water and in the diet (Table 2). These estimates have already been independently field validated by two

FIGURE 3. DOSE-RESPONSE PLOT FOR SELENIUM-CAUSED EMBRYO DEFORMATION IN WATERBIRDS



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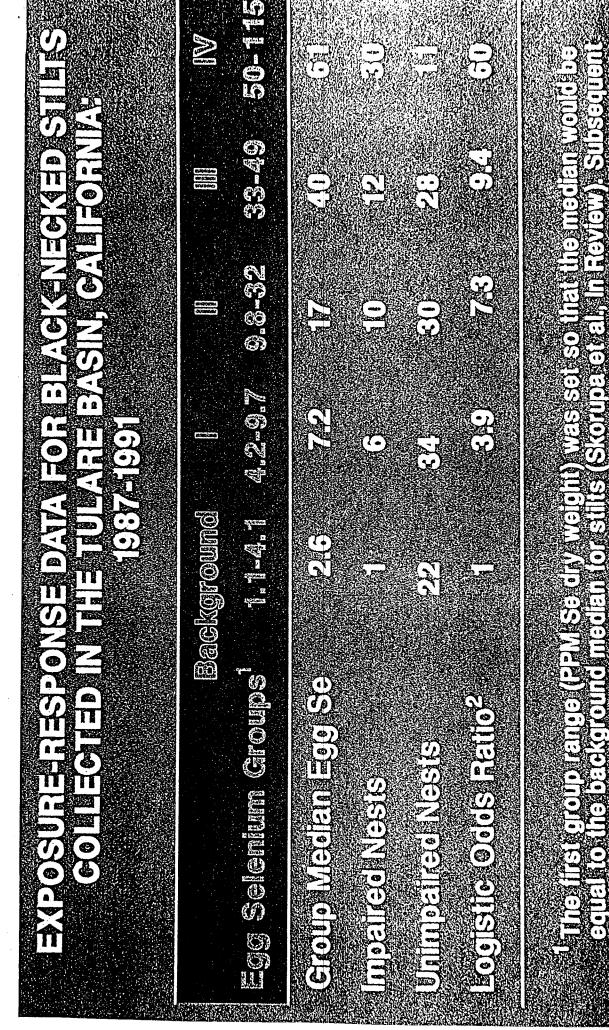
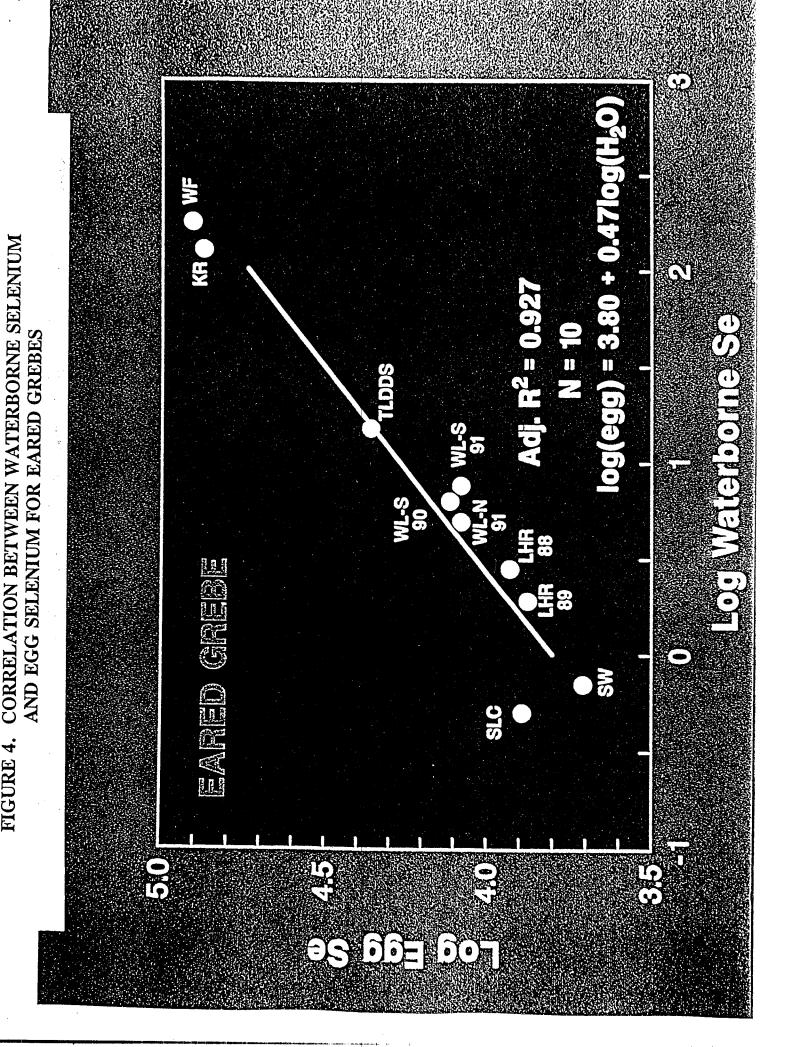


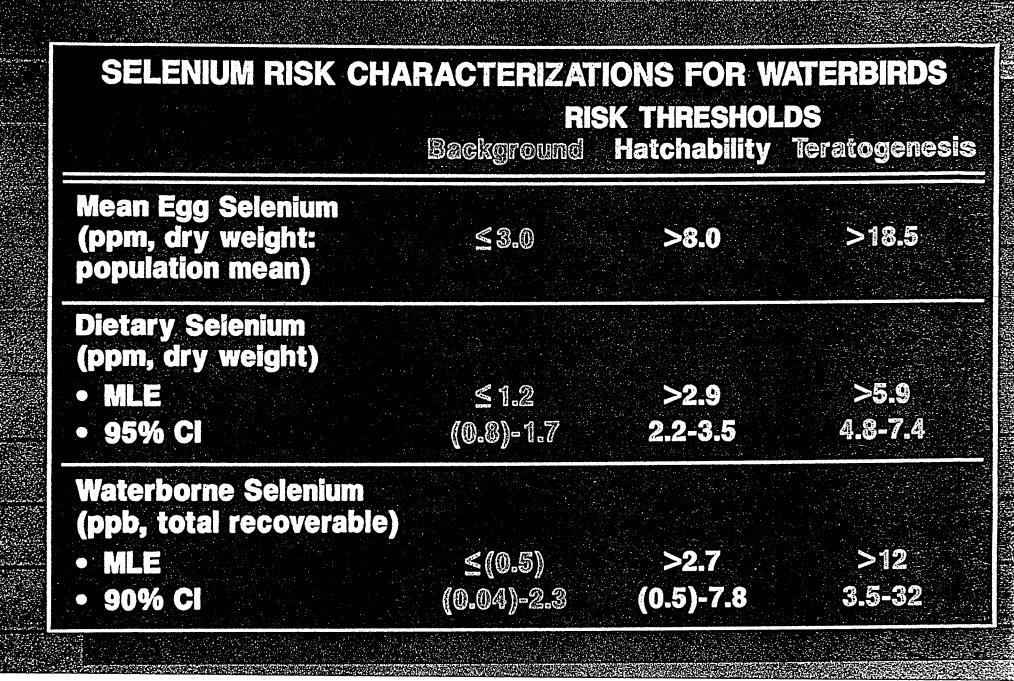
TABLE 1

²Hosmer and Lemeshow (1989)

group ranges are the istization of and 4th outsalles above background







recent studies of avian exposure to selenium.

Implications for Salton Sea Biological Systems

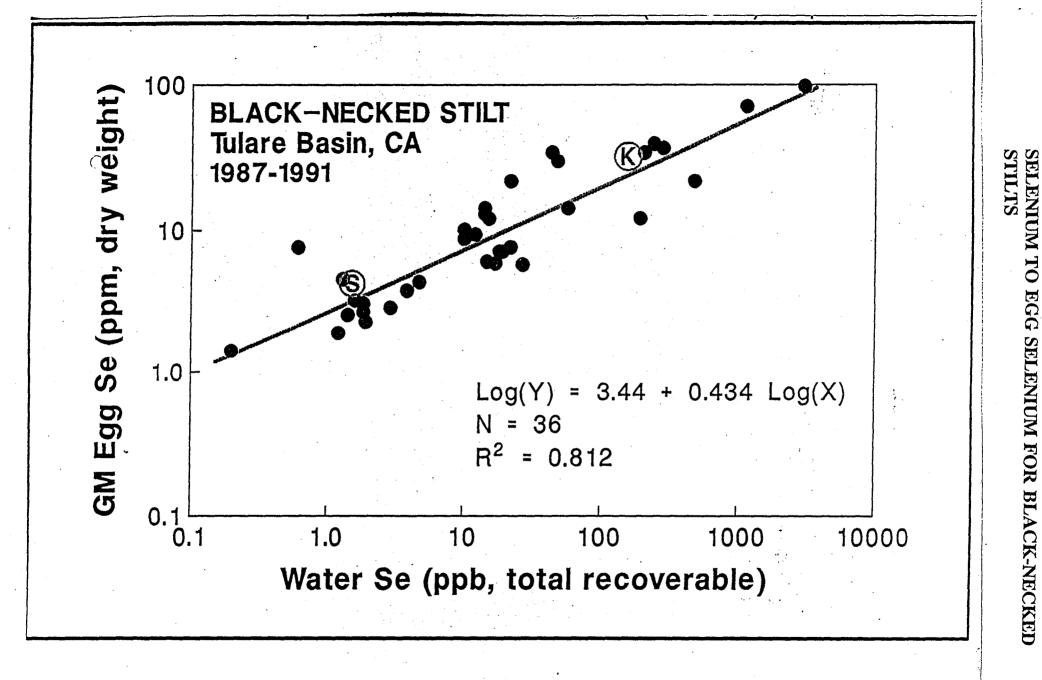
<u>Birds</u>

The general toxicological relationships documented for the San Joaquin Valley also appear to apply to data from the Salton Sea. For example, water-to-egg selenium ratios for black-necked stilts documented for the Salton Sea fall on the same regression line as data from Kesterson Reservoir and the Tulare Basin (Figure 5).

Data on egg selenium are available for only three species of birds at the Salton Sea, black-necked stilts (1988-1990), blackcrowned night-herons (1985), and great egrets (1985). Mean egg selenium for those three species ranged from 3.5 to 5.9 partsper-million (ppm) on a dry weight basis. To the extent that these results are representative of all species of breeding birds, all areas of the Sea, and current contaminant conditions, they indicate a low risk of selenium-caused embryo deformities. Among the 12**8** stilt eggs that were sampled between 1988 and 1990, about 5-6% contained selenium concentrations above toxicity threshold points for egg viability. Approximately 30-50% of the stilt eggs contained enough selenium to present a secondary

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toxicity hazard to egg-predators.

In general, egg data available from the mid and late 1980's clearly demonstrate that breeding birds were exposed to elevated levels of selenium, but that the risk of selenium-caused reproductive toxicity was low. These conclusions, however, must be qualified with at least two caveats. The most recent (1991-1992) sampling of eared grebe livers revealed about a tripling of selenium tissue burdens compared to 1988-1990 data suggesting the possibility that 1980's egg selenium data may substantially underestimate current levels of avian exposure to selenium risk that are presented here do not take into account or assess the potential for interactive effects due to exposure to multiple contaminants.

Finally, selenium has long been known to weaken animals' natural immune system defenses against disease, and preliminary studies with mallards suggest that avian exposure to concentrations of selenium that are not high enough to directly poison a bird or its eggs may nonetheless be sufficient to suppress various measures of avian immune response. Thus, virtually any time birds are exposed to above background concentrations of selenium, as they clearly are at the Salton Sea, the potential for increased susceptability to disease and other natural stressors exists.

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<u>Fish</u>

Toxicity criteria for saltwater fish have not been developed for selenium. As early as 1984 and 1985, however, tissue concentrations of selenium in sport fish commonly exceeded criteria judged by the California Department of Health Services as sufficient to warrant human health advisories. I am unaware of the existence of any data sufficient to assess whether sport fish selenium concentrations at the Salton Sea are changing over time.

General Sources for Further Reading

<u>Toxicology of Selenium: A Review</u> by Charles G. Wilber. Pages 171-230 in Clinical Toxicology, Volume 17, Number 2, (1980).

<u>Biogeochemical Cycling of Selenium in the San Joaquin Valley,</u> <u>California, USA</u> by Theresa Presser and Harry Ohlendorf. Pages 805-821 in Environmental Management, Volume 11, Number 6, (1987).

<u>Subchronic Effects of Sodium Selenite and Selenomethionine on</u> <u>Several Immune-Functions in Mallards</u> by Anne Fairbrother and J. Fowles. Pages 836-844 in Archives of Environmental Contamination and Toxicology, Volume 19, (1990)

<u>Elemental Concentrations in Fishes from the Salton Sea,</u> <u>Southeastern California</u> by Michael Saiki. Pages 41-56 in Water, Air, and Soil Pollution, Volume 52, (1990).

<u>Organochlorines and Selenium in California Night-Heron and Egret</u> <u>Eggs</u> by Harry Ohlendorf and Kathy Marois. Pages 91-104 in Environmental Monitoring and Assessment, Volume 15, (1990).

Copies of any of the above listed sources may be obtained by sending a written request to: Joseph Skorupa, U.S. Fish & Wildlife Service, 2800 Cottage Way, Rm E-1803, Sacramento, CA 95825.

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