0163-4984/89/2012-0023$02.00

Selenium in Animal Nutrition: The Oregon and San Joaquin Valley (California) Experiences—Examples of Correctable Deficiencies in Livestock

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Received May 19, 1988; Accepted September 3, 1988

ABSTRACT

White muscle disease and other selenium deficiency syndromes, once extremely common in young calves and lambs in Oregon, especially in the areas of volcanic origin east of the Cascade mountain range, prompted extensive investigations in the Oregon Agricultural Experiment Station that resulted in the implementation of large-scale selenium supplementation programs. Although selenium deficiency in livestock is consequently now rare in Oregon, selenium-deficient soils and attendant selenium deficiency conditions have been reported near the Kesterson Wildlife Refuge in the Northern part of the San Joaquin Valley, California, where, paradoxically, selenium toxicity in wildfowl, nesting near evaporation ponds, occurred and attracted wide attention. This review cites studies which explain why there is no evidence of selenium toxicity in livestock, but some selenium deficiency on the east side of the San Joaquin Valley. They also show that there is no threat to the food supply owing to excessive selenium in this area and that the consumption of meat and milk from the herds would not exceed the safe range of selenium for humans.

Text of a Presentation by the Author to the Klaus Schwarz 1988 Medal Award Symposium, held at the University of California, Berkeley, CA.

Index Entries: Selenium deficiency; selenium toxicity; white muscle disease; selenium supplementation; livestock; milk; meat; lambs; Oregon; Kesterson; San Joaquin Valley, California.

INTRODUCTION

Since its discovery, more or less by accident, by Berzelius in Sweden in 1818, selenium has been a topic of intense interest, often a subject of controversy, and sometimes an item of concern. In an age when we often seem obsessed with size of operations, it is refreshing to document an item of importance that functions on a small scale. Selenium performs its essential physiological functions in animal diets at levels of less than 1 ppm in the dry matter, which is a very small scale indeed. This suggests that this element, as is true for many other useful substances, may become toxic at higher concentrations, and, indeed, it does. T. H. Jukes (*1*) has estimated the difference between essential and toxic concentrations of selenium to be of about 20-fold magnitude. This difference is small, yet large enough to render cases of poisoning rare compared to diseases triggered by deficiency. In livestock, poisoning occurs only under unusual circumstances, i.e., when animals are forced to graze on pastures with plants like the wild vetch, *Astragalus*, that accumulate high concentrations of selenium from selenium-rich soils.

My personal experience with selenium goes back about 35 yr, when I was asked to serve as a member of a research team in the Oregon Agricultural Experiment Station to investigate "white muscle disease," (WMD) a myopathy that occurred extensively in very young calves and lambs in Oregon. The most troublesome incidence of WMD was in the upland area referred to as Oregon's high desert, just east of the Cascade mountain range, where the soils are generally of volcanic origin. Since this area produces much of the state's hay, under irrigation, and the hay is shipped widely, it has also contributed to outbreaks of WMD elsewhere.

The result of our investigation was the finding that WMD was a manifestation of selenium deficiency in animals, confirmed by evidence that minute amounts of selenium would entirely prevent or cure the problem (*2*). It's interesting to reflect, in retrospect, on why we included selenium in our investigations, since up to that time selenium had been known as a poisoner of livestock and not as a helpful element. We had experimented with vitamin E, which had been shown to be helpful in overcoming a similar problem called "stiff lamb disease", in experiments at Cornell (*3*) but under our conditions, vitamin E brought no relief. We added selenium as an alternate treatment, because of our knowledge of the interesting work with it, underway by Klaus Schwarz, who we honor this afternoon, and by others, including Karl Folkers at Merck and Com-

pany and the research team at Lederle Laboratories, who will be invested with the Klaus Schwarz medal.

We did our investigations with sheep, mainly for reasons of economy, but we felt the results would be applicable to both sheep and cattle. White muscle disease had not been a problem in our university flocks and herds, which included bloodlines common to Oregon's commercial ranches, so we assumed the disease was not genetically-based, but was likely nutritional. Our strategy was to import alfalfa hay from the WMD-prevalent areas in central Oregon, and to feed it to ewes during pregnancy, along with a small quantity of oats. We gave the animals distilled water, to guard against contamination from that source. Under these conditions, there was a high incidence of WMD in the test lambs, which was entirely prevented by feeding 0.1 ppm Se in the diet, provided by sodium selenite, but not prevented by supplementary vitamin E. I vividly remember the thrill of this discovery—that so little of this remarkable micronutrient could mean the difference between life and death in animals.

Prevention of death in calves and lambs was a powerful incentive for research with WMD in Oregon. I have personally visited ranches in the central part of the state where 80% of the calf crop was lost to it in a given year, and one cannot remain long in business that way. The average incidence, even in the most severely-deficient areas, was much lower, but still significant, and the economic cost of the problem in death losses alone was estimated at more than $1,000,000 a year in the late 1950's. Beyond this, of course, there are even greater losses in lowered productivity, in terms of growth depression of deficient animals that survive (*4*).

Some obvious questions occurred as a result of our early studies with selenium. Why should selenium be deficient in feedstuffs grown in certain areas, like central Oregon, and perfectly adequate elsewhere? And why should the young of the ruminant species, calves and lambs, be more susceptible than other species and types of livestock? Knowledge of the geology of the area threw light on the first question. The mountains of the Cascade range, which separates eastern from western Oregon, are nearly all volcanic, and surrounding soils are deficient because their indigenous selenium become volatile during the heat of eruption and passed off as a gas. What little selenium remained tended to be encased in glassy volcanic particles and was unavailable for plant growth. The eastern Oregon soils were more seriously deficient than the western ones, because the prevailing winds blow from west to east. The answer to the second question was similarly straightforward. Cattle and sheep are grazing animals and, as such, consume the local forage as virtually their entire diet. When the area in which the forage was grown is selenium-deficient, it follows that the consuming animals will become seriously deficient. The young are particularly susceptible to the deficiency, since they represent an added nutritional demand on top of that required

for maintenance of the mother animals. Other species or types, including dairy cattle, pigs, and poultry are more likely to be fed diets supplemented with feeds from other areas, many of which are not likely to be deficient.

Discovery of the effectiveness of selenium against white muscle disease soon led to identification of a number of other conditions in animals, world-wide, that were similarly selenium-responsive. In quick succession, these included hepatosis dietetica, a type of liver necrosis with petechial hemorrhaging in pigs; mulberry-heart disease, also found in pigs; exudative diathesis, a membrane-permeability problem that allows fluids to exude from the tissues and collect under the skin, and pancreatic degeneration, both of which occurred in poultry. The involvement of pigs and poultry in selenium deficiency problems reflects the common practice of feeding these species diets predominantly drawn from a single source—cereal grains—that increases the chance of deficiency. With so many evidences of specific deficiency signs, it followed logically that insufficient dietary selenium would affect life processes generally, and this was soon shown to be true. We demonstrated that surviving lambs born to selenium-deficient ewes grew significantly slower than lambs whose mothers had adequate selenium (*4*). New Zealanders showed that selenium deficiency contributed strongly to poor reproductive performance in sheep in that country (*5*). These findings on growth and reproduction related to the very base of the livestock industry and rapidly led to carefully controlled selenium-supplementation, world-wide, in areas of selenium-deficient soils. The results have been quite spectacular. Animals can now be profitably produced on vast land areas where previously they could not, and efficiency of production of animal protein for human food has received a significant boost.

In the course of these large animal investigations, some novel ways of providing selenium in needed amounts, in areas of natural deficiency, have been developed. The obvious way to correct a nutritional deficiency is to add the needed nutrient to the diet, but with free-grazing animals that are frequently given no prepared feed, this presents difficulties. Two effective alternatives have been developed, both of them "down under" in Australia and New Zealand. The first of these is the heavy pellet, a mixture of elemental selenium and iron filings that is dense enough to remain in the rumen for long periods of time, gradually releasing enough selenium to keep the animal in good health (*6*). This is possible because the very small amounts of selenium needed can be incorporated in a relatively small bolus that the animal can tolerate. Cobalt and copper can also be given in this way, and a soluble glass bolus that contains cobalt, copper, and selenium has recently been marketed in England, under the trade name 'Cosecure." A less efficient method of supplementation, in terms of the amount of selenium used, is to add selenium to the soil as a fertilizer amendment, thereby increasing the selenium content of forage

crops grown. We did some of the early investigations of this procedure (*7*) and it has been developed to the point of widespread aerial application in New Zealand, where much of the land is too rugged and mountainous for conventional fertilizing practice (*8*). Amendment of selenium to deficient soils has also been recommended by the national government in Finland.

The facility with which such tiny amounts of selenium could produce profound biological changes in animals naturally led to questions about how it performed its metabolic functions. Its trace element status suggested that it was probably acting enzymatically, and this has been proven to be the case. Some very elegant investigations at the University of Wisconsin (*9*), confirmed by equally-fine studies in Germany (*10*), proved that selenium was an integral part of the structure of glutathione peroxidase (GPX), an enzyme involved in protecting lipids from oxidative deterioration, particularly those of the cell membranes. Laboratory animals were used in these basic studies, and later examination of domestic species that were selenium-deficient showed their blood levels of this enzyme to be lower than in normal animals. Not all the observed effects of selenium in animal systems are readily explained by its role in GPX, however, and this is an active area of continuing research.

There are some good reasons, then, for providing additional animals with selenium in areas of deficiency and there are a variety of ways whereby this can be done effectively and safely. Dr. Allaway's laboratory at Ithaca (*11*) has charted selenium status of soils and plants in the US, and similar maps have been prepared in several other countries. Let me turn now to some comments on implications of selenium supplementation of livestock in California, which I know interests many people, concerns some, and has received considerable attention in the popular press.

In the San Joaquin Valley (SJV), which includes some of the most highly productive agricultural land in the US, it has been recognized for some time that the intensive irrigation practiced could lead ultimately to salination of the topsoil and loss of fertility. To combat this, deep drains were installed to drain off excess irrigation water after it had passed below crop level, and thus prevent it from carrying salts back to the surface. These various drains fed into the massive San Luis drain, which was planned to transport the salt-saturated water to the Sacramento River delta, or San Francisco Bay, where it would be discharged and immediately diluted. Funding was inadequate to finish the project, and the San Luis drain discharged instead into some shallow evaporation ponds on the Kesterson Wildlife Refuge, in the northern part of the Valley. Here selenium entered the picture, because as the salt-loaded water evaporated, its salt contents became concentrated, and selenium salts that were among them reached levels that were toxic to small fish and wildfowl (*12*).

This is a unique set of circumstances. The selenium in the SJV origi-

nated in some unusual alluvial fans, called the Panoche Hills, on the west side of the valley. Gradually, over many years, this leached downward to the Valley floor, where more recently it has been picked up in the irrigation water. Paradoxically, other soils in the immediate vicinity are selenium-deficient and cattle raised on them had sub-normal selenium status, as measured by blood selenium levels. The California Extension Service conducted interesting experiments at a ranch on the east side of the Valley, near the Fresno-Tulare county line (*13*). There, cows showed very low blood selenium levels, averaging less than 0.02 ppm (compared with "normal" levels of 0.08 ppm). Provision of selenium either to the cows by means of the heavy boluses already described, or to their calves by intramuscular injection, resulted in significantly-increased calf weights.

Persons familiar with the Kesterson situation have expressed concern about the use of selenium supplements in an area where selenium toxicity occurs and have wondered, generally, about the safety, from the point of view of selenium toxicity, of the crops produced for human consumption in the SJV. A recent study reported by the University of California Agricultural Issues Center, in Davis, allays some of the fears (*14*). It stated, as a result of intensive analyses in the area: (1) There was no evidence of selenium toxicity in livestock, but some of selenium deficiency, on the east side of the SJV; (2) Potential consumption of meat and milk from the herds studied would not exceed the safe range of selenium for humans, and (3) There is no evidence that selenium levels in commercial crops from the area constitute a threat to the food supply, and "there is a good deal of evidence indicating that it does not."

I must comment on the implications of selenium-nutrition, or malnutrition, for humans, because when we hear of the problems occurring in animals as a result of impaired selenium status, it immediately raises concerns whether similar conditions can occur in humans. Selenium deficiency and toxicity can, indeed, occur in humans, but it takes some quite unusual circumstances to cause either to happen, because of the variety of the human diet and the widespread transshipment of human foods, within and between countries, that tends to modify the total diet content of selenium. There have been examples of selenium deficiency, which is called, locally, "Keshan disease" and of selenium toxicity (*16,17*) in humans in China. Both situations involve individuals of very low economic status who are forced to rely entirely on their own production of food materials for their diet. The result can be compared with the reactions of grazing animals in areas of selenium-deficient soils. Beyond these general considerations, there is currently great interest in the protective action of selenium against certain forms of cancer, the basis for which has been recognized for a decade (*18*).

I think this is a good place to end my discussion, because it makes the point I wish to establish, rather neatly: Selenium is very much of a

two-edged sword (*19*) and its toxicity is well known. But it *is* an essential nutrient and the dangers of undersupplying it are just as real, and affect larger areas of the world, than do the dangers of oversupplying it. Selenium-supply situations need to be carefully monitored and problems that occur in unusual situations, such as those in the SJV, need to be dealt with, but we should not (indeed, we cannot) forgo the very real benefits of the proper use of selenium because of fears of its misuse. The selenium story has not yet been completely told: selenium research continues to offer intriguing challenges to enterprising biological scientists.

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