Selenium Requirements of Cattle and Means of Meeting Them

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It is becoming much easier to talk about selenium than it was and now one is more frequently faced with the problem of what to leave out, rather than what to put in. The literature on the element has expanded at a truly remarkable rate and no less than five books, completely devoted to it, have appeared in this country in the last dozen years. The first of these, a comprehensive text published in 1964 by Rosenfeld and Beath at the University of Wyoming entitled "Selenium: Geobotany, Biochemistry, Toxicity and Nutrition," successfully bridged the gap between the toxic and essential nutrient qualities of selenium. Two years later we assembled "Selenium in Biomedicine," which was the proceedings of an international symposium held at Oregon State University. In 1971 the Committee on Animal Nutrition of the National Research Council produced "Selenium in Nutrition" which was a summary of then-current knowledge. Ralph Zingaro at Texas A&M and Charles Cooper of Noranda Mines in Canada authored the text "Selenium" which was published by Van Nostrand in 1974 and the National Academy of Sciences came out this year with "Selenium: Medical and Biological Effects of Environmental Pollutants," which incidentally lists 845 reference citations.

In the midst of such abundance, however, there is still somewhat scanty information on the specific effects of selenium on beef cattle. The reason for this is largely economic. Beef cattle are expensive (or were, before the present critical market situation occurred) and with research funding as difficult to obtain as it has been, many investigators turned to sheep as a more economical test animal. Nevertheless, most that has been learned about selenium with sheep appears to be transferrable to cattle, and we are able to cope with selenium-responsive conditions in cattle with considerable success.

There is another side to the picture, and that is the legal one. It is ironical that in the country that has led the world in generating fundamental knowledge about selenium we are still unable to apply what we have learned freely in animal production practice. The Food and Drug Administration, while it has authorized selenium to be used in various forms, where needed, for pigs and poultry, has still not released it similarly for cattle, although it will be considering a petition to do so in a few weeks. The reason for the FDA concern is that they feel selenium may be carcinogenic, and if it is it would fall under the so-called Delaney Amendment to the Food and Drug Act which stipulates a "zero tolerance" for any such substances in the tissues of food animals. We have done considerable work, involving over 1,000 rats, at Oregon State University (Harr, et al., 1967) which has not indicated any involvement of Se in cancer formation (Harr, et al., 1967) while, interestingly, others have suggested that the element may have an anticarcinogenic function (Shamberger and Frost, 1972; Schrauzer, 1976).

The Biological Function of Selenium

One of the more perplexing of selenium's metabolic relationships has been that with vitamin E, and it has been the investigation of this that led ultimately to the present understanding of selenium's function. In the mid-1940's, Cornell workers showed that "stiff lambs" could be cured by the administration of vitamin E (Willman, et al., 1945) while our experiments (Muth, et al., 1958) implicated Se deficiency as the prime cause of white muscle disease involving similar clinical symptoms. Identification of a number of conditions in various animal species that would respond to Se, vitamin E, or both, followed, and the occurrence of these was summarized by Schwarz (1961). The question of why two such dissimilar substances should share a common metabolic effect was not resolved until the last decade. Wisconsin workers were able to show that Se was an integral part of the enzyme, glutathione peroxidase (Rotruck, et al., 1973) and this was quickly confirmed by Flohe in Germany (Flohe, et al., 1973). It now appears that the key item of metabolic damage is oxidative destruction of essential metabolites. Vitamin E is able to prevent this through its role as a "free-radical scavenger" (Tappel, 1967) and prevents peroxides from forming, while Se, *via* glutathione peroxidase, breaks down the peroxides before they cause tissue damage.

Essentiality of Selenium for Cattle

Selenium is now generally accepted to be an essential nutrient for cattle and for other species of domestic livestock (National Research Council, 1971). As such, it must be present in their diets in required quantities or they will exhibit deficiency symptoms. These symptoms include a myopathy

characterized by the light colored skeletal and heart muscle usually called "white muscle disease" (Andrews, Hartley and Grant, 1962). Lesions are most frequent in the very young calf, and as might be expected, have a depressing effect on growth rate (Jolly, 1960). While there are reports of serious lowering of the reproductive rate in selenium-deficient sheep (Hartley, 1963) this does not appear to have been duplicated in cattle; however, Se-deficient cows in the United States and elsewhere appear to have an increased incidence of retained placentas (Trinder, et al., 1969; Conrad, et al., 1976). There is also considerable evidence that selenium, given with vitamin E, can alleviate calf scours (Kendall, 1960).

Sources of Selenium

Normally Se is supplied to animals in sufficient amounts through their feed. It is clear, however, that soil-plant-animal relationships exist which may be upset at any point in the chain (Allaway, 1968a). Some of the areas of greatest risk in Se deficiency disease of animals have soils of fairly recent, volcanic origin (for example: the "volcanic plateau" of the central North Island of New Zealand and the volcanic pumice soils areas of northern California and central Oregon). The theory about such areas is that the Se originally brought to the surface during the volcanic eruptions became volatile and passed off, leaving a Se-deficient residue. This led Lakin and Davidson (1967) to comment that ". . . the soils derived from igneous rocks are most likely to be uniformly deficient in selenium." Deficiency of soil Se can also be aggravated by irrigation and it is noteworthy that one of the areas of most severe incidence of white muscle disease in cattle in Oregon, near Madras, has been subjected to intensive irrigation in recent years (Oldfield, 1972). There are Se-deficient areas that do not involve igneous rock-based soils, however, and the soil-plant relationships are less clear in them, probably because the different chemical forms in which Se is present in soils may have widely different availabilities to animals.

In some cases, reduced availability of soil Se may relate to presence of interfering factors in soils, one of which is sulphur. Antagonism between Se and S has been recognized for many years and has generally been attributed to competition between the two ions for an absorption site (Leggett and Epstein, 1956). We observed very early in our studies of white muscle disease that field incidence sometimes increased following the application of gypsum, $CaSO_4 \cdot 2H_2O$, to soils on which the animals grazed the forage (Muth, 1955). It appears that other trace elements such as iron may also limit the availability of soil Se, and Allaway (1968b) has noted that selenites are very strongly bound by hydrous oxides of iron, and that the iron oxide-selenite complexes are very insoluble from about pH 4 to pH 8.5.

There are reasonably direct relationships between levels of Se in plants and the incidence of Se deficiency symptoms, like white muscle disease, in animals (Allaway and Hodgson, 1964). Cattle and sheep appear particularly susceptible to Se deficiency, since, as grazing animals, their diet is often completely restricted to forage grown in local (deficient) areas.

In some cases plants have different capabilities for absorbing Se from the soil that have important implications in animal nutrition. The most dramatic example of this is the difference between the selenium-accumulator plants, which may take up many times as much Se as normal plants grown on seleniferous soil. For example, *Stanleya,* a Se-accumulator, has been reported as accumulating 2,380 ppm Se, as compared with sagebrush containing 6.8 ppm a few feet away on the same soil (Moxon, et al., 1950). Some variations in Se content have also been recorded among the more common forage species. New Zealand workers found that, among their indigenous pasture plants, browntop *(Agrostis tenuis)* consistently showed the highest Se concentration, and white clover *(Trifolium repens)* the lowest (Davies and Watkinson, 1966). It is interesting that the highest incidences of Se-responsive disease among New Zealand livestock have been on lush pastures rich in clover, rather than in unimproved "browntop country" (Cousins and Cairney, 1961). While we have found more white muscle disease among cattle fed alfalfa hay than other crops from Oregon's Se-deficient soil areas, this may reflect a dilution of available Se by the greater organic matter-yield of alfalfa, rather than any inherent problem in Se uptake.

Requirements for Selenium

The foregoing material has illustrated the difficulty in identifying a single, specific dietary level of Se as a requirement for cattle. The minimum requirement varies with the form of Se, both naturally present or supplemented, and with the nature of the rest of the diet, especially including its vitamin E content. As a result, there is considerable variation in the "minimally-adequate" levels of Se reported by different workers. We found, for example, that ewes needed 0.06 ppm Se to prevent white muscle disease in their lambs (Oldfield, et al., 1963) while Gardiner and Gorman (1963) reported from West Australia that lambs would become affected with white muscle disease on forage containing 0.05 ppm Se. On the other hand, there are reports from Florida of cattle that are apparently healthy after being maintained for two years on pastures containing only 0.03 ppm Se. It seems probable at this point that the absolute dietary minimum for Se in the diet of cattle is around 0.02 ppm, but that under certain conditions, where interfering factors are present, or where the level of vitamin E is inadequate, they may need several times this figure. In practical use, a dietary intake of 0.1 ppm Se seems to provide sufficient margin of safety against dietary variations and environmental stresses (Allaway and Hodgson, 1964). Although several salts have been used, experiments generally indicate that

sodium selenite, Na_2SeO_3, is the compound of choice in supplementing Se either as part of a feed mix or as a drench.

Alternate Methods of Selenium Administration

In addition to the obvious method of adding Se to the diet, or giving it by drench, there are a number of possible alternatives for overcoming or preventing Se deficiency in cattle. Parenteral administration has been widely followed, usually intramuscularly, and this, with Se accompanied by vitamin E, is the only supplementary procedure for cattle to win approval of the FDA in this country, as yet. Van Vleet (1975) has reported in detail on this route and preparation in terms of efficacy and residual tissue levels, using a Se concentration of 0.0825 mg (as selenite) and 5.61 I.U. of d-α tocopherol acetate 1 kg body weight. Field trials with such preparations have shown improved weight gains in cattle in Se-deficient areas of northern California (Norman, 1976). Although such administration raises Se concentrations in animal tissues over those found in a deficiency state, they do not exceed so-called "normal" levels from natural feeding situations where Se is adequate.

Selenium may also be administered to animals via their salt. To do this, one needs to estimate salt consumption and add Se, as selenite, to it to approximate the 0.1 ppm Se level in the total diet. Canadian workers have successfully used a level of 15 ppm Se in salt with calves and lambs (Jenkins, et al., 1974), and we have recently used a level of 20 ppm with lambs (unpublished data). If the salt method is followed, it is unwise to begin the treatment with animals that have not had ready access to salt previously, since they may overeat and expose themselves to a possible toxicity situation.

One of the more interesting means of Se supplementation that has been developed recently in Australia (Kuchel and Buckley, 1969) but is not yet available in this country is the use of so-called "heavy pellets." These are similar in principle to the "cobalt bullets" that were developed about 20 years ago. The selenium pellets for cattle weigh 30 grams each and contain 10% elemental Se, which is slowly available. The remainder of the pellet is compressed iron filings to give the needed weight and density to keep the pellets from passing out of the stomach. In practice two pellets are given together and contact between them in the rumen apparently prevents coatings from forming and interfering with Se absorption.

There are two other means of overcoming Se deficiency that may be of incidental interest to veterinarians. The first of these is pasture topdressing in which Se is added to fertilizers, such as superphosphate, that are then applied as soil amendments in deficient areas. In experiments near Madras, Oregon (Allaway, et al., 1966) we were able to raise the Se level in forage sufficiently to prevent white muscle disease by applying 336 g/hectare, on the assumption that this would provide 0.15 ppm Se in the top 18 cm of soil. Grant (1965) did similar work in New Zealand with much lower levels of Se: about 70 g per hectare. Although potentially useful, this method of Se administration has not been widely practiced because of difficulties and costs of securing the fairly large amounts of Se salts needed and of the uncertainties introduced by variables like soil mineral content, pH, etc.

The other possibility is interregional feed blending. It is known that forage and grain crops used as livestock feeds may contain elevated levels of Se when they originate in seleniferous areas (e.g., the Dakotas, Wyoming). In theory, such feeds could be brought to selenium-deficient areas as dietary supplements. Some such translocation of feeds undoubtedly occurs in practice but it has not been widely applied because of the difficulties in obtaining specific Se analyses and because of the cost of shipping large amounts of feed over long distances.

Selenium Toxicity

The very fact that Se is metabolically effective in minute doses of less than a part per million in the diet suggests its extremely high biopotency. At high levels Se is one of the most toxic substances known to man, and its use must always be planned with extreme caution. Preparations for either oral or parenteral use are usually highly concentrated, however, tolerance to an overdose is greater for the oral route than for injections.

A dramatic, documented example is the paper by Shortridge, et al. (1971), in which deaths of 376 out of 557 Angus calves treated, due to acute Se toxicity, were recorded over a five-week period. The calves had been given 5 ml of a sodium selenite solution which, in error, contained 100 mg actual Se rather than the desired 12 mg per injection. Eighteen of the animals died within 24 hours of treatment; testimony to the extreme toxicity of excess injected selenium.

An error of similar magnitude in orally-administered selenium, on the other hand, would not have been expected to have such drastic results. It has been suggested (Maag, et al., 1960; Frost, 1976; Bell and Bacon, 1976) that prolonged feeding of 100 times the recommended level (equivalent to a dietary level of about 10 ppm) would be expected to cause chronic Se toxicity but not to result in deaths within a five-week period.

Conclusion

Selenium is accepted to be an essential nutrient, required in minute quantities by cattle, as well as other species of domestic livestock. The predominant symptom of Se deficiency in cattle is a characteristic myopathy called white muscle disease, which usually occurs in the very young calf, although there may be other involvements. Soil selenium levels are not always linearly related to incidence of Se-deficiency disease in livestock due to a number of variables, but levels of Se in forage plants or grains usually bear a direct, inverse relationship to the problem. Selenium deficiency may be prevented or treated by ad-

ministering Se via feed, salt, drench or parenterally. Of these, injection with a vitamin E preparation is the only procedure currently approved by the FDA for use in this country with cattle. Great care needs to be exercised in Se administration due to its extreme toxicity in amounts in excess of physiological needs.

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Discussion

Question: Here in the higher mountain area of northern California we have all the problems you have talked about. One thing I'd like to know, can we blame any of our third trimester abortions on selenium deficiency? We have blamed everything on foothill and we are finding that some of these are nutritional. Would selenium have any effect on some of these abortions, as well as the weak calves?

Answer: I cannot give you a definite answer. I think some of the involvement of selenium in New Zealand in reproduction in sheep suggests that it may be involved, but so far as my knowledge is concerned I don't think this has definitely been proven to be the case in cattle in this country. Certainly it is something that ought to be looked into. The potential is there-could be.