

Beef Cattle Handbook



BCH-3410

Product of Extension Beef Cattle Resource Committee Adapted from the Cattle Producer's Library

White Muscle and Other Selenium-Responsive Diseases of Livestock

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A significant research finding reported from Oregon State University in 1958 was that the underlying cause of white muscle disease was a dietary deficiency of the trace element selenium (Se). The relationship between soil, plant, and animal factors involved is fairly clear cut. Certain soils, formed within the past 10,000 years or so as a result of volcanic action, contain practically no selenium. As a result, plants grown on them are seriously selenium-deficient, and the deficiency is passed onto animals eating them in the form of fresh forage, hay, or silage, as all or most of their diet.

In addition to having selenium available in original parent material in soil formation, Se supply is affected by intensive cropping practices. Irrigation tends to leach Se out of the topsoil, and the application of some commercial fertilizers, because they result in higher crop yields, reduces the percentage of selenium in the forage.

Effects of Selenium Deficiency

The first important evidence of Se deficiency has been aptly named "white muscle disease." It involves muscle damage (myopathy), in the course of which calcium salts may be deposited among the muscle fibers in parts of the bodies of affected animals. These salts give the involved areas a whitish appearance. Both skeletal muscles, such as those of the legs and back, and heart muscles may be affected. When skeletal muscles are affected, the animals may have difficulty walking and may be unable to rise and follow the herd. When the heart is affected, the animal may die from sudden heart failure. Some animals show signs of respiratory distress, which may lead the owner to treat them for pneumonia. They do not respond to antibiotic therapy, and death may occur as a result of fluid accumulation in the lungs. White muscle disease is most common in newborn lambs or calves, which will show clinical signs from birth to an age of 4 to 6 weeks. It occurs occasionally in yearlings or older animals.

Cows or ewes receiving a selenium-deficient diet during gestation may give birth to offspring suffering from this trace-mineral deficiency. Their calves or lambs may be born dead or weak, and may die during the first few days of life. In cases of extreme deficiency, permanent damage may occur, and the newborn animals will not respond to selenium administration. The dams need adequate selenium during gestation. This is accomplished by injecting the pregnant cows or ewes with carefully calculated amounts of Se salts in sterile solution, or by providing an adequate Se source in the diet.

The influence of Se supplementation on the livability of newborn calves has been observed in a number of beef herds. Some calves in selenium-deficient herds have impaired structure and function of the thyroid gland. This is an additional stress for the new born that can mean the difference between life and death.

A less acute deficiency of this trace element also contributes to unthriftiness of yearling and adult animals, often referred to as "ill thrift." Many of these animals will develop a profuse diarrhea, will be unable to use feed properly, and may die. Their hair coat may develop a blanched or "washed-out" appearance. Many animals so affected have a rough hair coat and do not shed normally in the spring to develop the normal, healthy, sleek appearance. An increased incidence of retained fetal membranes has been documented in dairy cows on selenium-deficient diets, and this also has been suspected in beef herds. The problem has been alleviated by adequate Se supplementation. The retention of fetal membranes beyond 24 hours contributes to infection of the uterus and impairs subsequent breeding ability.

Defining the Selenium Status of Animals

The occurrence of white muscle disease in one or more young animals is definite evidence of Se deficiency. But this is a costly method of detection in that it may entail some deaths and other production losses such as impaired growth of the young or lowered reproduction efficiency.

The amount of Se in blood is a reliable indicator of the Se status of animals. Blood samples should be collected in heparinized vacutainer tubes by your veterinarian and submitted to a reliable analytical laboratory for Se determination. We consider 0.04 ppm Se to be the minimum acceptable blood level for beef cows. Breeding ewes and dairy cows have a higher requirement for this mineral, and their respective blood levels should be over 0.05 and 0.07 ppm.

In a survey of beef cows in Oregon during 1975-76, two-thirds of the blood samples tested were Se deficient, as judged by these values. A minimum of five samples was collected from each herd, and the herds represented all major geographic areas in the state. This study also showed that cows grazing native range had nearly twice the blood Se level of cows grazing improved, irrigated, fertilized pastures (0.049 vs. 0.026 ppm Se, respectively).

Deficiency	Subclinical	Adequate	Toxic
<.05*	.05 to .10	>.	+5.0

*parts per million

Selenium is now known to act in the animal body as an integral part of the blood enzyme glutathione peroxidase (GSH-PX), so the level of this enzyme in the blood may be used to indicate an animal's Se status. When Se deficiency exists, this enzyme level is below 20 units of activity per milligram of hemoglobin. Determining GSHPX activity is faster and less expensive than determining Se itself, but the enzyme is unstable, and values may be affected by improper storage of blood samples before analysis.

Suppling Selenium to Your Animals

The oldest accepted method of providing needed selenium is by injection. Several preparations that contain both selenium and vitamin E are available commercially. The manufacturers of these have carefully calculated safe and effective dosage levels, and their recommendations should be followed closely.

Several other methods of selenium supplementation have been developed. Selenium may be given mixed in feed or in salt mixes offered to animals free choice. Based on research findings, the U.S. Food and Drug Administration (FDA) approved the addition of selenium to complete feeds at a level of 0.1 ppm in 1974. Subsequently, it has increased the allowable limit to 0.3 ppm, with corresponding levels in salt mixes calculated on the proportion of salt intake to the total feed intake. Selenium is usually obtained in the form of one of its salts, usually sodium selenite, Na2SeO3. Because the amounts of selenium needed are so small, mixing presents problems. Commercial supplement mixing is recommended over home mixing.

Selenium has also been given effectively in the form of heavy pellets, or "bullets" that lodge in the forestomach of ruminant animals and gradually release selenium to the animal.

Modern forage production practices tend to reduce the Se level in livestock feed. To combat this, research workers in New Zealand, Australia, Finland, and the U.S. include Se in commercial fertilizers at a level of 10 g Se per hectare of land. This is an alternative method of Se supplementation that may provide an easier answer to Se deficiency problems at some future date, since it avoids handling the animals. Plants in general do not require selenium for normal growth, but its involvement in animal health has been dramatic and significant.

Selenium, like some other nutrient materials, is dangerous to animals if given in excess. The problem is especially critical with Se because the animal's requirement is extremely low (in the area of 0.1 ppm of the diet dry matter; that is, 1/10 part Se per million parts of dry feed). Excessive amounts of selenium are known to cause toxicity problems in animals, including death.

Recent publicity has described problems of selenium toxicity, resulting from concentration of selenium in irrigation runoff water in the San Joaquin Valley of California. This is a reminder that excess selenium is dangerous and that administration of it to overcome deficiencies must be done carefully. Evidence has shown that levels identified in this publication are safe.

Adapted from CATTLE PRODUCER'S LIBRARY CL628

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This publication was prepared in cooperation with the Extension Beef Cattle Resource Committee and its member states and produced in an electronic format by the University of Wisconsin-Extension, Cooperative Extension. Issued in furtherance of Cooperative Extension work, ACTS of May 8 and June 30, 1914.

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