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Selenium, one of the most recently discovered essential nutrients, has special significance for Oregon livestock producers.

Selenium is a trace mineral—one of those elements that is present in the earth's crust in extremely small amounts—that is absorbed by plants from the soil and thus finds its way into the bodies of animals that eat either plants alone or plants and animal matter.

Much of Oregon's Central Plateau has soil of volcanic origin. Evidence suggests that in the heat of forming such soils, selenium became a gas and was lost, leaving the region almost completely seleniumdeficient.

As a result, much of the forage produced in central Oregon is very low in selenium. The result is that grazing cattle and sheep, which may receive no other feed, become deficient. Shipping forages grown on selenium-deficient soils to other areas greatly extends the effects of selenium deficiency.

## What animals are deficient? How can you diagnose the deficiency?

Animals grazing forages grown on deficient soils are most likely to suffer from selenium deficiency. Oregon experiences place beef cattle and sheep at the head of the susceptibility list.

The requirements for selenium are minute—usually less than a part per million in the diet—so any kind of purchased feed, frequently brought in from another area, can easily include enough selenium to protect the animals' total diet from any danger of selenium deficiency. Dairy cattle, hogs, and poultry are often fed protein supplements that contain enough selenium, though deficiencies in these species have occurred.

Very young animals are more likely to become selenium-deficient than mature ones. The stress of pregnancy, when the female has to provide for her own needs and those of her young, means that young may be born deficient in regions where selenium is in short supply. The normal demands of rapid growth in the young, superimposed on a low reserve of selenium at birth, can cause selenium deficiency symptoms to appear.

Since selenium supplementation represents an additional cost for livestock producers, it is useful to know when it is needed and under what conditions you can expect it to bring positive results. In Oregon, one of the main symptoms of selenium deficiency is white muscle



Lesions of white muscle disease, caused by selenium deficiency, in a calf heart. (Photo by O. H. Muth)

disease, which causes damage accompanied by a whitish coloration in muscles of the limbs or heart.

Such lesions are easily recognized in an animal's carcass. In the living animal, there may be varying degrees of stiffness, sometimes to the point where the animal can get up only with difficulty. If heart muscle is affected, there may be sudden deaths from heart failure if the young animals are exposed to unusual exertion. Spells of warm, bright, spring weather coinciding with calving or lambing times can bring on deaths from selenium deficiency for this reason. Such signs are fairly positive evidence of selenium deficiency.

Since selenium is involved in the normal processes of growth and reproduction, poor growth or reproductive performance may suggest selenium deficiency. However, such indications are by no means certain; they should be confirmed by blood analyses, either for selenium or for glutathione peroxidase (a selenium-dependent enzyme). You can have such analyses done at reasonable cost by the Department of Agricultural Chemistry at Oregon State University.

Other signs that have been linked with selenium deficiency include retained placentas (afterbirth), exudative diathesis (a collection of abnormal amounts of fluid in the body cavities and under the skin), and liver necrosis (marked by multiple degenerative spots on the liver surface).

## How do you supply selenium to your animals?

When a need for selenium has been established, there are a number of ways to supply it effectively. The



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oldest accepted method is by injection, and several preparations that include both selenium and vitamin E are available. Use of these preparations has been worked out carefully, so follow the manufacturer's recommendations closely.

It is now possible to add selenium to feeds, either complete rations or supplementary materials that include salt. In 1971 the National Academy of Sciences published a report, *Selenium in Nutrition*, that indicated an addition of 0.1 ppm (part per million) selenium was sufficient to overcome the deficiency and prevent selenium-responsive diseases. Subsequently, the U.S. Food and Drug Administration has approved feeding selenium at this level to various species of livestock.

In making the necessary calculations for supplementation, it is convenient to use the metric system, in which 1 ppm is translated to be 1 mg per kg of feed (2.2 lb = 1 kg; 1 ton = 909 kg). Thus to supplement a ton of feed at the approved level of 0.1 ppm selenium would require  $909 \times 0.1 = 90.9$  mg of selenium.

Selenium is not readily available in pure elemental form, however, so it usually is supplied as one of its salts—frequently sodium selenite, Na<sub>2</sub>SeO<sub>3</sub>. Sodium selenite contains 45.65% selenium. To supplement a ton (909 kg) of feed with this same 0.1 ppm of selenium, divide that 90.9 mg by 45.65% (or multiply 90.9 mg by 100 and divide by 45.65). The result is 199 mg of sodium selenite.

$$\frac{90.9 \times 100}{45.65}$$
 = 199 mg.

The amounts needed are thus extremely small; you must take great care in mixing to ensure thorough distribution. Preparing a premix first—dispersing the sodium selenite in a few pounds of finely ground feed—is recommended before attempting a final mix.

Adding selenium to salt requires knowledge of your animals' intake, both of salt and of total feed. Suppose, for example, beef cows are averaging a daily intake of 15 lb of hay as their complete winter feed (divide 15 by 2.2 to obtain kg); to supplement this at the 0.1 ppm level would require 1.49 mg of sodium selenite.

$$\frac{15 \times 0.1 \times 100}{2.2} = 1.49 \text{ mg}.$$

But suppose we provide this supplemental selenium mixed with salt and feed it free-choice rather than in the entire ration. If these same cows average a daily salt intake of 2 oz, then each 2 oz of salt should contain the needed 1.49 mg of sodium selentite. For 100 lb of salt, this would equal  $100 \times 2/16$  (lb)  $\times 1.49 = 1,192$  mg of sodium selenite (1.192 g). For a ton of salt (20  $\times$  100 lb), this would mean  $20 \times 1,192$  mg = 23.84 g of sodium selenite per ton of salt.

Commercial mineral feed and salt companies currently offer salt mixes that contain the required amounts of selenium for cattle and sheep.

## Caution

Although selenium in very small quantities is essential, in larger quantities it becomes highly toxic. Do your calculations of selenium supplementation very

carefully. Watch your animals' reactions carefully to avoid costly—or even disastrous—errors.

Selenium toxicity can be acute or chronic, depending on the amount consumed and the time period involved. Acute toxicity, which occurs when large overdoses of selenium are given over a short time, produces symptoms of labored breathing, garlicky breath, abnormal movement followed by prostration, diarrhea, and death in a few hours.

This can result from accidental provision of excessive amounts of pure selenium salts or from consumption of large quantities of selenium-accumulator plants, which grow in high-selenium soil areas.

Chronic toxicity occurs over a longer period of time and results in (1) "blind staggers," which involves nerve damage resulting in impaired vision; (2) stumbling and "locoing," frequently followed by death from respiratory failure; or (3) "alkali disease," which occurs over a longer period of time and results in weight loss, loss of hair, hoof damage, lameness, and cirrhosis of the liver.

Acute toxicity is unlikely in Oregon, since selenium-accumulator plants rarely grow here. Chronic toxicity can result when dietary intakes of more than 5 ppm selenium continue over a period of several weeks or months.

## Further reading

Greater detail on the complex involvement of selenium in metabolism is given in *Selenium in Nutrition*, rev. ed. 1983, available from National Academy Press, 2101 Constitution Avenue NW, Washington, DC 20418.

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