Selenium Deficiency and its Prevention in Grazing Ruminants

by
Jerry W. Spears, Ph.D.,
North Carolina State University

INTRODUCTION

Selenium was recognized for its toxicity before it was discovered to be required by animals. In the 1930s high forage selenium levels in some Western areas of the United States were associated with toxicity in grazing animals. Toxicity signs included loss of hair and lameness. The lameness was due to abnormal hoof growth that in some cases resulted in the hoof sloughing off.

Selenium was found to be required by rats to prevent liver damage in 1957 (McDowell, 2003). This discovery led to the findings that selenium could prevent certain disorders that were causing high death losses in animal agriculture. It soon became apparent that in many areas of the United States and around the world that cereal grains and forages produced were deficient in selenium.

Because of its known toxicity, selenium could not legally be supplemented to diets for several years after selenium was shown to prevent multiple disorders in domestic animals. During this time producers could inject selenium intramuscularly to treat selenium deficient animals but were unable to supplement selenium to prevent deficiency. Selenium was shown to be required for glutathione peroxidase, an enzyme important in the body’s antioxidant system, in 1973. Reduced activities of this enzyme were soon discovered to be associated with the occurrence of various disorders in animals that had previously been found to respond to supplemental selenium. The discovery that glutathione peroxidase required selenium resulted in Food and Drug Administration approval of supplemental selenium in animal diets and mineral supplements. Selenium is generally supplemented to animals as sodium selenite or selenized yeast, an organic form of selenium.

The selenium requirement of animals varies from 0.1 to 0.3 ppm of diet dry matter. Selenium is commonly supplemented to diets of pigs and poultry. Providing supplemental selenium for ruminants and horses grazing pasture is more of a challenge since pasture is oftentimes their sole source of energy and protein. This newsletter will describe selenium deficiency signs in grazing animals and discuss methods of providing supplemental selenium to grazing animals.

SELENIUM DEFICIENCY IN GRAZING ANIMALS

Muscular Dystrophy (White muscle disease)

A condition referred to as white muscle disease or muscular dystrophy is a major clinical sign of selenium deficiency in lambs, goats, calves, and foals (McDowell, 2003). This condition occurs in young animals and results from damage to skeletal and heart muscle. As the name
applies animals with white muscle disease have chalky white streaks in their muscles due to oxidative damage. This condition is most common in lambs and usually is noticeable around birth or between 3 and 8 weeks of age. Lambs affected at birth are born dead or die a few days after birth following physical exertion. In severely selenium deficient flocks death losses around birth can be over 50% of lambs born.

Lambs that appear normal as birth can also show signs of muscular dystrophy at 3 to 8 weeks of age, with muscular weakness being the major sign observed. Lambs affected walk with a stiff gait and arched back, and avoid movement. This leads to reduced body condition and usually death unless selenium is administrated.

Calves deficient in selenium are most likely to exhibit signs of muscular dystrophy between 2 and 16 weeks of age. The calves may appear normal when lying down but when they are urged to stand stiffness can be observed and animals have difficulty walking and perhaps even standing. Because of the pain involved in standing calves may refuse to nurse their mother and die from starvation. Death can also occur from heart damage due to damage to cardiac muscle. Animals suffering from muscular dystrophy generally respond to an intramuscular injection of selenium and vitamin E.

Unthriftiness and Reduced Performance

Unthriftiness characterized by diarrhea and loss of body weight may occur in calves and lambs deficient in selenium. The diarrhea does not respond to antibiotic treatment but animals rapidly recover when injected intramuscularly with selenium (Spears et al., 1986). Selenium deficiency can also decrease body weight gain in growing cattle and milk production in lactating cows grazing pasture (Wichtel, 1998). Supplementation of selenium to beef cows and their calves grazing selenium deficient pastures increased calf weaning weight by 82 pounds per calf in a Florida study (Morris et al., 1984), and by 19 pounds in a North Carolina study (Spears et al., 1986).

Impaired Reproduction

Selenium deficiency in sheep has been associated with high embryonic death, and reduced number of ewes becoming pregnant when exposed to rams (Underwood and Suttle, 1999). Wilkins and Kilgour (1982) found that selenium supplementation prior to breeding reduced ewes failing to lamb from 16 to 9% on sheep farms with low selenium pastures.

In dairy cows selenium deficiency increases the incidence of retained placenta. Cows normally expel the fetal placenta 2 to 8 hours after calving. Retained placenta occurs when placentas remain attached to the uterus for more than 12 hours after calving. Cows with retained placenta have a much higher incidence of uterine infections following calving. Selenium supplementation has greatly decreased the incidence of retained placenta in a number of studies. Conception rate following breeding can also be reduced by selenium deficiency in cows. Reduced viability of sperm has been observed in selenium-deficient bulls (Underwood and Suttle, 1999).

Reduced Immunity and Disease Resistance

A deficiency of selenium reduces the ability of the immune system to respond to various disease challenges that animals are exposed to. Selenium supplementation or administration has decreased death losses in calves and lambs fed diets low in selenium, even when clinical signs of white muscle disease were not apparent. In a 2-year study at New Mexico State University, providing supplemental selenium to ewes reduced lamb death losses from birth to weaning from 12.6 to 4.3% (Knott et al., 1983). Ewes in this study received feedstuffs that contained between 0.02 and 0.04 ppm of selenium. This level of selenium would be less than one-half of their requirement. Death losses in selenium-deficient lambs were largely attributed to weak lambs at birth, failure to nurse, and pneumonia. Injecting pregnant ewes at midgestation with selenium reduced lamb mortality rate from 31.3 to 13.8% in an Ohio study (Hamdy et al., 1968). Incidence of pneumonia was also reduced by selenium in this study. A 2-year study with beef cows and calves grazing pastures and being fed harvested feeds low in selenium (0.03 to 0.05 ppm) indicated that selenium administration reduced calf death losses.
from birth to weaning (Spears et al., 1986). These studies indicate that increased death losses due to selenium deficiency can easily go unnoticed under practical conditions.

Selenium status affects mammary gland health in lactating dairy cows. Mastitis is a major problem in high producing dairy cows. The inflammation to the mammary gland resulting from this disease increases the somatic or white blood cell count in milk. Low blood selenium concentrations were related to greater prevalence of intramammary infection in a study involving 32 dairy herds in Pennsylvania (Erskine et al., 1987). In a study involving 9 commercial dairy herds in Ohio, herds with high serum selenium concentrations had reduced rates of mastitis and low somatic cell counts (Weiss et al., 1990). In dairy cows fed feedstuffs low in selenium, intramuscular injection of selenium at 21 days prior to expected calving reduced the duration of clinical symptoms in cows with clinical mastitis by 46% (Smith et al., 1984).

Sanchez et al. (2007) evaluated the effects of selenium supplementation on udder health in dairy goats reared in a selenium-deficient area. Approximately 600 milking goats located on four commercial farms were used in this study. One-half of the animals on each farm served as controls and the remaining goats were injected subcutaneous with a slow-release selenium salt (barium selenate). Selenium administration reduced milk somatic cell counts and decreased the incidence of clinical mastitis from 18.2% to 4.0%.

METHODS OF PROVIDING SELENIUM TO GRAZING ANIMALS

A number of different methods have been used to supply supplemental selenium to animals grazing pastures. Injectable products containing selenium in the form of sodium selenite or barium sulfate have been effective in preventing selenium deficiency. Injectable forms of selenium are administered intramuscular or subcutaneous and must be given at critical times to prevent deficiency in areas where pastures are low in selenium. Critical periods for selenium administration would include prior to breeding and late gestation in sheep, and late gestation in cows. In some countries oral drenching with sodium selenium has been used to prevent selenium deficiency in sheep. Heavy pellets or soluble glass boluses that when inserted in the rumen release selenium over a period of weeks or months also have been used to provide selenium to grazing animals. An advantage of using injectable selenium or selenium containing boluses is that one can be assured that each animal receives a certain dose of selenium. A disadvantage of these products is that animals must be handled and restrained which increases labor costs unless animals are already being handled for other management practices such as vaccination.

Providing selenium in a salt-mineral mix is an effective and economical method of supplying selenium to grazing animals. Selenium can be provided in a block or loose trace mineral salt or a complete mineral that supplies some macrominerals such as calcium, phosphorus and magnesium in addition to salt and trace minerals. The natural appetite that animals have for salt will generally ensure adequate intake of selenium. Selenium is not included in all trace mineral salt blocks or loose salt mixtures. Therefore, it is important for livestock producers in selenium deficient areas to check the label of their trace mineral salt to ensure that selenium is present.

One of the first studies to evaluate trace mineral salt as a delivery system for selenium was conducted at the University of Wisconsin (Paulson et al., 1968). Approximately two weeks prior to lambing ewes were offered trace mineral salt or the same trace mineral salt supplemented with 132 ppm of selenium. This study was conducted on a farm where feedstuffs grown were low in selenium, and a high incidence of white muscle disease had traditionally been observed in lambs. Four of 13 lambs born to control ewes developed clinical signs of white muscle disease and almost all of their lambs were unthrifty in appearance from 4 to 8 weeks of age. Three lambs from control ewes died. None of the lambs from ewes receiving trace mineral salt supplemented with selenium died or showed signs of white muscle disease. During the first 8
weeks of life lambs from selenium supplemented ewes gained almost 8 pounds more weight than lambs from control ewes. Whanger and coworkers (1978) evaluated various methods of providing selenium to sheep receiving feeds severely deficient in selenium. They compared feeding selenium in a grain supplement daily, allowing ewes access to iodized or trace mineral salt containing 50 ppm of selenium, and administering 5 or 10 mg of selenium, twice during breeding and gestation, in a drench containing an anthelmintic. Providing selenium in either iodized or trace mineral salt was as effective in preventing white muscle disease as supplementing selenium in the grain or in the drench. Ewes receiving selenium in their salt or in their grain mix had similar blood selenium concentrations, while blood selenium concentrations were lower in ewes given selenium in the drench. Adding selenium to trace mineral salt has also been found to be an effective method of providing supplemental selenium in grazing cattle (Awadeh et al., 1998).

SELENIUM SUPPLEMENTATION IN HUMANS THOUGH SALT

In the 1970s a disease occurring in parts of China, characterized by damage to heart muscle and enlargement of the heart, was found to be caused by selenium deficiency. This condition known as Keshan disease occurred largely in children under 15 years of age and women of child bearing age, and was often fatal. Keshan disease occurred in certain rural areas of China where soil selenium concentrations were low and the people were dependent on locally produced food. In a large study with children, supplementation weekly with a sodium selenite tablet was found to prevent Keshan disease (Chen et al., 1980)

I attended a conference in China recently and found that selenium deficiency in humans was now being prevented by adding selenium to salt. Keshan disease has basically been eliminated by adding selenium to salt consumed by humans. It would be impossible to educate people in a country the size of China about selenium and the need for selenium supplementation. However, even in rural areas where people produce most of their food locally they usually purchase salt. Fortification of salt with selenium is an excellent way of providing widespread distribution of supplemental selenium to humans.

SUMMARY

Selenium is required by animals in low concentrations, generally between 0.1 and 0.3 ppm of diet dry matter. However, in many areas in the United States and around the world soils are low in selenium, and as a result pastures and other feedstuffs produced are deficient in selenium. A deficiency of selenium can result in high death losses in young ruminants due to white muscle disease or unthriftness. Selenium deficiency can also impair reproduction in sheep and cattle and reduce immunity causing animals to be more susceptible to disease, especially mastitis. In animals grazing pastures supplying selenium in a trace mineral salt or complete mineral mixture has proven to be an effective and economical method of preventing selenium deficiency.

LITERATURE CITED


