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Animal Nutrition Since 1912

SELENIUM AND VITAMIN E DEFICIENCIES OF LIVESTOCK

General

It has been commonly recognized that selenium and vitamin E are essential at every step of the production-marketing cycle, including breeding, gestation, lactation, survival of the newborn, the development of the offspring, and in the enhancement of the immune status of the animal.

Vitamin E and selenium are both micronutrients needed by food producing animals for normal health and development, which includes a major responsibility as antioxidants in animals.

Protective Role Of Selenium And Vitamin E

Biological cell membranes contain phospholipids which are subject to oxidative breakdown, leading to impairment of cell integrity and function. Vitamin E, because of its antioxidant action, decreases the amount of phospholipid breakdown into lipid hyperperoxides and hydrogen peroxide. Lipid hyperperoxides and hydrogen peroxide damage the integrity of cell membranes which lead to cell destruction and loss of function. Selenium, on the other hand, forms an integral part of the enzyme glutathione peroxidase (GSH-Px). Glutathione peroxidase is an important enzyme in destroying lipid hyperperoxides and hydrogen peroxide. The action of GSH-Px helps to maintain normal cell functions and prevents oxidative damage to cell membranes. All cells of the body, including those responsible for development of immunity, are protected from breakdown and loss of function by the integral actions of selenium and vitamin E.

Immunodeficiency results from destruction of immune cells which are highly dependent on a functional cell membrane. Selenium and vitamin E supplementation to animals enhances immunocompetence through improved cellular and antibody-mediated host defense systems.

Plants/Soil

When soils contain less than 0.2 ppm selenium, they are generally classed as deficient, in the sense that they will not, through plants grown on them, provide enough selenium to meet the requirements of animals. Results of a study of the selenium content of corn and soybean meal from 15 states over a period of two consecutive years and analyzed by five separate laboratories indicate that, in addition to a large variation in selenium among sources of corn and soybean meal, there also is considerable variation among laboratories in selenium analysis. Supplemental selenium in animal rations in areas of natural soil deficiency has resulted in significant increases in animal production, including elimination of deaths from selenium insufficiency.

Arguments against rigid definition of areas of selenium adequacy or deficiency may be made on the basis of shipment of livestock feeds from one area to another — a common practice. This shipment of feeds has been responsible for creating selenium deficiency in livestock in areas where it would not normally occur.

Selenium is absorbed and stored by plants in greater amounts in alkaline, well-aerated soils than in acid, poorly-aerated soils. Animals consuming forage or grain produced on these soils can develop blind staggers or alkali disease, characterized by emaciation, loss of hair, soreness, and sloughing of hooves, lameness, anemia, excess salivation, grinding of the teeth, blindness, paralysis, and death.

Another important factor in the generation of toxic levels of selenium is its accumulation, generally in range areas, by certain species of plants called "selenium indicators" or "selenium accumulators." These plants include types of Astragalus, a wild vetch; Stanleya, commonly called Prince's Plume; Machaeranthera, or woody aster; and Haplopappus, commonly called goldenweed. Some of

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these plants accumulate up to 1,500 ppm of selenium, while wheat grown in the same soil may contain only 6.3 ppm selenium. Stanleya has caused acute toxicity after snowstorms when it becomes the only vegetation visible.

Factors Leading To Deficiency

Factors leading to inadequate dietary selenium-vitamin E levels are often related to feeding levels, availability, and stability of these nutrients from natural sources. As grain production intensifies, soil selenium levels tend to decrease resulting in selenium deficient grains. Natural vitamin E sources (tocopherols) are unstable and are affected by heat, oxygen, moisture, mycotoxins, and oxidizing agents and fats added to the ration. Losses of natural vitamin E may vary from 10% to 60% in blighted, light test weight grain and artificially heat dried grain.

Intensified livestock production for faster gains, better feed conversion, and increased milk production may increase the metabolic demands for selenium-vitamin E. Intensified production may indirectly increase the needed levels of selenium-vitamin E by elevating stress. Other factors that may lead to inadequate vitamin E levels in feed or impaired ability of livestock to utilize selenium-vitamin E are restricted feeding practices and diseases affecting the gastrointestinal tract which may impair the intestinal absorption of these nutrients.

Signs Of Deficiency

Pigs

Mulberry heart disease (myocardial degeneration) is the most common condition associated with selenium-vitamin E deficiency in swine. The condition is associated with weakness and sudden death in rapidly growing pigs. Acute deaths are a direct result of cardiac insufficiency. Another less frequently observed condition in growing pigs is dietary liver necrosis (hepatosis dietetica). Dietary liver necrosis often goes unobserved in otherwise normal healthy-appearing pigs, only to be detected following an episode of sudden death. There appears to be an association between selenium-vitamin E deficiency and increased incidence of gastric ulcers in swine.

Cattle

Ruminants differ from other farm animals because the ruminal microbial populations and ruminal environment reduce availability and/or absorption of dietary selenium. Selenium-vitamin E deficiencies have been associated with an increased incidence of retained placenta, cystic ovaries, and mastitis in cattle, sheep, and goats. A deficiency in dairy animals may be associated with increased somatic cell counts and reduced milk production.

Selenium-vitamin E deficiency may also cause an increase in stillbirth rate or the birth of weak offspring. When stressed, selenium-vitamin E deficient feeder cattle are more susceptible to bovine respiratory disease complex and other related feedlot diseases.

Calves, Lambs, and Kids

The most common condition associated with selenium-vitamin E deficiency in calves, lambs, and kids is white muscle disease. White muscle disease in lambs is sometimes referred to as stiff lamb disease. These conditions result in weakness and/or stiffness associated with a bilateral degeneration of skeletal muscle groups and are recognized at necropsy as pale or white streaks in the affected muscle groups. White muscle disease occurs most often in young nursing animals. Symptoms vary depending upon which muscle groups are affected but usually include stiffness, weakness, lameness, inability to rise, and difficulty in breathing. White muscle disease is more prevalent in calves, lambs, and kids than in pigs or foals.

Horses

Conditions associated with selenium-vitamin E deficiency in horses include azoturia, myositis, and polymyositis (typing up syndrome). Supplemental selenium-vitamin E has also been used to treat infertility in mares, muscular weakness in foals, and to increase performance.

FDA Selenium Feeding Guidelines

The Food and Drug Administration has established feeding guidelines for additive selenium levels in rations. Those levels are not to exceed 0.3 parts per million in complete feeds for swine, sheep, and cattle.

Summary

With the exception of specific selenium-vitamin E deficiency diseases (white muscle disease, stiff lamb disease, mulberry heart disease, hepatosis dietetica, and azoturia-myositis), many of the losses incurred in animal production are associated with impaired immune function resulting in a higher level of disease and reproductive inefficiencies.