What is it?
White muscle disease (WMD) is a degenerative muscle disease found in all large animals. It is caused by a deficiency of selenium and/or vitamin E. Generally, it is not known which. Selenium (Se) deficiency is associated with selenium deficient soils and the inadequate uptake of selenium by forages grown on these soils. Certain areas of the U.S., including the Northeast, are considered low in selenium levels. Selenium deficiency occurs when the soil contains less than 0.5 mg Se/kg of soil and locally harvested feeds contain less than 0.1 mg Se/kg of feed.

Vitamin E deficiency is independent of soil type and more closely reflects forage quality. Grazing animals usually consume adequate amounts of vitamin E. This is because fresh legumes and pasture are good sources of vitamin E, whereas silage, oil seeds, root crops, cereal grains, and dry hays tend to be poor sources of vitamin E. Prolonged storage of feedstuffs results in a degradation of Vitamin E activity, as much as 50% per month.
In addition to WMD, selenium and vitamin E deficiencies can produce symptoms of ill thrift and reproductive losses: lower conception rates, fetal reabsorption, dystocia, retained placenta, reduced milk production, and reduced semen quality. They can cause poor rate of growth or ill thrift in young lambs throughout the growing period. Sheep consuming selenium-deficient diets produce low wool yields and have increased incidence of periodontal disease. Selenium and vitamin E also play key roles in the animal’s normal immune response.

**Symptoms**

All breeds of sheep and goats are susceptible to WMD, and the condition may develop under extensive or intensive management systems. WMD is most commonly found in newborns or fast growing animals. Kids are believed to be more susceptible than lambs, possibly because they have a higher requirement for selenium. The disease can affect both the skeletal and cardiac muscles.

When the skeletal muscles are affected, symptoms vary from mild stiffness to obvious pain upon walking, to an inability to stand. Lambs/kids may tremble in pain when held in a standing position. A stiff gait and hunched appearance are common. Affected lambs/kids may remain bright and have normal appetites, but eventually they become too weak to nurse. When the problem occurs in newborns, they are born weak and unable to rise. Sudden exercise may trigger the condition in older lambs and kids.

When the disease affects the heart, the animal shows signs similar to pneumonia, including difficult breathing, a frothy nasal discharge (may be blood stained), and fever. The heart and respiratory rates are elevated and often irregular. Skeletal and cardiac muscle disease may occur concurrently.

Selenium deficiency can be confirmed by measuring selenium levels in whole blood or tissues. A diseased animal will have less than 0.04 ppm of selenium in its blood. Breeding ewes require more selenium, and their blood levels should be over 0.5 ppm. At necropsy, the muscles of affected animals appear paler than normal and may show distinct longitudinal striations or a pronounced chalky appearance due to abnormal calcium deposition.
**Treatment**

Treating the heart form of WMD is usually ineffective and those that survive often do not thrive because of the residual cardiac damage. The muscle form of the disease can be successfully treated with supplemental selenium and/or vitamin E. Producers need to follow label directions carefully when using selenium for treatment. The concentrations of selenium (per ml) vary greatly with each product, and excessive or repeated injections can result in selenium toxicity and possibly death.

The commercially available selenium/vitamin E product(s) commonly used in the U.S. do not contain therapeutic levels of vitamin E. Additional vitamin E may need to be provided through an injection of vitamin E alone or through oral vitamin E products. Affected animals usually respond favorably to a single treatment of vitamin E and/or selenium in 24 hours, though recovery may not be complete, depending upon the severity of the condition. Animals which do not respond to treatment may be treated a second time. Treatment should not exceed two doses.

**Prevention**

Deficiencies occur when animals are fed poor-quality hay or straw or lack access to pasture. High concentrations of other minerals (e.g. calcium, sulfur, copper) and feed contaminants (e.g. nitrate, unsaturated fats, sulfates) may decrease absorption of selenium in the small intestine. Diets high in polyunsaturated fatty acids or deficient in Vitamin C and/or beta-carotene increase vitamin E requirements, whereas adequate dietary selenium is almost completely protective against vitamin E deficiency.

WMD can be prevented by supplementing the diet of susceptible animals with selenium and vitamin E. Since it occurs mostly in lambs and kids whose mothers were fed a selenium-deficient diet, supplementation of pregnant animals helps reduce disease in newborns. This is because selenium is transferred from dam to fetus across the placenta and also is present in the colostrum. While not much Vitamin E is transmitted across the placenta, colostral levels of Vitamin E increase with ewe/doe supplementation.

While pasture, hay, grain, and other supplements can be analyzed to determine the amount of selenium to be added to supplemental feeds, it is important to note that selenium supplementation is controlled by law. For sheep, selenium can be supplemented in a complete ration at a level up to 0.3 ppm, in a feed supplement so that the intake of selenium does not exceed 0.7 mg per head per day, and in salt/mineral mixes at 90 ppm as long as total daily consumption does not exceed 0.7 mg/head/day. Selenium supplementation of feed has not been approved specifically for goats.
Injectable selenium compounds are available to prevent WMD in at risk-animals; however, injections are a poor alternative compared to routinely providing adequate selenium and vitamin E in the diet. Ideally, the total diet for sheep and/or goats should contain 0.10 to 0.30 ppm of selenium.


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