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# Selenium Deficiency in Cattle

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## SUMMARY

Our knowledge of selenium deficiency as a cause of animal disease has increased since 1973, when the role of selenium (Se) in glutathione peroxidase was discovered.<sup>1</sup> Glutathione peroxidase (GSH-Px) works with vitamin E in the cytosol to prevent cell membrane damage. Selenium deficiency causes nutritional myopathy (white muscle disease) in young calves and yearlings. It is also implicated in a sudden death syndrome in calves less than three months old from acute myocardial degeneration. In adult cattle it is associated with muscle disease, retained placentas, and infertility. Se-deficiency diseases have been reported in Canada, Great Britain, New Zealand, Sweden, and the United States.<sup>2</sup>

## BIOLOGICAL AND PHYSIOLOGICAL ROLE OF SELENIUM

Selenium, in conjunction with vitamin E, prevents cell membrane damage due to excess organic hydroperoxide and hydrogen peroxide in the cytosol. These peroxides can react with and destroy cell membrane components by oxidation. While vitamin E acts directly, selenium works as part of GSH-Px.<sup>2</sup> This enzyme catalyzes the following reaction: 3, 4 Reduced Glutathion + H<sub>2</sub>O<sub>2</sub>  $\xrightarrow{\text{GSH-Px}}$  Oxidized Glutathione + H<sub>2</sub>O.

Through its metabolism of hydroperoxides, GSH-Px may be involved with the following: the synthesis of various prostaglandin derivatives; the normal function of lymphocytes, neutrophils, and macrophages; and maintenance of erythrocyte integrity.<sup>2,5,6,7</sup> Moreover, GSH-Px prevents sarcolemma damage and, therefore, muscle fiber damage.<sup>8</sup>

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A nonselenium-dependent GSH-Px is also present in tissue. In domestic animals the amount of this enzyme varies as well as its tissue location. In bovine, the liver, lungs, adrenal glands, testes, and kidneys contain both enzymes. However, in the spleen, erythrocyte, brain, thymus, adipose tissue, cardiac muscle, and skeletal muscle, only Se-dependent GSH-Px is found.<sup>9</sup> The presence of only Se-dependent GSH-Px helps to explain the location of lesions found in Se-deficient animals.

## AVAILABILITY

Selenium in soil is found in two forms: selenate and selenite. The selenite form is less available for plant uptake and is found in acidic, poorly drained soils. Plants grown on this type of soil are likely to be Se-deficient. Moreover, good weather and the proper fertilizer content of soil provide for a lush growth, further reducing Se content by dilution among the abundant growth.

Surveys of crops grown in the United States provide data needed to map areas of Se-deficiency. Deficient areas in the United States include the southeast, northeast, midwest, and far northwest.<sup>10</sup> These areas of low plant selenium (less than .05 ppm) correlate closely with the occurrence of Se-deficiency diseases.

In animal diets the availability of Se for absorption is important, since no mechanism for its storage has been recognized. Animals are able to utilize both the inorganic salts (selenite, selenate) and organic forms (plant, animal) of Se.<sup>11</sup> The organic form of plant origin has the best biological availability. However, inorganic selenite is the form most often used for supplementation, since it is least expensive.

Selenium availability is antagonized by high, nontoxic levels of certain elements in the diet. These include copper, silver, tellurium, zinc,

arsenic, and mercury.<sup>12,13</sup> Even though this antagonism has only been shown experimentally, it should be considered when Se-deficiency occurs in heavily supplemented herds.

### CLINICAL SIGNS

Selenium deficiency affects cattle of all age groups. In calves under six months of age, three syndromes of myopathy appear, depending on which muscle group is affected.

With cardiac involvement, sudden death can occur in calves up to two months of age and is associated with a period of excitement usually precipitated by feeding. On four farms in England, calves exhibited grunting and then died within one minute showing no other signs.<sup>14</sup> All herd mates had low selenium status, and no further deaths occurred after Se supplementation.

If the diaphragm and intercostals are the main muscles involved, the clinical signs simulate pneumonia. Dyspnea and abdominal breathing are observed.

When mainly skeletal muscle is involved, lameness, tremors, and symmetrical weakness are present.<sup>15</sup> The muscles of the hind limbs and back are usually involved. In acute cases the muscles are swollen and hard if palpated. However, in chronic cases they are hard, rubbery and somewhat atrophied. Since muscle involvement is usually generalized, the appearance of the preceding syndromes can overlap.

Another syndrome in calves, seemingly unrelated to myopathy, is a Se-responsive unthriftiness in dairy and beef calves.<sup>10</sup> This syndrome varies from a subclinical growth deficit to a sudden loss of condition, and is commonly associated with a profuse diarrhea of undetermined cause.

In yearlings and adult cattle, a nutritional myopathy with myoglobinuria is associated with Se-deficiency. It is more common in yearlings and is induced by stress due to transportation, bad weather, or turning out to pasture in the spring.<sup>8,16</sup> These factors, in conjunction with Se-deficiency, likely precipitate the problem. The main clinical signs are locomotor disturbance and myoglobinuria. Also in adult cattle, two reports describe a post-parturient downer cow syndrome resembling milk fever.<sup>16</sup> The affected cows were stiff, reluctant to walk, and recumbant in severe cases.

Other problems possibly linked to Se-deficiency in cows include higher incidence of retained placenta, abortion, and birth of weak

calves. Studies done in England, North Carolina, and Ohio indicate that Se/vitamin E supplements substantially decreased the incidence of retained placenta. These cattle were from herds with a history of retained placenta problems and were eventually found to have diets deficient in Se.<sup>17,18,19,20,21</sup> Other recent studies evaluated the effect of Se/vitamin E supplementation of dry cows on decreasing the incidence of retained placenta; no prevention of incidence could be shown.<sup>22,23</sup> Therefore, the role of Se/vitamin E deficiency in causing retained placenta is questionable.

### CLINICAL PATHOLOGY

A few reliably accurate methods may be used to determine the Se status of cattle. For samples of whole blood, tissues, and feed, a direct analysis for Se content is widely used. In whole blood, normal levels are 0.8–2.5 umol/liter, and clinical signs occur at levels below 0.4 umol/liter.<sup>26</sup> For liver and kidney samples, levels of below 3.0 and 30 umol/kg dry matter, respectively, indicate Se-deficiency. The liver is preferred for analysis, since in animals fed Se-deficient diets, the kidneys retain Se and therefore give falsely elevated values.<sup>27</sup> For a feed sample, a level below 100 ppb is considered Se-deficient.<sup>8</sup>

Indirect indicators of Se status are glutathione peroxidase and aspartate aminotransferase enzyme activity, which decrease and increase, respectively, in Se-deficient animals. GSH-Px levels are probably less affected by the daily diet than blood selenium, since GSH-Px is incorporated into erythrocytes during erythropoiesis.<sup>8</sup> Therefore, GSH-Px might be a more constant value and more reliable. Normal values for GSH-Px activity are 3,000 Units/ml of blood or 30 Units/mg of blood. Simple, qualitative tests for GSH-Px have been developed for field use. They are reported to be rapid, relatively inexpensive, and able to differentiate inadequate Se status of cattle.<sup>28,29</sup>

Two other serum enzymes are used to determine muscle damage. The first, creatine phosphokinase, is specific for myodegeneration. Levels for this enzyme rise quickly to 1,000–5,000 mU/ml, but can drop to normal levels in 3–4 days if there is no ongoing myodegeneration. With a second enzyme, glutamic oxaloacetic transaminase, the levels will rise and drop much slower, but this enzyme is not specific for myodegeneration. Overall, the moni-

toring of both these enzymes can indicate if the myodegeneration is ongoing or regressing.

### GROSS AND MICROSCOPIC LESIONS

The basic lesion associated with Se-deficiency is a hyaline degeneration of striated muscle that is usually more widespread than the gross lesions indicate. Grossly, whitish-yellow discolorations of muscle fibers are well defined in their long axis, but vary in distribution in the muscle bundle. These affected fibers are inelastic, friable, and dry, but may become chalky in texture if calcified. In skeletal muscles the gross lesions show bilateral symmetry and appear in the hind limbs, back, intercostal muscles, and the diaphragm.

Gross lesions in the heart appear anywhere in the myocardium, but frequently have an annular distribution around the left ventricle.<sup>15</sup> Unlike skeletal muscle, the lesion margins are irregular and undefined. Frequently, only a slight pallor appears grossly, while myocardial necrosis is demonstrated microscopically.<sup>14</sup> In severe cases of acute myocardial degeneration, pulmonary edema, hydropericardium, liver congestion, and a slight ascites are present.

In all striated muscle, the histological appearance is hyalinization with patchy segments of granular and liquefactive necrosis. This is commonly referred to as "Zenker's necrosis." Also, some fibrosis of the cardiac muscle occurs, which replaces damaged muscle fibers.

### DIAGNOSIS

Diagnosis is based on clinical, postmortem, and biochemical observations. Differential diagnoses to be considered in calves are pneumonia, other nutritional deficiencies, and causes of sudden death. Se-deficiency should be considered in cows if the herd has high incidence of retained placenta and fertility problems, or in heifers if abortion is prevalent and no etiology can be found.

A presumptive diagnosis is sometimes made when Se supplementation corrects the problem, but a definitive diagnosis must be based on Se levels of blood or liver.<sup>26</sup> It appears, however, that GSH-Px activity of blood might replace direct analysis, since it is reliable, quicker, and more useful in the field.<sup>28,29</sup>

### TREATMENT AND PREVENTION

The various manifestations of Se-deficiency described above can be treated or prevented by supplemental selenium in mineral mixes, pe-

riodical injections or oral dosing with Se salts, the use of Se-supplemented salt licks, or possible addition of selenium to soil to raise the forage content.

For intramuscular, subcutaneous, or oral routes, a dosage of 3–6 mg/100 lbs. B.W. of elemental Se is recommended.<sup>26</sup> This is the sodium or potassium salt, or selenite or selenate. In severely deficient areas, treatment may be repeated at one to three month intervals, as necessary. A common treatment regime used in studies for retained placenta prevention is 50 mg Se and 680 IU vitamin E intramuscularly at three to four weeks prepartum.<sup>23</sup>

Careful mixing and monitoring of levels in feed is necessary if supplementation is attempted, since toxic levels occur at 30–50 times the requirement levels.<sup>23</sup> Levels for dry cow ration are 0.23–0.92 mg/cow/day during the last four to eight weeks of gestation.

The use of Se-supplemented salt licks and slow release rumen pellets is frequently described in British literature. Levels recommended are 2.5–2.6 mg Se/kg of salt in the salt licks and 5% in the rumen pellets.<sup>31</sup>

Addition of selenium compounds to pasture or crop land is feasible, but whether it is efficient and cost-effective is questionable. Addition to pasture at 30–60 gm/acre is suggested. The protective effect is variable, but may last years. Since the uptake by plants is variable, regular monitoring of feed levels is recommended.

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