

Malnutrition and Trace Element Deficiencies

Trace Elements

Deficiencies of mineral substances have significant effects on metabolism and tissue structure. Trace elements are known as micro minerals and involved in the body's blood production, the structure of the hormones, vitamin synthesis, the formation of the enzymes, and are responsible for the integrity of the immune system and regulation of the reproductive system. Enzymes that become functional due to trace elements are present in all organisms, trace element deficiencies and imbalances have been reported to cause reproductive disorders and inadequacies in immune response. In female animals, especially in the postpartum period, the trace element support required for the regeneration process and milk yield of the endometrium must be performed appropriately. Excess amounts of minerals should be avoided; it should not be forgotten that the minerals that are given too much cause problems like the ones given less. In contrast, manufacturers think that excess amounts will be more useful and often do not know that it causes problems.

Trace element deficiencies generally depend on the soil structure and the geography of the breeding region. The amount of a particular mineral in any plant consumed by animals is dependent on the soil on which it grows, its concentration in the soil, the type of the plant and environmental factors in the developmental period. On the other hand, one way feeding of animals may cause mineral deficiencies. Selenium, cobalt, manganese, copper and iodine deficiencies are an important problem in various regions of our country.

Trace elements are effective on reproduction on their own and as well as depending on their interaction with each other. They show their effects by affecting each other and the absorption mechanism. Therefore, they must be present in certain proportions when adding in rations.

Trace elements in rations are generally used as inorganic salt form, sulfate form, oxide form and chlorite form. Organic forms are reported to increase feed conversion, growth, reproduction and immune response. These effects are due to their higher bioavailability compared to their inorganic forms. It was found that the addition of copper, zinc, manganese, iron, selenium and magnesium reduced uterine infections, embryonic deaths and endometrial injuries and increased postpartum involution and tone in pregnant horn.

- NAME OF THE DISEASE:

SELENIUM-VITAMIN E deficiency

Selenium deficiency is seen together with vitamin-E deficiency. It is an important reproductive trace element that causes muscular dystrophy in kids, calves and lambs. This trace element deficiency is usually due to the lack of its amounts in rations; the most important factor in this is the soil structure of the geography where the breeding is made.

-SYMPTOMS:

Vitamin E and selenium (Se) deficiency, and their antioxidant effects due to lack of free radicals play a role in the etiology. White muscle disease is a nutritional disease characterized by hyaline degeneration of the heart and skeletal muscles, causing economic losses in different parts of the world. It can cause weight loss, low yield and death in lambs, kids and calves. In terms of

reproductivity, selenium is important for fetal and embryonic development, milk yield, metritis and ovarian cyst formation.

-PREVENTION:

Vitamin E is one of the important fat-soluble vitamins. It stabilizes biological membranes and protects the body against lipid peroxidation formed by free radicals. Green fresh pastures are the best source of vitamin E, especially pregnant animals should be given rations formed with green grass and cereals. Selenium must also be administered to animals in appropriate doses. In addition, lambs should be avoided rotten feed containing large quantities of unsaturated fatty acids.

-DIAGNOSIS:

The problem is specific to the region and there may be sudden or slow findings. In cases of sudden effects, animals may die without showing symptoms. In some calves, sudden onset of blindness, severe breathing difficulties and lying may be seen. Animals do not even stand on the chest with help. Death can occur within 6-12 hours with the onset of symptoms. In slow progressive cases, the sick animals lie on the rib cage. They want to get up, but fall. Muscle tremors and weakness are noticeable in those who can get up. Body temperature is normal. In addition to clinical findings, the disease is diagnosed by serum selenium and vitamin E quantity analysis and AST, CPK, LDH enzyme activities, which are the parameters of serum biochemistry.

-TREATMENT:

The course of the disease is favorably improved by administering oral or per oral preparations containing combinations of selenium and vitamin E. Trace element and vitamin premixes that can provide supplementation in ration are used.

-NAME OF THE DISEASE:

ZINC-VITAMIN A deficiency

Zinc plays an important role in the recognition and continuity of pregnancy by the mother by taking part in the genetic structure in the stages of cell division.

Vitamin A is an essential, fat-soluble vitamin in ruminants. It is a necessary vitamin for cell replication and epithelial integrity. It plays an important role in tight junction between cells. It acts as an antioxidant in the body. Many enzymes involved in vitamin A metabolism are regulated due to zinc. Therefore, growth retardation due to indirect vitamin A deficiency is seen in animals with zinc deficiency and the mechanism of epithelial protection is affected.

-SYMPTOMS:

Energy imbalance due to decrease in feed consumption in vitamin A deficiency has a negative effect on fertility. It also increases susceptibility to infections by weakening the cellular immunological mechanism. Since zinc is a very important element for normal growth and health in animals, it can lead to various offspring abnormalities (malformations) and decrease in mating functions.

Since zinc has a very important role in fertilization as well as in the attachment of the embryo to the uterus, embryonic development and pregnancy process, death and hereditary defects may occur in the embryo. In addition, deficiency in the uterine muscles can affect the estrogen density and cause delays in delivery. Visual disturbances, weight loss, neurological and dermatological problems are among the other findings.

-PREVENTION:

In areas where zinc deficiency is seen, it is important to add zinc and vitamin A in appropriate proportions to the rations of the animals. Oral or injection applications before breeding periods are recommended.

-DIAGNOSIS:

In addition to unsuccessful pregnancy and childbirth processes showing stubbornness without any cause of infection, findings such as increased skin (dandruff, folding, thickening etc.) and nail problems, blown offspring births and swelling of testes in sheep should be considered.

In addition to clinical findings, the diagnosis of the disease is made by analyzing serum zinc and vitamin A amount and ALP enzyme activities from serum biochemistry parameters.

-TREATMENT:

The course of the disease is favorably improved by administering oral or per oral preparations containing zinc and vitamin A supplements. Trace element and vitamin premixes that can provide supplementation in ration are used.

-NAME OF THE DISEASE:

KOBALT - Vitamin B12 deficiency

As long as there are cobalt sources, the ruminants are different from other animal species because of the microorganisms in the rumen can synthesize vitamin B12. Since cobalt can be stored in ruminants to a limited extent, cobalt needs of the animals must be met continuously in order to avoid vitamin B12 deficiency. In other words, deficiency of vitamin B12 in animals is due to insufficient cobalt intake. Cobalt is taken from meadow and soil. In deficiencies, failure of protein mechanism, nervous system effects and reproductive problems may occur.

-SYMPTOMS

In case of symptoms such as abnormalities in the blood (anemia), coarseness of the skin and hair, dermatitis, coordination disorders, offspring, weak calving, pica, loss of appetite and chronic growth retardation, the disease should be suspected.

-PREVENTION:

The cobalt deficiency in the soil causes the deficiency of the plants in terms of this element. Addition of cobalt to ration is important in preventing problems that may arise due to B12 deficiency.

-DIAGNOSIS:

Diagnosis of the disease is made by clinical findings such as unexplained loss of appetite, weight loss, cachexia, anemia characterized by wilt in mucous membranes, decrease in wool quality in sheep, infertility and intense tear discharge in the last stages of the disease, ataxia, as well as serum cobalt and vitamin B12 quantity analyzes.

-TREATMENT:

The course of the disease is favorably improved by the regular addition of cobalt to ration and the use of oral or per oral preparations containing vitamin B12 supplementation when the sudden symptoms of the disease occur. Trace element and vitamin premixes that can provide supplementation in ration are used.

-NAME OF THE DISEASE:

COPPER deficiency

Copper acts as an element of connective tissues, blood and enzyme systems in the body. Copper has to be taken from the outside and its deficiency is related to the feeds grown in the poor areas of copper. This element is found in the brain, kidney, heart, hair and wool in high concentrations. Copper is present in the blood attached to ceruloplasmin at a rate of 90%, while 10% of it found in erythrocytes. It has related with iron and is responsible for the conversion of iron to hemoglobin, formation of white blood cells and activities.

-SYMPTOMS:

Lack of copper leads to hair and fleece disorders, growth retardation, anemia, diarrhea, joint disorders and fertility disorders. These fertility disorders are decreases in conception rate, early embryonic deaths and retention secundinarum cases. It has an effect on the development of the central nervous system, especially during embryo development. As it is known, neonatal ataxia cases are seen in offspring born in regions with copper deficiency. Usually the occurrence of diseases coincides with the spring and summer months.

-PREVENTION:

Copper deficiency can be caused by the lack of copper in the rumen due to the presence of

cadmium, molybdenum, zinc, iron, lead and sulfur, as well as copper deficiency in the pasture. In the long term, it is important to ensure that sufficient copper is added to the feed mix of the animals to avoid recurrence of the problem.

-DIAGNOSIS:

Losing control of hind legs and fall to the ground and sit like dog sitting (ataxia) are important symptoms for diagnosis. In addition to these problems with walking, enlarged joints, walking on nail tips due to flexion tendon pulls and partial paralysis of lambs are important clinical signs of the disease.

-TREATMENT:

The problem can be prevented by using commercial preparations in the early stages of the disease, but if the disease is not noticed and progressed, it can cause irreversible damage, especially in the nervous system and circulatory system. In the areas where copper deficiency and related diseases occur, urgent intervention is recommended for the use of commercial preparations containing copper orally or peroral. It is important to keep copper licking stones all the time, to sprinkle copper on pasture soils and to provide copper support in the second half of the pregnancy, especially in areas where the disease leads to large yield losses.

-NAME OF THE DISEASE:

MANGANESE deficiency

Manganese is involved in the activation of many enzyme systems. It is important for the synthesis of cholesterol. Thus, they participate in the release mechanism of steroid hormones. It supports the production of progesterone by participating in the synthesis of cholesterol and therefore the Mn level in the advanced corpus luteum is maximized. It plays a role in the sensitization of the uterus by preparing for estrogen. Manganese is transmitted from the mother to the offspring through the placenta and plays a role in bone formation during organogenesis by participating in the structure of chondroitin sulfate which is important in the bone formation and mucopolysaccharides that form the bone matrix.

-SYMPTOMS

Deficiency causes cycle irregularities, calm oestrus, increases in ovarian cysts and decreases in embryo retention rates. It may also cause high abortion rates. Bone deformations are seen in offspring as it is effective in the development of bones.

-PREVENTION:

Manganese deficiency also occurs as a result of overconsumption of beet pulp and corn silage. The resorption of manganese from the plants is directly related to the soil pH values. When the pH values of the soil are high, decreases in Mn values occur. In order to prevent the disease,

manganese supplements should not be ignored in case of the mentioned feeding programs.

-DIAGNOSIS:

The foremost signs of manganese deficiency especially in cattle are infertility and abortion. In addition to these, symptoms like birth anomalies, growth retardation in calves and lambs, hunchback, drying of the skin, bending of the joints, shortness of the bones, undetermined infertility in females should suggest manganese deficiency. The definitive diagnosis is made by determining the serum manganese values.

-TREATMENT:

It is recommended to use manganese-containing commercial preparations which can be given orally or peroral in cases where urgent intervention is required in areas where copper deficiency and related diseases occur. In manganese supplementation, it is necessary to pay attention to the dose given in order not to cause different element deficiencies since the excess manganese given to the animals affects the cobalt and zinc absorption negatively.

Sample Selection and Sending Method for Trace Element Analysis

Blood serum is used for the determination of mineral and vitamin deficiencies. If the offspring is alive after the abortion, the blood should be taken from the offspring and if it is dead, the blood taken from the mother should be sent to the laboratory. Blood around 5-6 cc should be taken from the V. jugularis to the tubes without anticoagulant and serum should be separated and sent. Serum samples should be at least 2 ml, not mixed with blood or hemolysed. The label with the animal's information (ear tag number or name etc.) must be attached on the tubes where the serum is placed. Serum should be shipped in a suitable packaging in cold chain. If serum separation conditions are not available, the whole blood should be delivered to the laboratory as soon as possible while maintaining the cold chain conditions. Whatever deficiency is suspected, the requested analysis should be written in the Morbid Substance Protocol (such as Cu, Zn, Se etc.) or the information of the disease in the animal should be clearly stated in the protocol. It should be kept in mind that the serum composition changes if the serum of the blood is not separated.