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EFFECTS OF MINERAL DEFICIENCY ON THE HEALTH OF YOUNG RUMINANTS

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Abstract

Twenty-two elements have been identified as essential to the growth and health of animals. They include 7 macroelements and 15 microelements, which play four key functions in the body: structural, physiological, catalytic and regulatory. Mineral deficiencies can result from low quality feed, impaired absorption or assimilation in the body or increased demand for minerals during intensive growth, pregnancy and lactation. Mineral-deficient feed and diets with an unbalanced mineral content impair the growth and development of young animals, decrease appetite, lower nutrient absorption, decrease immunity and increase susceptibility to contagious diseases. This paper discusses the consequences of low levels of macronutrients and micronutrients that are required for the optimal growth of calves, lambs and kids, including calcium, phosphorus, magnesium, selenium, cobalt, iron, zinc, copper, sodium, potassium and chloride.

Key words: calves, lambs, kids, microelements, macroelements, deficiency.

WPŁYW NIEDOBORU PIERWIASTKÓW MINERALNYCH NA ZDROWIE MŁODYCH PRZEŻUWACZY

Abstrakt

Przyjmuje się, że 22 pierwiastki są niezbędne do prawidłowego funkcjonowania organizmów zwierzęcych. Należy do nich 7 makro- i 15 mikroelementów, które w organizmie pełnią 4 zasadnicze funkcje: strukturalną, fizjologiczną, katalityczną oraz regulacyjną. Przyczyną niedoboru pierwiastków mineralnych może być niewłaściwa jakość paszy, nieodpowiednia absorpcja lub asymilacja pierwiastków w organizmie lub zwiększone zapotrzebowanie zwierzęcia na składniki mineralne (okres wzrostu, ciąża, karmienie potomstwa). Niedobór pierwiastków mineralnych w paszy lub ich nieodpowiedni stosunek przyczynia się w głównej mierze do: zahamowania wzrostu i rozwoju młodych zwierząt, zmniejszenia apetytu oraz gorszego wykorzystania składników pokarmowych lub spadku odporności i większej zapadalności na choroby zakaźne. Celem artykułu jest omówienie skutków niedoboru najbardziej istotnych makro- i mikroelementów, takich jak: wapń, fosfor, magnez, selen, kobalt, żelazo, cynk, miedź, sód, potas oraz chlor, niezbędnych do prawidłowego rozwoju cieląt, jagniąt i koźląt.

Słowa kluczowe: cielęta, jagnięta, koźlęta, mikroelementy, makroelementy, niedobór.

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INTRODUCTION

The advancements made in molecular biology in the late 20th century contributed to our understanding of the functions and transformations of mineral elements in living organisms and the complex mechanisms responsible for the transport of trace elements across cell membranes (Mayland, Shewmaker 2001). The minerals found in body tissues and fluids of adult animals originate mainly from exogenous sources and constitute approximately 4% of the animal's body weight. Subject to their content in the body, minerals are generally divided into macroelements with concentrations higher than 50 mg kg^{-1} BW and trace elements or microelements with concentrations below 50 mg kg^{-1} BW. A list of 22 elements essential to animal life was developed in 1981. It included 7 macroelements: calcium, phosphorus, potassium, sodium, chloride, magnesium and sulphur, and 15 microelements: iron, iodine, zinc, copper, manganese, cobalt, molybdenum, selenium, chromium, tin, vanadium, fluoride, silicon, nickel and arsenic (Bednarek, Bik 1994). Macronutrients and micronutrients play four key roles in the body: structural, physiological, catalytic and regulatory. The structural function involves elements that build organ and tissue structures (calcium, magnesium, phosphorus, silicon in bones and teeth, phosphorus and sulphur in muscle proteins). The physiological function is responsible for the supply of electrolytes to body fluids and tissues in order to regulate osmotic pressure, maintain the acid-base balance, regulate membrane permeability and nerve impulse transmissions (sodium, potassium, chloride, calcium, magnesium). The catalytic role of minerals is probably the most important function. Macronutrients and micronutrients act as catalysts in enzyme and endocrine systems; they can act as coenzymes to initiate enzyme and endocrine functions, and they can constitute integral and specific structural elements of metalloenzymes and hormones. In living organisms, mineral elements are also responsible for cell replication and differentiation. Zinc influences transcription, whereas iodine is a component of thyroxine, a hormone responsible for thyroid function and energy processes (Suttle 2000).

In ruminants, mineral deficiency can impair or even inhibit metabolic pathways required for normal body function, and produce clinical symptoms of different intensity. Severe macroelement or microelement deficiencies are manifested by symptoms corresponding to the function of the deficient element in the body, thus contributing to an accurate diagnosis of the health problem. In a minor deficiency, the symptoms are non-specific, often transient and difficult to diagnose due to low intensity. Mineral deficiency generally leads to impaired immunity, inhibited growth, reproductive disorders and lower productivity in animals. In ruminants, subclinical deficiencies are observed more frequently than severe ones, hence the interest in the role of minerals in animal production on behalf of breeders, feed manufacturers, veterinary practitioners as well as researchers.

Mineral deficiencies can result from low quality feed, impaired absorption or assimilation of minerals in the body or increased demand for minerals during intensive growth, pregnancy and lactation.

CALCIUM AND PHOSPHORUS

Calcium (Ca) is the most abundant mineral in the body, and 99% of this element is found in bones. Calcium is essential to key body processes such as ossification, blood coagulation, cardiac rhythm control, cell membrane permeability, nerve and muscle excitation, activation and secretion of hormones and enzymes (Soetan et al. 2010). Phosphorus (P) is the second most abundant mineral in animals, and 80% of this element is located in bones and teeth. Phosphorus works together with calcium in ossification processes, and is a constituent of energy rich compounds and nucleic acids. Calcium and phosphorus levels in the body are regulated by vitamin D3 and parathyroid hormones: parathormone and calcitonin (Sobiech et al. 2010). Phosphorus deficiency, which may be exacerbated by low vitamin D levels, contributes to changes in the skeletal system and development of rickets in young animals. In most cases, vitamin D deficiency results from a poor diet and insufficient exposure to sunshine (UV radiation induces the conversion of vitamin D into active metabolites). Phosphorus deficiency is caused by a diet low in this element, which distorts the Ca:P ratio (1.5-2:1), inhibits Ca and P absorption and their transport to the skeletal system (Iqbal et al.2005). According to the literature, cattle are more sensitive to phosphorus deficiency than sheep (Dittmer, Thompson 2010). Defective bone mineralization accompanied by inhibited necrosis of cartilaginous cells and osteoblast penetration leads to the loss of bone elasticity and bone deformation. Another consequence is the compensatory proliferation of cartilaginous tissue and inhibited bone elongation. Fluorosis can also contribute to rickets. Fluoride stimulates bone formation, increases the demand for calcium and aggravates vitamin D deficiency (Kurland et al. 2007).

Inhibited growth and allotriophagy are among the earliest symptoms of rickets. The disease may be accompanied by the characteristic thickening of epiphyses and circumferential rib segments, which leads to the formation of the rachitic rosary. In animals, rickets may also result in curvature of legs, impaired mobility, lameness and recumbence. Inhibited dental development and enamel loss are also observed (Dittmer, Thompson 2010).

MAGNESIUM

Magnesium (Mg) is the second most abundant intracellular cation in mammals after potassium. Bones and muscles are the main magnesium

pools in the body. Magnesium plays vital roles in nearly all physiological processes and participates in many cellular metabolic pathways. It activates nearly 30 enzymes and participates in the metabolism of carbohydrates, nucleic acids and proteins. Magnesium stabilizes DNA structure and influences RNA transcription as well as the formation of ribosomal subunits. The presence of Mg^{2+} ions is required in all processes involving ATP. The ability of magnesium to stabilize cell membranes is one of its most important functions (Soetan et al. 2010). Magnesium and calcium remain in a dynamic equilibrium, and a higher intake of magnesium than calcium can stilt the bone growth (Zimmermann et al. 2000). Magnesium improves potassium absorption and protects cardiac muscle cells and neurons against free radicals and toxic substances. It activates classical and alternative complement pathways (McCoy, Kenney 1992).

In adult ruminants, particularly in animals fed green fodder, even a minor magnesium deficiency can lead to nerve and muscle excitation, and a further drop in Mg concentrations increases the risk of hypomagnesemic-tetany (Martens, Schweigel 2000). In young ruminants, hypomagnesemia affects sucklings and animals fed solely milk or milk replacers deficient in magnesium. Other factors that increase the demand for magnesium or lower its availability, such as exercise, low temperature, decreased appetite/fasting and diarrhoea, also raise the risk of the disease (Robson et al. 2004). In calves, hypomagnesemictetany affects mostly rapidly growing animals at the age of 1.5 to 4 months, but in animals with bowel inflammations, the disease may occur as early as the second or third week of life. The first symptoms of nerve and muscle excitation are observed after several days or weeks of hypomagnesemia. They include anxiety, twitching of the ears, bulging eyes, jerking movements of the head, kicking at the abdomen, stiff and unsteady gait (Naik et al. 2010). The above stage is not always observed, and it is followed by contraction episodes lasting 5 to 20 minutes during which the animal lies on its side with stiffly stretched limbs, makes rowing movements, salivates profusely, makes chewing motions and grinds its teeth. The head and the neck are thrown backward (*opisthotonus*), the eyes are bulged, the heart rate and the respiratory rate are significantly elevated. After each episode, the animal remains weak, may exhibit tremor and signs of allotriophagy. Contractions may reappear 1-2 days later, and they can lead to death (Soni, Shukla 2012).

SELENIUM

In higher animals, selenium (Se) is closely linked with vitamin E and sulphur-containing amino acids. The physiological significance of selenium can be attributed to its presence in more than 30 selenoproteins, most of which are enzymes. Glutathione peroxidase (GSH-Px), one of the major sele-

noproteins, protects haemoglobin and fatty acids against oxidation and scavenges free radicals. Other selenoproteins include iodothyroninedeiodinase, which regulates the conversion of thyroxine (T_4) to 3,5,3'-tri iodothyronine (T_3), thioredoxin reductase, which works with electrons from NADPH to reduce oxidized thioredoxin in a catalytic reaction that transfers the reducing capacity from thioredoxin to cell proteins, selenoprotein P, which protects endothelial cells, and selenoprotein W, which protects myoblasts against oxidative stress and participates in muscle differentiation and development (Żarczyńska et al. 2013). Selenium influences immune processes. Selenium compounds participate in humoral immune responses and increase the concentrations of immunoglobulin M (Maggini et al. 2007). Selenium deficiency is generally associated with a low selenium content of soils. High environmental concentrations of sulphur, an antagonist of selenium, further decrease selenium concentrations. Selenium deficiency affects all animal species, but ruminants, in particular sheep and goats, seem to be at a higher risk.

Nutritional muscular dystrophy (NMD), also known as white muscle disease, is the most common disorder caused by selenium and vitamin E deficiency. NMD involves hyaline degeneration of skeletal muscles in various regions of the body, including the diaphragm, heart muscle and tongue (Beytut et al. 2002). The disease is most often diagnosed in healthy calves, lambs and kids younger than 6 months. The most common symptoms include an incorrect posture with widely spread limbs, hunched up spine, stilted gait and recumbency. The disease affects mostly thigh and crus muscles. Changes in tongue muscles prevent sucking, swallowing and lead to milk discharge through the nostrils. Pathological processes spread to the diaphragm and the heart muscle, leading to dyspnoea, higher respiratory rate, pulmonary murmur and cough (Ghany-Hefnawy, Tortora-Perez 2009). NMD affecting the cardiac function leads to mass animal deaths (up to 90% mortality), but it is far less lethal when only muscles are affected (Ramirez-bribiesca et al. 2005). Young animals with hyposelenosis are more susceptible to respiratory and gastric infections. Lower body gains are also reported (Aleman 2008).

COBALT

Cobalt (Co) catalyzes biochemical reactions in various metabolic pathways, including hydration, hydrogenation and desulphurization. This element participates in the biosynthesis of nucleic acids and stimulates the production erythropoietin in the kidneys, which is required for healthy erythropoiesis in living organisms. Cobalt activates gluconolactonase and participates in the pentose-phosphate pathway, an important process of tissue oxidation, which is independent of the Krebs cycle. Glucose oxidation is most intensive in the mammary gland (Kennedy et al. 1994). Half of the cobalt

supplied to the rumen is used to synthesize vitamin B_{12}, which contains up to 4.5% cobalt and is therefore referred to as cobalamin. In ruminants, cobalt is stored in small amounts mainly in the liver, kidneys and heart, and its concentrations range from 3.0 to 220.0 μg g^{-1} of fresh tissue weight (Sivertsen, Plassen 2004). Cobalt is essential for the optimal development and function of ruminal microflora, and ruminants, in particular lambs, sheep, goats, calves and adult cattle, are most susceptible to cobalt deficiency (Sharman et al. 2008).

The symptoms of cobalt deficiency are relatively non-specific. Low levels of cobalt contribute to changes in the composition of ruminal microflora and function, which inhibit the decomposition of cellulose and transformation of pyruvic acid, disrupts the production of methane, ammonia and volatile fatty acids and contributes to chronic indigestion and metabolic disorders. In lambs, clinical symptoms of cobalt deficiency generally appear after weaning in the grazing season (summer to autumn). Early stages of the disease are accompanied by a higher feed conversion ratio and decreased appetite. Growth is inhibited, and symptoms of wasting may be observed. In extreme cases, animals look starved (Sharman et al. 2008). Cobalt deficiency leads to hyperchromic macrocytic anaemia (Digest 2007). It lowers the immune function in ruminants, in particular resistance to parasitic infections of the gastrointestinal tract, and decreases the ability of neutrophils to kill bacteria (Schwarz et al. 2000). Other symptoms include poor coat condition and skin changes. Severe cobalt deficiency can also be manifested by the nervous system disorders, mostly depression, aimless wandering and pushing against obstacles (Fisher, MacPherson 1991).

IRON

Iron (Fe) is essential for the maintenance of systemic homeostasis. Iron is the fourth most abundant element on the earth, but environmental reactions such as oxidation prevent its full biological use (Mohri et al. 2004). Iron compounds play various roles in the body, the major ones being oxygen transport (haemoglobin) and storage (myoglobin). As well as regulating phagocytosis and immunoglobulin production, iron-containing enzymes participate in immune responses by conditioning the non-specific immunity of acute phase proteins, transferrin, haptoglobin and lactoferrin (Walter 1997, Benito, Miller 1998). The mechanisms that regulate Fe levels are based on iron absorption, but they do not evacuate excess iron from the body. Iron has to be reduced to Fe^{+2} in order to be absorbed in the intestines. Under physiological conditions, iron absorption from food is controlled by iron levels in the body. Iron uptake increases when its concentrations in body tissues decrease, whereas iron buildup inhibits absorption. When enterocytes are absorbed by the mucous membrane, iron is oxidized to Fe^{+3}, which binds to apoferritin to

produce ferritin. Excessive iron concentrations prevent binding with apoferritin and lead to the formation of haemosiderin, an insoluble iron-storage complex. The highest iron concentrations are found in haemoglobin, followed by porphyrin respiratory enzymes, ferritin, myoglobin and cytochromes. Iron released from degraded erythrocytes is reused and stored.

In young ruminants, the risk of anaemia posed by iron deficiency is not as high as in piglets, where the rate of erythropoiesis and conversion of fetal to adult haemoglobin is very high and requires iron supplementation. Subclinical anaemia, which leads mainly to inhibited growth, is rarely observed in suckling ruminants. Clinical symptoms generally subside after the introduction of a solid diet (Heidarpour, Bami et al. 2008). In young ruminants, the risk of iron deficiency is determined by iron concentrations stored in the liver and spleen during fetal development. Iron reserves are depleted in the first 3-4 weeks of life.

Anaemia is the most common manifestation of iron deficiency, and although it is generally subclinical, it can be aggravated by secondary diseases resulting from a weaker immune response and lower efficiency of circulatory and respiratory systems. Clinical symptoms are intensified when the Fe deficit cannot be compensated. In calves, the first symptoms are non-specific, including loss of appetite, inhibited growth, apathy, fatigue and allotriophagy, and they appear at the age of approximately 2 months. Mucosal pallor, increased respiratory rate and heart rate are noted in severe cases. In young animals, iron deficiency rarely leads to death, and cases of mortality are observed mostly in animals with inflammations of the respiratory (bronchi, lungs) tract or the gastrointestinal (diarrhea) system. In older ruminants, sideropenia can result from high dietary concentrations of Fe antagonists, such as phosphates and divalent ions, including copper, manganese, zinc, cobalt, selenium and nickel ions, which impair iron absorption in the proximal portion of the small intestine (Benito, Miller 1998).

Iron deficiency is rarely reported in traditional ruminant production systems, but it is noted in calves that are fed only milk or milk replacers with a low Fe content to obtain "white veal". The above leads to acute anaemia with haemoglobin concentrations of up to 50% of the physiological norm, muscle pallor and an underdeveloped gastrointestinal system. In those animals, the contents of forestomachs and the entire alimentary tract account for only 1-3% body weight. The production of calves for white veal has been banned in Poland and other countries.

COPPER

Copper (Cu) is found in many enzymes and plays a very important regulatory role in biochemical processes. Copper is a component of two key aerobic metabolism enzymes: cytochrome C oxidase and superoxide dismutase.

The discussed element influences the prooxidative-antioxidative balance and exerts antioxidative effects. Copper participates in the formation of myelin sheaths surrounding nerve fibers, haematopoiesis, and in skeletal development; it regulates the synthesis of collagen, elastin, melanin and catecholamines; it influences the color and waviness of the hair coat. Copper stimulates iron absorption and metabolism (Hostetler et al. 2003). In ruminants, Cu is absorbed partially in forestomachs with the involvement of microflora, and in the stomach and the small intestine, where copper ions are bound by metallothioenins, low-molecular-weight proteins with a high content of cysteine residues (Gooneratne et al. 1989). In calves, Cu absorption is high at 70-80%, but it decreases to >1.0–10.0% after the forestomach development (Spears 2003). Copper is accumulated already during the fetal development. Gooneratne and Christensen (1989) observed significantly higher copper concentrations in the livers of cattle fetuses than in the mothers. The Cu content of the liver in a fetus was found to increase during pregnancy (Hostetler et al. 2003).

Ruminants' sensitivity to copper-deficient feed decreases with age because copper is particularly important to rapidly growing calves, lambs and kids. Primary copper deficiency is observed mainly in regions where the copper content of soil is low or where the analyzed element is not available to plants. Grass and green fodder from such pastures and fields are unable to meet the animals' demand for copper, and young individuals are not supplied with adequate amounts of Cu in milk. Secondary copper deficiency occurs when the presence of copper antagonists, such as molybdenum, sulphur, cadmium, lead, zinc, calcium and iron, inhibits the availability of copper from feed. Diseases that inhibit the availability of dietary copper, in particular gastrointestinal disorders, can also contribute to secondary Cu deficiency.

Copper plays a multitude of roles in the body, and its deficiency produces a variety of symptoms, which is why Cu deficiency in calves is known under different names, such as "falling disease" in Australia and "pine" in Scotland. In lambs, clinical symptoms also vary in different regions of the world. In Great Britain, copper deficiency is manifested by swayback, in Australia – by anaemia and low wool quality, whereas in New Zealand – by bone mineralization disorders. In young cattle, the most common symptoms of primary and secondary copper deficiency include inhibited growth and development, bone abnormalities such as osteophytes on distal metacarpal and metatarsal growth plates, which lead to stiffness of the fetlock joint and steppage gait. Calves are also affected by lameness and transient hind limb ataxia (which subsides after rest) accompanied by sudden falls and/or dog-sitting position, progressing weakness that leads to wasting and allotriophagy (in particular dirt eating). Changes in the coat color and hypochromic macrocytic anaemia are more frequently noted in adult cattle (Gooneratne et al. 1989). Banton et al. (1990) identified two forms of copper deficiency in lambs and kids: congenital (swayback) and late (enzootic ataxia). The congenital form develops already in the fetus, and clinical symptoms are visible at birth. They include

recumbency, weakness, head shaking, tremor and ataxia. The symptoms of nervous system disorders are caused by an absence of or damage to white matter in cerebral hemispheres. Gelatinous malacic foci and cavitation are observed in the white matter. Demyelination is noted in motor pathways of white matter in the spinal cord. The late form occurs in lambs and kids at the age of 1 week to 6 months, and it is characterized by incoordination, ataxia and paresis of hind limb. Anatomopathological changes are limited to enlarged brainstem and spinal cord neurons, and cerebellar changes have also been reported in young goats (Banton et al. 1990).

ZINC

Zinc (Zn) is a component or an activator of nearly 300 enzymes and it plays a variety of biological roles in the body (Al-Saad et al. 2010, Miao et al. 2013). It is a structural element of superoxide dismutase, alkaline phosphatase, carbonic anhydrase, lactate dehydrogenase, RNA and DNA polymerases (Kavas et al. 2013). Zinc is essential for hormonal functions, including the growth hormone (GH), thyroid stimulating hormone (TSH), glucagon, insulin, follicular stimulating hormone (FSH), luteinizing hormone (LH) and adrenocorticotropic hormone (Alves et al. 2012). The discussed element influences the immune response, the development and activity of neutrophils and NK cells, and lymphocyte gene expression (Kincaid 1999, El-Far 2013).

Similarly to other microelement deficiencies, Zn deficiency can be both primary, when milk and feed do not supply animals with sufficient quantities of the element, and secondary, when feed contains adequate levels of zinc, but its availability from feed is reduced by Zn antagonists (copper, magnesium, calcium, phosphates, divalent iron compounds) and amino acid deficiency. In cattle, mostly Holstein-Friesian, Aberdeen-Angus, Simmental and Shorthorn breeds, impaired zinc absorption may be an autosomal recessive congenital defect (lethal trait A46) (Yuzbasiyan-Gurkan, Bartlett 2006).

Zinc deficiency has been studied extensively in calves and sheep. Mild deficiency is accompanied by non-specific symptoms such as impaired appetite, higher feed conversion ratio and lower weight gains. Delayed testicular development and hypogonadism leading to oligospermia and testosterone deficiency were observed in bull calves with low zinc levels (Bedwal, Bahuguna 1994). Severe Zn deficiency may contribute to skin changes, including peeling, scabbing, itching and hair loss. These changes are initially observed around the eyes, lips, nostrils, on the neck and at the back of the neck, on limbs (in particular on the inner side), udder skin, scrotum skin, in the area of the rectum and vulva. Swelling may appear in the affected areas. This type of Zn deficiency may also be accompanied by inflammations of oral and nasal cavities with excessive salivation, swelling of the gums and teeth grinding (Machen et al. 1996, Hosnedlová et al. 2007). The above changes are

symptoms of parakeratosis, a disorder which is probably caused by impaired protein synthesis and activation of Zn-dependent enzymes that are essential for carbohydrate, lipid and nucleic acid metabolism (Al-Saad et al. 2010, Alves et al. 2012). A severe form of the disease is observed in calves with inherited lethal trait A46, where skin changes are accompanied by atrophy of the thymus and significant impairment of T cell-dependent immune response. In the absence of dietary zinc supplementation, the affected calves usually die at the age of 4-6 months (Yuzbasiyan-Gurkan, Bartlett 2006).

SODIUM, CHLORIDE AND POTASSIUM

Sodium and potassium ions are the major cations responsible for the maintenance of physiological blood pH values in animals. Their content differs considerably in intracellular and extracellular fluids (plasma and extracellular, extravascular fluid). Potassium concentrations are higher than Na concentrations inside cells, whereas the reverse is observed in plasma, where Na accounts for 90% of all mineral elements. Sodium and potassium regulate osmotic pressure in body tissues. Different concentrations of Na and K ions on the two sides of the cell membrane contribute to the maintenance of stable resting membrane potential and nerve conduction. Na and K ions affect muscle contractility and tension. Physiological Na concentrations support monosaccharide and amino acid absorption. Bicarbonates and Cl ions constitute the major anions in plasma and interstitial fluid, and Cl ions play a key role in maintaining the acid-base balance in the body. Chloride is a component of hydrochloric acid that is produced by parietal cells for digesting nutritive compounds. Acid production and Cl absorption increase in the perinatal period. In lambs, the amount of hypochloric acid produced in the abomasum increases rapidly and lowers abomasal pH from 7 on day 142 of fetal development to 3 in 2-day-old infants (Gilloteau et al. 2009).

The discussed macroelements are supplied mainly with feed, including milk and milk replacers in young animals and solid feed in adult individuals. The quality and quantity of drinking water are also important. Salt licks can compensate for Na and Cl deficiency resulting from poor quality feed. Na, K and Cl are excreted mainly by the kidneys with urine, and they are evacuated in smaller amounts *via* the gastrointestinal system and the skin. Physiological levels of the discussed macroelements, mostly Na and K, are regulated by the nervous system, including the hypothalamus and the renin-angiotensin-aldosterone (RAA) system, and hormones, including thyroid hormones, and atrial natriuretic factors (Dratwa et al. 2004).

In calves, stress factors such as high stocking density, heat stress and transport, increase the demand for electrolytes. The above can be attributed to the elevated secretion of ACTH from the pituitary gland and higher re-

lease of aldosterone and corticosterone from the adrenal cortex. Aldosterone is the key hormone that regulates Na absorption in the kidneys and Na secretion with urine (Fitzsimons 1998). The discussed hormones also increase water and Na uptake through the angiotensin II neuropeptide. In calves, sodium deficiency intensifies stereotypies, which are altered behavioral responses to stress. Phillips et al. (1999) demonstrated that increased Na concentrations in feed improve feed and water uptake, increase body gains and reduce stereotypies such as cross-sucking in calves. The use of electrolyte solutions is recommended to reduce stress before animal handling operations such as transport (Schaefer et al. 1997).

Na, K and Cl deficiencies resulting from low concentrations of dietary macroelements are rarely reported in young ruminants. In calves, lambs and kids, the first symptoms of Na and Cl deficiency include allotriophagy, sparse hair coat, low body gains and lower feed uptake. Other symptoms involve abnormal licking behavior, dirt eating, drinking own or other animals' urine. In ruminants, electrolyte deficiency can be exacerbated by infectious and non -infectious diseases with diarrhoea (Bazeley 2003). Symptoms of dehydration, including weaker skin tension, deep eye sockets, tachycardia and decreased diuresis, are also observed (Guzelbektes et al. 2007). Water and electrolyte deficit treatments generally improve animals' health condition and eliminate clinical symptoms (Naylor 1990, Constable 2003).

CONCLUSIONS

In young ruminants, macroelement and microelement deficiencies contribute to metabolic disorders and clinical changes in individuals and in the herd. They impair the immune response and increase susceptibility to disease, in particular contagious diseases (diarrhoea, respiratory disorders). The mineral content of feed should be regularly monitored to improve animal health and promote healthy growth and development of animals.

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