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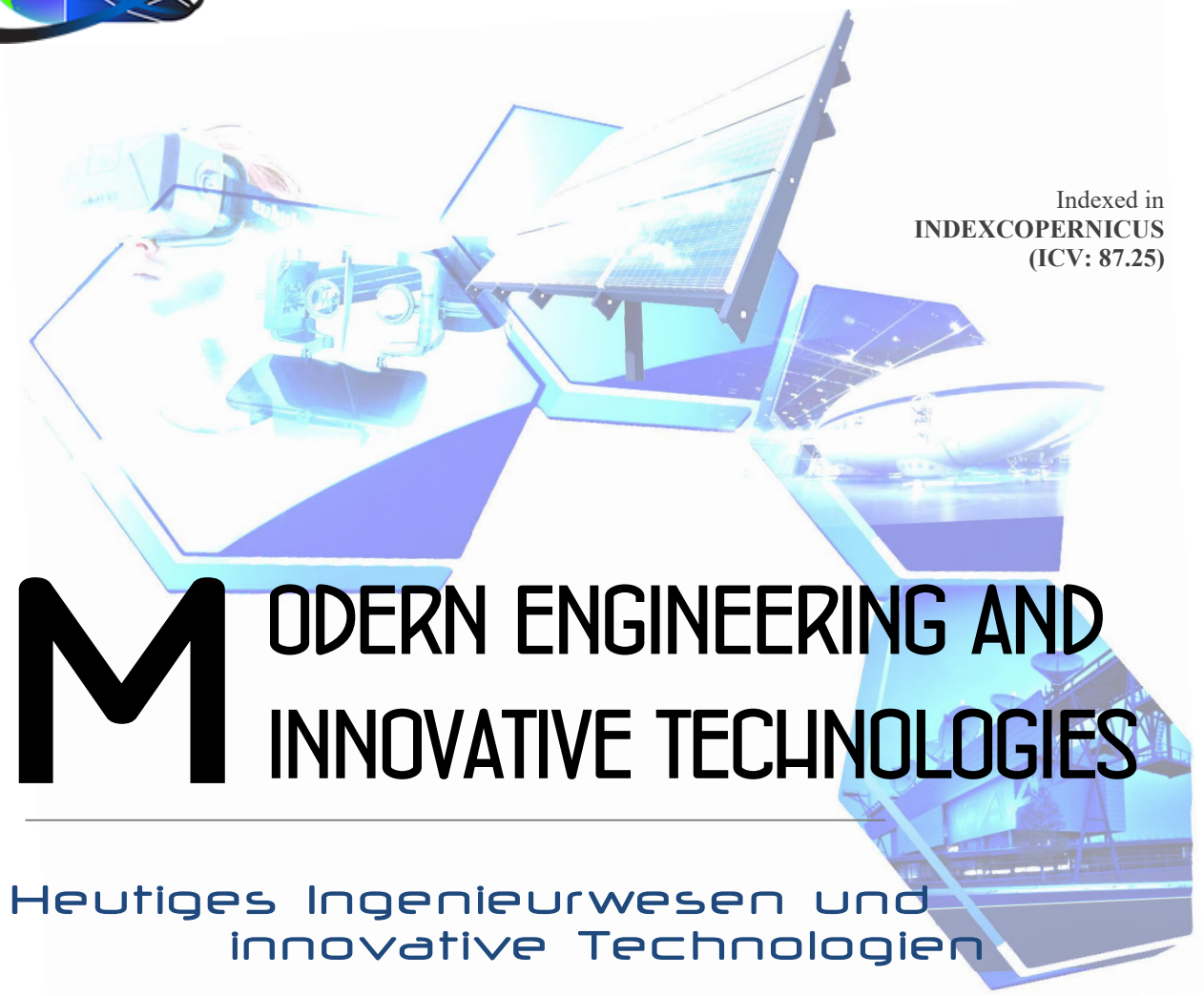

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EFFECT OF CHELATE COMPOUNDS OF MICROELEMENTS ON THE ORGANISM OF AGRICULTURAL ANIMALS

ВПЛИВ ХЕЛАТНИХ СПОЛУК МІКРОЕЛЕМЕНТІВ НА ОРГАНІЗМ СІЛЬСЬКОГОСПОДАРСЬКИХ ТВАРИН

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Abstract. *The study of the doctrine of "biogeochemical provinces" clarified the specific differences between animals and plant organisms in different areas and areas of the earth's surface, soils and waters, which are characterized by a lack or excess of some trace elements. This work provided an understanding of a number of local endemic human and animal diseases and played a major role in disease control.*

The optimal content and ratio of vital trace elements in the body of farm animals determines the normal course of metabolic processes, good health and high productivity.

Key words: *rations, farm animals, microelements,*

Introduction.

With a lack or excess of trace elements in the body there are diseases called trace elements. The most common hypomicroelementosis, which occurs due to a lack of essential trace elements in animals. Hypermicroelementosis as a consequence of an excess of microelements in an organism meets much less often. These diseases both in our country and abroad are still insufficiently studied, especially the issues of pathogenesis, clinical diagnosis and prevention and veterinary and sanitary quality of the products [1,2].

In the conditions of intensification of animal husbandry the role of high-grade feeding which provides display of genetic potential of productivity of animals, reception of high-quality production at decrease in expenses of forages especially grows. Complete feeding of animals is based on knowledge of their needs for energy, nutrients and biologically active substances, among which an important place is occupied by minerals, in particular trace elements. They give structure and strength to the skeleton, act as a component of organic compounds, increase the activity of the enzyme system of the body. Minerals are also necessary for the synthesis of hormones. They also control the water balance in the body, determine the number of positively and negatively charged compounds and thus regulate the balance of the acidic environment, cause muscle contraction, the movement of nerve impulses. In addition, they are used by animals for digestion of food, affect the course of digestion, support the protective functions of the body and neutralize metabolic products [2,3].

Lack or excess of certain micronutrients, violation of the optimal ratio between them in the diet leads to a decrease in metabolic processes, digestibility and nutrient

utilization of feed, animal productivity, and in long-term insufficiency to hypomicroelementosis.

Trace elements in animals are enzootic (local) diseases, because they are caused by insufficient or excessive content of mobile forms of trace elements in soils, water sources and plants of the relevant areas. They are found in farm animals more often in biogeochemical zones and provinces. Diseases cause significant economic damage to livestock. In animals suffering from microelementosis, due to metabolic disorders in the body not only reduces productivity but also resistance [4,5,6].

Iron in its content in the body of animals can be considered as a micro- and macronutrient. However, in terms of biochemical properties and physiological role in the body, it should be considered a trace element from the group of heavy metals.

Most iron is found in erythrocytes (60-73% of hemoglobin). In addition, 15-16% of it is part of iron-protein complexes, myoglobin (3-5%), enzymes and tissues (up to 0.1%). Iron, which is part of hemoglobin, cytochrome oxidase, peroxidase, catalase, is called hemin. Tissue iron contained in hemosiderin, ferroascorbate, ferritin, is called non-heme. There are also muscle iron, which is part of myoglobin, and serum iron - siderophylline (transferrin). Physiological depot in the body is the liver, spleen, bone marrow [2,7].

Copper is involved in the synthesis of hemoglobin, accelerates the mobilization of deposited iron and its transfer to the bone marrow, promotes the transition of mineral forms of iron in organic. Most copper is found in the liver, which is the physiological depot of this trace element. Much of it is in the blood, especially in erythrocytes [7,8,9].

Manganese is associated with enzymes, hormones and vitamins. In enzymatic systems, it acts as a non-specific activator or an indispensable metal component in the enzyme molecule. Manganese activates phosphatases of blood and tissues: phosphoglucomutase, prolidase, carboxylase, succinate dehydrogenase, etc. Thus, manganese significantly affects the vital functions of animals, regulates protein, carbohydrate, fat, vitamin and especially mineral metabolism. It also has a positive effect on the growth and development of animals, hematopoiesis and reproductive functions. Manganese is deposited in the body mainly in the liver. In addition, it is abundant in the bones, brain, kidneys, spleen [1,4].

Cobalt is part of vitamin B12 and is a major factor in blood formation. It promotes the formation of erythrocytes and hemoglobin synthesis. By regulating metabolic processes in animals, cobalt increases its protective properties, stimulates growth, development and productivity. The main depot of cobalt in the body is also the liver [8,9,10].

Zinc activates hormones of the anterior pituitary and pancreas. Zinc is part of the pancreatic hormone insulin, while regulating carbohydrate metabolism. Close connection with hormones, enzymes and vitamins determines its regulatory effect on reproductive function, metabolism of carbohydrates, proteins, fats, hematopoietic system, growth and development of animals. Thus, it is involved in the processes of cellular respiration and oxidation of carbohydrates [1, 2].

Iron enters the animal's body with food. Many foods contain iron in the most easily digestible form. Some plant foods are also rich in iron, but their digestibility is

more difficult. It is estimated that the body absorbs up to 35% of iron, while other sources report that this figure is less than 3%. Large amounts of iron are found in beef, beef liver, fish (tuna), pumpkin, oatmeal, cocoa, peas, leafy greens, brewer's yeast, figs and raisins [5,8].

The adult body contains about 3-5 g of iron, almost two thirds of this amount is part of hemoglobin. It is estimated that the optimal intensity of iron intake is 10-20 mg / day. Iron deficiency can occur if the intake of this element in the body is less than 1 mg / day. The threshold of iron toxicity for a living organism is 200 mg / day.

An important role of iron for the body was established in the XVIII century. The main function of iron in the body is the transfer of oxygen and participation in oxidative processes. Iron is part of hemoglobin, myoglobin, cytochromes. Most of the iron in the body is found in red blood cells, a lot of iron is in brain cells. Iron plays an important role in the processes of energy release, in enzymatic reactions, in ensuring immune functions, in cholesterol metabolism. Saturation of cells in the tissue with iron is carried out using the protein transferrin, which is able to carry ions of ferric iron. Iron ligand complexes stabilize the genome, but in the ionized state can be inducers, cause DNA damage and provoke cell death. Both iron deficiency and excess negatively affect the health of animals [12,13].

There are many factors that can help reduce iron content. The reasons for the low content of iron in the body can be its insufficient intake with food, destruction of metabolism, impaired absorption in the gastrointestinal tract. Situations associated with relative or absolute iron deficiency can occur when the body needs more of this bioelement. Such situations include pregnancy, lactation, periods of growth and development. Finally, the cause of iron deficiency can be acute or chronic blood loss [11,12].

In turn, iron deficiency is one of the most common causes of anemia, major bleeding, weakening of the body, impaired neuropsychiatric function.

In some hereditary and chronic diseases, with excessive intake, iron can accumulate in the body. The body with excess iron suffers from physical weakness, loses weight, often gets sick. At the same time, getting rid of excess iron is often much harder than getting rid of its deficiency.

With severe iron poisoning, the intestinal mucosa is damaged, liver failure develops, nausea and vomiting appear. Keep in mind that iron is an oxidizing agent (that is, it can cause free radicals that can destroy tissues), so do not take too much iron. In cases of iron deficiency, the intake of iron-containing drugs should be combined with the intake of antioxidants: vitamin C and E, as well as copper.

Copper is a trace element that is part of a living cell and is necessary for the normal functioning of the body. She is involved in the biochemical processes involved in redox reactions. Copper, like other chemical elements, is widespread in the biosphere, but quite unevenly. The level of mobile forms of copper in soils varies from 1 to 5 mg / kg (average 3 mg / kg). The high concentration of copper in the upper layers of soils compared to the parent rocks is associated with the biological activity of plants and its accumulation [4,5,8].

Copper belongs to the elements of high biogenicity and accumulates in humus horizons. The degree of its penetration in these layers averages 14% and ranges from

5.2 to 22.2%. It is believed that the largest percentage of copper in soils falls on the silt fraction. The level of mobile forms of microelements is subject to significant fluctuations during the vegetative period of plant life.

Copper is one of the most important essential trace elements necessary for human and animal life. The largest amount of this element is found in the lungs, intestines, spleen, skin and hair. All endocrine organs contain the largest amount of copper. The blood contains an average of 100 mcg of copper, of which in erythrocytes and leukocytes 60 mcg. A significant amount of copper in blood plasma is found in ceruloplasmin, the most important copper-containing protein. Copper is found in the superoxide dismutase of erythrocytes and leukocytes.

Copper intake with food should be 2-5 mg / day, with a daily intake of less than 2 mg, which is dangerous due to the possibility of developing a deficiency. About 30% of the daily intake of copper is increased, and the rest of the copper in the gastrointestinal tract is converted into insoluble compounds, which are excreted in the feces. Of the total amount of resorbable copper, about 80% is excreted in the bile and about 16% by the walls of the gastrointestinal tract. About 4% of absorbed copper is excreted in the urine. A small amount of this element is excreted with sweat [8,9,10,11].

The key role in copper metabolism is played by the liver and its structural elements - hepatocytes. By entering them through the portal vein system, copper is primarily bound to metallothionein, found in the liver of humans and most of the animals studied. The opposite of the nature of the protein that binds copper in the liver is due to its oxidation during excretion. The synthesis of metallothionein is regulated by the content of zinc and copper in the liver at the level of mRNA transcription. Thionein performs the functions of detoxification of copper and its intracellular transport. Increasing the copper content can lead to amplification of thionein genes and a sharp increase in the synthesis of this protein. The content of copper in plasma is regulated by neurohumoral mechanisms, and differently at the person and various animals.

In humans and all studied animal species, the vast majority of copper that enters the body is excreted in the feces. Most of this ME, present in the feces - not absorbed copper, a smaller part - endogenous copper, which is isolated from the bile and the walls of the gastrointestinal tract. Copper deficiency (hypocuprose) was first found in laboratory animals in 1927. Endemic diseases of sheep and cattle were soon reported in a number of countries due to copper deficiency in grazing plants and the preventive inclusion of small amounts of this element in the diet of animals [10]. These studies have shown that copper, in addition to participating in hematopoiesis, is also necessary for the normal course of many biochemical processes, pigmentation and keratinization of hair, myelin formation, synthesis of various tissue production compounds and more. There is no doubt that in the near future the list of copper deficiency syndromes and diseases, which is already large enough, will increase, and most importantly, be clarified.

This shows that copper plays a major pathogenetic role in the onset of the main symptoms of the disease. Copper deficiency causes CNS damage in farm and laboratory animals, as exemplified by the endemic ataxia of lambs described in

various countries around the world [12]. The reason for its occurrence is both the primary deficiency of copper in the pasture and the secondary one - caused by the lack of physiological antagonists of this metal - molybdenum, sulfates, lead, cadmium, etc. The symptoms of copper deficiency are varied and depend on the species and age of the animals. Experimental copper deficiency in animals leads to delayed skeletal growth and development, anemia, depigmentation, alopecia and dermatoses, as mentioned above. With acute copper deficiency in feed, animals develop anemia, which is accompanied by diarrhea and exhaustion. With a deficiency of copper in feed, deformation of joints and tubular bones of the extremities, demineralization of the brain and spinal cord.

Copper, entering the body of an animal with food or water, is absorbed in the gastrointestinal tract. Recently, there are no significant differences in the assimilation of copper by animals from plant foods and inorganic salts. The main site of absorption of copper in the gastrointestinal tract is the upper part of the small intestine. The absorption of copper in ruminants decreases sharply with an excess in the diet of copper antagonists such as molybdenum, sulfates and zinc. It has been found that excess zinc can inhibit intestinal absorption, transport and accumulation of copper. Impaired absorption of copper leads to an increase in the gastrointestinal tract production of metallothionein [13].

It should be noted that in ruminants the absorption of copper is better than in animals with a single chamber stomach. Obviously, this is due to their higher need for this element, especially in the initial period of lactation. Copper is necessary for the normal functioning of the microflora of the ruminant pancreas. Under the influence of the microflora and the juices of the rumen from the food that got into the pancreas, partially becomes part of the rumen fluid. A significant part of this water-soluble copper in the contents of the scar is the microflora of the scar. Copper, which is absorbed in the upper part of the small intestine, penetrates the liver, bone marrow, spleen and pancreas. It is believed that the main organ of copper deposition in the body, as mentioned above, is the liver. The level of copper in the liver is an indicator of assimilation and provision of this element of the body's needs. Endogenous copper is excreted mainly through the gastrointestinal tract with bile. The concentration of copper in bile is higher than in blood. Excretion of copper with bile is considered as one of the main ways to maintain homeostasis of this element in the body (gastrointestinal tract - blood - liver - bile).

It has been established that copper has different effects on the absorption of minerals, sugar and amino acids of glycol, as well as on the excretion of nitrogenous substances and motility of the small intestine. It stimulates the absorption of potassium, calcium and glucose, but inhibits the absorption of sodium and phosphorus. Copper is actively involved in blood formation, synthesis of hemoglobin and other blood hemoporphyrin compounds, such as cytochrome, catalase and cytochrome oxidase. The latter catalyzes the incorporation of iron into the structure of heme and thus promotes the maturation of erythrocytes in the early stages of their development. It was found that the addition of copper to the diet of rats increased the content of cytochromes and increased the activity of cytochrome oxidase and bone marrow. There is a biochemical relationship between erythropoiesis and cytochrome

oxidase activity between hematopoiesis and cytochrome oxidase activity. These processes are regulated by copper. It is known that copper affects the synthesis of porphyrin compounds. It combines with some of them to form iron-copper-nucleoprotein complexes, which are precursors of hemoglobin and an important source in the copper metabolism chain in the body. Copper ions in the free state act as oxidants, like oxidase, catalase and peroxidase. However, especially high biological activity of copper as an oxidant is manifested when it combines with proteins.

Copper is also involved in osteogenesis. It increases the body's protective functions and forms hair pigment, which promotes keratinization of hair and feathers. In addition, it is part of proteins, activates their enzymatic functions. Copper is a component of a number of enzymes such as tyrosinase, ascorbinase, urease, ceruloplasmin, cytochrome oxidase, galactose oxidase, uricase, beta-hydrolase, diamine oxidase, monoamine oxidase, benzylaminooxidase, xanthine oxidase, xanthine oxidase, xanthine oxidase. Divalent copper is a specific activator of certain enzymes and also supports the activity of unstable pituitary hormones in the blood. Copper is directly related to vitamins. There was a significant increase in B vitamins in products of animal and plant origin with increasing levels of copper in feed. The concentration of vitamin C in the body depends on the level of copper in the diet. This is due to the fact that copper ions dramatically accelerate the oxidation of ascorbic acid and reduce its concentration in tissues and organs. Ascorbic acid and copper thus correlate with each other. In addition, a correlation was found between the level of copper and vitamin A in animals [1,2,3].

Another thing to note is that they are interdependent: metabolism, health, productivity and reproductive ability of animals based on the action of copper and hormonal status of the body. Copper plays a role in the biosynthesis of hormones and their effects on the living organism. It is known that the latter increases the effectiveness of insulin in diabetes. Carbohydrates are mostly used in animals and glycogen breakdown is limited. In addition, its content in the liver increases [11].

In recent years, it has become important to study the sensitivity of ruminants to copper. It is quite low in the development of toxicosis. The reasons for this phenomenon are - overdose of copper salts or their uncontrolled use, feeding copper additives to animals without taking into account the amount of microfertilizers that were introduced into the soil. In addition, the content of copper in feed is not always taken into account, the use of copper sulfate for deworming of animals. Chronic copper poisoning leads to necrosis of liver cells, methemoglobinemia, hypercuprema, bilirubinemia and hemolysis of erythrocytes. It is important to emphasize that the toxic effects of copper in animals decrease with increasing levels of zinc in their diet [8].

Thus, copper is one of the many bioelements that plays an extremely important role in almost all bioprocesses of a living organism. Lack or excess of copper in the body of animals causes a variety of diseases, which, in turn, significantly affects the productivity and ability of animals to reproduce [4].

Manganese was found in animal tissues more than 70 years ago, but its vital role for the animal body was established only 18 years ago, when it was shown to be necessary for the growth and development of rats and mice. It was soon found that

this IU prevents the development of skeletal abnormalities. Manifestations of manganese deficiency were then found in almost all studied laboratory and farm animals. Normally in the human body weighing 70 kg is 10-20 mg (0.18-0.36 mmol) of manganese. Normally, according to L.S. Hurley et al. (1987), in bones, especially tubular, and also in a liver and kidneys there are higher concentrations of manganese, than in other bodies. This is true for humans, cattle and rats. The manganese content in the muscles is very low, even lower in whole blood and especially in plasma. In the liver of a healthy body, regardless of age, is 6-8 mg / kg (based on dry weight). Data on the amount of manganese in whole blood is very variable. Its level in the blood serum increases with heart disease, infections and some psychoses. Elevated levels of manganese in whole blood have been reported with excessive intake of this IU, as well as with rheumatoid arthritis and iron deficiency [8].

Manganese is vital for brain function. Its highest concentration is in the pineal gland, the median elevation of the hypothalamus and in the basal ganglia. It accumulates primarily in melanin-containing structures of the CNS, such as in the substantia nigra. It is characterized by a higher content in the cells of organs rich in mitochondria. Manganese deficiency leads to selective damage of these organelles, they are prolonged, the correct orientation of these crystals is broken. This obvious specific mitochondrial damage can be prevented by including manganese salts in food [12].

Manganese metabolism was studied using its isotope 54Mn with a half-life of 312 days. Manganese in the form of a divalent cation is absorbed equally throughout the small intestine. The mechanism of this process is little studied. It is known that normally only a small (3-4%) part of this IU is absorbed in cattle, regardless of its content in the diet. This process does not play a major role in maintaining the homeostasis of manganese, which, unlike iron, is controlled by the mechanisms of secretion of this element [13].

In the portal vein, most of the manganese is bound to protein. Free and protein-bound manganese is effectively captured by the liver. A small portion is oxidized to Mn3 +, bound by transferrin, and travels from the bloodstream to various body tissues [13].

Manganese leaves the bloodstream very quickly. Only 10% of this isotope can be found in the blood 10 minutes after 54Mn. Once in the cell, this IU is involved mainly in the mitochondria, so the cells of the liver, kidneys, pancreas, rich in these organelles, contain increased amounts of manganese. Glucocorticoids cause the transfer of manganese from the liver to other organs and tissues.

Manganese is excreted in small amounts in the urine. Normally, this trace element enters the bile, excreted from the body mainly in the feces. But at receipt in the increased quantities it is eliminated also through a wall of intestines, and first of all through proximal department. Some manganese is also excreted by the pancreas, which in the case of cholestasis may even become the main organ of excretion of this IU. The rate of excretion of manganese is its content in food and does not depend on ions of other metals. The release of manganese from the body of animals increases only with the introduction of its stable isotope. Manganese, which is excreted with bile, is subject to partial reabsorption, in this regard, the enterohepatic circulation of

this element is established [11].

Divalent manganese is predominantly present in solutions and biochemical structures. It, like ferric iron, has significant similarity to imidazole depending on the divalent cations of copper, zinc and cadmium, predominant in the SH group, and does not replace these ions in their complexes with proteins. Manganese and other vital transition metals act as tightly bound components of enzyme molecules, which in this case are true metalloenzymes, or serve as activators of enzymes, creating easily decomposing complexes with them. Manganese acts as an activator of a number of enzymes. In the case of reactions that are activated by manganese, the metal ion reacts with the substrate, which contains a phosphate residue, forming a chelate, or reacts directly with the protein. Manganese is chemically close to magnesium. Based on this, the activation of most enzymatic reactions by this element is nonspecific and it can be replaced by magnesium. But manganese interacts with all three phosphate groups and the pyridine ring or water molecule. Despite the fact that the activation of enzymes by metals is of a common non-specific nature, a number of phenomena observed in manganese deficiency can be associated with dysfunction of enzymes that are activated by manganese. Manganese has a marked effect on the processes of gluconeogenesis and regulation of blood glucose levels. Pyruvate carboxylase and phosphoenolpyruvate carboxykinase are involved in the processes of gluconeogenesis. In fed animals in the first of these enzymes, manganese can be replaced by magnesium. However, in hungry animals, the activity of the enzyme in manganese deficiency is significantly reduced. Subsequent experiments have shown that manganese is required for normal insulin secretion, and the degree of suppression of the secretory mechanism in animals with manganese deficiency increased as the stimulation of the secretion of this hormone. The first observations on the effect of manganese on lipid metabolism have shown that it interacts with choline to prevent excessive deposition of fat in the liver. At different doses of choline in animals with manganese deficiency in hepatocytes found more fat than in animals that received adequate amounts of this element. The lipotropic effect of manganese was much stronger at low choline content, which indicates their close interaction [11,12].

However, the involvement of manganese in lipid metabolism is related to its role in cholesterol synthesis. This process is an important step in the synthesis of lanosterol - the precursor of cholesterol. The enzyme shows its activity only in the presence of metal, and at low concentrations of manganese is very effective. Hypocholesterolemia has been reported in manganese deficiency. It is possible that in violation of the synthesis of cholesterol - the precursor of sex hormones, explains the effect of manganese on the reproductive function of animals.

The effect of manganese on lipid metabolism can be realized through its action on cell membranes. It is concentrated by mitochondria and in the absence of this IU clearly visible damage and even the absence of the outer chondrial membrane, as well as disorders in the system of mononuclear phagocytes.

Manganese deficiency in animals is reflected primarily in the formation of the skeleton in both intrauterine and postnatal periods. These phenomena are caused by a violation of chondrogenesis. Detection of ME deficiencies that occur in a pathology of bone and cartilage tissue could contribute to a clinical study of the content and

synthesis of glycosaminoglycans and manganese levels in tissues and body fluids. In vivo, manganese deficiency in cattle has a number of skeletal abnormalities and reproductive dysfunction, most of which are also associated with defective glycosaminoglycan synthesis. When manganese deficiency in utero is born offspring with signs of ataxia, which is characterized by loss of balance, impaired coordination reflexes, tilting the head. The cause of these defects is the abnormal development of otoliths in the inner ear, necessary for the normal functioning of the vestibular apparatus. Manganese deficiency also affects brain function.

The biological role of manganese in the CNS may be related to the normal structure and stability of membranes. This ME is also necessary for the normal synthesis of biogenic amines. Biogenic amines cause an increase in the concentrations of manganese in the blood due to the formation of complexes with this metal, which are involved in their transport, binding and deposition. Of the enzymes that are activated by manganese, it is worth noting glutamine synthetase, which plays an important role in the detoxification of ammonia. The modified form of the enzyme is much more subject to the regulatory action of a number of products of glutamine metabolism than the original form. There is a possibility that manganese performs a regulatory function and a number of other enzymes. Among the enzymes that are activated by manganese is also lactose synthetase. Manganese can accelerate the transcription process by activating RNA polymerase. The main route of supply of manganese in the production environment is the respiratory tract. The enteral pathway and to a lesser extent the inflow through the skin can be of great importance. Manganese has pronounced cumulative properties, accumulating in the liver, kidneys, endocrine glands, in smaller quantities it accumulates in the bones, brain and spinal cord. It should be emphasized that this trace element freely penetrates the blood-brain barrier and has tropism to the subcortical structures of the brain. And its pathogenic effect is connected with it.

A severe manifestation of manganese CNS is the stage of functional disorders of the nervous system with characteristic psychopathological symptoms in the form of weakening of associative processes. This stage is followed by the second, for which the typical increase in signs of toxic encephalopathy. It should be emphasized that CNS damage in chronic manganese is accompanied by signs of gonadal suppression, as well as functional disorders of the thyroid gland, liver and gastrointestinal tract. The content and retention of manganese in the blood and matter of the brain depends on the form in which this metal enters the body.

Perhaps the role of manganese in the etiology and pathogenesis of diseases of the bone and cartilage, in the development and functioning of the CNS will be revealed very quickly. Due to the need for optimal manganese content for the formation of otoliths, it is believed that genetic, congenital and some acquired vestibulopathies are associated with a violation of manganese homeostasis. The hypothesis of maintaining the optimal state of otoliths through the prophylactic use of manganese-containing drugs is noteworthy.

Cobalt is one of the most important trace elements. This element is physiologically active, affects hematopoiesis and metabolism. It is a component of a number of metalloenzymes: transcarboxylase isomer, glycylglycine dipeptidase. The

most important role belongs to cobalt in the endogenous synthesis of vitamin B12 (cyanocobalamin). In ruminants, the synthesis occurs in the pancreas, and in monogastric - in the cecum and colon. Once in the bloodstream, cobalt is deposited in the liver of animals, which is the richest in cyanocobalamin food, then in other organs: pancreas and thymus, kidneys, spleen, adrenal glands. This vitamin is a complex of Co3 +, it is involved in the synthesis of hemoglobin and its deficiency causes anemia.

The content of cobalt in soils determines the amount of this element in plants and the level of its entry into the body of herbivores. If the content of cobalt in the soil is 2 · 10-6%, there is a severe specific disease of cattle, due to its insufficiency (enzootic insanity, coastal disease, shrub disease). Lack of cobalt in the diet of animals leads to acobaltosis (growth slows down, productivity decreases, anemia appears). The diseases are endemic and occur in areas with low cobalt content in soil and plants. The introduction of additional amounts of cobalt in animal feed leads to their recovery.

The effect of cobalt on the body is not limited to hematopoiesis. It is associated with the activity of enzymes, vitamins, hormones, and also affects protein, fat, carbohydrate and mineral metabolism, promotes the accumulation of vitamins in the organs and tissues of animals, stimulates the growth and reproduction of rumen microorganisms and their synthesis of B vitamins. cations, cobalt is involved in the reactions of glycolysis and the tricarboxylic acid cycle, activates dipeptidases and phosphatases, interacts with other minerals, while performing various functions. For example, carboxylase may contain manganese or magnesium, cobalt, calcium, iron. There is a relationship between Co, Mn, Zn and Cu in the interaction with B vitamins, manganese and cobalt with vitamin E.

Traces of cobalt are found in all foods, but its highest content is found in vegetable leaves. Consumption of cobalt with iodine diet is usually 170-440 mcg / day, and with water up to 10 mcg / day. 90% of the total amount of cobalt is contained in plant products. Cobalt is excreted in the feces (about 80%) and urine (10%).

Cobalt is part of the cyanocobalamin molecule, is actively involved in enzymatic processes and the formation of thyroid hormones, inhibits iodine metabolism, promotes water excretion by the kidneys. Cobalt increases iron absorption and hemoglobin synthesis, is a powerful stimulant of erythropoiesis. The process of hematopoiesis in humans and animals can take place only with the normal interaction of three bioelements - cobalt, copper and iron. It should be noted that the mechanism of cobalt influence on hematopoiesis continues to be studied. It is known that the introduction of cobalt into the bone marrow increases the formation of young red blood cells and hemoglobin. But this requires the presence of sufficient iron in the body.

Vitamin B12, in addition to its effect on hematopoiesis, has a very effective effect on metabolism, especially on protein synthesis, and also has the ability to restore -S-S-groups involved in the processes of blocking and utilization of toxic elements. Assessment of cobalt content in the body is based on the results of blood, urine and hair.

From the gastrointestinal tract, cobalt enters the blood, where its content varies from 0.07 to 0.6 μmol / l and depends on the season and time of day. It is slightly higher in summer due to the animals eating fresh green fodder, which is rich in this IU. The concentration of cobalt is much higher in erythrocytes than in plasma. According to T. Gunther and sang. (1974), in serum cobalt binds to the albumin fraction. The absorption of vitamin B12 is different from the absorption of cobalt ions. It depends on the so-called intrinsic factor - one of several mucopolyproteins produced by the gastric mucosa. The complex is an internal factor - vitamin B12 enters the cells of the mucous membrane, where the vitamin is released by a special enzyme, and the factor is either subject to proteolysis, or enters the intestinal lumen. In pernicious anemia there is a genetic defect in the synthesis of this factor. Impaired absorption of vitamin B12 is also observed during gastrectomy and infection with helminths, competing with the host for the available vitamin B12.

In the blood, vitamin B12 is transported by special proteins - transcobalamin, of which transcobalamin II transports it in the blood of the portal system, and transcobalamin I - in the plasma of the great circle of blood circulation. It is believed that transcobalamin I binds vitamin B12 more strongly, performs the function of deposition. Transcobalamin III is also known and its function is being studied.

Vitamin B12 is converted in the liver to hydroxycobalamin (Co3 +), then in the mitochondria cobalt is reduced enzymatically to Co +, after which the vitamin is converted by deoxyadenosyltransferase into a coenzyme. Vitamin B12, like cobalt ions, is excreted mainly in the urine. A unique feature of the structure of vitamin B12 is the bond between cobalt and a carbon atom 0.205 nm long, which is not found in any natural organometallic compound.

Vitamin B12 deficiency in ruminants occurs when its content in the scar fluid is below 5 ng / l and in the blood - below 0.2 ng / l. Vitamin B12, which is synthesized by the microflora of the cecum and colon, is not absorbed by ruminants. The rumen microflora produces many vitamin B12-like compounds, and the synthesis of their biologically active form takes place with relatively low efficiency, which is cobalt deficiency of about 15% of the total number of compounds containing this element, and with adequate content in the diet - only 3% .

The second feature of ruminants is their marginal ability to absorb vitamin B12, which is only about 3-5% of a given dose. Thus, ruminants use cobalt very inefficiently both in the synthesis of vitamin B12 and in the process of its assimilation by the body. At the same time, they have an increased need for vitamin B12, due to the peculiarities of their energy metabolism.

In ruminants, the main source of energy is not glucose, but lower fatty acids - acetic, propionic, to a lesser extent butyric and others, which are formed by the microflora of the rumen during the fermentation of feed. The content of these acids in the urine of ruminants can serve as a reliable cobalt deficiency long before the onset of clinical signs of acobaltosis. Thus, ruminants have a high need for vitamin B12 due to the low efficiency of its synthesis and assimilation, making them particularly sensitive to cobalt deficiency compared to other animal species. These defects are prevented by methionine injections. Methionine deficiency can also cause the slow absorption of nitrogen, which is observed in vitamin B12 deficiency, and act as a

factor that limits the growth of animals and their hair. A further consequence of vitamin B12 deficiency is a marked decrease in the level of folic acid in the liver, which is restored with the introduction of methionine. These observations suggest that the effect of vitamin B12 on the metabolism of lipids and folic acid is through methionine, which increases folic acid stores, improving its entry into the hepatocyte.

It was found that at physiological concentrations cobalt is required for the synthesis of thyroid hormones. In animals suffering from acobaltosis, giving this IU leads to a decrease in the size of thyroid follicles and an increase in the height of the epithelium lining them. Endemic thyroid dysfunction is observed in farm animals and humans from biogeochemical provinces with low levels of cobalt in the environment or its unfavorable ratio with iodine.

A similar pattern is established between diseases of the circulatory system and low levels of this trace element. The ability of cobalt to inhibit tissue respiration, including bone marrow cells, is associated with the development of compensatory polycythemia with severe bone marrow hyperplasia and the formation of foci of extramodular hematopoiesis. One of the possible mechanisms of erythropoiesis stimulation is the effect of cobalt on the formation of erythropoietins. This effect of cobalt is explained by the blockade of SH-groups of some oxidoreductases, which lead to oxygen starvation of the bone marrow, which stimulates it to increased activity or through increased synthesis of erythropoietin. The latter is produced in the blood of an inactive precursor in response to hypoxia under the influence of erythropoietin formed in the kidneys. In this regard, the use of cobalt as a stimulant of hematopoiesis can be justified only in cases of low blood erythropoietin.

Cobalt has low toxicity to the studied experimental animals and humans. It should be emphasized that a 1000-fold excess of cobalt almost completely inhibits the absorption of iron. The subcellular distribution of these metals in duodenal enterocytes was similar. Based on these data, the authors concluded that cobalt inhibits the absorption of iron not by affecting the binding sites on the apical surface of erythrocytes, but by acting on the release processes, occurs in the basal part of the cells. Organic cobalt compounds have a coronary dilating effect. Drugs that contain this IU, promote the formation of iron and contribute to the beneficial effect on immunological reactivity.

It has been established that cobalt compounds can cause sensitization of the body, which can cause dermatitis with characteristic hyperkeratosis, as well as interstitial pulmonary fibrosis. Cobalt can have toxic effects on the pregnant woman and the developing fetus. The connection between this metal and carcinogenesis remains controversial and controversial. Indeed, in the tumor tissues of humans and animals, in the blood of cancer patients cobalt content is increased by one and a half - two and a half times compared to normal. In addition, in patients with leukemia, the concentration of this IU in whole blood is reduced. The most pronounced decrease in its level was observed in undifferentiated and lymphoblastic forms of leukemia. During remission, the level of cobalt approaches the control figures, with the deterioration of patients - is significantly reduced in serum, erythrocytes and whole blood. However, in animal experiments, the introduction of cobalt at a concentration of 0.01 mg / kg inhibited the growth of Pliss lymphosarcoma in rats and Ehrlich's

ascites carcinoma in mice. Thus, the possible blastomogenic role of cobalt requires further research.

The biological role of zinc was established about 120 years ago, when J. Raulin showed that this element is necessary for the growth of Aspergillus niger. This position was later confirmed for plants and animals. Zinc has been found to be a component of carbonate dehydrogenase. The study of zinc deficiency in animals revealed the following symptoms: changes in appetite, disturbance of animal behavior and loss of training properties, growth retardation and puberty, blocking estrus, infertility, lymphopenia and increased hematocrit. Zinc deficiency was first identified as a syndrome of hypogonadism and dwarfism. Zinc reserves are small - 22.9-30.6 mmol, ie 1.5-2 g. It is found in all organs and tissues, but its amount varies widely: from 0.15 to 3.3 mmol per 1 kg of raw tissue. Skeletal muscles are the richest in zinc, accounting for 62.6% of all IUs. It is important to emphasize that the release of zinc from its tissue depots is facilitated by glucocorticoids. The total content in the body is 2300 mg, of which in soft tissues - 1800 mg.

At the cellular level, zinc stimulates the formation of polysomes, inhibits iron-catalyzed free radical oxidation. It is shown that the presence of zinc is necessary for the transition from one phase of the cell cycle to another, its lack blocks this process. Particular interest in zinc in the last 15 years is associated with the discovery of its role in nucleic metabolism, transcription processes, stabilization of nucleic acids, proteins and especially components of biological membranes, as well as in the metabolism of vitamin A.

The main regulatory mechanism of zinc homeostasis is its absorption, which takes place in the small intestine.

In humans and rats, zinc, like calcium, is absorbed mainly in the small intestine. In cattle, about 1/3 of a single dose of zinc is absorbed in the rennet.

Zinc is absorbed in two phases, one of which may be related to energy expenditure, which is not equally recognized by all authors (Solomons N.W., Cousins R.J., 1984). The first phase is fast and reflects the flow of zinc, while the second, slower, phase characterizes the transport of this element across the basement membrane. There are two different mechanisms of zinc absorption, one of which operates at low concentrations of this element. Due to the quantitative intake of zinc in the epithelium of the mucous membrane may be similar at both low and high content of this element in the intestinal contents.

Increased protein content in the diet improves the absorption of zinc due to the formed amino acids. With a low-protein diet, the absorption of zinc, on the contrary, decreases. Lysine, cysteine, glycine and glutamate stimulate this process, while the positive effect of histidine is not recognized by all authors. Elevated levels of calcium in the diet make it difficult to absorb zinc and often cause parakeratosis. A number of studies have suggested that the lipid fraction of milk is important for zinc absorption, especially in newborns. The main role was played by essential fatty acids.

Up to 85% of the total amount of this element is absorbed from food poor in zinc, and only 10-30% from ordinary food. Assimilation of zinc from different feeds is different. As part of corn, it is available by 52%, wheat - by 60%, peas, barley, beans - by 66-68% and lupine - by 80%. Of the semi-synthetic casein diet, which

contains 18 mg of zinc, its absorption was 83%. Absorption of zinc is significantly reduced in inflammatory processes under the influence of leukocyte endogenous mediator IL-1, which causes a simultaneous decrease in the level of zinc in blood plasma and its accumulation in the liver.

It has been shown that zinc deficiency in the liver disrupts the synthesis of retinol-binding protein required for the transport of vitamin A in the bloodstream. Particularly strong influence is on the simultaneous deficiency of both nutritional factors that lead to disruption of homeostatic regulation of the body. This explains the seasonal nature of some hypomicroelementosis, such as endemic bovine parakeratosis, which occurs in early spring, and endemic icterohemoglobinuria in astrakhan sheep, which disappears during their transition to green fodder.

The next stage of zinc absorption is its interaction with intracellular enterocyte ligands.

It is possible that the physiological antagonism of copper and zinc is played out in part at the level of metallothionein. The synthesis of this protein is induced by these two elements, but zinc is more active, and copper forms stronger complexes with this protein. Therefore, when giving a moderate amount of zinc, it is realistic to expect, first of all, the formation of copper-thionein and reduce the entry of this element into the blood. The interaction of both metals may have clinical significance, for example, when taking massive doses of zinc in Wilson-Konovalov disease.

The transfer of zinc across the basolateral membrane is an active process that requires the presence of oxygen and energy expenditure. It is inhibited by metabolic toxins and may also depend on the availability of metal-binding portions on plasma albumin. Zinc is contained in plasma and erythrocytes in a ratio of 1: 8 or 1: 9. In erythrocytes, it is present mainly as a component of carbonic anhydrase and to a lesser extent superoxide dismutase. Some zinc binds to the membrane, helping to stabilize its structure. Leukocytes contain up to 0.3% zinc, which, unlike erythrocyte zinc, does not exchange with its plasma reserves and does not respond to a deficiency of this element. The main transport protein of blood plasma, which carries 2/3 of metabolically active zinc - is albumin. Plasma zinc is closely correlated with the amount of zinc bound by albumin. For some time, it has been suggested that zinc is transported in the portal vein by transferrin, but it is noted that the binding to albumin is stronger than to transferrin, and in the perfusion of isolated intestinal segments, absorbed zinc does not bind to transferrin but to albumin. The degree of absorption of zinc correlates with the content of albumin, not transferrin. Thus, the level of circulating albumin may be important in the absorption of zinc. Moreover, it is noted that hepatocyte culture absorbs zinc only from media that contain amino acids and albumin, but not transferrin.

These observations suggest that albumin is the major physiological ligand that transports zinc from the intestine to the liver. It was noted above that albumin also plays an important role in the transport of copper in the portal vein system, but it was found that the binding sites of both elements on albumin do not coincide.

Over the past 15 years, a great deal of research has been done on the intracellular volume of zinc. Glucocorticoids have been shown to increase zinc levels in hepatocytes, and this accumulation is associated with the synthesis of

metallothionein, in the selective stimulation of the synthesis of which insulin and glucagon are also involved. The release of zinc from the cell is due to the breakdown of its intracellular ligands. The outflow of zinc from cells depends on the plasma content of albumin and amino acids.

Basically, zinc is excreted in the feces. Zinc, which is excreted in the feces, consists of undigested zinc, and the amount of the latter reflects the level of intake of this element with food and plays an important role in homeostasis [14,15].

With tissue breakdown, such as burns, surgery and other injuries, starvation, the zinc content in the urine increases significantly. Hyperzincuria is also observed in hepatic porphyria, postalcoholic cirrhosis and chelation therapy []. Zinc enters the urine mainly from plasma ultrafiltrate. In the distal renal tubules, it is usually 95% reabsorbed and its amount in the urine correlates well with its volume and creatinine content. With increasing urine volume and pregnancy, there is some increased zinc secretion associated with increased tubular flow. Significant release of zinc occurs during infusions of amino acids, especially cysteine and histidine [16,17].

The largest amount of this element is also released with hair and nails. Detection of zinc in hair is an important diagnostic test for latent forms of its deficiency.

Zinc plays an important role in the synthesis of protein and nucleic acids, and also plays an important role in skeletal development. In zinc deficiency there is an inhibition of alkaline phosphatase in the chondrocytes of the pineal cartilage, which is the main biochemical defect in bone development. There is no doubt that zinc is involved in calcification processes, but the disclosure of the specific mechanism of its action is a matter of the future.

A number of manifestations of the biological activity of zinc are due to its high affinity for sulfhydryl groups, which are important determinants of the structure and function of proteins. Because zinc ions do not participate in redox reactions, they help stabilize sulfhydryl groups, preventing their oxidation by copper and iron ions. The lack of this IU in laboratory and farm animals is accompanied by inhibition of antibody production, a decrease in the number of lymphocytes circulating in the blood, and a significant decrease in thymus mass.

Vitamins A and B6 are needed for better absorption of zinc by the body. Assimilation of zinc interferes with copper, manganese, iron and calcium (in large doses). Cadmium can displace zinc from the body.

Zinc is a cofactor of a large group of enzymes involved in protein and other types of metabolism, so it is necessary for the normal course of many biochemical processes. This element is required for the synthesis of proteins, including collagen. Zinc is involved in the processes of cell division and differentiation, the formation of T-cell immunity, the functioning of dozens of enzymes, pancreatic insulin, the antioxidant enzyme superoxide dismutase, the sex hormone dihydrocorticosterone. Zinc plays an important role in the processes of skin regeneration, hair and nail growth, secretion of sebaceous glands. Zinc promotes the absorption of vitamin E and maintains a normal concentration of this vitamin in the blood. It plays an equally important role in the body's processing of alcohol, so zinc deficiency can increase the chance of developing alcoholism.

Zinc is part of insulin, a number of enzymes involved in hematopoiesis. Zinc is

needed to keep the skin in good condition, as well as in wound healing, as it plays an important role in protein synthesis. Zinc strengthens the body's immune system and has a detoxifying effect - promotes the release of carbon dioxide from the body.

Conclusion. The biological effect of chelate complexes on the body of animals is determined by their stability and properties of the ligands that are part of the complex. At this stage, several hypotheses are known regarding the role of chelated compounds in the absorption and transport of trace elements. According to some authors, the constant formation of various compounds can be a test indicator of their sorption. The chelated agent has a positive effect only when it forms a sufficiently stable compound with a metal, but the stability constant should be lower than in compounds of the corresponding metals with biologically active substances in the body's metabolic processes.

It is established that chelated metal compounds have an effect on almost all types of metabolism. Thus, the zinc complex increases the intensity of protein and carbohydrate metabolism, copper and cobalt, and zinc compounds - the activity of reamination enzymes. The adequacy of the action of trace elements and their chelates contributes to the manifestation of the following physiological effects: increased activity of transaminases, metalloenzymes (ceruloplasmin, glutathione peroxidase, cytochrome oxidase, catalase, etc.), antioxidant, protein-synthesizing systems, erythropoietin.

Due to the gradual rupture of chelated bonds, the drugs have a prolonged effect. When cleaving trace elements, protein ligands are effectively used by the body. All this makes it possible to reduce the dose of trace elements dozens of times, to positively solve environmental and economic problems.

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Анотація. *Дослідження вчення „Про біогеохімічні провінції" дало роз'яснення специфічних різниць тварин і рослинних організмів в різних зонах і областях земної поверхні, ґрунтів та вод, які характеризуються нестачею або надлишком деяких мікроелементів. Ця робота дала можливість зрозуміти ряд місцевих ендемічних захворювань людей і тварин та відіграла велику роль у боротьбі із захворюваннями.*

Оптимальний вміст і співвідношення життєво необхідних мікроелементів в організмі сільськогосподарських тварин зумовлює нормальний перебіг обмінних процесів, добрий стан їх здоров'я і високу продуктивність.

Ключові слова: *раціони, сільськогосподарські тварини, мікроелементи, хелатні сполуки, метіонати.*

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