University of Mosul
Lecture No.: 7
College of Veterinary Medicine
Date: second term/ 2025
Unit of Scientific Affairs
Website: https://uomosul.edu.iq/veterinarymedicine/

Lecture title: **Animal nutrition: Zinc and Manganese**

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Summary:

Zinc

Zinc has been found in every tissue in the animal body. Zinc does not donate or accept electrons, and therefore zinc is not involved in oxidation–reduction reactions. free zinc reacts rapidly with water to yield an insoluble zinc hydroxide,It's tends to accumulate in the bones (29%) rather than the liver (5%). Zinc is involved in cell replication and differentiation, particularly in nucleic acid metabolism. Thus, it regulates gene expression in cells. Zinc interfere with other physiological functions like production, storage and secretion of hormones, Also, involvement in the immune system and electrolyte balance. Several enzymes in the animal body are known to contain zinc; these enzymes include:

- Carbonic anhydrase
- Pancreatic carboxypeptidase
- Lactate dehydrogenase
- Alcohol dehydrogenase
- Alkaline phosphatase
- Thymidine kinase

Absorption of zinc

In gastric uid with an acidic environment, zinc can exist as a free ion. In the lumen of the small intestine with an alkaline solution, zinc binds to gastroferrin to increase its solubility. Zinc is absorbed into the enterocyte. zinc binds primarily to a cytosolic protein called cysteine-rich intracellular protein and possibly a nonspeci c binding protein (NSBP) for transport to the basolateral membrane. This mineral may also bind to cytosolic metallothionein for temporary storage in enterocytes. Then enters the portal circulation. Intestinal absorption of zinc is reduced by various minerals (e.g., calcium and copper) and phytate. Absorbed zinc enters the portal circulation. In the plasma, 60% and 30% of the zinc binds to albumin and α2-

macroglobulin, respectively, and about 10% of the zinc binds to other serum factors. Once entering the cells, zinc is assimilated into various metalloproteins, including metallothionein. Metallothionein may act as a reservoir for zinc, and as a detoxifying agent for toxic heavy metals, such as cadmium. Excretion of zinc occurs predominantly via pancreatic secretions and the faeces.

Sources of zinc

Zinc is widely distributed. Yeast is a rich source, and zinc is concentrated in the bran and germ of cereal grains. Animal protein by-products, such as fishmeal, are usually richer sources of zinc than are plant protein supplements. Forage-based diets for ruminants and horses are generally de cient in zinc, and, therefore, are usually supplemented with a free-choice mineral premix.

Deficiency symptoms

Zinc deficiency is widespread in farm animals if their diets are not supplemented with adequate zinc. Zinc is essential for many metabolic pathways (including protein synthesis and cell division), in animals, zinc deficiency results in reduced food intake, growth restriction, and poor food utilization; gastrointestinal ulcerative colitis, diarrhea, and anorexia, low sperm counts, fetal abnormalities, and infertility; skin abnormalities, dermatitis, and impaired wound healing; impaired growth and development of the cells that mediate innate immunity and of T- and B-lymphocytes, leading to impaired immune function; impaired activation of the extracellular signal-regulated kinases .In the brain, leading to neurological dysfunction; eye lesions and photophobia; and behavioral change

Gross signs of zinc deficiency include the following:

1. in chicks, retarded growth, foot abnormalities, ‘frizzled’ feathers, parakeratosis and a bone abnormality referred to as ‘swollen hock syndrome’.

2. in calves include inflammation of the nose and mouth, stiffness of the joints, swollen feet and parakeratosis.

3. In dairy cows' low dietary zinc concentrations are associated with high somatic cell counts in their milk. **Feeding** a diet low in zinc during pregnancy reduces survival of the newborn lamb.

Excess of zinc

zinc toxicity includes reduced feed intake and impaired growth; gastric irritation, abdominal pain, nausea, vomiting, and diarrhea; impaired absorption and abnormal metabolism of dietary iron and copper and anemia; increased risk for infectious disease; cardiovascular and neurological dysfunction; and impaired reproduction in both males and females.

Manganese

The amount of manganese present in the animal body is extremely small. Newborn calves and newly hatched chicks contain 65–70 mg and 10–20 µg Mn, respectively. Most tissues contain traces of the element. the highest concentrations occurring in the bones, liver, kidney, pancreas and pituitary gland. Manganese is important in the animal body as an activator of many enzymes for glucose metabolism, and ammonia detoxification, such as hydrolases and kinases. Also it acts as a constituent of arginase, pyruvate carboxylase and manganese superoxide dismutase**.** Manganese, through its activation of glycosyl transferases, is required for the formation of the mucopolysaccharide that forms the organic matrix of bone.

Absorption of manganese

Less than 10% of ingested manganese is absorbed by the small intestine of animals. In the small intestine, Mn^{2+} is absorbed into enterocytes. divalent metals, such as $Ca^{2+,}$ Cu^{2+}, Mg^{2+}, and Zn^{2+}, negatively inhibits Mn^{2+} absorption by the small intestine.

Sources of manganese

most forages contain 40–200 mg/kg DM. Seeds and seed products contain moderate amounts, except for maize, which is low in the element. Yeast and most foods of animal origin are also poor sources of manganese. Rich sources are rice bran and wheat offals. Most green foods contain adequate amounts.

Deficiency symptoms

Manganese deficiency has been found in ruminants and poultry. It includes:

1. retarded growth
2. Abnormal metabolism of AAs, glucose, and lipids
3. skeletal abnormalities

4. ataxia of the newborn
5. reproductive failure.
6. fatty infiltration of the liver
7. In cows and goats delay oestrus, conception and increase abortion**.**
8. In young chicks causes perosis or 'slipped tendon', a malformation of the leg bones.
9. in breeding birds reduces hatchability and shell thickness, and in chicks causes head retraction.

Excess of manganese

High concentrations of manganese in tissues, particularly the brain, result in neurological disorders in animals. Symptoms of manganese toxicity include reduced feed intake and growth restriction, muscle rigidity, leg cramps, and irritation. Excessive manganese 0.32 mg/mL in ruminal fuid adversely alters the microbial population and the concentrations of total short-chain fatty acids.