THE VALUE OF MINERAL ZINC IN ANIMAL PHYSIOLOGICAL FUNCTIONS

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ABSTRACT

Zinc is one of the most important trace elements in the body, and it is indispensable to the growth and development of microorganisms, plants, and animals. This review work looks at the value of zinc in animal physiological functions. In domesticated species, experimental zinc deprivation was produced first in chicks and pigs. Zinc - responsive disorders were induced by excess dietary calcium in poultry attributed to a potent and complex interaction with the major source of phosphorus in feed grains (phytate). Fetal growth is especially affected by lack of zinc.Zinc deprivation increases the expression of the gene for the appetite-regulating hormone cholecystokinin and also increases the susceptibility of endothelial cells to oxidant stress. The role of zinc is probably multi-factorial. Zinc regulates genes involved in signal transduction, responses to stress, reduction–oxidation reactions, growth and energy utilization.Itis also a potent inducer of metallothionein (MT), a zinc and thiol - rich metalloprotein that scavenges free radicals and could act as a reduction– oxidation sensor or active signaling switch in cells. Zinc aids in proper assimilation of vitamins, normal growth and development.Zinc supplementation also cured and prevented parakeratosis, a thickening and hardening of the skin found in pigs given grain-based diets, rich in calcium. It is therefore important that the value of zinc in animal physiological functions be assessed and deficiency corrected in some chronic animal diseases.

Keywords: animal physiological functions, mineral supplementation, pigs, poultry, zinc

INTRODUCTION

Zinc (Zn) is a ubiquitous trace element. It is one of the most important trace elements in the body, and it is indispensable to the growth and development of microorganisms, plants, and animals. It is involved in DNA replication and RNA transcription. The first unequivocal evidence that zinc is necessary for growth and health was obtained in laboratory animals (Todd *et al*., 1934) at a time when associations were being noted between zinc and carbonic anhydrase, an enzyme eventually shown to contain zinc (Keilin and Mann, 1940). In domesticated species, experimental zinc deprivation was produced first in chicks (O'Dell and Savage, 1957) and pigs (Stevenson and Earle, 1956). In all species, the disorder was characterized by loss of appetite, growth depression, abnormalities of the skin or its appendages and reproductive failure. In the field, zinc supplementation cured and prevented parakeratosis, a thickening and hardening of the skin found in pigs given grain-based diets, rich in calcium (Tucker and Salmon, 1955). Zinc - responsive disorders were also induced by excess dietary calcium in poultry and were eventually attributed to a potent and complex interaction with the major source of phosphorus in feed grains, phytate (Py) (Ziegler *et al*., 1961).

The quest to understand the pathogenesis of zinc deprivation continues and has led to the measurement of free intracellular zinc at femtomolar concentrations (Outten and O'Halloran, 2001), while therapeutic properties have been found for zinc at near-molar concentrations (Hill *et al*., 2001). Fear of zinc deprivation is being exploited commercially by feed industry which offers wide variety of mineral additives, including organically bounded trace elements as compounds for the enhanced bioavailability of a variety of 'organic' zinc supplements (Suttle, 2010).

GENE EXPRESSION

Fetal growth is especially affected by lack of zinc (Hurley, 1981) and this may reflect the roles that it plays in DNA synthesis and, nucleic acid and protein metabolism. Zinc regulates genes involved in signal transduction, responses to stress, reduction–oxidation reactions, growth and energy utilization, including a T-cell cytokine receptor and uroguanylin (Cousins *et al*., 2003). Teratogenic defects may mostly derived from the primary effects of zinc deprivation on gene expression – effects that are most marked when cells are rapidly dividing, growing or synthesizing (Chesters, 1992).

APPETITE CONTROL

Since O'Dell and Reeves (1989) reviewed the role of zinc in the regulation of appetite, advances have continued to come from studies with rats. The pattern and rate of food intake is affected and acute zinc deprivation causes rats to nibble at food rather than 'meal eat'. Little addition of zinc (3–6 mg Zn kg−1 dry matter (DM) removes the complication, but description of such diets as 'marginal' in zinc (e.g. Kwun *et al*., 2007) is inappropriate because the diets unequivocally provide insufficient zinc for normal growth and development.

Zinc deprivation increases the expression of the gene for the appetite-regulating hormone cholecystokinin (Cousins *et al*., 2003), but the role of zinc is probably multi-factorial. There is also increased expression of leptin, a cytokine hormone whose secretion from adipocytes acts as a satiety signal (Kwun *et al*., 2007), and reduced expression of pyruvate kinase (Beattie *et al.,* 2008), which is regulated by insulin. Changes in the concentrations of neurotransmitters in the brain have been reported in zinc deprivation, but may be secondary consequences of appetite reduction (Kwun *et al.,* 2007). In what might more accurately be called acute zinc deprivation, the reduction in appetite is selective, with carbohydrate being avoided and protein and fat preferred (Kennedy *et al*., 1998).

FAT ABSORPTION

When low plasma and liver concentrations of vitamin E and fat-soluble vitamin A were reported in zinc - deprived animals, attention became focused on the possible role of zinc in fat absorption. The pancreas secretes zinc - dependent phospholipase A2 (Kim *et al.,* 1998). This hydrolyses phosphatidyl choline, facilitating its absorption and the formation of chylomicrons, which are crucial for the absorption of fat micelles (Noh and Koo, 2001). Duodenal infusion of hydrolysed phosphatidyl choline greatly increases the absorption of fats and the fat-soluble vitamins A and E in well grown rats that are 'marginally deprived' of zinc. Partial alleviation of the effects of heat stress in quail by supra-nutritional levels of zinc (Sahin and Kucuk, 2003) may be mediated, in part, by secondary improvements in vitamin status.

ANTIOXIDANT DEFENSE

In addition to the contribution of a superoxide dismutase, CuZnSOD, in protecting cells from the superoxide radical, *in vitro* studies suggest that zinc may afford protection from iron - induced lipid peroxidation by blocking iron-binding sites at cell surfaces, acting synergistically with vitamin E (Zago and Oteiza, 2001). Zinc deprivation increases the susceptibility of endothelial cells to oxidant stress (Beattie and Kwun, 2004). In humans, zinc supplementation decreases the expression of anti-inflammatory cytokines (Prasad, 2007), but it is unclear whether this is beneficial to health. Zinc is also a potent inducer of metallothionein (MT), a zinc and thiol - rich metalloprotein that scavenges free radicals and could act as a reduction– oxidation sensor or active signalling switch in cells (Beattie and Trayhurn, 2002).

Zinc aids in proper assimilation of vitamins, normal growth and development. It is found in all the fluids in the body. It assists in the maintenance of body tissue, sexual function, immune system, chemical detoxification, synthesis of DNA and helps in reducing healing time. It is stored in the thyroid, pancreas, liver, kidney, bone, voluntary muscle, prostate, sperm cells, skin, hair, nails and white blood corpuscles and parts of the eyes (Sanchez, 2010). Zinc plays an important role in poultry, particularly layers, as a component of a number of metalloenzymes such as carbonic anhydrase which is essential for eggshell formation in the hens shell gland (Scheideler, 2008). Other important zinc metalloenzymes in the hen include carboxypeptidase and DNA polymerases. These enzymes play important roles in the hen's immune response, in skin and wound healing, and for hormone production (testosterone and corticosteroids). Classic deficiency symptoms of a zinc deficiency in poultry could include a suppressed immune system, poor feathering and dermatitis, infertility and poor shell quality (Scheideler, 2008).

Lack of zinc during embryonic growth grossly disturbs skeletal development and severe abnormalities of the head, limbs and vertebrae have been found in chick embryos from the eggs of severely deprived hens (Kienholz *et al*., 1961). Zinc deficiency in breeder diets reduces egg production and hatchability. Proper zinc supplementation has proven to be important in reducing early mortality of turkey poults (Larson, 2005).

CONCLUSION

Zinc is one of the most important trace elements in the organism, with three major biological roles, the catalytic, the structural, and the regulatory one. It is a multifunctional metal compatible with satisfactory growth, health, and well-being. It is essential for the structure and function of various proteins and cellular components and plays an important role in animal physiology from its involvement in the proper function of the immune system to its role in cellular growth, cell proliferation, cell apoptosis, as well as in the activity of numerous zinc-binding proteins. It is therefore important that the value of zinc in animal physiological functions be assessed and zinc deficiency be corrected in some chronic animal diseases.

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