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Review Article

METAL TOXICOSIS IN POULTRY – A REVIEW

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Abstract: At high levels all mineral elements can produce adverse effects in animals. Thus, the essential minerals must be provided in the diet at concentrations sufficient optimum response and the dietary intake of other minerals must be low enough to provide complete safety for both the animals and also the human population which will consume the meat and eggs. Dietary maximum tolerable levels have been generated which can serve as guidelines in ensuring safety for both domestic animals and humans. Although there are several potential dietary sources of toxic elements for poultry, adequate quality control programs at the feed mill coupled with good manufacturing practices and a knowledgeable nutritionist should greatly decrease the likelihood of significant contamination. Contaminated ground water from hazardous waste sites and other forms of industrial pollution as well as municipal waste systems probably represent the most likely sources of contamination in the United States at the present time. (Henry and Miles., 2001).

INTRODUCTION

All minerals can cause toxicosis in animals, when consumed in large quantities. The margin of safety between the minimum amount required to animal feed and the amount that causes adverse effects in animal health varies for different minerals. However, there are many minerals that do not participate in any known function of the animal body weight and, in fact, are harmful-toxic. Many heavy metals are used as a trace elements and feed additives in poultry feed. These metals are common in our environment some of these (iron, copper, manganese, zinc, etc) are essential for good health, however; other (Arsenic, mercury, lead, cadmium, etc) are poisonous and deleterious for health (Jadhav *et al.,* 2007).

ARSENIC

Arsenic is most important and usually found in the environment in organic and inorganic forms with different bioavailability. Phenyl arsenic acids (organic) are used as poultry feed additive as it controls coccidiosis, improves production performance (Ghosh *et al*., 2012) and

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increase body weight (BW) in pigs. Usually organic arsenic compounds (arsenobetaine, arsenocholine, trimethylarsine and arsalinic acid) exhibit very low toxicity. Dietary vitamin C eliminates heavy metal toxicity as it binds with free sulfhydryl group of heavy metals in different tissues and restores enzyme level (Rana *et al.,* 2010). Generally trivalent arsenic compounds are more toxic than the pentavalent forms. It produces toxicity when the concentration of arsenic compounds is 2-10 times higher than the recommended dose, which is usually 100 mg/kg complete feed. Most of the toxic effects arise from exposure to inorganic As from ommercial uses such as wood preservatives, herbicides and termiticides. Its toxicity usually attributed to low methylating ability

Changes of toxicity includes decrease in hemoglobin, packed cell volume, erythrocytic count and total leukocytic counts, heterophils and lymphocytes (Halder *et al*., 2009), lipid peroxidation and damage cell membranes. Histopathologically intoxication produces moderate to severe hyperemia, hemorrhages cellular swelling with granular cytoplasm, degeneration and coagulative necrosis in liver, intestines, kidneys and spleen. Then, all the inorganic and organic arsenic compounds accumulate to various tissues (higher to lower concentration: kidneys > lungs > urinary bladder > skin > blood > liver). However, several weeks later, arsenic is translocated to hair, nails and skin because of the high concentration of sulfur-containing proteins in these tissues (Agency for Toxic Substances and Disease Registry, 2000). Moreover, inorganic arsenic has been shown to be embryotoxic and teratogenic.

Litter produced by chickens fed roxarsone (organic arsenics) amended food contains total arsenic concentrations ranging from 2.9 to 77 mg/kg. Litter from chicken fed food not amended with roxarsone contains only trace levels of arsenic (< 1 mg/kg). Once ingested by animals, roxarsone can be degraded into inorganic forms of arsenic (arsenite and arsenate) within the animal's digestive tract and in animal waste. Arsenite and arsenate are both known to cause cancer in humans (NAS 1999; NAS 2001).

Clinical signs of Arsenic toxicity in birds

Rabia Sharaf *et al.,* reported that as exerts clinical signs birds including decreased body weight and feed intake, dullness, open mouth breathing, increased thirst, ruffled feathers, pale comb, skin irritation and watery diarrhea.

Arsenic use promotes antibiotic resistance

Exposing bacteria to arsenic in feed can inadvertently cause an increase in antibiotic resistance. Infectious disease concerns are heightened by the fact that poultry producers

routinely use feed additives that include both antibiotics and arsenic components. Vitamin C partially ameliorated the toxic effects of arsenic in broiler birds.

LEAD

Lead is a common cause of poisoning in domestic animals throughout the world. Lead poisoning can occur in all domestic animals including horses, poultry and dogs (Khan et al., 2008). Lead is a nonessential trace element and animal health can be adversely affected (oncology actions, causing liver, skin and lung cancer and change hematological parameters). Broiler chickens are vulnerable to Pb intoxication. Lead or zinc poisoning can happen quickly or build up slowly over time depending on how much of the metal a bird ingests, how much of the toxin is present, and other factors. Behavioral changes, such as screaming, might happen because the bird is in pain and otherwise uncomfortable. It is also extremely toxic to birds.

Sources

Common sources of lead include **l**ead paint, lead fishing weights, curtain weights, lead frames of stained glass windows and tiffany lamps, foil from champagne bottles, lead solder, old pewter, lead batteries, weighted ashtrays and toys, twist ties, tooth brushes, plastic / vinyl coverings, cardboard boxes with dyes, imported candy / food packaging wrapping/labels, crystal, fishing sinkers, some artist paints, lead weights / lead hardware, stainless glass windows / solder in stained glass/tiffany style lamps, venetian blinds and gasoline fumes (paints used in poultry equipment such as drinkers and feeders to prevent rusting, with either lead based paint or lead-free paint with leaded drying agent. Also, majority of litter material that comes from woods Painted with lead base paints (North & Bell, 1990), constitute another source of lead poisoning to birds.

Toxicity

Absorbed lead is retained by soft tissues and eventually by bone and is slowly excreted through the kidneys. Lead affects the CNS, renal, hematopoietic, neurologic and gastrointestinal systems. Lead can cause cerebral edema and neuronal damage, demyelination and decreased peripheral nerve conduction peripherally. Lead can cause anemia through increasing RBC fragility. Bone marrow suppression is also a potential effect.

As tiny as 1.0 ppm Pb in the diet can cause significant growth suppression in chickens and consistent decline in blood D-aminolevulinic acid dehydratase, an erythrocyte enzyme sensitive to Pb consumed by chickens is accumulated in bones, soft tissues, and eggs. Pb

bone levels are by far the uppermost, followed by kidney and liver. The lower most Pb concentration is detected in skeletal muscle. Ingested lead has resulted in poisoning.

Symptoms / Clinical Signs

Clinical signs of toxicity includes depression, weakness; falling of perch; unable to walk, stand or fly straight, anorexia, abnormal droppings / blood in droppings, polydipsia, greenish-black diarrhea, ataxia, head tilt, seizures, blindness and death. In broiler chickens,

Lead poisoning significantly reduced body weight and body weight gain. Erdogan *et al.* (2005) showed that 200 mg lead/kg diet reduced growth in term of body weight and body weight gain, poor performance and death in animals (McDowell,1992; Gurer & Ercal,2000). Bakalli *et al.* (1995), also reported that feed conversion ratio was significantly poor at a level of 10 mg lead/kg feed. Lead accumulation in kidney and liver of broiler was reported by Erdogan et al. (2005), Chickens tolerate lead concentrations up to 500 mg/kg, without affecting the rate of weight gain. When in the diet the level of calcium increases a small concentrations of lead acetate, by 250-400 ppm, can determined adverse effects, on the performance of broiler chickens.

CADMIUM

Cadmium (Cd) is an abundant, non-essential element that is widely used in electroplating and galvanizing, as a colour pigment in paints and in batteries. The increased release of cadmium from industrial process, waste disposal and cigarette smoke in the environment lead to a general concern for the potential toxic effects of cadmium. The most likely source of contamination in the animal feed industry is in conjunction with the use of zinc sulfate or poorly processed zinc ores as sources of supplemental zinc. Other potential sources include mining and smelting operations, corrosion of metal-plated iron, discarded cadmium-chloride products, and the use of urban sewage sludge's to fertilize pasture or croplands (NRC, 1980). The extremely long biological half-life of cadmium essentially makes it a cumulative toxin, so long past exposure could result in direct toxic effects of the residual metal Adema chl *et al.* (1991).

Cadmium intake resulted in changes to all indicators of oxidative stress studied and the changed activities of SOD, catalase and GST and levels of blood glutathione and MDA is a reliable sign of imbalance between oxidative and antioxidative capacity of blood. Cadmium stimulates the formation of beta 2-microglobulin in urine which induces renal tubular dysfunction. Keda *et al.*(2005) Yadav *et al.*(2006) reported cadmium on the antioxidant defense system and lipid per oxidation (LPO) of erythrocytes of adult poultry birds. It was

observed that the activities of erythrocyte superoxide dismutase (SOD) and catalase (CAT) increased during sub-acute cadmium toxicity.

The level of blood glutathione (GSH) was significantly decreased in cadmium exposed birds. However, the lipid per oxidation (MDA) was significantly increased in the cadmium exposed birds could be concluded that cadmium exposure altered the activities of antioxidant enzymes of erythrocytes and produce oxidative stress by disturbing the oxidative and anti oxidative balance of the adult poultry birds. Kant *et al*. (2011). Ability of cadmium to induce oxidative stress in blood of adult poultry bird, as evidenced by increased lipid per oxidation and altered activities of antioxidant enzymes. It was observed that increased activities of erythrocyte SOD and catalase in all the cadmium exposed groups might be due to the activation of antioxidant defense system of the body. Antioxidants reduce oxidative radical-induced reactions and have protective effect on stabilization of metabolic processes in erythrocytes that prevent the development of oxidation stress and hypoxia. Antioxidants reduce oxidative radical-induced reactions and have protective effect on stabilization of metabolic processes in erythrocytes that prevent the development of oxidation stress and hypoxia. GSH appears to be important in protecting the cell against Cd toxicity Singhal *et al*.(1987). Cadmium exposure produce oxidative stress by disturbing the oxidative and anti oxidative balance of the adult poultry birds.

The cadmium sulphate at 100 mg/L in drinking water can be used to induce oxidative stress in adult poultry birds .Kant *et al*. (2011) Zinc administration recorded beneficial effects in reduction of cadmium residues in broiler tissues and reduced cadmium toxicity. By enhancing the liver and kidney functions and the hematological parameters. Naveen *et al*. (2011). Maximum tolerable Cd level for poultry was 0.50ppm. The high mean Cd content (0.12-1mg) in the poultry feeds may have negative consequences on the environment when poultry manure is used for fertilizing plants (or) as soil amendment in combination with other organic materials. The results obtained were compared with the maximum permissible hygiene limits for Cd in meat (0.1mg/kg) and liver (0.5 mg/kg) according the Codex Alimentorum.

Induction of synthesis of the protein metallothionein in the intestinal tract is the primary protective mechanism the animal has to prevent absorption of toxic amounts of cadmium. The element is sequestered by the protein and the epithelial tissue subsequently sloughed and eliminated in feces. When japanese quail were fed a diet containing 1 ppm radio-cadmium for 1 week, followed by 50 days on a basal diet, less than 4% of the initial

dose remained (Jacobs et al., 1978). Approximately 25% was found in liver and another 25% in kidneys with 12% in the intestinal tract, probably in conjunction with metallothionein. The anemia observed during cadmium toxicosis can be alleviated to a certain extent by addition of iron to diets (Hamilton and Valberg, 1974). Addition of dietary ascorbic acid and selenium had the greatest protective effects on broiler kidney damage from cadmium consumption (Rambeck and Kollmer, 1990)

VANADIUM

The maximum tolerable level designated for vanadium in diets for poultry is 10 ppm. As opposed to fluorine which is less toxic to poultry. Romoser et al. (1960) reported vanadium concentrations as high as 6,000 ppm in some rock phosphate deposits. vanadium in a commercial tricalcium phosphate. Addition of the former source at 2% in the total diet for poultry would add 120 ppm vanadium, while addition of the latter would provide 28 ppm. Vanadium concentrations in commercial phosphate sources ranged from 26 to796 ppm for individual samples with most values falling within the range of 50 to 200 ppm. Vanadium also exists in a peroxide form which can mimic the action of insulin (Fantus et al., 1989). Deterioration of internal egg quality has been speculated to be mediated by an inhibition of magnum motility during egg formation (Eyal and Moran, 1984). Growth depression has been reported in birds consuming diets containing greater than 10 ppm vanadium for more than 21 to 28 days. Poorer albumin quality has been reported in eggs from laying hens consuming as little as 6ppm vanadium in a dicalcium phosphate (Sell et al., 1982). Hatchability was decreased by feeding 25 ppm vanadium as calcium orthovanadate fed for 20 weeks (Kubena et al., 1980), whereas 40 ppm were required to affect egg weight and egg specific gravity (Ousterhout and Berg, 1981). Kubena and Phillips (1982) reported no mortality in hens fed 50 ppm vanadium as calcium orthovanadate for five consecutive 28-day periods. The detrimental effect was generally overcome by addition of 5% cottonseed meal to the diet. Ousterhout and Berg (1981) had indicated that 20% cottonseed meal or 0.4 to 0.5% ascorbic acid in the diet protected hens from toxic effects of 40 ppm vanadium. A significant decline in Haugh units was observed within 3 days following feeding of a diet containing 20 ppm vanadium as ammonium metavanadate (Toussant and Latshaw, 1994). The detrimental effect on egg interior quality induced by feeding hens 10 ppm vanadium was partially improved by addition to the diet of the antioxidants ascorbic acid (100 ppm), vitamin E (200 IU/kg), or carotene (500ppm)(Miles et al.,1997). Hill (1988) reported that addition of 400 ppm copper or 100 ppm mercury overcame the growth depression observed in broiler chicks

fed for 19 days. Egg weight, shell thickness and shell breaking strength were not affected by vanadium. The percentage of outer thin albumen increased and the percentage of inner thin albumen decreased with increasing dietary vanadium.

MERCURY

Maximum tolerable level of mercury for poultry is only 2 ppm. Poultry can tolerate greater amounts of inorganic mercury than the organic methylated form. However, biomethylation of inorganic forms can occur in the environment or in the animal and increase the potential for toxicity. There are conflicting reports concerning the efficacy of selenium to reduce the toxicity of mercury (NRC, 1980). The dietary concentrations of selenium needed to have an effect (5 to 8 ppm) are above the maximum tolerable levels for this element and hence of little practical importance.

ZINC

It is extremely toxic to birds. Sources include galvanized cage wire, clips or staples, bird toy naps, zippers, keys, nails, plumbing nuts, nuts on animal transport cages, hardware cloth, padlocks, chrome, and some antirust paints, shampoos and skin preparations.

Symptoms / Clinical Signs

Clinical signs includes lethargy, shallow respiration, anorexia / reduced appetite, decreased body weight, weakness; falling of perch; unable to walk, stand or fly straight, diarrhea, hemolytic anemia, kidney dysfunction, cyanosiss, possible liver and pancreatic abnormalities, feather pickings, pale mucous membranes and death.

COPPER

It is potentially toxic to birds although avian toxicity from this metal is less common. Acidic foods stored in copper containers may leach out copper, and occasionally copper piping for water is a potential source of increased copper in the diet if the water is slightly acidic and has been allowed to remain in contact with the piping for some length of time. Allowing the water from the tap to run for a few minutes before filling the water dishes will prevent this problem. **Tin** (not galvanized), **steel and iron** (not treated with antirust paints) are not toxic to birds. **Brass** is an alloy of copper and zinc. Both of these metals are potentially toxic to birds. Brass padlocks are probably not a problem for cages of small birds that are unlikely to chew the padlock. However, they should be avoided around larger birds that are able to chew them.

MAXIMUM TOLERABLE LEVELS (PPM) OF TRACE MINERAL ELEMENTS FOR POULTRY

ELEMENT	MAXIMUM TOLERANCE(ppm)
Aluminium	200
Arsenic	50
Inorganic Organic	100
Cadmium	0.5
Chromium	1000
Chloride Oxide	3000
Copper	300
Fluoride	150,200
Lead	30
Mercury	2
Molybdenum	100
Nickel	300
Vanadium	10
Zinc	1000

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