Introduction

Poultry require at least 36 dietary nutrients at appropriate concentrations And balance. Worldwide, the majority of poultry diets are based on soymeal as the primary protein source, and grains such as corn, sorghum, or wheat, as the primary energy source. To prevent nutrient deficiencies, grain-soy based diets are typically supplemented with concentrated sources of methionine, sodium chloride, calcium, phosphorus, zinc, manganese, copper, iron, selenium, and all of the essential vitamins. Nutrient deficiencies or toxicities most often result from errors in diet formulation or milling and these supplemented nutrients are the most probable causes of nutritional problems. Marginal deficiencies or toxicities often result in suboptimal growth, impaired resistance to infectious diseases, decreased egg production, or lowered hatchability, but rarely result in clear deficiency symptoms.

Water

Poultry can survive much longer without food than without water. An insufficient amount of water results in decreased growth, egg production, and resistance to heat stress. More specific signs include hemoconcentration, increased erythrocyte osmotic fragility, visceral Urates and renal disease.

The quantity of water consumed by chicks is correlated directly with The salt (NaCl) and protein content of their diet. Sodium or potassium bicarbonates cause similar increases in water intake of broiler chicks. Chlorine and phosphorus also increase water intake, but not as much as sodium or potassium, and calcium has little effect. Excess water excretion often results in wet litter, which predisposes birds to foot pad dermatitis

Proteins and Amino Acids

Commercial diets usually are formulated using a “least cost” approach, and meeting the essential amino acid requirements greatly impacts the cost of the diet. For this reason, the limiting amino acids in the diet are typically supplied with very little margin of safety. The protein requirement represents the collective need for 10 absolutely **essential amino acids (arginine, histidine, isoleucine, leucine, lysine, methionine, phenylalanine, threonine, tryptophan, and valine)**, 2 amino acids (cysteine and tyrosine) that can be synthesized from essential amino acids, 2 amino acids that are essential for the young chick (glycine or serine and proline), plus additional amino acids to satisfy the nitrogen requirement for synthesis of nonessential amino acids, purines, pyrimidines, and other nitrogenous compounds.

Practical ingredients usually are limiting in 1 or more amino acids. Grains such as corn, milo, and wheat are most deficient in lysine, whereas soybean meal is most deficient in methionine. It is often cost effective to supply these limiting amino acids in the form of pure amino acids, especially lysine and methionine. Threonine, tryptophan, arginine, and isoleucine also may be supplied as pure amino acids when economically advantageous.

The effects of essential amino acid deficiencies are nonspecific: **reduced growth reduced feed consumption, lower meat yield, decreased egg production and egg size, and loss of body weight in adults. The decrease in feed intake due to a severe deficiency occurs within hours of consumption of a deficient diet. Some amino acids have additional effects. Methionine deficiency may exacerbate choline or vitamin B12 deficiencies due to its role in methyl group metabolism. Lysine deficiency causes impaired pigmentation of bronze turkey**

poults and can result in stunting and retarded development in chicks. Arginine deficiency tends to cause the wing feathers to curl upward, giving the chick a distinct ruffled appearance. Several other amino acids have been reported to affect feather growth and structure.

Carbohydrates

Starch is the primary source of metabolizable energy in practical poultry Diets. Non starch polysaccharides (NSP) are not digested by the endogenous enzymes and may have either positive or negative effects on Intestinal health depending on their chemical and physical properties.

Some grains (e.g., wheat, barley, and rye), legumes, have NSP that increase the susceptibility of poultry to several important bacterial and parasitic pathogens. Grain NSP have been implicated in susceptibility to bacterial infection, *Clostridium perfringens*,and *Salmonella*. This effect of NSP is thought to be due to increased viscosity of the digesta in the ileum and ceca, inducing epithelial inflammation and providing nutritional substrates that facilitate microbial overgrowth. In recent years, the diminishing use of antibiotics in the feed of poultry has exacerbated this problem unless exogenous enzymes are added to break down the NSP. **Intestinal lactase activity is low in chickens**; this limits the amount of lactose that can be tolerated. Milk byproducts such as whey, are excellent sources of protein and some vitamins but excessive levels in the diet cause growth depression and severe diarrhea.

Fats

Fats are important in the diet of poultry as concentrated sources of **energy** and sources of the essential nutrients **linoleic acid and linolenic acid**.

Linoleic acid deficiency in young chicks results in suboptimal growth and enlarged fatty livers, and in laying hens results in lowered egg production, egg size, and hatchability .

Unsaturated fatty acids may undergo oxidative rancidity, with multiple

effects: Essential fatty acids are destroyed; aldehydes that are formed may react with free amino groups in proteins, reducing amino acid availability; and reactive oxygen intermediates generated during rancidification may destroy activities of vitamins A, D, and E and water soluble vitamins such as biotin. The addition of synthetic antioxidants or plethoric levels of tocopherols to poultry feeds provides protection to essential fatty acids and other essential nutrients.

Vitamins

- ***Vitamins*** are organic compounds required in minute amounts for normal growth, production, reproduction, and health. The vitamins function as cofactors for enzymes, hormones (e.g., vitamins A, D), or antioxidants (previtamin A and vitamin E).

  Note:

- Vitamins regulate body processes, help the body resist disease, and improve health in general.
- Vitamins are either ***fat soluble*** **like A,D,E.K** (stored in body fat and released when needed), or ***water soluble*** **(**not stored in tissues, must have constant supply) **like B, B1, B2, B6 & B12,Niacin, Folic Acid and C**
- Essential vitamins include A, D3 , E, K, Biotin, Choline, Folacin, Niacin, Pantothenic Acid, Riboflavin, Thiamin, B-6, and B-12.

The deficiency of vitamins symptoms is often similar. Usually the overall growth rate of young birds is impaired and tissues that turn over rapidly are most affected, including the growth plate of bones, feather follicles, the epithelia surfaces of skin, and hematopoietic tissues. Common symptoms that arise from defects in these tissues are dyschondroplasia, chondrodystrophy, dermatitis, poor feathering, anemia, and increased susceptibility to infectious diseases. For

example, chondrodystrophy (“perosis”) occurs in young birds when the diet is deficient in choline, nicotinic acid, pyridoxine, biotin, folic acid, zinc, or manganese. Chondrodystrophy is an anatomic deformity of leg bones of young birds that is characterized by decreased linear bone growth, enlargement of the tibiometatarsal joint, and twisting or bending of the distal end of the tibia and proximal end of the metatarsus. It appears that chondrodystrophy occurs because the growth plate is an avascular tissue and chondrocyte growth and maturation depends on nutrient diffusion into the connective tissue matrix, which makes this region very susceptible to deficiencies of a variety of nutrients that have unrelated biochemical functions. The fat-soluble vitamins A, D, and E are stored relatively well and birds can withstand long periods of depletion before deficiency symptoms manifest. Birds with adequate stores of most of the vitamins also can withstand feed restriction without deficiency symptoms because tissue catabolism liberates vitamins from muscle, fat, and other tissues. Thus, vitamin deficiencies are not usually important determinants of the clinical signs of feed restriction or starvation. The level of deposition of the vitamins into the egg directly reflects the amount in the diet. Marginal deficiencies of vitamins may not decrease egg production but are likely to influence embryonic development.

Excretion rates of vitamins A and D are limited and toxicity of these vitamins is a frequent problem. Water-soluble vitamins are not stored to a large extent, excesses are excreted mostly in urine, and they are relatively nontoxic.

Vitamin A

From a nutritional perspective, vitamin A is the most challenging of the vitamins because it is deficient in most feedstuffs and is among the most likely to become toxic upon over-supplementation. Corn has moderately high levels of beta carotene, which is converted to retinol, so diets that are high in corn are less likely to cause severe deficiencies compared to diets based on wheat or sorghum.

With the **exception of its role in vision**, retinol and retinal must be converted to retinoic acid for its required function that regulate cell replication, differentiation, and death. Retinoic acid is among the most important early-stage embryo, the induction of digit formation, and the connection of the extra-embryonic blood supply to the forming heart. Retinoic acid also important in epithelial differentiation, bone modeling, spermatogenesis, and leukopoesis. For example, retinoic acid induces the differentiation of epithelial basal cells into the cubodial, columnar, and goblet cells characteristic of the soft, moist epithelia. When vitamin A is deficient, the basal cells differentiate into squamous cells (squamous metaplasia), which may become stratified and keratinized to form a hard, dry epithelia characteristic of the skin. Vitamin A deficiency can affect the epithelia of the esophagus, intestines, cloaca, respiratory tract, conjunctiva of the eye, ureters, cloacal bursa (bursa of Fabricius), and vaginal region of the oviduct.

Retinal, the aldehyde form of retinol, covalently binds to opsins in sensory cells of the retina and plays an essential role in the detection of light, especially in dim lighting .

Vitamin A Deficiency

Clinical Signs.

Vitamin A deficiency in the embryo results in a grossly abnormal cardiovascular system, Embryos often die during the first week of incubation. Those embryos that survive to term may be too weak to hatch or die shortly thereafter

In growing chicks and poults, severely deficient birds display decreased growth, depression, inappetence, unthriftiness, increased mortality due to infections, unsteady gait, and postural imbalance . In the absence of accompanying infectious diseases, deficiencies of vitamin A may cause epithelial damage in the oropharynx and esophagus without marked growth depression. **In broilers**, severe deficiencies result in hyper proliferation and decreased maturation of enterocytes, a decrease in the number of goblet cells, and blunting of villi. Impaired growth rate of deficient broiler chicks appears to be secondary to diminished digestive function. In acute vitamin A deficiency, lacrimation usually occurs, and a caseous material may be seen under the eyelids. Deficiencies of vitamin A are deleterious for development of the cloacal bursa and thymus.

In layer ruffled feathers, a sharp drop in egg production, increased incidence of blood spots in eggs, and increased susceptibility to infections, followed by swelling of the nictitating membrane and a watery discharge from the nostrils and eyes .As the deficiency continues, milky white, caseous material accumulates in the eyes, displacing the nictitating membrane, and eyelids may become stuck together. When chronic, infection of the eye results in necrosis and irreversible blindness.

Gross lesion.

Vitamin A-deficiency lesions first appear in the esophagus and pharynx and are confined largely to mucous glands and their ducts. The original

epithelium is replaced by a keratinizing epithelium (i.e., squamous metaplasia;) that blocks ducts of the mucous glands, causing them to become distended with secretions and necrotic materials. Small, white nodules may be found in the nasal passages, mouth, esophagus, and pharynx and may extend into the crop. Nodules range in size from microscopic lesions to 2 mm in diameter . As the deficiency progresses, lesions enlarge, are raised above the surface of the mucous membrane, and have a depression in the center. Small ulcers surrounded by inflammatory products may appear at the site of these lesions.

This condition resembles certain stages of fowl pox, and the 2 conditions can be differentiated only by microscopic examination. infectious coryza, fowlpox, or infectious bronchitis. In vitamin A deficiency, thin diphtheritic membranes and nasal plugs usually are limited to the cleft palate and its adjacent epithelium. They may be removed easily without bleeding. Atrophy and degeneration of the respiratory mucous membrane and its glands occur. Later, the original epithelium is replaced by a stratified squamous keratinizing epithelium. In early stages of vitamin A deficiency in chickens.

Exudate also may fill sinuses and other nasal cavities, causing swelling of one or both sides of the face. Mucous membranes, cleared of inflammatory products, appear thin, rough, and dry. Chronic vitamin A deficiency causes damage to the kidney tubules, which leads to visceral urate deposits (e.g., “visceral gout”) in severe cases .

Treatment of Deficiency.

Poultry found to be severely deficient in vitamin A should be given a stabilized vitamin A preparation at a level of approximately 10,000 IU vitamin A/ kg of ration. It also may be provided in the water or via injection. Absorption of vitamin A is rapid; therefore, chickens or turkeys not in advanced stages of deficiency should respond promptly, except for

blindness due to xerophthalmia, which is often permanent.

Hypervitaminosis A

Mistakes in formulation of vitamin premixes can result in in broilers signs of toxicity include slow growth, an unsteady gait, reluctance to walk, and a higher incidence of tibial dyschondroplasia as indicated by widened epiphyseal growth plates with irregular tunneling by blood vessels. Severe toxicity results in anorexia, conjunctivitis, adhesions of the eyelids.

Vitamin D

Vitamin D acts as a hormone to regulate calcium and phosphorus metabolism in poultry and important for the formation and maintenance of a normal skeleton and for strong egg shells. Vitamin D3 (cholecalciferol) can be synthesized from 7-hydrocholesterol in the skin under the influence of ultraviolet light. Although this synthesis can reduce the dietary requirement for vitamin D. functions Vitamin D3 (1,25(OH)2D3).in the regulation of calcium metabolism by stimulating the intestinal absorption of calcium, influencing osteoblast and osteoclast activity, which is important in bone mineralization. And increasing renal tubular reabsorption of alcium, differentiation in the immune system, kin, and growth plate of the bone also which is important in calcium absorption and egg shell mineralization.

Vitamin D Deficiency

Clinical Signs.

Hatchability is reduced markedly by vitamin D deficiency. Embryonic Mortality is greatest late during incubation.

The first signs of vitamin D deficiency in **growing chicks** or poults are Slower growth. As the deficiency advances rickets become evident as severe fragility and bending of long bones due to poor mineralization

Beaks and claws become soft, feathering is poor, and birds walk with obvious effort and take a few unsteady steps before squatting on their hocks, which they rest upon while swaying slightly from side to side. Many of the signs of a vitamin D deficiency may be similar to calcium Deficiency, and analysis of levels of these two nutrients in the diet, or 25- (OH)D3 in blood plasma, will confirm the cause.

In confined **laying hens**, signs of deficiency begin to occur as soon as 2 Weeks after they are deprived of vitamin D. The first sign is a marked Increase in the number of **thin-shelled and soft-shelled eggs, followed soon after by marked decrease in egg production**.

During periods of extreme leg weakness, hens show a characteristic posture that has been described as a "*penguin-type squat." Later, beak, claws, and keel become very soft and pliable. The sternum usually is bent, and ribs lose their normal rigidity and turn inward at the junction of the sternal and vertebral portions, producing a characteristic inward curve of the ribs along the sides of the thorax.*

Pathology or gross lesion

In laying and breeding chicken and turkey hens receiving deficient vitamin D, characteristic changes observed on necropsy are confined to bones and parathyroid glands. The latter become enlarged from hypertrophy and hyperplasia. Bones are soft and break easily. knobs are present on the inner surface of the ribs at the costochondral junction (rachitic rosary) .Many ribs show evidence of pathologic fracture in this region. In chronic vitamin D deficiency, marked skeletal distortions become apparent. The spinal column may bend downward in the sacral and coccygeal region; the sternum usually shows a lateral bend and an acute cavity near the middle of the breast. The beak may be soft and

pliable. Bones of vitamin D-deficient chicks have reduced calcium content lead to loss of strength leads to fractures.

Treatment of Deficiency.

Feeding a single massive dose of 15,000 IU vitamin D3 kg of ration. In giving massive doses to rachitic chicks, it should be remembered that excess vitamin D can be toxic.

Hypervitaminosis D

Activate calcium mobilization. Elevated rates of calcium absorption and mobilization from the bone because elevated levels of calcium in body fluids, resulting in soft-tissue calcification, cellular degeneration, and inflammation.

Vitamin E

The term vitamin E refers to 2 groups of compounds that have antioxidant activity in cellular membranes: the tocopherols and the tocotrienols . Vitamin E is usually supplemented to poultry diets as α-tocopherol. Vitamin E is a very effective antioxidant it protects unsaturated fatty acids, including the essential fatty acids vitamin E and selenium have interactive effects on cell viability.

Deficiency of Vitamin E

During vitamin E deficiency, oxidative damage may cause **encephalomalacia, exudative diathesis, and nutritional myopathy (muscular dystrophy)** in chicks Meat and eggs from vitamin E-deficient birds have increased susceptibility to oxidative rancidity during processing and storage. vitamin E and selenium have interactive effects on cell viability and antioxidant.

Clinical Signs, and Pathology.

Encephalomalacia in Chicks.

Encephalomalacia is a nervous syndrome characterized by ataxia or paresis , backward or downward retractions of the head (sometimes with lateral twisting), forced movements, decreasing coordination, rapid contraction and relaxation of the legs, and finally complete prostration and death. Low levels of dietary selenium or high levels of dietary long-chain polyunsaturated fatty acids, especially linoleic acid, increase the severity of encephalomalacia .

The cerebellum, striatal hemispheres, medulla oblongata, and mesencephalon are affected most commonly. In chicks killed soon after the appearance of signs of encephalomalacia, the cerebellum is softened and swollen, and the meninges are edematous . Minute hemorrhages are often visible on the surface of the cerebellum.

A day or 2 after signs of encephalomalacia appear, necrotic areas present a green-yellow opaque appearance. One or 2 days later, the cerebellum may become pale and shrunken.

Histologically, lesions include circulatory disturbances (ischemic necrosis), demyelination, and neuronal degeneration .Meningeal, cerebellar, and cerebral vessels are markedly hyperemic, and a severe edema usually develops. Capillary thrombosis often results in necrosis of varying extent. Degenerative neuronal changes occur everywhere but are most prominent in Purkinje cells.

Exudative Diathesis.

Exudative diathesis is an edema of subcutaneous tissues associated with abnormal permeability of capillary walls. In severe cases, chicks stand with their legs far apart as a result of accumulation of fluid under the ventral skin. This green-blue viscous fluid is seen easily through the skin

and usually contains some blood components from slight hemorrhages that appear throughout the breast and leg musculature and in the intestinal walls. In laying hens, the thigh muscles are more susceptible to deficiency lesions than the breast muscles and show degenerative muscle fibers including calcium deposits, Vitamin E deficiency in the presence of adequate dietary selenium does not result in severe signs of exudative diathesis. The combined deficiency appears to be due to their complementary antioxidant functions.

Nutritional Myopathy (Muscular Dystrophy).

When vitamin E deficiency is accompanied by a sulfur amino acid deficiency, chicks show signs of nutritional myopathy, particularly of the breast muscle, at about 4 weeks-of-age. The condition is characterized by light-colored streaks of easily distinguished affected bundles of muscle fibers in the breast .A similar dystrophy occurs throughout all skeletal muscles of the body in vitamin E-deficient ducks. The initial histologic change is hyaline degeneration. Mitochondria undergo swelling, coalesce, and form intracytoplasmic globules. Later, muscle fibers are disrupted transversely. Extravasation separates groups of muscle fibers and individual fibers.

Treatment of Deficiency.

If not too far advanced, exudative diathesis and nutritional myopathy in chicks are readily reversed by the administration of vitamin E and selenium by injection, by oral dosing, or in feed. Encephalomalacia may or may not respond to treatment with vitamin E, depending on the extent of damage to the cerebellum.

Vitamin K

Vitamin K is required is a cofactor in the Prothrombin function, osteocalcin, and several other calcium binding proteins. In the absence of

vitamin K, an abnormal prothrombin lacking γ-carboxyglutamic acid is secreted into the blood by the liver.

Because prothrombin is an important part of the blood-clotting Mechanism, deficiency of vitamin K results in markedly prolonged Blood-clotting time.

Clinical Signs and Pathology of Deficiency

In growing chicks, signs of vitamin K deficiency occur as early as 2–3 weeks; hemorrhages appear externally at areas that receive abrasions such as the feet and wings. Internally, petechial hemorrhages in the liver and erosion of the kaolin lining of the gizzard may occur. Chicks show an anemia that may result partly from loss of blood . Although blood-clotting time is a fairly good measure of vitamin K deficiency, a more accurate one is obtained by determining prothrombin time. Inadequate vitamin K in breeder diets causes increased embryo mortality late in incubation . Dead embryos appear hemorrhagic.

Treatment of Deficiency

Within 4–6 hours after vitamin K is administered to deficient chicks, blood clots normally, but recovery from anemia or disappearance of hemorrhages cannot be expected to take place promptly.

Thiamin (Vitamin B1)

The active form of thiamin is thiamin pyrophosphate, which is an important cofactor in decarboxylation, dehydrogenase, and transketolase reactions. These enzymes are critical in the metabolism of carbohydrates, lipids, and branched-chain amino acids. Deficiency of thiamin leads to extreme anorexia, polyneuritis, and death. Adequacy can be tested by the erythrocyte transketolase activation coefficient assay or thiamin concentrations in blood .

Clinical Signs and Pathology of Deficiency

Anorexia is usually the first sign of deficiency and can quickly become severe, resulting in weight loss and leg weakness. Polyneuritis is observed in mature chickens approximately 3 weeks after they are placed on a thiamin-deficient diet. In young chicks, it may appear before 2 weeks of age. Onset is sudden in young chicks but more gradual in mature birds. Adult chickens often show a blue comb. As the deficiency progresses paralysis of muscles occurs, beginning with the flexors of the toes and progressing upward, affecting the extensor muscles of legs, wings, and neck. The chicken characteristically sits on its flexed legs and draws back the head in a "**stargazing**" position . Retraction of the head is due to paralysis of the anterior muscles of the neck. The chicken soon loses the ability to stand or sit upright, and it falls to the floor, where it may lie with the head still retracted.

Treatment of Deficiency

Chickens suffering from thiamin deficiency respond in a matter of a few hours to oral administration of the vitamin. Because thiamin deficiency causes extreme anorexia, supplementing feed with the vitamin is not a reliable treatment until after chickens have mostly recovered.

Riboflavin (Vitamin B2)

Riboflavin is a cofactor in more than 50 enzymes, many of which are vitally associated with oxidation-reduction reactions involved in cell respiration.

Clinical Signs and Pathology of Deficiency

When chicks are fed a diet deficient in riboflavin, they grow very slowly and become weak and emaciated, and diarrhea develops between the first and second weeks. Chicks may not walk except when forced to, and then they frequently walk on their hocks with the aid of their wings. Leg paralysis may be more prevalent than curled-toe paralysis . **Toes curl**

inward when both walking and resting. Chicks are usually found in a resting position with drooping wings. Leg muscles are atrophied and flabby, and the skin is dry and harsh. Young chicks in advanced stages of deficiency do not move around but lie with their legs sprawled out. Riboflavin deficiency in young turkeys is characterized by poor growth, poor feathering, leg paralysis, and encrustations in the corners of the mouth and on the eyelids. Severe dermatitis and edematous swelling may occur on the feet and shanks.

In severe cases of riboflavin deficiency, chicks show marked swelling and softening of sciatic, cervical, and lumbar spinal nerves . Sciatic nerves usually undergo the most pronounced changes, sometimes reaching a diameter 4–5 times normal size. Histologically, affected nerves show degenerative changes in myelin sheaths of the main peripheral nerve trunks .

Treatment of Deficiency

Two 100-µg doses of riboflavin should be sufficient for treatment of riboflavin-deficient chicks or poults, followed by incorporation of an adequate level in the ration. When the curled-toe deformity is of long standing, however, irreparable damage has occurred and administration of riboflavin no longer cures the condition.

Pantothenic Acid (vitamin B_5)

Pantothenic acid is a component of coenzyme A, which is involved in the formation of citric acid in the Krebs cycle, synthesis and oxidation of fatty acids, oxidation of keto acids resulting from deamination of amino acids, acetylation of choline, and many other reactions.

Clinical Signs and Pathology of Deficiency

Signs of pantothenic acid deficiency in chicks are difficult to differentiate from those of a biotin deficiency; deficiencies of either result in dermatosis, ruffled and broken feathers, chondrodystrophy, poor growth,

and mortality. Chicks are emaciated and crusty, scab-like lesions appear in corners of the mouth . Eyelid margins are granular, and small scabs develop on them. Eyelids frequently are stuck together by a viscous exudate and vision is restricted. There is slow sloughing of the keratinizing epithelium of the skin. small cracks and fissures appear at these points. These cracks and fissures enlarge and develop, so chicks move about very little. In some cases, skin layers of the feet of deficient chicks cornify, and wart-like protuberances develop on the balls of the feet. Necropsy shows the presence of a pasty substance in the mouth and an opaque gray-white exudate in the proventriculus. The liver is hypertrophied and may vary in color from a faint to dirty yellow. The spleen is atrophied slightly. Kidneys are somewhat enlarged. Nerves and myelinated fibers of the spinal cord show myelin degeneration. These degenerating fibers occur in all segments of the cord down to the lumbar region.

Embryos from pantothenic acid-deficient hens have high mortality and show hemorrhages and severe edema.

Treatment of Deficiency

Pantothenic acid deficiency appears to be completely reversible, if not too far advanced, by oral treatment or injection with the vitamin followed by restoration of an adequate level in the diet.

Nicotinic Acid or Niacin (vitamin B3)

Nicotinic acid is the vitamin component in 2 important coenzymes, nicotinamide adenine dinucleotide (NAD) and nicotinamide adenine dinucleotide phosphate (NADP), that are required for more than 200 reactions involved in the metabolism fatty acids, carbohydrates, amino acids, and nucleic acids. Niacin can be synthesized from tryptophan, Excessive supplementation should be avoided because causes decreased bone thickness and strength.

Clinical Signs and Pathology of Deficiency

Severely deficient chicks show anorexia, slow growth, inflammation of the oral cavity, dermatitis, poor feathering, and an enlargement of the hock joints (**chondrodystrophy**) and bowing of the legs (**perosis**). The perosis caused by a niacin deficiency is not as severe as that due to a manganese deficiency and the Achilles tendon rarely slips from its condyles. The tongue of deficient chicks may turn black due to necrosis. Poults and ducklings may develop diarrhea in addition to the above symptoms. however, niacin is needed to optimize egg weight and hatchability .

Treatment of Deficiency

Supplementing a deficient ration with required amounts of nicotinic acid has little or no effect on cases that have progressed to the extent that the tendon has slipped from its condyles (chondrodystrophy) or on advanced cases of enlarged hock disorder in adult tom turkeys.

Pyridoxine (Vitamin B6)

Pyridoxal phosphate and pyridoxamine phosphate are cofactors involved in transamination and decarboxylation of amino acids as well as glycogen mobilization.

Clinical Signs and Pathology of Deficiency

Pyridoxine deficiency is relatively rare and most likely to occur with diets containing high levels of protein and especially methionine . Severely pyridoxine-deficient chicks show depressed appetite, poor growth, **chondrodystrophy** resulting in bone curvature,and characteristic nervous signs .These signs may be distinguished from those of encephalomalacia (vitamin E deficiency) by the relatively greater intensity of activity of the chicks during a seizure, which results in complete exhaustion and often death. Clinical signs of pyridoxine deficiency in ducklings include poor growth and food consumption, weakness, anemia,convulsions, and death .

A pyridoxine deficiency causes a defect in collagen fibers in cortical bone and articular cartilage matrix and increased solubility of proteoglycans and collagen . These structural defects apparently cause chondrodystrophy and osteoarthritis in deficient chicks. In adult birds, pyridoxine deficiency causes marked reduction of egg production and hatchability, as well as anorexia and weight loss.

Biotin

Biotin is a cofactor in carboxylation and decarboxylation reactions involved in the metabolism of lipids, glucose, and some amino acids.

Clinical Signs and Pathology of Deficiency

The signs of a biotin deficiency are variable due to the marked influence of other dietary factors, especially the amount and type of fat. In chicks and poults, a deficiency causes slow growth, ruffled feathers, chondrodystrophy, and dermatosis of the feet and skin around the beak and eyes . Many of these signs are similar to those of a pantothenic acid deficiency and a differential diagnosis requires the analysis of vitamin levels in the diet. Dermatitis of the footpads without involvement of the head (footpad dermatitis) has become an economically important disease of broilers and poults. Footpad dermatitis can be exacerbated by a biotin deficiency but is most often caused by environmental factors, such as wet litter, and is not usually prevented by extra dietary biotin .

Defects in bone growth due to biotin deficiency include shortened tibiae, higher bone density and bone ash, and an abnormal pattern of bone modeling characterized by the mid-diaphyseal cortex being thicker than the lateral side.

In broiler chicks and poults, a biotin deficiency may lead to sudden death without external lesions. Chicks become lethargic and develop hepatic and decreased plasma glucose, increased plasma free fatty acids, and increased ratio of fatty acids in liver and adipose tissue. This

condition is known as fatty liver and kidney syndrome (FLKS) and can occur with a marginal deficiency. Biotin has been suspected of having a role in “acute death syndrome” (or “sudden death syndrome”) in broiler chickens .Biotin deficiency alters the unsaturated fatty acid profile in tissue lipids in a manner suggestive of impaired conversion of linoleic acid to arachidonic acid.

Treatment of Deficiency

Injection or oral administration of a few micrograms of biotin prevents biotin deficiency signs in deficient chicks and turkey poults, but reversal of existing signs has not been explored.

Folic Acid (Folacin) B9

Folic acid is a cofactor of the enzyme system involved in the transfer of methyl groups of such important metabolites as choline, methionine, purines, and uric acid. Folic acid is required for normal nucleic acid metabolism and formation of the nucleoproteins required for cell replication. There are more biologically active forms of folate than any other vitamin, making their quantification in foods difficult.

Clinical Signs and Pathology of Deficiency

Folic acid deficiency in chicks is characterized by poor growth, very poor feathering, macrocytic anemia, leukopenia, and chondrodystrophy. Poorly developed feathers with weak and Folic acid are required for pigmentation in feathers of Rhode Island Red and black leghorn chicks.

Treatment of Deficiency

A single intramuscular (IM) injection of folic acid returns hemoglobin values and growth rates to normal within 1 week. Addition of 5 mg folic acid/kg feed gives similar results. Folate absorption is relatively well regulated and high dietary levels are well tolerated.

Vitamin B12 (Cobalamin)

Vitamin B12 is involved in nucleic acid, carbohydrate, and fat

metabolism. One of its main enzyme functions involves isomerization of methylmalonyl coenzyme A to form succinyl CoA.

Clinical Signs and Pathology of Deficiency

Vitamin B12 deficiency results in slow growth, decreased efficiency of feed utilization, mortality, and reduced egg size and hatchability . Signs of a folate deficiency also may occur due to trapping of folate. Vitamin B12 deficiency has been reported to cause myelin degeneration in chicks. Chondrodystrophy may occur in vitamin B12-deficient chicks or poults when their diets lack choline, methionine, as sources of methyl groups. Vitamin B12-deficient embryos have a peak in mortality at day 17 of incubation and show reduced size, myoatrophy of the legs, diffuse hemorrhages, chondrodystrophy, edema, and fatty liver .

Treatment of Deficiency

IM injection of 2 µg vitamin B12/hen increases hatchability of eggs from hens with a deficiency of vitamin B12. Oral dosing also is effective. Similarly, B12 injections of young chicks followed by supplementation of

the diet reverses the deficiency signs.

Choline

Choline has 3 primary functions in birds: **structural as part of phospholipids**; **neurotransmitter as part of acetylcholine**; and **methyl donor, via betaine, in the synthesis of methionine, creatine, carnitine, and N-methylnicotinamide**. Choline can be synthesized by chickens but the rate is insufficient for growth or egg production.

Clinical Signs and Pathology of Deficiency

In addition to poor growth, the most consistent lesion of choline deficiency in chicks and poults is **chondrodystrophy** and . Young

turkeys have a high requirement for choline and, therefore, will show a high incidence of severe chondrodystrophy.

Chondrodystrophy is first characterized by pinpoint hemorrhages and a slight puffiness about the hock joint, followed by an apparent flattening of the tibiometatarsal joint caused by rotation of the metatarsus. The metatarsus continues to twist and may become bent or bowed until it is out of alignment with the tibia. When this condition exists, the leg cannot adequately support the weight of the bird. The articular cartilage is deformed and the Achilles tendon (tendo calcaneous) may slip from its condyles.

Treatment of Deficiency

If choline deficiency is noted in chicks or poults before severe signs of chondrodystrophy have developed, the deficiency can be treated by supplementing the ration with sufficient choline to meet the requirements. After the tendon has slipped in chicks or poults suffering from choline deficiency, the damage is irreversible.

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Calcium and Phosphorus

Calcium (Ca) and phosphorus (P) are closely associated in metabolism, particularly in bone formation. The major portion of dietary calcium is used for bone formation in growing chicks or poults and for eggshell formation in mature hens. Calcium also is essential for clotting of blood and it is required along with sodium and potassium for normal contraction of cardiac muscle. Calcium is an integral constituent of cell signaling and regulatory pathways. In addition to its role in bone formation, phosphorus is an essential component of phospholipids, purine nucleotides, and many other macromolecules, and is involved in the regulation of many cellular and metabolic processes including acid-base

balance. The availability of phosphorus can be increased by dietary phytase of microbial origin.

Efficient utilization of calcium and phosphorus depends on the presence of an adequate amount of vitamin D in the diet. An excess of calcium induces a phosphorus deficiency. Likewise, an excess of phosphorus induces a calcium deficiency.

Calcium and Phosphorus Deficiency

Deficiencies of calcium and phosphorus in the diet of growing broiler chicks cause **rickets**.

A phosphorus deficiency results in similar calcium deficiency In severe cases of rickets, chicks display a spraddle-legged posture, folding fractures, and bowing or rotation of the tibiotarsus. In a study of field rickets in broilers, calcium deficiency typically causes accumulation of proliferating chondrocytes, whereas a phosphorus deficiency causes accumulation of hypertrophic chondrocytes in the metaphyseal zone .

Severely phosphorus-deficient chicks have increased respiratory rates, polycythemia, ascites, and decreased CO2 and O2 levels, presumably due to poor rib strength and infolding, which interfere with respiratory movements of the rib cage .

In laying hens, calcium deficiency results in reduced egg production and thin-shelled eggs as well as calcium mobilization from bones, first by complete removal of the medullary bone, followed by a gradual removal of the cortical bone . Finally, bones become so thin that spontaneous fractures may occur, especially in vertebrae, tibia, and femur. This condition may be associated with a syndrome commonly termed “**cage layer fatigue.”** Although a marginal calcium deficiency has often been found to be a triggering agent in cage layer fatigue, the syndrome is not due to a simple deficiency of calcium but also involves other etiologic factors .

Excess Calcium or Phosphorus

Excess calcium causes a phosphorus deficiency and the development of rickets in broilers. In leghorn pullets, excess calcium causes urolithiasis, nephrosis, and visceral urate deposition.

An excess of calcium interferes with the digestibility of other minerals in addition to phosphorus, including magnesium, manganese, and zinc; this may result in secondary deficiencies. High dietary levels of these minerals decrease the toxicity of calcium. Excess phosphorus causes a thinning of the egg shell independent of calcium status.

Magnesium

Magnesium (Mg) acts as a cofactor or an activator of many critical enzymes including the reactions involving ATP that energize all major metabolic pathways. Magnesium levels in many feedstuffs are adequate, so magnesium is not heavily supplemented to poultry diets and deficiencies are rare.

Deficiency

Acute magnesium deficiencies are severe, beginning with lethargy, slow growth, and hypomagnesemia, followed by hyperexcitability if disturbed. Tibiae have decreased magnesium and increased calcium content and exhibit abnormalities including thickening of trabeculae, increased retention of cartilage cores, and the occurrence of elongated and inactive osteocytes in the metaphysis. Deficient chicks have thickening of the cortex, the presence of elongated inactive osteocytes, and enlargement of Haversion canals within the diaphysis. The epiphyseal plate, however, appears normal. The parathyroid appears hyperactive, perhaps in response to the hypocalcemia that is characteristic of magnesium deficiency.

Excess Magnesium

Under certain conditions rations may contain excess magnesium, producing detrimental effects including reduced growth rate and bone ash

in chicks and decreased egg size, eggshell thinning, and diarrhea in hens. The maximum tolerable level of magnesium in the diets of poultry is about 0.5% for growing birds and 0.75% for laying hens .Diets with marginal levels of phosphorus enhance the sensitivity of hens to toxicosis

Sodium and Chlorine (Salt)

Sodium (Na) is found chiefly in extracellular fluids. Sodium is connected intimately with maintenance of membrane potentials, cellular transport processes, and the regulation of the hydrogen ion concentration of blood. Chloride, the major mineral anion in extracellular fluids, plays a role in fluid, ionic, and acid-base balance. Many feedstuffs are deficient in sodium and chloride so salt is supplemented to most poultry diets.

Clinical Signs and Deficiency

Chicks receiving diets deficient in sodium not only fail to grow but also develop softened bones, corneal keratinization, adrenal hypertrophy, and decrease in both plasma and special fluid volumes. Cardiac output drops, mean arterial pressure falls, the hematocrit increases, elasticity of subcutaneous tissue decreases, adrenal function is impaired, and a state of shock results, which if uncorrected, terminates in death. Lack of salt in the diet of laying hens results in an abrupt decrease in egg production, reduced egg size, loss of weight, and cannibalism. In turkeys, low dietary salt impairs egg production and hatchability. A chloride deficiency in chicks causes extremely poor growth rate, high mortality, hemoconcentration, dehydration, reduced blood chloride, and nervous signs characteristic of Cl deficiency. When startled, chicks fall forward with their legs outstretched behind them and lay paralyzed for several minutes, then appear quite normal until frightened.

Excess Salt

Large amounts of salt in the ration are toxic to chickens and turkeys. The lethal dose is approximately 4 g/kg bodyweight. Young chicks appear to

be more susceptible to toxic effects of salt than are older chickens. Poultry are much less tolerant to salt supplied via the water than by the diet. Signs of salt intoxication include intense thirst, pronounced muscular weakness, inability to stand, and convulsive movements preceding death. Hemorrhages and severe congestion occur in the gastrointestinal tract, muscles, liver, and lungs. Excess sodium results in ascites, right ventricular hypertrophy, and right ventricular failure in broiler chickens and turkeys ,and contributes to spontaneous cardiomyophathy in turkeys. Matterson et al. fed day-old poults graded quantities of salt for 23 days and observed edema and mortality at 4% salt but none at 2%. Young turkey poults have signs of salt toxicity at 1.85% salt, including respiratory distress, ascites, hydropericardium, hydrothorax, and sudden death . High levels of salt also cause the excretion of dilute urine and wet litter.

Potassium

Potassium (K) is found primarily in the intracellular compartment of the body. The soft tissues of the chicken contain more than 3 times as much potassium as sodium. Potassium has an essential role in the maintenance of membrane potential and cellular fluid balance. Potassium participates directly in numerous biochemical reactions and is necessary for normal heart activity, reducing contractility of the heart muscle and favoring relaxation. Many feedstuffs contain adequate levels of potassium so deficiencies are relatively rare.

Clinical Signs and Signalment of Deficiency

The main effect of potassium deficiency is overall muscle weakness characterized by weak extremities, poor intestinal tone with distention, cardiac weakness, and weakness of the respiratory muscles and their ultimate failure. Severely affected individuals may exhibit tetanic seizures followed by death. Low levels of potassium in laying diets cause

decreased egg production and eggshell thinning. In one experiment, a diet low in potassium produced low plasma potassium concentrations simultaneous with a high incidence of sudden death syndrome at the onset of egg production in restricted fed broiler breeder pullets . The hypothesis that low dietary potassium leads to this syndrome, however, requires additional testing.