Deficiency of fat-soluble vitamins

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Vitamin A Deficiency

- Deficiency depending on liver stores, adult birds could be fed a vitamin A–deficient diet for 2–5 months before signs of deficiency develop. Deficiency does develop because of either inadvertent omission of the vitamin A supplement or inadequate feed preparation.
- Vitamin A is required for the health of the membranes of the digestive, urinary, reproductive and respiratory systems.
- A vitamin A deficiency can result when the level in the diet is inadequate or the vitamin added to the diet is oxidized by rancid fat in the diet. Additionally, neomycin, a common antibiotic, decreases the absorption of vitamin A.
- Vitamin A is a fat-soluble vitamin and an inadequate level of fat in the diet could also limit its absorption, even if in the diet at adequate levels.

In Chicks:

- Depending on the quantity of vitamin A passed on from the breeder hen, day-old chicks reared on a vitamin A–deficient diet may show signs within 7 days. However, chicks with a good reserve of maternal vitamin A may not show signs of a deficiency for up to 7 wk.
- Signs in chicks include anorexia, growth retardation, drowsiness, weakness, incoordination, emaciation, and ruffled feathers. If the deficiency is severe, the chicks may become ataxic. The yellow pigment in the shanks and beaks is usually lost, and the comb and wattles are pale.
- A cheesy material may be noted in the eyes, but xerophthalmia is seldom seen because chicks usually die before the eyes become affected. Secondary infection may play a role in many of the deaths noted with acute vitamin A deficiency.

In Adult:

- Eventually, birds become emaciated and weak with ruffled feathers.
- Egg production drops markedly, hatchability decreases, and embryonic mortality increases. As egg production declines, there will likely be only small follicles in the ovary, some of which show signs of hemorrhage.
- A watery discharge from the eyes may also be noted. As the deficiency continues, milky white, cheesy material accumulates in the eyes, making it impossible for birds to see (xerophthalmia).
- The eye, in many cases, may be destroyed.

Lesions:

- The first lesion usually noted in adult birds is in the mucous glands of the upper alimentary tract. The normal epithelium is replaced by a stratified squamous, keratinized layer. This blocks the ducts of the mucous glands, resulting in necrotic secretions.
- Small, white pustules may be found in the nasal passages, mouth, esophagus, and pharynx, and these may extend into the crop.
- Breakdown of the mucous membrane usually allows pathogenic microorganisms to invade these tissues and cause secondary infections.

Chronic vitamin A deficiency:

- Young chicks with chronic vitamin A deficiency may also develop pustules in the mucous membrane of the esophagus that usually affect the respiratory tract.
- Kidneys may be pale and the tubules distended because of uric acid deposits, and in extreme cases, the ureters may be plugged with urates. Blood levels of uric acid can rise from a normal level of 5 mg to as high as 40 mg/100 mL.
- Vitamin A deficiency does not interfere with uric acid metabolism but does prevent normal excretion of uric acid from the kidney.

Histologic findings include atrophy of the cytoplasm and a loss of the cilia in the columnar, ciliated epithelium.

- No gross lesions are found in the brain.
- The livers of ataxic vitamin A–deficient chicks contain little or no vitamin A.

Treatment:

- Vitamin A supplement up to 2 times the normally recommended level, should be fed for 2 wk. Vitamin A can be administered through the drinking water, and such treatment usually results in faster recovery than supplemtation via the feed.
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Vitamin D 3 Deficiency

- Vitamin D3 is required for the normal absorption and metabolism of calcium and phosphorus.
- A deficiency can result in rickets in young growing chickens or in cage layer fatigue (osteomalacia) and/or poor eggshell quality in laying hens, even though the diet may be well supplied with calcium and phosphorus.
- Abnormal skeletal development is discussed under calcium and phosphorus imbalances and manganese deficiency.

Rickets in young growing chickens

- **Signs:**
- Retarded growth and severe leg weakness are the first signs noted when chicks are deficient in vitamin D3. Beaks and claws become soft and pliable. Chicks may have trouble walking and will take a few steps before squatting on their hocks. While resting, they often sway from side to side, suggesting loss of equilibrium.
- Feathering is usually poor, and an abnormal banding of feathers may be seen in colored breeds. With chronic vitamin D3 deficiency, marked skeletal disorders are noted.
- The spinal column may bend downward and the sternum may deviate to one side. These structural changes reduce the size of the thorax, with subsequent crowding of the internal organs, especially the air sacs.

- **Lesions:**
- The finding in chicks is a beading of the ribs at the junction of the spinal column along with a downward and posterior bending. Poor calcification can also be seen at the epiphysis of the tibia and femur. By immersing the split bone in a silver nitrate solution and allowing it to stand under incandescent light for a few minutes, the calcified areas are easily distinguished from the areas of cartilage.

Cage layer fatigue (osteomalacia) and/or poor eggshell quality in laying hens:

- **Laying hens** fed a vitamin D3–deficient diet show loss of egg production within 2–3 wk, and depending on the degree of deficiency, shell quality deteriorates almost instantly. Using a corn-soybean meal diet with no supplemental vitamin D3, shell weight decreases dramatically by 150 mg/day throughout the first 7 days of deficiency. The less obvious decline in shell quality with suboptimal, rather than deficient, supplements is more difficult to diagnose, especially because it is very difficult to assay vitamin D3 in complete feeds.
- signs of gross pathology are usually confined to the bones and parathyroid glands. Bones are soft and easily broken, and the ribs may become beaded. The ribs may also show spontaneous fractures in the sternovertebral region. Histologic examination shows decreased calcification in the long bones, with excess of osteoid tissue and parathyroid enlargement.

Treatment:

- Adding synthetic 1,25 (OH)2D3 to the diet of susceptible chicks reduces the incidence of this condition. Although response is variable, results suggest that some leg abnormalities may be a consequence of inefficient metabolism of cholecalciferol.

- Dry, stabilized forms of vitamin D3 are recommended to treat deficiencies.

- In cases of severe mycotoxicosis, a water-miscible form of vitamin D3 is administered in the drinking water to provide the amount normally supplied in the diet. In cases of impaired liver function, metabolites of vitamin D are the usual choice for treatment.

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Vitamin E Deficiency

The three main disorders seen in chicks deficient in vitamin E are encephalomalacia, exudative diathesis, and muscular dystrophy. The occurrence of these conditions depends on various other dietary and environmental factors. It usually occurs between 15-30 days of age.

- Encephalomalacia '(crazy chick disease' or encephalomalacia ,softening of the brain)is seen in commercial flocks if diets are very low in vitamin E, if an antioxidant is either omitted or is not present in sufficient quantities, or if the diet contains a reasonably high level of an unstable and unsaturated fat.

- Exudative diathesis to occur, the diet must be deficient in both vitamin E and selenium.

- Muscular dystrophy is rare in chicks, because the diet must be deficient in both sulfur amino acids and vitamin E. Because the sulfur amino acids are necessary for growth, a deficiency severe enough to induce muscular dystrophy is unlikely to occur under commercial conditions.

1- Encephalomalacia

- **Sign:**
- Chicks hatched from breeders that are given additional dietary vitamin E seem less susceptible to lipid peroxidation in the brain.
- The classic sign of encephalomalacia is ataxia. Rapid contractions and relaxation of the legs resulting in what is known as 'crazy chick disease'
- The results from hemorrhage and edema within the granular layers of the cerebellum, with pyknosis and eventual disappearance of the Purkinje cells and separation of the granular layers of the cerebellar folia.
- Because of its inherently low level of vitamin E, the cerebellum is particularly susceptible to lipid peroxidation.

Prevention of encephalomalacia

- In prevention of encephalomalacia, vitamin E functions as a biologic antioxidant. The quantitative need for vitamin E for this function depends on the amount of linoleic acid and polyunsaturated fatty acids in the diet.
- Over prolonged periods, antioxidants have been shown to prevent encephalomalacia in chicks when added to diets with very low levels of vitamin E or in chicks fed vitamin E- depleted purified diets. The fact that antioxidants can help prevent encephalomalacia, but fail to prevent exudative diathesis or muscular dystrophy in chicks, strongly suggests that vitamin E is acting as an antioxidant in this situation.

2- Exudative diathesis

- Exudative diathesis results in a severe edema caused by a marked increase in capillary permeability.

- Electrophoretic patterns of the blood show a decrease in albumin levels, whereas exudative fluids contained a protein pattern similar to that of normal blood plasma.

3- Muscular dystrophy

- Vitamin E deficiency accompanied by sulfur amino acid deficiency results in severe muscular dystrophy in chicks by 4 wk of age. This condition is characterized by degeneration of the muscle fibers, usually in the breast but sometimes also in the leg muscles.
- **Histologic examination** shows Zenker's degeneration, with perivascular infiltration and marked accumulation of infiltrated eosinophils, lymphocytes, and histocytes. Accumulation of these cells in dystrophic tissue results in an increase in lysosomal enzymes, which appear to function in the breakdown and removal of the products of dystrophic degeneration.

Prevention and treatment

- Initial studies involving the effects of dietary vitamin E on muscular dystrophy show that the addition of selenium at 1–5 mg/kg diet reduced the incidence of muscular dystrophy in chicks receiving a vitamin E–deficient diet that was also low in methionine and cysteine, but did not completely prevent the disease. However, selenium was completely effective in preventing muscular dystrophy in chicks when diet contained a low level of vitamin E.
- Studies with chicks on the interrelationships between antioxidants, linoleic acid, selenium, and sulfur amino acids have shown that selenium and vitamin E play supportive roles in several processes, one of which involves cysteine metabolism and its role in prevention of muscular dystrophy in chickens.
- Glutathione peroxidase is soluble and located in the aqueous portions of the cell, whereas vitamin E is located mainly in the hydrophobic environments of membranes and in adipose tissue and other lipid storage cells.

- The overlapping manner in which vitamin E and selenium function in the cellular antioxidant system suggest that they spare one another in prevention of deficiency signs.
- Adequate levels of stabilized vitamin E should be used in conjunction with a commercial antioxidant and at least 0.3 ppm selenium.
- Signs of exudative diathesis and muscular dystrophy due to vitamin E deficiency can be reversed if treatment is begun early by administering vitamin E through the feed or drinking water. Oral administration of a single dose of vitamin E (300 IU per bird) usually causes remission.
- Signs of exudative diathesis and muscular dystrophy can be reversed in chicks by supplementing the diet with liberal amounts of vitamin E, assuming the deficiency is not too advanced.
- Encephalomalacia may respond to vitamin E supplementation, depending on the extent of the damage to the cerebellum.

Vitamin K Deficiency

- Impairment of blood coagulation is the major clinical sign of vitamin K deficiency. With a severe deficiency, subcutaneous and internal hemorrhages can prove fatal. Vitamin K deficiency results in a reduction in prothrombin content of the blood, and in the young chick, plasma levels are as low as 2% of normal. Because the prothrombin content of newly hatched chicks is only 40% that of adult birds, young chicks are readily affected by a vitamin K–deficient diet.
- A vitamin K deficiency in poultry may be related to low dietary levels of the vitamin, low levels in the maternal diet, lack of intestinal synthesis, extent of coprophagy, or the presence of sulfur drugs and other feed additives in the diet.
- Chicks with coccidiosis can have severe damage to their intestinal wall and can bleed excessively. Antimicrobial agents can suppress intestinal synthesis of vitamin K, rendering the bird completely dependent on the diet for its supply of the vitamin. Synthesis of vitamin K does occur in the bacteria resident in the bird's digestive tract; however, such vitamin K remains inside the bacterial cell, so the only benefit to the bird arises from the bacterial cell digestion or via coprophagy.

Signs and lesions

- Hemorrhagic syndrome in day-old chicks has been attributed to a deficiency of vitamin K in the diet of the breeder hens. Gross deficiency of vitamin K results in such prolonged blood clotting that severely deficient chicks may bleed to death from a slight bruise or other injury. Borderline deficiencies often cause small hemorrhagic blemishes.
- Hemorrhages may appear on the breast, legs, wings, in the abdominal cavity, and on the surface of the intestine.
- Chicks are anemic, which may be due in part to loss of blood but also to development of hypoplastic bone marrow. Although blood-clotting time is a reasonable measure of the degree of vitamin K deficiency, a more accurate measure is obtained by determining the prothrombin time.
- Prothrombin times in severely deficient chicks may be extended from a normal of 17–20 sec to 5–6 min or longer. No major heart lesions are seen in vitamin K–deficient chicks.

Prevention and control

- The inclusion of menadione at 1–4 mg/ton of feed is an effective and common practice to prevent vitamin K deficiency.
- If signs of deficiency are seen, the level should be doubled.
- A number of stress factors (eg, coccidiosis and other intestinal parasitic diseases) increase the requirements for vitamin K.
- Dicumarol, sulfaquinoxaline, and warfarin are antimetabolites of vitamin K.