

## **CHAPTER III**

### **VITAMINS IN POULTRY NUTRITION**

Vitamins are defined as organic compounds which are required in small quantities for the maintenance and normal growth of animal life. Deficiency of vitamins leads to the deficiency disease specific with a particular vitamins. The term vitamin was derived from 'vital amines'. But interestingly, only few vitamins contain the amines. The vitamins have been divided into two groups namely fat soluble and water soluble. The various vitamins important in animal feeding are as follows: Fat soluble vitamins are A, D<sub>2</sub>, D<sub>3</sub>, E and K whereas water soluble vitamins are B<sub>1</sub>, B<sub>2</sub>, B<sub>3</sub>, B<sub>5</sub>, B<sub>6</sub>, B<sub>12</sub>, Choline, Biotin, Folic acid and vitamin C.

#### **Disease and Condition due to Vitamin Deficiency**

Vitamin deficiencies are most commonly due to inadvertent omission of a vitamin premix from the birds' diet. Multiple signs are therefore seen, although in general, problems with deficiencies of the B vitamins appear first. Because there are some stores of fat-soluble vitamins in the body, it often takes longer for these deficiencies to affect the bird.

Treatment and prevention rely on an adequate dietary supply, usually microencapsulated in gelatin or starch along with an antioxidant. Vitamin destruction in feeds is a factor of time, temperature, and humidity. For most feeds, vitamin efficiency is little affected over 2-month storage within mixed feed.

#### **Vitamin A deficiency**

Adult birds, depending on liver storage, could be fed a vitamin A-deficient diet for 2–5 mo before signs of deficiency develop. Eventually birds become emaciated and weak with ruffled feathers. Egg production drops markedly, hatchability decreases, and embryonic mortality with incubated eggs increases. As egg production declines, there will likely be atretic follicles in the ovary, some of which show signs of haemorrhage. 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.

The first lesion usually noted in adult birds is in the mucous glands of the 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 may extend into the crop. Breakdown of the mucous membrane may allow pathogenic microorganisms to invade these tissues and cause secondary infections.

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. Gross signs in chicks include anorexia, growth retardation, drowsiness, weakness, incoordination, emaciation, and ruffled feathers. If the deficiency is severe, the chicks may become ataxic, as with vitamin E deficiency. 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. Infection may play a role in many of the deaths noted with acute vitamin A deficiency.

Young chicks with a chronic vitamin A deficiency may also show pustules in the mucous membrane of the oesophagus that may also affect the respiratory tract. Kidneys may be pale and the tubules distended due to the uric acid deposits. In extreme cases, the ureters may be filled with urates. Blood levels of uric acid can rise from a normal level of ~5 mg to as high as 40 mg/100 ml of blood. Vitamin A deficiency does not interfere with uric acid metabolism but does prevent normal excretion of uric acid from the kidney. Histological findings include atrophy of the cytoplasm and a loss of the cilia in the columnar, ciliated epithelium.

While vitamin A-deficient chicks can be ataxic, similar to those with vitamin E deficiency, no gross lesions are found in the brain of vitamin A-deficient chicks as compared with degeneration of the Purkinje cells in the cerebellum of vitamin E-deficient chicks. Also, the livers of ataxic vitamin A-deficient chicks contain little or no vitamin A.

Because stabilized dry, vitamin A supplements are almost universally used, it is unlikely that a deficiency will be encountered. However, if a deficiency does develop due to inadvertent omission of the vitamin A supplement or poor mixing, up to 2 times the normally recommended level should be fed for ~2 week. The dry, stabilized forms of vitamin A are the feed supplements of choice. Forms that can be administered through the drinking water are available and usually result in fast recovery than medication of the feed.

### **Vitamin D deficiency**

Abnormal development of the bones is discussed under calcium and phosphorus imbalances. Vitamin D<sub>3</sub> is required for the normal absorption and metabolism of calcium and phosphorus. A deficiency can result in rickets in young growing chicken or in osteoporosis and poor eggshell quality in laying hens, even though the diet may be well supplied with calcium and phosphorus.

Laying hens fed a vitamin D<sub>3</sub>-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 D<sub>3</sub>, shell weight decreases dramatically by about 150 mg/day within 7 days. The less obvious decline in shell quality with suboptimal supplements is more difficult to diagnose, especially because it is very difficult to assay vitamin D<sub>3</sub> in complete feeds.

There is a significant increase in plasma  $1,25(\text{OH})_2\text{D}_3$  of birds producing good versus poor eggshells. Feeding purified  $1,25(\text{OH})_2\text{D}_3$  improves the shell quality of these inferior layers, suggesting a potential inherent problem with metabolism of cholecalciferol.

Retarded growth and severe leg weakness are the first signs noted when chicks are deficient in vitamin  $\text{D}_3$ . 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 coloured breeds. With chronic vitamin  $\text{D}_3$  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. A characteristic 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 dipping the split bone in a silver nitrate solution and allowing it to stand under an incandescent light for a few minutes, the calcified areas are easily distinguished from the areas of cartilage. Adding synthetic  $1,25(\text{OH})_2\text{D}_3$  to the diet of susceptible chicks reduces the incidence of this condition. Although the response is variable, results suggest that some leg abnormalities may be a consequence of inefficient metabolism of cholecalciferol.

In the laying hen, 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 deficiency of calcification in the long bones, with excess of osteoid tissue and parathyroid enlargement.

Enough vitamin D is added to commercial diets to provide 3 times the normally recommended level for a period of  $\sim 3$  wk. Dry, stabilized forms of vitamin  $\text{D}_3$  are recommended to treat deficiencies. In cases of severe mycotoxicosis, a water-miscible form of vitamin D is administered in the drinking water to provide the amount normally supplied in the diet.

### **Vitamin E deficiency**

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 dietary and environmental factors.

Encephalomalacia is seen in commercial flocks if diets are 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, unsaturated fat. For exudative diathesis to occur, the diet must be deficient in both vitamin E and selenium. Signs of muscular dystrophy are rare in chicks, as the diet must be deficient in both sulphur amino acids and vitamin E. Because the sulphur amino acids are necessary for growth, a deficiency severe enough to induce muscular dystrophy is unlikely to occur under commercial conditions. Signs of exudative diathesis and muscular

dystrophy can be reversed in chicks by supplementing the diet with liberal amounts of vitamin E, if the deficiency is not too advanced. Encephalomalacia may respond to vitamin E supplementation, depending on the extent of the damage to the cerebellum.

The classical sign of encephalomalacia is ataxia. The results from haemorrhage and oedema within the molecular and granular layers of the cerebellum, with pyknosis and eventual disappearance of the Purkinje cells and separation of the molecular and granular layers of the cerebellar folia. Due to its inherently low level of vitamin E, the cerebellum is particularly susceptible to lipid peroxidation. 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. Chicks hatched from breeders that are given additional dietary vitamin E are also less susceptible to lipid peroxidation in the brain. 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. Exudative diathesis results in a severe oedema 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.

Vitamin E deficiency accompanied by sulphur amino acid deficiency results in severe muscular dystrophy in chicks by ~4 wk of age. This condition is characterized by degeneration of the muscle fibres, 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. Initial studies involving the effects of dietary vitamin E on muscular dystrophy showed 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 low in methionine and cysteine, but did not completely prevent the disease. However, selenium was completely effective in preventing muscular dystrophy in chicks when the diet contained a low level of vitamin E, which alone had been shown to have no effect on the disease.

Studies with chicks on the interrelationships between antioxidants, linoleic acid, selenium, and sulphur 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 the prevention of muscular dystrophy in the chicken. Glutathione peroxidase is soluble and is located in the aqueous portions of the cell, while vitamin E is located mainly in the hydrophobic environments of membranes and in 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 the prevention of deficiency signs.

Only stabilized fat should be used in feeds. Adequate levels of stabilized vitamin E should be used in conjunction with a commercial antioxidant and up to 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 PO or through the feed. Oral administration of a single dose of 300 IU of vitamin E per bird usually causes remission.

### **Vitamin K deficiency**

Impairment of blood coagulation is the major clinical sign of vitamin K deficiency. With a severe deficiency, subcutaneous and internal haemorrhages 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 carryover of vitamin K from the dam to eggs, and subsequently to hatched chicks, has been demonstrated, so breeder diets should be well fortified. 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 a prolonged blood clotting time that severely deficient chicks may bleed to death from a slight bruise or other injury. Borderline deficiencies often cause small hemorrhagic blemishes. Haemorrhages 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 the development of hypoplastic bone marrow. Although blood-clotting time is a fairly good measure 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 such as those that occur in pigs.

A vitamin K deficiency in poultry may be related to low dietary levels of the vitamin, low levels in the maternal diet, degree of intestinal synthesis, extent of coprophagy, or the presence of sulphur 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. In poultry, there is little intestinal synthesis due to the short digestive tract. 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 coprophagy.

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.

### **Thiamin (Vitamin B<sub>1</sub>) deficiency**

'Polyneuritis' in birds represents the later stages of a thiamin deficiency, probably caused by buildup of the intermediates of carbohydrate metabolism. Because the brain's immediate source of energy results from the degradation of glucose, it is dependent on biochemical reactions involving thiamin. In the initial stages of deficiency, lethargy and head tremors may be noted. A marked decrease in appetite is seen in birds fed a thiamin-deficient diet. Poultry are also susceptible to neuromuscular problems, resulting in impaired digestion, general weakness, star-gazing, and frequent convulsions.

Polyneuritis may be seen in mature birds ~3 week after they are fed a thiamin-deficient diet. As the deficiency progresses, birds may sit on flexed legs and draw back their heads in a star-gazing position. Therefore, this condition is also referred as 'star gazing disease'. Retraction of the head is due to paralysis of the anterior neck muscles. Soon after this stage, chicken lose the ability to stand or sit upright and topple to the floor, where they may lie with heads still retracted. Thiamin deficiency may also lead to a decrease in body temperature and respiratory rate. Testicular degeneration may be noted, and the heart may show slight atrophy. Birds consuming a thiamin-deficient diet soon show severe anorexia. They lose all interest in feed and will not resume eating unless given thiamin. If a severe deficiency has developed, thiamin must be force-fed or injected to induce the chicken to resume eating.

Thiamin deficiency is most common when poorly processed fish meals are used, because they contain thiaminase enzyme. In such situations, adding extra thiamin may be ineffective. In regular diets, deficiency is prevented by supplements of thiamin at 4 mg/kg.

### **Riboflavin (Vitamin B<sub>2</sub>) deficiency**

Many tissues may be affected by riboflavin deficiency, although the epithelium and the myelin sheaths of some of the main nerves are major targets. Changes in the sciatic nerves produce 'curled-toe paralysis' in growing chicken. Egg production is affected, and riboflavin-deficient eggs do not hatch. When chicks are fed a diet deficient in riboflavin, their appetite is fairly good but they grow slowly, become weak and emaciated, and develop diarrhoea between the first and second weeks. Deficient chicks are reluctant to move unless forced and then frequently walk on their hocks with the aid of their wings. The leg muscles are atrophied and flabby, and the skin is dry and harsh. In advanced stages of deficiency, the chicks lie prostrate with their legs extended, sometimes in opposite directions. The characteristic sign of riboflavin deficiency is a marked enlargement of the sciatic and brachial nerve sheaths; sciatic nerves usually show the most pronounced effects. Histologic examination of the affected nerves shows degenerative changes in the myelin sheaths that, when severe, pinch the nerve. This produces a permanent stimulus, which causes the curled-toe paralysis.

Signs of riboflavin deficiency in the hen are decreased egg production, increased embryonic mortality, and an increase in size and fat content of the liver. Hatchability declines within 2 wk when hens are fed a riboflavin-deficient diet, but returns to near normal when

riboflavin is restored. Affected embryos are dwarfed and show characteristically defective “clubbed” down. The nervous system of these embryos shows degenerative changes much like those described in riboflavin-deficient chicks.

Signs of riboflavin deficiency first appear at 10 days of incubation, when embryos become hypoglycemic and accumulate intermediates of fatty acid oxidation. Although flavin-dependent enzymes are depressed with riboflavin deficiency, the main effect seems to be impaired fatty acid oxidation, which is a critical function in the developing embryo. An autosomal recessive trait blocks the formation of the riboflavin-binding protein needed for transport of riboflavin to the egg. While the adults appear normal, their eggs fail to hatch regardless of dietary riboflavin content. As eggs become deficient in riboflavin, the egg albumen loses its characteristic yellow colour. In fact, albumen colour score has been used to assess riboflavin status of birds.

Chicks receiving diets only partially deficient in riboflavin may recover spontaneously, indicating that the requirement rapidly decreases with age. A 100- $\mu$ g dose should be sufficient for treatment of riboflavin-deficient chicks, followed by incorporation of an adequate level in the diet. However, when the curled-toe deformity is longstanding, irreparable damage occurs in the sciatic nerve, and the administration of riboflavin is no longer curative.

Most diets contain up to 10 mg riboflavin/kg. Treatment can be given as two 100  $\mu$ g doses for chicks or poults, followed by an adequate amount of riboflavin in feed.

### **Niacin (nicotinic Acid) (Vitamin B<sub>3</sub>) deficiency**

There is considerable evidence that poultry – even chick and turkey embryos – can synthesize niacin, but at a rate that is too slow for optimal growth. It has been claimed that a marked deficiency of niacin cannot occur in chicken unless there is a deficiency of tryptophan, a niacin precursor.

Niacin deficiency is characterized by severe metabolic disorders in the skin and digestive organs. The first signs are usually loss of appetite, retarded growth, general weakness, and diarrhoea. There is conflicting evidence as to whether broilers respond, in terms of growth and feed utilization, to niacin supplementation. However, it has been clearly established that chicks do have a requirement for niacin. Deficiency produces enlargement of the tibiotarsal joint, bowing of the legs, poor feathering, and dermatitis on the head and feet.

Niacin deficiency in chicks can also result in “black tongue.” At ~2 wk of age, the tongue, oral cavity, and esophagus become distinctly inflamed. In the niacin-deficient hen, weight loss, reduced egg production, and a marked decrease in hatchability can result. Turkeys, ducks, pheasants, and goslings are much more severely affected by niacin deficiency than are chicken. Their apparently higher requirements are likely related to their less efficient conversion of tryptophan to niacin. Ducks and turkeys with a niacin deficiency show a severe bowing of the legs and an enlargement of the hock joint. The main difference between the leg seen in niacin

deficiency and perosis seen in manganese and choline deficiency is that with niacin deficiency the Achilles tendon seldom slips from its condyles.

Niacin deficiency in chicken may be prevented by feeding a diet that contains  $\geq 30$  mg niacin/kg; however, many nutritionists recommend 2–2.5 times as much. An allowance of 55–70 mg/kg of feed appears to be satisfactory for ducks, geese, and turkeys. Ample niacin should be provided in poultry diets so that the birds do not have to synthesize it from tryptophan.

### **Pantothenic acid (Vitamin B<sub>5</sub>) deficiency**

Pantothenic acid is the prosthetic group of coenzyme A, an important coenzyme involved in many reversible acetylation reactions in carbohydrate, fat, and amino acid metabolism. Signs of deficiency relate to general avian metabolism.

The major lesions of pantothenic acid deficiency involve the nervous system, the adrenal cortex, and the skin. Deficiency may result in reduced egg production; however, a marked drop in hatchability is usually noted prior to this event. Embryos from hens with pantothenic acid deficiency can have subcutaneous haemorrhages and severe oedema, with most other mortality showing up during the later part of the incubation period. In chicks, the first signs are reduced growth and feed consumption; poor feather growth, with feathers becoming ruffled and brittle; and a rapidly developing dermatitis. Corners of the beak and the area below the beak are usually the worst affected, but the condition is also noted on the feet. In severe cases, the skin of the feet may cornify, and wart-like lumps occur on the balls of the feet. The foot problem often leads to bacterial infection.

Liver concentration of pantothenic acid is reduced during a deficiency, with the liver becoming atrophied. A faint to dirty yellow colour may be noted. Nerve fibres of the spinal cord may show myelin degeneration. Pantothenic acid-deficient chicks show lymphoid cell necrosis in the bursa of Fabricius and thymus, together with lymphocytic paucity in the spleen. The foot condition in chicks and the poor feathering are difficult to differentiate from signs of a biotin deficiency. In a pantothenic acid deficiency, dermatitis of the feet is usually noted first on the toes; in contrast, a biotin deficiency primarily affects the foot pads and is usually more severe. Ducks do not show the usual signs noted for chicken and turkeys, except for in retarded growth, but mortality can be quite high.

Most poultry diets contain supplements of calcium pantothenate. Periodically, growing chicken fed practical diets develop a scaly condition of the skin, the exact cause of which is not known. Treatment with both calcium pantothenate (2 g) and riboflavin (0.5 g) in the drinking water (190 litre) for a few days has been successful in some instances.

### **Pyridoxine (Vitamin B<sub>6</sub>) deficiency**

A vitamin B<sub>6</sub> deficiency causes retarded growth, dermatitis, and anaemia. Because a major role of the vitamin is protein metabolism, deficiency can result in reduced nitrogen retention. Dietary protein is not well utilized and thus nitrogen excretion increases. Increased

iron levels and decreased copper levels are noted in the serum, and iron utilization appears to be markedly decreased. The resulting anaemia is likely due to a disturbance in the synthesis of protoporphyrins. Anaemia is often noted in ducks but is seldom seen in chicken and turkeys. Young chicks may show nervous movements of the legs when walking and often undergo spasmodic convulsions, leading to death. During convulsions, the chicks may run about aimlessly, flapping their wings and falling with jerking motions. The greater intensity of activity, resulting from pyridine deficiency, distinguishes these signs from those of encephalomalacia. Gizzard erosion has been noted in vitamin B<sub>6</sub>-deficient chicks. It can be prevented by inclusion of 1% taurocholic acid in the diet, leading to the speculation that pyridoxine is involved in taurine synthesis and is important for gizzard integrity. In pyridoxine deficiency, collagen maturation is incomplete, suggesting that this vitamin is essential for integrity of the connective tissue matrix. A chronic deficiency can result in perosis, with one leg usually being crippled and one or both middle toes bent inward at the first joint.

In adult birds, pyridoxine deficiency results in reduced appetite, leading to reduced egg production and a decline in hatchability. Severe deficiency can cause rapid involution of the ovary, oviduct, comb, and wattles, and of the testis in cockerels. Feed consumption in B<sub>6</sub>-deficient hens and cockerels declines sharply. Although a partial molt is observed in some hens, normal egg production returns within 2 wk following provision of a normal dietary level of pyridoxine.

Deficiency can be prevented by adding 3–4 mg pyridoxine/kg feed.

### **Folic acid (folacin) deficiency**

A folacin deficiency results in a macrocytic (megaloblastic) anaemia and leukopenia. Tissues with a rapid turnover, such as epithelial linings, GI tract, epidermis, and bone marrow, as well as cell growth and tissue regeneration, are principally affected.

Poultry seem more susceptible to folacin deficiency than other farm animals. Deficiency results in poor feathering, slow growth, an anemic appearance, and perosis. As anaemia develops, the comb becomes waxy white and pale mucous membranes in the mouth are noted. Elevated erythrocyte phosphoribosylpyrophosphate concentration can be used as a diagnostic tool in folate-deficient chicks. There may also be damage to liver parenchyma and depleted glycogen reserves. While turkey poults show some of the same signs as chicken, mortality is usually higher and the birds develop a spastic type of cervical paralysis that results in the neck becoming stiff and extended.

The abnormal feather condition in chicken leads to weak and brittle shafts. Depigmentation develops in coloured feathers due to a deficiency of the vitamin. While a folacin deficiency can result in reduced egg production, the main sign noted with breeders is a marked decrease in hatchability associated with an increase in embryonic mortality, usually during the last few days of incubation. Embryos have deformed beaks and bending of the tibiotarsus. While birds may exhibit perosis, the lesions seen differ histologically from those that develop due to

choline or manganese deficiency. Abnormal structure of the hyaline cartilage and retardation of ossification are noted with folacin deficiency. Increasing the protein content of the diet has been shown to increase the severity of perosis in chicks receiving diets low in folic acid, as there is an increased folacin demand for uric acid synthesis.

Signs of folic acid deficiency in poultry can be prevented by ensuring diets contain supplements of up to 1 mg/kg.

### **Biotin deficiency**

Biotin deficiency results in dermatitis of the feet and the skin around the beak and eyes similar to that described for pantothenic acid (*see* p 2533). Perosis and footpad dermatitis are also characteristic signs. While signs of classic biotin deficiency are rare, occurrence of fatty liver and kidney syndrome (FLKS) is important to commercial poultry producers. FLKS was first described in Denmark in 1958, but was not a major concern until the late 1960s, especially in Europe and Australia. Chicks ~3 wk of age become lethargic and unable to stand, then die within hours. Mortality is usually quite low at 1–2% but can reach 20–30%. Postmortem examination reveals pale liver and kidney with accumulation of fat.

The condition was usually confined to wheat-fed birds and was most problematic in low-fat, high-energy diets. High vitamin supplementation in general corrected the problem, and biotin was isolated as the causative agent. It is now known that biotin in wheat has exceptionally low availability. The trigger of high-energy diets led to investigation of biotin in carbohydrate metabolism. Chicks with FLKS are invariably hypoglycemic, emphasizing the importance of biotin in 2 key enzymes: pyruvate carboxylase and acetyl Co-A carboxylase. Acetyl Co-A carboxylase appears to preferentially sequester biotin, such that with low biotin availability and need for high *de novo* fat synthesis (high energy, low-fat diet), pyruvate carboxylase activity is severely compromised. Even with this imbalance, birds are able to grow. However, with a concurrent deprivation in feed intake or increased demand for glucose, hypoglycemia develops, leading to adipose catabolism and the characteristic accumulation of fat in both liver and kidney. Birds with FLKS rarely show signs of classic biotin deficiency.

Plasma biotin levels <100 ng/100 ml has been reported as sign of deficiency. However, recent evidence suggests that plasma biotin levels are quite insensitive to the birds' biotin status, and that biotin levels in the liver or kidney are more useful indicators. Plasma pyruvic carboxylase is positively correlated with dietary biotin concentration, and levels plateau much later than does the growth response to biotin.

Embryos are also sensitive to biotin status. Congenital perosis, ataxia, and characteristic skeletal deformities may be seen in embryos and newly hatched chicks when hens are fed a deficient diet. Embryonic deformities include a shortened tibiotarsus that is bent posteriorly, a much shortened tarsometatarsus, shortening of the bones of the wing and skull, and shortening and bending of the anterior end of the scapula. Syndactyly—an extensive webbing between the third and fourth toes—in biotin-deficient embryos has been noted. Such embryos are

chondrodystrophic and characterized by reduced size, parrot beak, crooked tibia, and shortened or twisted tarsometatarsus.

A number of factors increase biotin requirements, including oxidative rancidity of feed fat, competition by intestinal microorganisms, and lack of carryover into the newly hatched chick or poul. It is good practice to add 150–200 mg biotin/ton feed, especially when wheat or wheat byproducts are used.

### **Choline deficiency**

In addition to poor growth, the classic sign of choline deficiency in chicks and poults is perosis. Perosis is first characterized by pinpoint haemorrhages and a slight puffiness about the hock joint, followed by an apparent flattening of the tibiometatarsal joint caused by a rotation of the metatarsus. The metatarsus continues to twist and may become bent or bowed so that 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 displaced, and the Achilles tendon slips from its condyles. Perosis is not a specific deficiency sign; it appears with several nutrient deficiencies.

Although choline deficiency readily develops in chicks fed diets low in choline, a deficiency in laying hens is not easily produced. Eggs contain ~12–13 mg of choline/g of dried whole egg. A large egg contains ~170 mg of choline, found almost entirely in the phospholipids. Thus, there appears to be a considerable need for choline to produce an egg. In spite of this, producing a marked choline deficiency in laying hens has been difficult even when highly purified diets essentially devoid of choline were provided for a prolonged period. The choline content of eggs was not lowered, suggesting synthesis by the bird.

Diets that contain appreciable quantities of soybean meal, wheat bran, and wheat shorts are unlikely to be deficient in choline. Soybean meal is a good source of choline, and wheat byproducts are good sources of betaine, which can perform the methyl-donor function of choline. Other good sources of choline are distiller's grains, fishmeal, liver meal, meat meals, distiller's solubles, and yeast. A number of commercial choline supplements are available, and choline is routinely added to most poultry feed.

### **Cyanocobalamin (vitamin B<sub>12</sub>) deficiency**

Vitamin B<sub>12</sub> is an essential part of several enzyme systems, with most reactions involving the transfer or synthesis of methyl groups. While the most important function of vitamin B<sub>12</sub> is in the metabolism of nucleic acids and proteins, it also functions in carbohydrate and fat metabolism.

Poor feathering and mortality are the most obvious symptoms of a vitamin B<sub>12</sub> deficiency, and gizzard erosions may also appear. In growing chicken, a deficiency of vitamin B<sub>12</sub> results in reduced weight gain and feed intake, along with poor feathering and nervous disorders. While deficiency may lead to perosis, this is probably a secondary effect due to a dietary deficiency of methionine, choline, or betaine as sources of methyl groups. Vitamin B<sub>12</sub>

may alleviate perosis due to its effect on the synthesis of methyl groups. Other signs reported in poultry are anaemia, gizzard erosion, and fatty infiltration of the heart, liver, and kidneys. Laying hens appear to be able to maintain body weight and egg production; however, egg size is reduced. In breeders, hatchability can be markedly reduced, although several months may be needed for signs of deficiency to appear. Changes noted in embryos from B<sub>12</sub>-deficient breeders include a general hemorrhagic condition, fatty liver, fewer myelinated fibres in the spinal cord, and high incidence of embryo deaths at 17 days incubation.

Deficiency of vitamin B<sub>12</sub> is highly unlikely, especially for birds grown on litter or where animal-based ingredients are used. Treatment involves feeding up to 20 µg/g feed for 1–2 wk.

### **Disease and Condition due to Mineral Deficiency**

#### **Potassium, sodium and chlorine deficiencies**

A deficiency of potassium results in high mortality and retarded growth of chicks and causes reduced egg production and eggshell thickness in laying hens. It is not usually necessary to add potassium to practical feed formulations, since such formulas generally contain about 0.7–1% potassium. A deficiency of sodium in chicken diets results in poor growth, increased adrenal weight, and decreased egg production. Frequently, sodium supplementation is minimized to reduce the moisture level in the excreta. Signs of chlorine deficiency in chicks include poor growth, mortality, haemoconcentration, and reduced blood chlorine level. Chlorine-deficient chicks show a nervous condition resembling tetany and fall forward with legs extended backward when stimulated by a sharp noise.

#### **Calcium and phosphorus deficiencies**

Bone formation is highly dependent on the dietary concentrations of calcium and phosphorus as well as on adequate intake of vitamin D<sub>3</sub>. Deficiency of any one of these nutrients will result in rickets. Poor growth may also be a sign of calcium or phosphorus deficiency. Dietary excesses of either calcium or phosphorus should be avoided because such excesses can hinder the intestinal absorption of other mineral elements. The phosphorus that comes from plant products (i.e., phytin) should not be depended on to fulfill the phosphorus requirement for two reasons: it is not readily available in its natural form to the bird, and it may bind calcium, zinc, iron, and manganese so as to render them unavailable. Pullets at the beginning of the laying period undergo considerable metabolic stress associated with adjustment to the need to supply approximately 2.4 g of calcium daily to the oviduct for shell formation. Some birds mobilize large amounts of calcium from their skeleton during this period, and the bones may become so demineralized that the birds are unable to stand and appear paralyzed. The sternum and rib bones are frequently deformed, and all bones are easily broken. Dietary management to prevent this condition (generally termed **cage-layer fatigue** but more precisely described as **osteoporosis**) has not been devised.

### **Magnesium deficiency**

When fed a diet very deficient in magnesium, chicks grow slowly for about 1 week and then stop growing and become lethargic. Chicks fed diets marginal in magnesium may grow quite well but exhibit reduced levels of plasma magnesium and symptoms of neuromuscular hyperirritability when disturbed. Chicks show a brief convulsion and then enter a comatose state from which they usually recover, but sometimes death occurs. A magnesium deficiency in laying hens results in a rapid decline in blood magnesium level, withdrawal of magnesium from bone, decline in egg production, and, eventually, a comatose state and death. Magnesium content and hatchability of eggs also are reduced when hens are fed magnesium-deficient diets. Increasing either the calcium or the phosphorus content of the diet accentuates magnesium deficiency. Normally, adequate magnesium is present in the natural ingredients of practical diets to meet the requirements of poultry.

### **Iron deficiency**

Iron deficiency in chicken and turkeys causes an anaemia in which the red blood cells are reduced in size and low in haemoglobin. In red-feathered chicken, pigmentation does not occur when the diet is deficient in iron.

### **Zinc deficiency**

Zinc has many biochemical functions. Deficiency causes retarded growth and frayed feathers. The extent of fraying varies from almost no feathers on the wings and tail to only slight defects in the development of some of the barbules and barbicels. The long bones of the legs and wings are shorter and thicker than normal. The hock joint may be enlarged. Layer and breeder diets deficient in zinc reduce egg production and hatchability.

### **Manganese deficiency**

Manganese deficiency in chicks and poults results in perosis or slipped tendon. Deficiencies of other nutrients, such as choline and biotin, may also be involved in inducing perosis. The usual signs of perosis are swelling and flattening of the hock joint, with subsequent slipping of the Achilles tendon from its condyles. The tibia and the tarsometatarsus may exhibit bending near the hock joint and lateral rotation. One or both legs may be affected. A shortening and thickening of the long bones of the wings and legs are also observed. The disorder, insofar as manganese is concerned, is aggravated by excess dietary calcium and phosphorus. In laying and breeding birds, manganese deficiency results in lowered egg production, reduced eggshell strength, poor hatchability, and reduced fertility. Manganese-deficient embryos exhibit shortening of the long bones, 'parrot beak', and wiry down.

### **Copper deficiency**

Copper deficiency in poultry causes an anaemia in which the red blood cells are small and low in haemoglobin. Bone deformities can occur. Pigmentation of feathers in New Hampshire and Rhode Island Red chicken is reduced. Copper is required for the activity of the

enzyme needed for the cross-linking of lysine in the protein elastin. Dissecting aneurism of the aorta occurs in birds deficient in copper because of the defect in elastin formation. Copper deficiency also results in marked cardiac hypertrophy.

### **Iodine deficiency**

Iodine is necessary for the synthesis of thyroid hormones. Iodine deficiency results in goitre, which is the enlargement of the thyroid glands. The glands may increase to many times their usual size. If the deficiency is not too severe, the increased efficiency of the enlarged gland in “trapping” iodine from the bloodstream may compensate for the low dietary concentration. When this is the case, the production of thyroid hormones is normal, although the thyroid glands are enlarged. Inadequate production of thyroid hormones results in poor growth, egg production, and egg size. Iodine deficiency in breeders results in low iodine content of the egg and, consequently, decreased hatchability and thyroid enlargement in the embryos.

### **Selenium deficiency**

Selenium is closely associated with vitamin E and other antioxidants in practical feed formulation. The principal sign of deficiency in chicks is exudative diathesis. A requirement for selenium supplementation, even in the presence of vitamin E, is demonstrated by the poor growth, muscular dystrophy, and mortality of chicks fed purified diets or diets based on grains produced on low-selenium soils. Selenium is required for prevention of myopathies of the gizzard and heart in turkeys. Pancreatic fibrosis, with resultant reductions in the pancreatic output of lipase, trypsinogen, and chymotrypsinogen, has also been associated with selenium deficiency. Selenium is a structural component of glutathione peroxidase, an enzyme needed to quench peroxides generated during metabolism. There is wide variability in the amount and availability of selenium in the soils of different geographic areas. Consequently, cereals and plant-derived feedstuffs are variable sources of selenium. Grains from some areas contain sufficient selenium to render them toxic to chicks. The amount of supplementary selenium permissible in diets is regulated in the USA and Canada.