VITAMIN E DEFICIENCY

DEFINITION

Three distinct disorders (syndromes) related to or caused by vitamin E deficiency have been recognized in poultry. Each disorder usually occurs alone, although there are occasional overlaps. The three disorders are:

1. Encephalomalacia (crazy chick disease).
2. Exudative diathesis.

Although each of these syndromes is associated to some degree with vitamin E deficiency, each can be prevented by dietary changes unrelated to the vitamin E content of the ration. There is some interaction with synthetic antioxidants, selenium and sulfur-containing amino acids, especially in preventing encephalomalacia, exudative diathesis and muscular dystrophy respectively.

OCCURRENCE

Vitamin E deficiencies usually are seen in young chicks or turkey poults but also occur in ducklings and, perhaps, in other poultry. Deficiencies usually occur in birds raised in confinement i.e., birds compelled to eat only what is offered to them. Most outbreaks occur in birds fed rations that are high in polyunsaturated fats (e.g., cod liver oil, soy bean oil), that oxidize and become rancid. Vitamin E is very unstable with oxidative destruction enhanced by minerals and polyunsaturated fats in diet.

ETIOLOGY

1. Vitamin E and the selenium-containing enzyme glutathione peroxidase prevent cell membrane destruction caused by peroxides and other powerful oxidants produced as metabolic by-products.
2. There is evidence that vitamin E, selenium, and sulfur-containing amino acids perform separate functions but still act together to prevent the accumulation of harmful peroxides in tissue. Peroxides are derived, in part, from polyunsaturated acids in feeds.
3. The following facts are of interest in considering etiology:
   A. Encephalomalacia can be prevented by adding synthetic antioxidants to the feed.
   B. Exudative diathesis can be prevented by adding selenium to the feed.
   C. Muscular dystrophy can be prevented by adding cysteine, a sulfur-containing amino acid, to the feed.

CLINICAL SIGNS

Vitamin E is involved in several metabolic functions but mostly play a role of natural antioxidant.

Encephalomalacia

Signs are those associated with lesions of the central nervous system and include ataxia, loss of balance, falling over backwards while flapping the wings, sudden prostration on the side with legs outstretched, toes flexed, and head retracted [Fig. 1; Vit E deficiency; NCSU]. Birds that show clinical signs often continue to eat.
VITAMIN E DEFICIENCY

The deficiency usually occurs between the 15th and 30th day of life; however, it may occur as early as the 7th and as late as the 56th day.

Exudative diathesis

There is a severe edema caused by increased capillary permeability. This edema is located along the ventrum of the thorax, the abdomen, and perhaps under the mandible. Birds with extensive edema may have difficulty in walking and may stand with their legs far apart because of accumulation of subcutaneous fluid ventral to the abdomen.

Muscular dystrophy

Signs are usually inapparent but there may be locomotor problems.

LESIONS

Encephalomalacia

The swollen cerebellum often contains congested, hemorrhagic, or necrotic areas visible on the surface [Fig. 2; Vit E deficiency; NCSU]. Lesions occur less frequently on the cerebrum. Lesions are accentuated by formalin fixation for a few hours. In turkeys, poliomalacia of the lumbar spinal cord is often found microscopically.

Exudative diathesis

There is green-blue blood-stained viscous edema in the skin and subcutis of the ventrum. Muscular dystrophy occasionally is apparent in breast or leg muscles of the same birds. Distention of the pericardium with fluid has been the cause of sudden deaths in birds.

Muscular dystrophy

In chicks white to yellow degenerative muscle fibers give a streaked appearance to skeletal muscles of the breast or legs. In poult s the musculature of the gizzard may contain gray areas of muscle degeneration [Fig. 3; Vit E deficiency; NCSU].

DIAGNOSIS

1. The diagnosis can usually be made on the basis of typical signs and gross lesions.

2. Examination and analysis of the ration may indicate rancidity or likelihood of deficiency of vitamin E and/or selenium. Feed analysis for vitamin E activity is time consuming and expensive, therefore care should be taken to submit truly representative samples. Storage temperature and duration are very important in evaluating the quality of the vitamin E ingredient.

3. Microscopic examination of typical lesions is of considerable value in confirming suspected vitamin E deficiency, especially with encephalomalacia or muscular dystrophy.

CONTROL

1. Mix new batches of feed at frequent intervals. Use only high quality ingredients. Avoid storage of mixed feeds for periods longer than 4 weeks. If prolonged storage is necessary, add chemical antioxidants.

2. Use only stabilized fats in the feed.

3. Store feeds in a cool, dry place to reduce vitamin and other quality losses.
VITAMIN E DEFICIENCY

4. Avoid improperly compounded do-it-yourself -type rations. Most well-known, commercially prepared feeds are superior in quality to unplanned, self-mixed feeds.

TREATMENT

1. Recommended vitamin E levels are 30 to 150 mg/kg in the diet. Be sure an antioxidant (0.25kg of BHT or santoquin per 1000kg of feed) is in the feed if storage is long or environmental temperatures high. However the newest forms of vitamins are enveloped hence more resistant to heat treatments, humidity and storage. A dose of 0.3 ppm of selenium is recommended in the broiler chicken and turkey diets. Zero to 3 week-old chicks and 0 to 6 week-old turkeys should receive half of this selenium in an organic form which is more readily available to the bird.

2. Oral administration of a single 300 IU of vitamin E per bird will often cure exudative diathesis or muscular dystrophy. Birds with encephalomalacia do not usually respond well to treatment.