International Poultry Production

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Broiler Breeder 2019

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Vitamin A III

Pathology

The first lesions to appear for vitamin A deficiency are those in the oropharynx and oesophagus and tend to be confined to the mucous glands and their ducts. Here the original epithelium is replaced by a keratinising epithelium that blocks the ducts causing the mucous glands to swell with secretions and necrotic debris.

Small white nodules can be found in the nasal passages, mouth, oropharynx and oesophagus and, on occasions, in the crop. Small ulcers can accompany these lesions and because the integrity of the mucous membrane has been lost there can be accompanying localised viral and bacterial infections.

Clinical signs and lesions associated with vitamin A deficiency in the respiratory system are variable and often difficult to differentiate from infectious coryza, fowl pox, infectious bronchitis and other tracheal infections. In vitamin A deficiency thin diphtheritic membranes are usually confined to the hard palate and its cleft.

Chronic vitamin A deficiency can cause damage to the renal tubules in the kidney and this can ultimately lead to a visceral gout.

Diagnosis

Diagnosis is based on clinical and post mortem findings and demonstration of deficient feed and/or tissues from affected birds. Since affected/deficient birds respond quickly to vitamin A, diagnosis can be confirmed by administering vitamin A via the water and seeing the response to treatment.

Hypervitaminosis A

Mistakes in poultry ration formulation by as little as x3.5 the recommended level of vitamin A can result in toxicity or hypervitaminosis.

The signs of vitamin A toxicity in broilers include slow growth, an unsteady gait, reluctance to walk and an elevated incidence of tibial dyschondroplasia.