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Review

Production and growth related disorders and other metabolic diseases of poultry – A review

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Abstract

In humans, metabolic complaints may be associated with a failure in one of the body hormone or enzyme systems, a storage disease related to lack of metabolism of secretory products because of the lack of production of a specific enzyme, or the breakdown or reduced activity of some metabolic function. Some of these disorders also occur in poultry, as do other important conditions such as those associated with increased metabolism, rapid growth or high egg production that result in the failure of a body system because of the increased work-load on an organ or system. These make up the largest group of poultry diseases classified as metabolic disorders and cause more economic loss than infectious agents.

Poultry metabolic diseases occur primarily in two body systems: (1) cardiovascular ailments, which in broiler chickens and turkeys are responsible for a major portion of the flock mortality; (2) musculoskeletal disorders, which account for less mortality, but in broilers and turkeys slow down growth (thereby reducing profit), and cause lameness, which remains a major welfare concern. In addition, conditions such as osteoporosis and hypocalcaemia in table-egg chickens reduce egg production and can kill.

Keywords: Metabolic; Musculoskeletal; Cardiovascular; Ascites; Osteoporosis

1. Introduction

Metabolic disorders may be classed as illness associated with a failure in one of the body hormone or enzyme systems, storage disease related to lack of metabolism of secretory products because of the lack of production of a specific enzyme, or the failure or reduced activity of some metabolic function. In humans these conditions frequently have a genetic cause (Stanbury et al., 1983). Known classical genetic loci in poultry have been included in a recent article by Romanov et al. (2004). There are numerous genetic, metabolic disorders in poultry (Migaki, 1982) but these are rare in commercial poultry.

In poultry it is usual to include under the heading of 'metabolic disorders' those conditions associated with increased metabolism, rapid growth rate or high egg production that result in the failure of a body system because of the increased work-load on that organ or system. A broad interpretation of metabolic disorders within the general areas shown below, will be used for this review, but because of the extensive literature on some of the disorders not all of the references will be listed.

(1) Metabolic disorders that result from an increase production of, or deficiency of or failure in the production, synthesis, or transport of an enzyme, hormone or secretory mechanism.
(2) Metabolic disorders that result from high nutrient intake, rapid growth, high metabolic rate, pulmonary or systemic hypertension, and high egg production or a rapid increase in egg production.
(3) Other conditions that could be classed as metabolic disorders related to: (a) management defects; (b) nutritional deficiency or excess; (c) infectious agents; (d) toxins.

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2. Metabolic disorders that result from the failure or deficiency in the production, synthesis or transport of an enzyme, hormone or secretory mechanism

Migaki (1982) listed 17 inherited metabolic diseases of birds including pigeons, quail and 'pet birds'. In poultry he reported two defects of amino acid metabolism in chickens, tyrosinase-positive albinism and protein binding riboflavinuria; one defect of lipid metabolism, hyperlipidaemia in chickens; two defects in connective tissue, muscle and bone, inherited muscular dystrophy in both chickens and turkeys; two defects in transport, ADH-responsive (kidney) nephrogenic diabetes and uric acid hyperuricaemia, both in chickens. He also reported four defects in circulating enzymes and plasma proteins: C_1 deficiency in chickens; dysgammaglobinaemia in chickens; α_1-antitrypsin deficiency in turkeys and catalase acatalasaemia in ducks. Other researchers have also reported some of these defects in chickens: Benoff and Buss (1976) described hereditary diabetes, Cole and Austic (1980) hereditary uricaemia, and Wilson et al. (1988a,b) inherited muscular dystrophy.

Other defects reported include high density lipoprotein deficiency (Poernama et al., 1990); thyroiditis in obese strain chickens (Wick et al., 1989; Brown et al., 1991); trimethylamine taint in eggs (Butler et al., 1984) caused by an inherited inability to synthesize adequate amounts of trimethylamine oxidase; an enzymatic abnormality in the skin of Sebright roosters causing an increased conversion of androgens to oestrogens in the skin resulting in a female feather pattern (George and Wilson, 1980); ascorbic acid diabetogenesis (Meglasson and Hazelwood, 1982), and a genetic defect in hens that were unable to deposit riboflavin in their eggs. The embryos died at about day 13 because of impaired fatty acid oxidation (Abrams et al., 1995). There was decreased activity of flavin adenine dinucleotide (FAD)-dependant medium-chain acyl CoA dehydrogenase in liver and heart.

2.1. Fatty liver and kidney syndrome (FLKS) in broilers

FLKS is a biotin deficiency-related metabolic disease in broiler chicks, 2–3 weeks of age, resulting in impaired hepatic gluconeogenesis and increased fat deposition. The problem is caused by low activity of the biotin dependent enzyme pyruvate carboxylase. Birds die from hypoglycaemia and the clinical signs and death are related to hypoglycaemia (Whitehead et al., 1978). The condition usually occurs suddenly, as an outbreak, associated with some management, feed (perhaps fat level) or environmental change that affects feeding. Affected broilers are usually well grown. Clinical signs include aphagia, lethargy and weakness with uncoordinated behaviour and head movement (sometimes classed as nervous signs). The chicks may lie on their breast with their neck and legs extended (Butler, 1976). Mortality can vary from 5% to 35%. At necropsy the liver and kidneys are markedly enlarged, pale and fatty. Adequate dietary biotin will prevent FLKS and the condition is no longer seen in broilers on commercial rations.

2.2. Fatty liver and kidney syndrome in turkeys

Fatty liver and kidney syndrome has been produced experimentally in young turkeys (Whitehead and Siller, 1983).

2.3. Spiking mortality syndrome in broiler chickens

Spiking mortality is the name given to a metabolic disorder of previously healthy, normal appearing, broiler chickens that experience a sudden increase in mortality between 12 and 18 days of age. Live chicks are found recumbent and uncoordinated, frequently lying on their breasts with legs extended. Reported nervous signs are related to the inability to rise and head movement. The clinical signs and death are caused by hypoglycaemia (Davis and Vasilatos-Younken, 1995). This hypoglycaemia is a metabolic disorder that may be related to melatonin deficiency caused by lack of a long dark period. Melatonin does have an effect on metabolism (Apeldoorn et al., 1999) and the use of a long daily dark period will usually prevent this problem. There are several reports of spiking mortality caused by infectious viral agents such as arenaviruses (Davis, 2003). It is unlikely that infectious agents cause this condition, but sub-clinical infection by a variety of biological organisms, or other stresses, could be a predisposing cause.

What has been called 'spiking mortality' in turkey poults may now be referred to as 'poult enteritis-mortality syndrome' (Barns and Guy, 2003).

2.4. Tip-over in turkey poults

Over the past 18 years there has been an increase in the percentage of newly hatched turkey poults that are found on their backs in the litter, unable to right themselves. When righted they may recover or they may tip over again. Various aetiologies have been postulated, such as a large yolk sac, but the cause is not clear. Friedman and Pantanowitz (1994) have reported that the poults have ketosis that may cause the disorientation. The incidence is reported to be higher in poults from younger breeder flocks (P. Gazdzinski, personal communication).

2.5. Dwarfism

Using dwarf female breeders may affect growth rate of progeny but there is no evidence that it causes