NON-INFECTIONOUS FORM OF LEG WEAKNESS IN COMMERCIAL BROILERS –AN OVERVIEW

Dr. G. Raj Manohar Ph.D. and Dr. P. Kanagaraju Ph.D
Tamil Nadu Veterinary and Animal Sciences University
E-mail: rajmanovet@gmail.com

Diseases of the locomotor system are gaining economic significance in poultry industry. A normal gait is an integrated function of the nervous, muscular and skeletal systems. A failure in any of the above component will clinically result in leg weakness or lameness. This will cause increased culling rate or mortality in broilers.

(a) Genetic selection

Genetic selection appears to be a most effective means of preventing non-infectious skeletal disorders and in recent years has had a major impact on decreasing the incidence of disorders such as tibial dyschondroplasia (TD). Kestin et al. (2001) observed that genetic selection for modern fast growing birds resulted in increased leg disorders.

(b) Growth rate

Rate of growth has long been shown to affect the incidence of leg disorders (Su et al., 1999). Male broilers which grow faster than females have been reported to show about twice the incidence of skeletal disorders as well as a poorer walking gait.

(c) Feed restriction

Quantitative feed restriction throughout the growth period has been shown to cause a proportional reduction in skeletal disorders, in addition to a reduced growth rate. A restriction of feed sufficient to produce a large decrease in growth rate has been shown to virtually abolish skeletal disorders (Riddell, 1983). Qualitative feed restriction has also been used to reduce the incidence of leg disorders i.e. reducing the metabolizable energy density of the feed (Hulan and Proudfoot, 1987). Broilers fed a low energy diet were able to catch up with full-fed controls before market-age. Plavnick and Hurwitz (1990) found that broilers fed a reduced protein diet during the second week of life failed to catch up with full-fed controls by market age.

Lighting regimes

Continuous or near-continuous lighting is the norm for commercial broiler production, in order to maximise food intake and hence growth rate. It has been suggested...
that changing the photo period: scoto period from the commonly used 23 h light: 1 h dark to a more natural light: dark pattern may limit expression of skeletal deformities (Sorensen et al., 1999).

**Food conversion efficiency and body conformation**

Genetic selection was found to be responsible for 30 % improvement in FCE. The term ‘biomechanical forces’ is normally used to describe the pressures and strains that act upon developing chondrocytes and bone. The skeleton may be subjected to excessive forces as a consequence of a disproportionate development of muscle: bone coupled with immaturity of the leg bones in birds that achieve a high body weight rapidly. The magnitude of biomechanical forces alone may not, however, be sufficient to produce skeletal disorders.

**Exercise (activity)**

Increased exercise (activity) is believed to prevent leg problems in chickens. A number of factors such as genetic origin of the birds, manipulation of the environment, manipulation of different feeding strategies and lighting regimes affect the activity of birds.

Broilers housed in cages whose exercise is restricted show a high incidence of skeletal disorders when compared with group-housed birds with a litter floor (Rizk et al., 1980). The effect of activity level indicates that leg disorders may be reduced by encouraging locomotion and increased activity early in the growing period.

**Circadian rhythms**

The circadian rhythms are primarily under endogenous control, by means of an internal body clock. However, the rhythms can be modified by certain environmental stimuli, the most important of which in poultry is light. Classen (1992) has suggested that reduction of the 23 or 24 hour commercial photoperiod by adding an appreciable period of darkness may have a beneficial effect on skeletal growth and development.

**Nutrition**

Edwards (2000) indicated that dietary content of eight vitamins, thirteen elements and six amino acids and protein and energy may be directly involved in leg disorders or skeletal problems in poultry.

Vitamin A toxicity causes an osteodystrophy characterised by abnormal thickening of the growth plate zone, depressed calcification and lameness. Feeding fast-growing broilers low in calcium or high in phosphorus produces a high incidence of both rickets and tibial dyschondroplasia (Riddell and Pass, 1987).
Calcium and phosphorus imbalances of Ca: P ratio with an excess of phosphorus increases the incidence of dyschondroplasia (DC). When too much phosphorus is present in the ration, the Ca: P ratio is disturbed. To balance the excessive amounts of blood phosphorus, calcium reserves are drawn upon in an attempt to maintain an approximate 2:1 ratio within the blood. This mobilisation of calcium results in decalcification of the bones.

Perosis occurs due to the deficiency of manganese, choline, niacin, folic acid, biotin, pyridoxine and zinc in broilers characterised by a failure of longitudinal growth, but the growth in width and mineralisation is normal. The legs are shortened and thickened with enlarged hock joints. Because of the still and deformed legs, there is a loss of motion and the bird dies of starvation.

Manganese (Mn) deficiency in bone cause ‘slipped tendon’ disease in chicken. High calcium rations are supposed to increase manganese requirement of birds. The excess of calcium from limestone has no effect on manganese utilization but calcium from other sources viz. oyster shell, defluorinated rock phosphate and dicalcium phosphate will decrease the utilization of manganese in chick rations.

Niacin deficiency will result in deformed legs. There is lateral or medial deviation of the distal end of the tibio-tarsal bone with a corresponding deviation in the tarsal metatarsal region. This causes a change in the direction of the legs and results in a hock-out condition. This leg deformity is also called twisted leg, slipped tendon or perosis.

Riboflavin deficiency may appear in chicks fed exclusively on cereals and their by-products. There is diarrhoea, emaciation and development of “Curled-toe paralysis” which causes the chicks to walk on their hocks with their toes curled inwards. Pantothenic acid and Pyridoxine deficiencies in chicks cause myelin sheath-degeneration resulting in incoordination and inability to move. The birds have a ‘jerky’ movement of legs when they try to walk.

Magnesium (Mg) is of importance in bone development (especially bone hardness). Excessive amounts of magnesium in the ration interfere with proper bone formation. The bones are soft because magnesium has an antagonistic action against calcium and therefore ossification is not proper. It is usually observed as leg weakness or as leg deformity of the chickens. The disease in chicken is associated with the feeding of large amounts of dolmitic limestone, because of its high magnesium content.

Fluorine poisoning may occur when rock phosphate, which contains considerable fluorine is being used to correct a phosphorus deficiency. Bones may be unusually hard with
exostoses or they may show osteomalacia and osteoporosis. Because of the stiffness in bone, lameness is often observed in birds.

**Stocking density**

High stocking density can increase the incidence and severity of leg disorders.

Sorensen *et al.* (2000) studied the effect of age and stocking density on leg weakness in broiler chickens and concluded that the lower stocking density substantially reduced the prevalence of leg weakness.

**REFERENCES**


