Mycotoxins affect bone structure and leg weakness

Feed contaminated with mycotoxins plays an important role in causing leg weakness in poultry. For this reason, controlling polar and nonpolar mycotoxins with available strategies will undoubtedly lower the amount of leg weakness incidences.

By Devendra S. Verma

Mycotoxins in feed may cause skeletal disorders found in poultry.

A number of factors are known to influence normal bone development in fast growing meat birds as well as laying hens. Interestingly, the incidence of leg problems is generally higher in males than in females. Nutrition, genetics, sex of the bird and growth rate, all play a major role in the development of bone disorders. The anatomical difficulty in free movement and the dominance of normal birds make the lame birds deprived of normal feeding and watering. In addition, broilers with lameness are difficult to eviscerate properly with the automatic eviscerating machines, thereby rendering them liable for discard, despite better body weights.

Birds can be afflicted with varying degrees of skeletal disorders at some time during their productive life cycle. These problems are pronounced when fast growth rate or an infectious agent, such as bacteria, viruses and mycoplasma, is involved.

Normal bone development

The leg bones are one of the fastest growing bones in the skeleton and, coupled with their weight bearing characteristics, it is not surprising that femur, tibiotarsus and tarso metatarsus bone problems are so prevalent. In broilers and turkeys, leg problems occur early in life and lead eventually to morbidity with failure to eat and drink or in extreme cases, mortality.

Bone is a cartilaginous connective tissue with the unique property of being mineralised *(Figure 1)*. The mineral component is essentially calcium phosphate. The calcium-phosphorous ratio of bone is approximately 2:1. This is, essentially, the reason why to maintain this important ratio during feed formulation.

Bone is an extremely important mineral reserve in the body, representing approximately 99% of calcium, 88% of phosphate, 80% of bicarbonate, 50% of magnesium and 35% of total mineral body reserves.

Potential contributors of disorders

Besides nutrition and management, skeletal disorders found in poultry can also be caused by mycotoxins in the feed. Some of the major skeletal disorders wherein involvement of mycotoxin has been implicated are presented in *Table 1*.

Tibial dyschondroplasia (TD)

TD is characterised by an abnormal cartilage mass in the proximal head of tibiotarsus, most commonly seen in broiler chickens. Symptoms are first seen at 21 – 35 days. Birds are reluctant to move and when forced to walk, they do so with swaying motion or with stiff gait. TD relates to disruption of the normal metaphyseal blood supply in the proximal tibiotarsal growth plate, where the resulting disruption in nutrient supply means that the normal process of ossification does not occur. A typical cartilage plug therefore develops. As the bone grows, a lateral displacement of the growth plate appears, causing characteristic bowing or bending of the legs. The proximal tibiotarsus shows the greatest development of TD between 21-24 days.

Among causes such as genetics and electrolyte balance, mycotoxins also play

Prevalence of various mycotoxins in feed

Mycotoxin surveys from around the world indicate that protein sources such as rapeseed meal, cottonseed meal, groundnut cake, sunflower cake, copra meal and palm kernel meal are more susceptible to mycotoxin contamination than conventional raw materials such as soybean meal. Owing to high prices of conventional raw materials during certain years, feed manufacturers have been forced to opt for alternatives to soybean meal, therefore increasing the potential for mycotoxicoses for many livestock species. Similarly, the cost of maize has forced a look at other energy sources, including by-products such as rice bran, wheat bran and screenings. Many mycotoxins are concentrated in the outer covering of the seeds and therefore the chances of mycotoxin related problems are increased when such materials are used in animal rations. For example, during the milling process, DON (deoxynivalenol) was found in the highest concentration in the bran and lowest in the flour. Mycotoxins from these by-products in combination with mycotoxins from more traditional ingredients can result in toxicological interactions.

Table 1 - Skeletal disorders and the involvement of mycotoxins

Skeletal Disorders	Mycotoxin involvement
Tibial Dyschondroplasia	yes
Cage layer Fatigue	_
Rickets	yes
Chondrodystrophy	_
Spondylolisthesis/ Kinky Back	_
Femoral Head Necrosis	_
Foot Pad Dermatitis	_
Articular Gout	yes

Figure 2 - Aflatoxin blocking vitamin D absorption

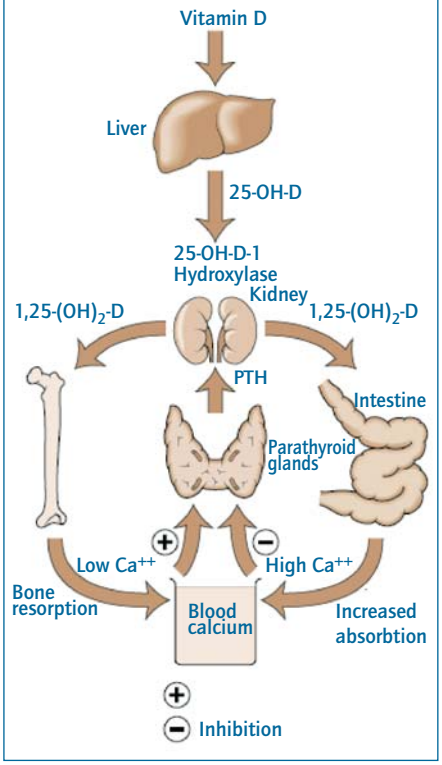
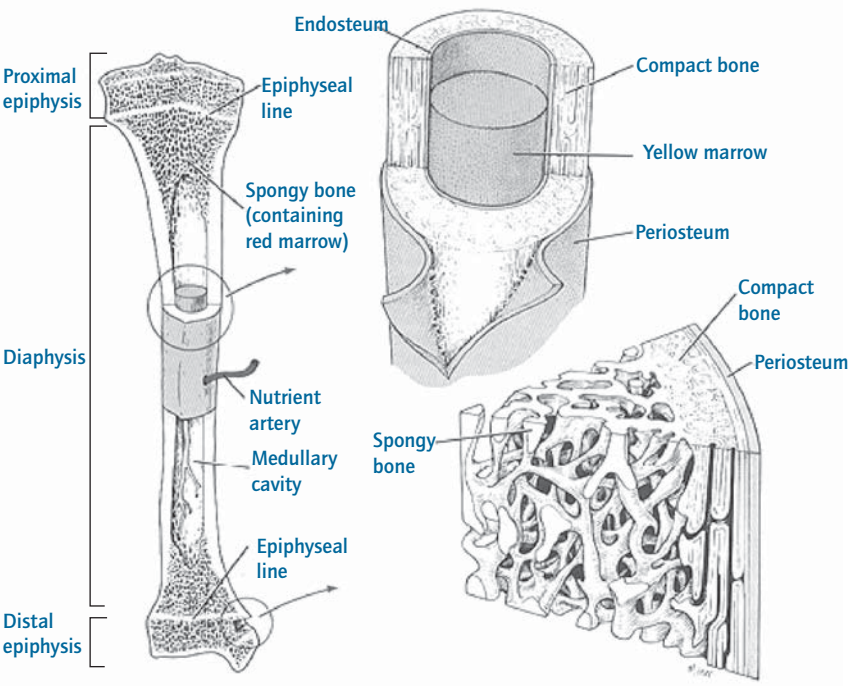


Figure 1 - The structure of a bone with its unique mineralised properties



an important role in the development of TD. Mycotoxins produced by various Fusarium molds are known to affect TD.

The water soluble mycotoxin fraction isolated from Fusarium roseum in oats has been found most problematic. One of the six major components of this fraction, known as TDP-1, was found to cause 100% TD when fed at 75ppm. TDP-1 MBA has since been isolated as Fusarochromanone.

More recently it was indicated that moderately high levels (75ppm) of fusarochromanone caused 100% incidence of TD in broilers. The minimum dietary level of this toxin needed to produce leg problems was 20ppm.

Rickets

Rickets (hypocalcaemic) most commonly occurs in young meat birds, the main characteristic being inadequate bone mineralisation. Calcium deficiency is the main problem,induced by feed deficient in Calcium, Phosphorous or vitamin D3. In most field outbreaks, abnormal bird behaviour is seen between 7-10 days old. Weak bones and rib beading can be see at around 10-14 days old, where 10 -100% of the flock is affected. Their bones are rubbery and the rib cage is flattened or beaded at the attachment to the vertebrae.

In most cases of rickets, a deficiency of vitamin D3 is often suspected. This can be due to a simple dietary deficiency, inadequate potency of D3 supplement or other factors that reduce the absorption of vitamin D3. Rickets is often more problematic when diets contain mycotoxins and especially aflatoxins. Normally, Vitamin D2 & D3 is converted to 25-hydroxycholecalciferol (25-OH), which is a circulatory form of Vitamin D3. This is then converted to 1,25 di hydroxycholecalciferol (1,25-OHD) or calcitrol, the most biologically active form of vitamin D.

Aflatoxin reduces vitamin D absorption and liver damage prevents conversion to the active 25-OH form of vitamin D3 *(Figure 2)*. It is unclear, however, if the mycotoxins create a specific metabolic deficiency of vitamin D3 and of other nutrients or if they simply affect the bird by reducing the feed intake. In mycotoxin contaminated diets, especially those from Fusarium molds, it is recommended to increase levels of vitamin D3.

Articular Gout

Articular Gout refers to the condition in which high plasma uric acid leads to precipitation of urate crystals, either in synovial fluid and tendon sheaths of various joints, especially the hock joint, or on serous surface of various visceral organs when kidneys are dysfunctional. Sustained hyperurecemia is most commonly caused by decreased renal clearance of urate. Deposition of such urate crystals at the joints is referred to as Articular Gout. Mycotoxins like aflatoxin, ochratoxin, and citrinin are all known for their kidney dysfunction mechanism. When feeding aflatoxin at levels up to 5ppm of diet, one may, after 21 days, observe thickening of glomerular membranes of glomerular apparatus of chickens leading to articular gout.

Another mycotoxin, namely oosporein (formed from mold, Chaetomium spp) not only severely affects epithelium of proximal tubules of the nephron but also the basement membranes. These changes in the kidney lead to hyperurecemia and ultimately leading to gout.

Economic losses

It is believed that leg abnormalities cause more economic losses than any other abnormality in chickens. It has been estimated that 2-6% of all broilers display some observable signs of skeletal problems. However, many more chickens are affected in a less visible way. Leg abnormalities result in reduced feed utilization and growth rate, mortality, and down-grading in the processing plant, followed by harsh economic losses. Unlike in North America, where efforts have been made to accurately monitor the losses incurred in the livestock industry due to mycotoxicoses, no such detailed information is available for the Asia-Pacific region. Losses due to mycotoxicoses have been estimated

at more than $1billion in Canada and over $2.5billion in the US during the 1990s.

Loss of broilers

Broilers have a life span of approximately 5 - 7 weeks after which they are slaughtered for meat. The important production parameters that a disease may affect include weight gain, culling and mortality rates and condemnation at slaughter. In terms of weight gain, no published data has been found on the effect of skeletal disorders on weight gain although they are said to have an impact. Regarding mortality and culling, a UK trial showed that 0.8% of broilers between 15 days old and age of slaughter, were culled for lameness. The suggested mortality/culling rates for skeletal problems reach a maximum of 3% of broilers placed per year.

At a large poultry processing plant in 1992, 2.09% of broiler carcasses were rejected. Of these, joint lesions were responsible for 0.31% with rejection rates of 3% or more. It is noted, however, that the 19.5% of condemnations for emaciation were probably the result of leg weakness. If the assumption is true, around 20% of condemnations in broilers from farms with high condemnation rates were directly or indirectly attributable to leg weakness (skeletal problems). If the relative importance of skeletal problems is the same in flocks with average condemnations rates, then around 0.3% (20% of 1.57%) of broilers slaughtered are likely to be condemned for skeletal problems. Between 0.2% and 0.5% of broilers are slaughtered per year.

Loss of breeder and laying flocks

Studies of losses due to disease in egg laying birds tend to be concentrated on birds in lay. Skeletal problems, however, also cause losses during the rearing period in birds destined for breeder and commercial egg laying flocks. The life span of egg laying birds has been divided into 0 - 19 weeks growing period and 20 - 70 weeks laying period. Mortality/culling is the most important production parameter affected by skeletal disorders in egg laying birds. Little data has been found on mortality and culling rates for skeletal disorders in growing birds in breeder and layer flocks. In a survey of mortality in chicks from 0 - 70 days old in nine breeder flocks, 1.3% of birds that died had deformed hocks to give an overall mortality rate from the condition of around 0.02%. However, as the study only covers the early part of the birds' lives, it is an underestimate. The same rate applied proportionately over 19 weeks is 0.04%. The suggested mortality/culling rates for skeletal problems in birds aged 0 - 19 weeks is a minimum of 0.04%, and in birds aged 20 weeks (point of lay) to 70 weeks (slaughter) is a minimum of 0.1% and a maximum of 1.0% of birds in lay.

From the above it is evident that feed contaminated with mycotoxins plays an important role in inducing leg weakness.
Differential diagnosis will always allow us to determine what actions are needed to control such incidences. Controlling all mycotoxins (polar and nonpolar) with available control strategies will undoubtedly bring down the leg weakness incidences and benefit the poultry industry as a whole. ■