

«Review»

## Leg Weakness Pathology in Broiler Chickens

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The aim of the present work is to make a review on the literature concerning the incidence of some major pathologies of leg skeleton in broiler chickens and broiler breeders, related to poor animal welfare. On the basis of data on the incidence of leg skeletal pathology published by us and by other researchers, this review goes through some reports on femoral head necrosis (FHN), tibial dyschondroplasia (TD), rupture of the gastrocnemius tendon (RGT) and rickets. The welfare of broiler chickens and breeder flocks continues to be closely related to leg skeletal pathology. Although some reports establish that most abnormalities related to legs in chickens were largely overcome by selection work and improvement of production systems by the end of the 20<sup>th</sup> century, the problems still occupy an important place in broiler production.

Apart published literature data, own results from the last decade (2001–2011) were also used.

**Key words:** broilers, leg's skeleton, pathology, welfare

*J. Poult. Sci.*, 49: 63–67, 2012

### Introduction

#### *Femoral Head Necrosis*

Disorders related to the extremities were a serious problem during the 1980's; however they have been overcome to a great extent through genetic selection and improving breeding technologies. Diseases of the extremities can however occur for various reasons, for example bacterial infections, which could cause necrosis of the femoral head

From a chronological viewpoint, the majority of conditions considered to be important causes of lameness have been associated with non-infectious etiology (McNamee and Smyth, 2000). Osteomyelitis was first reported as a cause for lameness in commercial broiler chickens in Australia (Nairn and Watson, 1972). The etiological agent was determined to be *Staphylococcus aureus*. The condition, named at that time bacterial chondronecrosis with osteomyelitis (BCO), was consequently reported in broilers from various parts of the world: Australia, USA, Canada, and Europe (Griffiths *et al.*, 1984; Riddell and Springer 1985; Thorp *et al.*, 1993; Thorp and Waddington, 1997; McNamee *et al.*, 1998). Studies on commercial broilers in Canada showed that deformations of long bones were the primary causes of lameness, while arthritis and osteomyelitis were only associated with 10% of lameness-related losses during the last week before slaughter (Riddell and Springer, 1985). A decade

earlier, a study by Thorp *et al.* (1993) in the UK, which planned for possible cases of birds with thigh lesions, showed a high prevalence of BCO. Also, osteomyelitis was usually diagnosed in birds with lameness, studied by Thorp and Waddington (1997).

Naturally occurring bacterial osteomyelitis with synovitis was found in turkeys from Australia (Nairn and Watson, 1972) and the USA (Nairn, 1973), where *S. aureus* was the commonest isolate. Consequently, Wyers *et al.*, (1991) described osteomyelitis associated with *S. aureus* in male broiler parents in France. There are data for a complex of femoral head necrosis, epiphysiolysis, osteomyelitis, and tendovaginitis, associated with *S. aureus* in male broiler parents in Bulgaria and reproduction of the condition with the isolate in broiler chickens (Dinev, 2009).

Nairn and Watson (1972) reported that the prevalence of lameness caused by BCO in broiler chickens could reach 50%, with over 5% mortality in the herd. The relative prevalence of the pathogens in cases of BCO in broiler flocks was studied at the end of the last century in Great Britain and Northern Ireland (Thorp *et al.*, 1993; McNamee *et al.*, 1998). Thorp *et al.* (1993) isolated bacteria from the proximal end of the femurs with lesions, indicative of BCO. From the lesions samples, coagulase-positive staphylococci (22.2%), coagulase-negative staphylococci (11.1%), and *E. coli* or mixed cultures (13.3%) were isolated. Riddell (1997) reported *Staphylococcus* spp. as the most commonly isolated bacterium from arthritis/tendinitis/osteomyelitis in broilers from west Canada. He also noted an increase in the prevalence of musculoskeletal infection associated with *E. coli*.

The results from recent large-scale investigations regard-

Received: September 24, 2011, Accepted: November 29, 2011

Released Online Advance Publication: January 25, 2012

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ing the incidence of FHN (Dinev, 2009) confirm the tendency observed by Riddell (1997) concerning increased incidence of musculoskeletal problems associated with *E. coli* infections among broilers in West Canada. In the previous work taking into consideration that no attempts have been made to reproduce the disease with isolated *E. coli* strains, authors could hardly reflect on whether the participation of the infectious agent was a primary cause or it was a secondary event in FHN cases. Actually, hypotheses could be true as in one part of FHN cases (25%), bacteriological results were negative, and histologically, osteochondrosis but not osteomyelitis was detected. These results are closer to the view of authors assuming that the majority of skeletal disorders causing lameness are due to disorders in the growth of tubular bones, often affecting the growth plate or manifested by bone deformities (Poulos *et al.*, 1978).

On the most of FHN cases (75%) with present osteomyelitic lesions and with evidence of infectious agent (mostly *E. coli*) are a serious precondition to assume an infectious cause. It is believed that the infection spreads via the blood circulation from the respiratory or the alimentary tract (Ross Tech 01/40 – Leg health in broilers). This is further confirmed by the concurrent signs of colisepticemia (polyserosites, cellulites) in about 1/3 of chickens with FHN and osteomyelitis. According to Julian (1985), osteomyelitis is the commonest cause of long bone necrosis in chickens and turkeys.

Apart the expressed view that dyschondroplastic lesions could play the role of a core for bacterial necrosis and consequently, for osteomyelitis (Wyers *et al.*, 1991), was point out that the rickets had a definite predisposing role with this regard, as in half of observed cases with fractures, there was evidence for rickets as well (Dinev, 2009). In the last report was not find a case of osteomyelitis in broilers without concurrent FHN. A significant difference in the cases of osteomyelitis that was detected and those described so far in broiler chickens, was the bacterial etiology. In more than 90% of the bacteriologically examined samples with FHN in association with osteomyelitis, *E. coli* was isolated (Dinev, 2009). This etiogenetic trait is different from reviewed cited facts where the etiology of FHN and osteomyelitis in broiler chickens was associated with *S. aureus* (Thorp *et al.* 1993; Thorp and Waddington, 1997; McNamee *et al.*, 1998; McNamee and Smith, 2000). On the basis of those data it could be summarized that in broiler chickens, *E. coli* had a primary role in the etiology of osteomyelitis and FHN unlike the growing broiler parents where *S. aureus* was mainly involved. It could not provide an explanation for the differences in the affinity to either agent in the respective category of poultry. Instead, on the basis of obtained results, was hypothesize that *S. aureus* is not always the commonest bacterial agent detected in bone infections of commercial broiler chickens as reported by many authors (Nairn and Watson, 1972; Griffiths *et al.* 1984; Riddell, 1997; McNamee *et al.*, 1988).

The age when the lesions were usually seen in chickens (about the age of 35 days), was associated with the effect of

intensive muscle growth by that time. Thus, the negative impact of the increasing weight could be used to predict experimentally induced lesions.

In conclusion, femoral head necrosis in broiler chickens was associated with lesions in the metaphysis, femoral head, proximal femoral growth plate and joint. Necrosis was most commonly caused by osteomyelitis, which was caused predominantly by *E. coli* infection (Dinev, 2009).

### Tibial Dyschondroplasia

Studies on commercial broilers performed in 1965 and 1978 showed that most skeletal abnormalities causing lameness were associated with long bones growth disorders, often affecting the growth plate or manifested as bone deformations (Poulos *et al.*, 1978). Consequently, tibial dyschondroplasia (TD) (Siller, 1970) and angular limb deformities (Julian, 1984) were pointed out as the primary cause of lameness. Dyschondroplasia is a fairly common cartilage defect in fast-growing young broiler chickens and turkeys, with the pre-hypertrophic cartilage of the growth plate being retained in the metaphyse (Poulos, 1980; Leach and Monsonego-Ornant, 2007). The lesion is most common in the proximal tibiotarsus and can be extensive without causing significant deformation or lameness. However, a greater mass of cartilage could lead to metaphyseal enlargement, bone weakness due to bending or fracture, or the formation of a sequestrum secondary to avascular necrosis (Julian and Bhatnagar, 1985).

TD is the most common skeletal anomaly associated with fast growth in numerous bird species, the result of which is the occurrence of bone deformation and lameness (Farquharson and Jefferies, 2000). It usually develops between 3 and 5 weeks of age (Bradshaw *et al.*, 2002). Leach and Nesheim (1965) reported that male chickens are more susceptible to TD, while others did not establish variations for the two genders regarding the prevalence of TD (Riddell *et al.*, 1971). Economic losses caused by TD through mortality and discarded material are enormous, reaching up to 30% during outbreaks (McNamee *et al.*, 1998). Birds with heavy lesions are predisposed to fractures during transportation to slaughterhouses, further increasing economic losses. Apart from that, specifically for broilers, TD-associated lameness is a major cause for the poor bird welfare in the flocks (Bradshaw *et al.*, 2002).

Selection for a shorter and faster fattening period results in increased occurrence of this skeletal disorder. This could be related to a transient deficiency during the phase of fast growth of long bones, especially the tibia, because the proximal tibia is the site of the most fastly growing growth plate (Angel, 2007). Since it was first described by Leach and Nesheim (1965), TD was established to be mostly prevalent in broiler chickens (Leach and Lilburn, 1992) and turkeys (Wyers *et al.*, 1991) during the development of their maximum genetic potential.

Recent large-scale field investigations have attempted to define the frequency of TD lesions incidence as a cause for lameness among three different hybrid broiler-chickens

(Dinev *et al.* unpublished). The results of these studies showed that the highest prevalence of TD in broilers could be observed in the hybrid Pureline (27.7%), followed by Cobb 500 (26.5%), and Ross 308 (24.22%). The data showed that the variations in TD prevalence for the different hybrids were relatively small, compared to the results by Edwards (1984), who showed a variation of TD prevalence between 27 and 40% in a study of 5 broiler hybrids. The small differences in the prevalence of TD are probably due to the continuous selection aimed at improvement of the hybrids in this regard (Leach and Nesheim, 1965; Riddell, 1992; Kestin *et al.*, 1992).

Studies of Dinev *et al.* (unpublished) revealed a significantly earlier occurrence of macroscopic TD lesions, respectively at the age of 18 to 34 days (23.7 days in average) for Ross 308; from 16 to 23 days (19.6 days in average) for Cobb 500, and from 22 to 34 days (28.8 days in average) for Pureline, compared to results from studies into the problem in commercial broiler flocks of Ross and Cobb, in which the lesions became apparent between 28 and 42 days of age (35 days in average) (McNamee *et al.*, 1998). The earlier by 11 days (mean value for all 3 hybrids) occurrence of macroscopic lesions in that study was a reason to believe that this fact was due to genetic selection aimed at faster growth (Poulos, 1978; Bradshaw *et al.*, 2002).

Also did not establish dependence between the parent flocks' age and the occurrence of TD in their hybrid broiler offspring (Dinev *et al.* unpublished). This correlates with the position of Nelson *et al.* (1992), based on results derived under experimental conditions. The data from that study also showed that the variations in the weight of 1-day-old chickens of younger or older parents had no influence on the prevalence of TD (Kestin *et al.*, 1992).

The relatively high prevalence of metaphyseal deformations of proximal tibiotarsus established in the study of Dinev *et al.* (unpublished) in association with TD lesions, was considered to be an important factor in the occurrence of broiler lameness (Thorp and Waddington, 1997). It was also considered that the consequences of deformations – fractures, morbidity, and mortality resulted in significant economic losses, as outlined by a number of authors (Orth and Cook, 1994; Farquharson and Jefferies, 2000). No significant differences in the prevalence of TD, related to the birds' gender could be found, as established by other authors as well (Riddell *et al.*, 1971), in contrast to the results of Leach and Nesheim (1965), who believed that males were more susceptible. Even though the fractures associated with TD were predominantly in males, the relatively low overall prevalence of this lesion did not allow for making conclusions on gender dependences. There have been reports that dyschondroplastic lesions were observed in most hollow bones – femur, humerus (Farquharson and Jefferies, 2000) yet this was not observed in the study of Dinev *et al.* (unpublished). According to that report, the observed TD lesions were located only within the proximal tibiotarsus, which correlates with the observations by Lynch *et al.* (1992), yet there were no lesions in the distal tibial metaphyse, as reported earlier

(Leach and Lilburn, 1992). The results from that analysis present current data on the detection of macroscopic TD lesions at significantly lower ages for different commercial broiler hybrids, a relatively high prevalence of TD-related deformities of the proximal tibiotarsus with the possible consequences as a cause for lameness, as well as the prevalence of TD for both sexes (Dinev *et al.* unpublished).

### Rickets

In other our work was performed a review of bibliographic data regarding the spreading of rickets forms, as well as of its associations with certain pathological conditions in broiler chickens that had gone through rickets (Dinev, 2012). The analysis of the literature review suggested that that the occurrence of subclinical and clinical rickets forms within broiler flocks are a possible prerequisite for a number of pathological conditions such as FHN, osteomyelitis, fractures, TD, vertebral column deformations with clinical expressions indistinguishable from spondylolisthesis, chronic respiratory disease (CCRD), ascites syndrome, etc. related to morbidity within the flocks. The gross lesions in the proximal tibiotarsus can be used for differentiation of clinical hypocalcaemic and hypophosphataemic rickets in broiler chickens. Macroscopically, hypocalcaemic rickets is hardly distinguished from tibial dyschondroplasia because the latter condition may be a consequence of the former in etiological aspect.

### Rupture of the Gastrocnemius Tendon

The rupture of the gastrocnemius tendon (RGT) is a recognized cause of lameness in broiler chickens and broiler breeders. In earlier reports, the state was thought to be rather congenital than acquired (Riddell, 1991). Later, the problem has been associated with viral (reoviral) tenosynovitis (Kibenge *et al.*, 1982; Jones and Kibenge, 1984). It was assumed that rupture of tendon occurred consequently to tenosynovitis and that both lesions were components of one disease (Itakura *et al.*, 1977). However, some viral isolates used in experimental studies produced lesions in tendons other than the gastrocnemius and viruses were not always reisolated from macroscopically changed tendons (Duff and Randall, 1986). It is further hypothesized that reoviruses could not provoke ruptures by themselves, without the involvement of other predisposing factors (Duff and Randall, 1986).

Mycoplasmata and staphylococci were also associated with the problems but it is known that bacteria could colonize the tendon secondary to a viral tenosynovitis (Kibenge *et al.*, 1983; Kleven, 2003).

In some investigations, the tendon rupture was associated with several non-infectious factors as deficiency states, variations in tensile strength, glucosaminoglycan content, cellular structure of the tendon in the various avian species etc. (Cook *et al.*, 1983a, b). Other reports assume a traumatic aetiology, supported by histological data (Hill *et al.* 1988).

Riddell (1983) did not observe tenosynovitis in 4 broilers with ruptured tendons and presumed the involvement of

more than one cause. Similar cases of RGT in the absence of tenosynovitis were observed by other investigators as well (Randall, 1991). According to bibliographic data, the problem is observed in both genders and in both broilers for fattening and broiler breeders (Riddell, 1991).

The results of studies of the lesions in field cases in female broiler breeders Chaver minibro provided evidence for spontaneous rupture of the gastrocnemius tendon (Dinev, 2008). The findings were further confirmed by the lack of simultaneous tenosynovitis or arthritis. The absence of RGT in male birds, reared together with females during both the growth and productive periods supported the thesis for a non-infectious etiology in that case. Moreover, Duff and Randall (1986) concluded that in tenosynovites, especially those due to reoviruses, other tendons and ligaments of legs are also ruptured. The negative results of bacteriological and serological studies provided another proof for the lack of an infectious aetiology in that cases.

The dystrophic-necrobiotic alterations in tendons, the hemorrhagies and the fibroblastic-reparative tissue are suggestive for traumatic aetiology (Hill *et al.*, 1988). The author has, however, observed such lesions only in male birds unlike other study, where females were affected only (Dinev, 2008). According to Duff (1986), the dystrophic and necrobiotic changes adjacent to intratendinous hematoma could result from a compression atrophy.

Duff and Anderson (1986) conclude that in some birds there is a congenital predisposition of tendons to rupture. This observation is contrary to that of Devos (1963) and makes clear that synovitis and tenosynovites are not a prerequisite for rupture of tendons in birds. Thus, the assumptions about tendon ruptures and tenosynovites being one disease entity, are obviously inconsistent (Itakura *et al.*, 1977).

The tensile strength of the common tendon of the gastrocnemius muscle is lower in meat-type chickens compared to those from the egg-laying type (Riddell, 1991). This could therefore predispose to spontaneous tendon ruptures. According to Van Walsum (1979) the tissue of the gastrocnemius tendon in meat-type chickens was less organized and in many instances, there was a hypovascular zone just above the tarsal joint. This zone was associated with dense chondrocyte masses and lipid deposits in the tendon. Such changes could also predispose to non-infectious tendon ruptures.

Recently performed field studies by Dinev (2008) supported the opinion of investigators claiming that in some birds, there was a predisposition towards a spontaneous rupture of the gastrocnemius tendon (Duff and Anderson, 1986; Riddell, 1991; Duff and Randall 1986).

### Conclusions

The analysis of the overview indicates that the most common problems are related to skeletal leg pathology among modern hybrid broiler chickens, namely FHN, TD and rickets. FHN was most commonly caused by osteomyelitis, which was caused predominantly by *E. coli* infection.

Cases of TD are related to significantly younger chickens, approximately 24 days, among modern hybrid broiler chickens and are predominantly associated with insufficient Ca.

Subclinical and clinical rickets forms within broiler flocks are a possible prerequisite for a number of pathological conditions such as FHN, osteomyelitis, fractures, TD, vertebral column deformations with clinical expressions indistinguishable from spondylolisthesis, CCRD, ascites syndrome, etc. related to morbidity within the flocks.

The overview analysis also indicates that RGT is the most common problem related to the skeleton of the legs among parent flocks of broiler breeders. This condition could be related to some anomalies which have not been overcome at a high level of selection work.

### Acknowledgment

The author, Ivan Dinev, has applied for a grant, supporting publishing in peer-reviewed journals with impact factor - PROJECT BG051PO0013.3-05-001 "Science and Business", financed by Operational Programme "Human Resources", funded by the European Social Fund, the European Union.

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