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Bradshaw, R.H., Kirkden, R.D. and Broom, D.M. 2002. A review of the aetiology and pathology of leg weakness in broilers in relation to their welfare. *Avian Poult. Biol. Rev.* 13, 45-103.

A review of the aetiology and pathology of leg weakness in broilers in relation to welfare

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Summary

Leg disorders in broilers are a major economic and welfare problem. The aetiology of many disorders is complex but includes genetics, growth rate (due to feed restriction or lighting regime), feed conversion efficiency and body conformation, exercise, circadian rhythms, nutrition and stocking density. These categories are not mutually exclusive as one aetiological factor may affect another. Many studies of leg disorders fail to identify the specific pathological condition underlying the observed lameness. However, disorders may be classified according to underlying pathology as infectious, developmental and degenerative. This classification is difficult because these categories are not mutually exclusive. Infectious conditions include bacterial chondronecrosis with osteomyelitis (BCO or femoral head necrosis, FHN), tenosynovitis and arthritis, infectious stunting syndrome (ISS) and viral induced neoplasia. Developmental conditions include varus valgus disease (VVD), rotated tibia, tibial dyschondroplasia (TD), rickets, chondrodystrophy and spondylolisthesis. Degenerative disorders include osteochondrosis (often TD), epiphyseolysis (often classified as FHN), degenerative joint disease (DJD), spontaneous rupture of the gastrocnemius tendon and contact dermatitis. BCO, TD, dermatitis and VVD are the most common disorders. Outbreaks of leg disorders are often site / context specific. The welfare of broilers with leg disorders may be impaired due to pain from the condition, an inability to walk leading to frustration and associated problems of being unable to feed and drink due to immobility (which may result in starvation). In assessing welfare, the individual broiler must be considered irrespective of the frequency of occurrence of the disorder. Most studies of welfare in relation to leg disorders have used a subjective gait scoring method (0 is normal walking and 5 is unable to walk). Gait scoring is a practical method for assessing broiler lameness in the field. The method provides a useful tool to employ in the field without recourse to pathological investigation and, while the method conflates conformity with pathology, it is a helpful and constructive additional method to assist in welfare studies. For birds with scores greater than 3, lameness may be viewed as severe enough to potentially impair welfare. It is difficult to assess all disorders in relation to frequency of occurrence and their impact on welfare due lack of evidence. BCO (or FHN and BCN) is the most common disorder and is often severe in form. TD (incl. epiphyseolysis) and rickets is common, often sub-clinical but when severe is a considerable impact on welfare. Contact dermatitis may be common under certain conditions and causes poor welfare when severe. Gastrocnemius tendon slippage, tenosynovitis, DJD and spondylolisthesis are not so common but are likely to cause poor welfare when they occur (i.e. pain and prevention of certain behaviours). VVD and rotated tibia can be common but tend not to be directly painful unless another condition is present. However, they can cause poor welfare if the bird is not culled and lead to an inability to walk, feed, drink and perform other behaviours. ISS, viral induced neoplasia and chondrodystrophy have only a small impact on welfare in the UK flock, the last because it is no longer encountered. To decrease the prevalence of leg disorders, growth rate needs to be decelerated: meal feeding, feed restriction during the early period of rearing, lower stocking densities and increased activity can result in a considerable reduction in leg problems. Continuous lighting should be avoided; overall the light period should be reduced. There also needs to be careful management of litter to prevent dermatitis. Factors in the diet are also important, particularly Ca, P and D3 which can prevent certain disorders

(e.g. TD). Further research is needed to establish the prevalence of disorders, underlying pathology in relation to subjective gait scores and analgesics in relation to pain mechanisms. A forum for discussion of these issues should be established and further control-trials conducted to investigate these factors.

Introduction

Welfare concepts

In recent years, the welfare of animals has come to be regarded as something which can be assessed scientifically. This has occurred as a result of many studies which have been carried out in which careful measurements have been made of the effects of various treatments and conditions on animals.

As explained by Broom (1986, 1988, 1993, 1999, 2001), Broom and Johnson (1993), the welfare of an animal is its state as regards its attempts to cope with its environment. As this definition implies, welfare is a characteristic of the animal, not something which is given to it. Welfare varies on a scale from very good to very poor and can be measured.

Direct measurements of good welfare are difficult to make (Table 1). However, specific behavioural measures give some guidance and physiological measures may also be of value.

Table 1 Measures of good welfare

Variety of normal behaviours shown
 Extent to which strongly preferred behaviours can be shown
 Physiological indicators of pleasure
 Behavioural indicators of pleasure

Measures of poor welfare include both those which indicate a failure to cope with the environment, in that the individual dies or fails to grow, and those which show how much difficulty the animal is having in trying to cope. Just as in man, the welfare of an individual chicken is poor, both when growth is impaired, and when severely depressed or injured in a way that does not threaten life or growth. In Table 2, the range of measures of poor welfare which can be used are listed.

Table 2 Measures of poor welfare

Reduced ability to grow or breed
 Body damage
 Disease
 Immunosuppression
 Physiological attempts to cope
 Behavioural attempts to cope
 Behaviour pathology
 Self narcotization
 Extent of behavioural aversion shown
 Extent of suppression of normal behaviour
 Extent to which normal physiological processes and anatomical development are prevented

Broiler breeders and meat production birds

The major fundamental problems of broiler production which result in poor bird welfare are a consequence of selecting birds for a short, very fast growing life. The adult birds will tend to be too heavy for normal locomotion and tend to develop leg disorders. Since leg disorders are disabling and often associated with inflammation of joints, hocks and bone, it is clear that their incidence may cause a major welfare problem (Webster 1994). There is a study showing that leg pain is considerable in very heavy breeding male turkeys (Duncan et al 1991). Recent findings support the suggestions of Sorensen (1989) and Webster (1994) that lame broiler chickens are in pain when they walk (Danbury et al 2000; Kestin et al 1994; McGeown 1999) and that this pain causes them distress from which they seek relief through the self administration of the analgesic drug carprofen (McGeown 1999; Danbury et al 2000). Immobility associated with lameness is also likely to impinge on four of the five freedoms which farmed animals are expected to enjoy (FAWC 1992).

Recognition of the problem

In 1992 when the Farm Animal Welfare Council (FAWC) assessed the welfare of broilers. They visited many broiler producers and came to the following conclusion:

'Leg problems of varying degrees of severity were seen by the Working Group on nearly every farm visited during this study. Sometimes these involved only a slight abnormality and the birds were able to walk easily; but in the worst cases the birds were only able to move with great difficulty, using their wings to balance. Birds in the latter category were obviously distressed and at this stage they had problems in reaching food and water. We believe that any such bird should be humanely killed immediately. Between these extremes many birds were seen exhibiting gait abnormalities. Producers should be aware that in the most severe cases they may be in contravention of the Agriculture (Miscellaneous Provisions) Act 1968 by causing unnecessary pain and unnecessary distress. We had considerable difficulty in deciding when leg problems become unacceptable on welfare grounds. It is very difficult to decide easily whether individual birds are in pain; and whether that pain is a result of leg abnormalities. notwithstanding this, the Council considers it unacceptable for any bird to suffer pain or distress as a result of a leg problem.' (FAWC 1992).

More recently, the European Commission Scientific Committee on Animal Health and Animal Welfare produced a report on the welfare of chickens kept for meat production (broilers). This EC (2000) report addressed the issue of skeletal disorders, specifically leg weakness. The report concluded:

'Leg disorders are a major cause of poor welfare in broilers. Gait scoring surveys have shown that large numbers of broilers have impaired walking abilities and there is evidence that birds with score 3 or higher experience pain or discomfort. However, the subjective nature of the scoring system leads to difficulties in making direct comparisons between different studies and there is a strong need to develop objective measurement systems and to carry out systemic epidemiological studies. Femoral head necrosis is an important cause of poor welfare. Developmental disorders resulting from dyschondroplasia or other bone growth abnormalities represent less severe but more widespread problems. Continued effort is needed to improve genetic, nutritional and management methods of minimising these problems.'

Terminology regarding leg disorders

Leg weakness has been a portmanteau term used to describe a number of pathological states affecting broilers whose walking ability is impaired (Butterworth 1999). The terms 'lameness' and 'leg weakness' are often used to describe a series of infectious and non-infectious disorders which can affect the skeleton, muscle, tendons, skin or nervous system. While these terms may be useful in assessing overall prevalence of the condition, they focus on symptoms (pathology) and not

underlying causes (aetiology). It is therefore of little value in understanding the aetiology of specific disorders or relevant preventative measures (Thorp 1994).

As pointed out by Butterworth (1999), the Oxford Dictionary defines the terms 'weakness' and 'lameness' as follows:

Weakness = deficient in strength or power, easily broken, bent or defeated, weak point, defect.

Lameness = disabled by injury or defect in a limb especially foot or leg, limping or unable to walk normally.

It has become customary to describe the group of pathologies which result in impaired walking ability as 'leg weakness' (Butterworth 1999). While this term is a vague one used to describe a series of debilitating conditions, of infectious and non-infectious origin (Sorensen et al 1999) it is preferable because it includes all possible incidences without recourse to discussion concerning whether bird is truly 'lame' (i.e. disabled). This convention will therefore be used in this review except in the section on subjective gait scoring in which direct reference to lameness is more relevant.

Prevalence and cost

Most leg disorders in modern broilers are linked to skeletal abnormality (Kestin et al 1992) but it is difficult to accurately determine the cost or the nature of these losses (Sullivan 1994). Leg disorders can increase mortality and the number of culls, increase condemnations and increase downgrades from trimming (Morris 1993). Riddell and Springer (1985) reported the incidence of skeletal abnormalities in flocks of Canadian broilers. Their data revealed that the average incidence of chickens with skeletal disorders was 1.72% which included 1.10 % mortality culled in the field and 0.62% of the flock was condemned or trimmed as carcasses due to leg disorders. Morris (1993) conducted a national survey in the US and estimated leg problems cost between 80 and 120 million dollars. Overall, 1.1% mortality was attributed to leg problems and an additional 2.1 % of birds were reported to have been downgraded from leg disorders (Morris 1993). McNamee (1998) found 0.52 % of male and 0.38 % of female broilers were culled due to leg weakness. A more recent survey (which involved 2500 birds) commissioned by the British Chicken Association found that the proportion of birds with subjective gait scores of 3 or above was nearer 3% (cited EC 2000). The details of this study remain unclear and unpublished. This makes comparison between studies difficult and the reason for the differences between the studies remains unclear.

Studies of commercial broilers from 1965 to 1978 showed that most skeletal abnormalities causing lameness were associated with long bone growth disturbances, frequently involving the growth plate (Prasad et al 1972). Subsequently a MAFF/ADAS working party identified tibial dyschondroplasia and angular limb deformities as predominant causes (see Leach and Nesheim 1965, Siller 1970 for TD; see Julian 1984, Duff and Thorp 1985a for angular limb deformities). Su et al (1999) and Sorensen et al (1999) also found considerable TD. Pattison (1992) conducted a commercial post-mortem trial and found femoral head necrosis was the most common condition (38%) followed by infected hocks (13.1 %), twisted leg (11.1 %) and tibial dyschondroplasia (7.2 %). Riddell and Springer (1985) showed in commercial Canadian flocks that long bone deformity was the main cause while arthritis and osteomyelitis accounted for 10% of losses due to lameness (osteomyelitis was also low on the list for the MAFF working party). In 1992 FAWC indicated that osteomyelitis was increasingly recognised as a cause of leg disorders while Thorp et al 1993 targeted UK birds suspected of having a hip lesion and showed a high incidence of femoral head necrosis. This was also a common diagnosis in Thorp and Waddington (1997). Bacterial chondronecrosis with osteomyelitis or BCO (which McNamee and Smyth (2000) argue is usually the condition described as femoral head necrosis) was also found to be the most common cause of lameness in commercial birds in a systematic large scale study of lame broilers from commercial flocks (McNamee et al 1998; 1999a). Finally, Sanotra et al (2001) surveyed 28 broiler flocks in Denmark (8% of the total number of flocks in the country) and recorded incidences of tibial

dyschondroplasia, varus-valgus disease, crooked toes and foot pad burns and asymmetrical development of the tarsometatarsal (but not femoral head necrosis of BCO). The mean prevalence of each disorder was 57.1%, 37.0%, 32.6% and 42.0% respectively.

It is important to realise that only very recent data on leg disorders is relevant since the genetics of the birds and the conditions under which they are being reared changes frequently. Thus the problems of just five years ago may not be the problems of today.

Structure of review

The first section of this report will discuss general aetiological factors i.e. the common causes of lameness. This approach is appropriate insofar as disorders can be meaningfully classified according to their aetiology and pathogenesis. It is demanded by the fact that a proportion of the literature on the aetiology of skeletal disorders fails to specify the particular disorders occurring in the birds studied. For each individual pathological condition a description will then be provided of its aetiology, classified based on the recent EC (2000) report on broiler welfare. Emphasis will be placed on any preventative actions which may reduce incidence of specific leg-disorders. This classification will provide a framework within which to describe the various conditions but it is important to realise that much of the classification is somewhat arbitrary. A bird with one particular disorder may carry another and the first or the second disorder may lead to an angular deformity. Thus classifying birds as if they have one particular disorder or another is misleading. Also the severity of disorders varies. These points will be discussed further below.

Once specific pathologies have been reviewed, a section will examine the phenomenon of subjective gait scoring and other methods in relation to studies of welfare. The last sections will draw conclusions on the prevalence of particular disorders, their impact on welfare, how they can be prevented and make recommendations on specific areas which require further research.

Aetiology of skeletal disorders

Factors which have been implicated in causing lameness include infectious agents (e.g. bacteria and viruses), genetic selection, growth rate, food conversion efficiency and body conformation, exercise and nutrition. This section will be restricted to non-infectious aetiology classified as follows: genetics, growth rate, body conformation, exercise, circadian rhythms, nutrition and stocking density. The main causes of non-infectious and non-nutritional leg disorders are genetic susceptibility and management factors (Reiter and Bessei 1998a). Infectious agents which are thought to result in severe problems, will be discussed in the next section of this report classified according to each specific aetiological agent. It should be noted from the outset that the aetiology of leg problems is frequently difficult to establish and problems identified by veterinarians are frequently site and / or context specific.

Non-infectious categories are not mutually exclusive. For example genetics may be the primary reason underlying differences in growth rate. However, such a classification provides a basis upon which to order our discussion of aetiological factors.

Genetic selection

Since the 1950's, commercial breeding companies have been selecting broilers for increased growth rate and food conversion efficiency. Havenstein et al. (1994a) compared the performance traits of a 1991 broiler strain with those of a 1957 strain. They found a 420% increase in body weight at 42 days of age, about 85-90% of which was attributable to genetic selection. The remainder was attributed to the modern diet. There was also a 30% improvement in food conversion efficiency as a result of genetic strain and a very considerable increase in mortality for the 1991 strain, especially after 21 days of age, due to leg problems, sudden death syndrome and ascites. After 21d, mortality for the 1957 strain had fallen to almost 0%, while a high level of

mortality in the 1991 strain was maintained throughout the growth period. Walking ability was significantly poorer in the 1991 strain. Comparing strains or breeds within different growth rates several authors have concluded that leg disorders are the result of increased growth rate (Wise and Jennings 1972; Reiland et al 1978).

While several studies have found a strong genetic correlation between increased growth rate and increased valgus-varus deformity (VVD) incidence (Leenstra et al., 1984; Mercer & Hill, 1984), others have failed to find a connection (Dämmrich & Rodenhoff, 1970; Haye & Simons, 1978). The relationship between VVD and growth rate therefore remains uncertain. Twisted leg (rotated tibia) is heritable to some extent but selection to minimise the condition is often hampered by low frequency (Sorensen 1992). Pattison (1992) stated that the incidence of VVD, rotated tibia (twisted leg), spondylolisthesis and tibial dyschondroplasia (TD) has been reduced over the last decade in the UK by a programme of selection against them by breeding companies. However, he went on to say that if the incidence of skeletal disorders is to be reduced to a minimum, then this would entail some compromise of production traits. Julian (1997) pointed out that growth related disease can be reduced or eliminated by decreased feed intake without affecting final body weight. The incidence of tibial dyschondroplasia (Yalcin 2000a, b), rickets (Austic et al., 1977), and spondylolisthesis (Riddell, 1973) can all be increased by genetic selection (reviewed by Sorensen, 1992) while the incidence of TD may be increased by selection for increased growth rate, indicating a strong genetic connection between this condition and growth rate (Sorensen, 1992).

Kestin et al (1992) found that the random breeding of a broiler strain for 11 generations virtually eliminated leg problems as measured by subjective walking gait (discussed further below in section of gait scoring), despite the employment of a typical intensive-type photoperiod, diet and stocking density, which have been found to exacerbate leg problems in commercial strains. Barbour et al (1996) investigated the performance of two breeds of meat poultry breeders and found the mean percentage lameness at 35 - 45 days of age was significantly higher in one of the breeds studied. Hocking et al (1996) found lameness and musculoskeletal disease were more frequent in two particular strains of broilers. In addition, selection in the broiler parent male-strain had clearly increased the prevalence of disorders. Kestin et al (1999) found there were large differences in some important traits associated with leg weakness among four crosses of commercial broiler lines. Three crosses had similar prevalence of TD while one cross had a much lower incidence. There were large differences in walking ability and prevalence of foot pad, hock burn and angulation of the hock joint amongst crosses. Bizeray et al (2000) investigated incidence of leg-disorders in fast growing and slow-growing strains of broilers. Their study principally investigated locomotor behaviour (activity). They concluded that genetic factors were probably involved in the expression of locomotor behaviour in very young chicks and that selection of young mobile broiler chicks might increase activity at a later age and therefore reduce the occurrence of leg abnormalities. Kestin et al (2001) selected thirteen genotypes of poultry and concluded that genetic selection for modern fast growing birds has resulted in increased leg disorders. Reiter and Kutritz (2001) compared behaviour, angulation of the proximal tibiae and walking ability of three fast growing breeds and one with a low growth rate. The duration of locomotor activity, standing, scratching and walking ability was higher and duration of lying and angulations of proximal tibiae was lower in broilers with a low growth rate.

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 TD.

Growth rate

Recently, Kestin et al (2001) investigated the relationship between lameness, liveweight, growth rate and age. Thirteen genotypes of poultry were selected to represent a wide range of growth rates and were fed either a non-limiting or label Rouge diet. The results indicated that liveweight and growth rate were important determinants of lameness.

Rate of growth has long been shown to affect the incidence of leg disorders (Julian 1997; Sue et al 1999). Male broilers, which grow faster than females, have been reported to show about twice the incidence of skeletal disorders (Riddell & Springer, 1985; Classen & Riddell, 1989), as well as a poorer walking gait (Kestin et al. 1994). Correlative evidence of a connection between growth rate and leg disorders may be derived from studies involving alternative feeding (feed restriction) and lighting programmes (decreasing photoperiod) respectively. Each of these approaches will be reviewed in turn.

(1) 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 (Duff & Thorp, 1985b; Classen & Riddell, 1990; Robinson et al 1992). A restriction of feed sufficient to produce a large decrease in growth rate has been shown to virtually abolish skeletal disorders (Riddell, 1983). The particular importance of restricting growth early in life may relate to the very high rate of growth that occurs during the first few weeks of life (Marks, 1979), and the lower bone ash content early in life (Wise, 1970b). Murthy et al. (1986), working with rats, have suggested that feed restriction may work by reducing the growth rate of muscle tissues, thereby increasing the relative growth rate of the skeleton. This hypothesis is supported by the finding of Pinchasov et al. (1985), that feed restriction increased body ash content. In other words, feed restriction counteracts the deleterious effects of a high meat: bone ratio, that broilers have been continually selected for by breeding companies (Pattison, 1992). Riddell (1983) noted that feed restriction throughout the growing period would be commercially impractical because of the delay in reaching market-weight. Programmes of short-term, early feed restriction have therefore been investigated.

Yu & Robinson (1992) reviewed the effect of short-term early feed restriction on production traits. While several studies found no difference in body weight between early restricted and full-fed broilers at 8 weeks of age (Plavnick & Hurwitz, 1985, 1990; Plavnik et al., 1986; McMurtry et al., 1988), eleven other studies cited in this review found that broilers failed to completely catch up in body weight by market age. However, there were usually improvements seen in food conversion efficiency and leanness. Given also the reduction in mortality and culling which result from a reduction in leg disorders, they concluded that short-term feed restriction was a very promising technique.

Robinson et al (1992) restricted feed availability to maintenance levels during the second, third or fourth weeks of life in broilers and roasters. In all cases, the incidence of skeletal disorders was reduced. Restriction during the second week was the most effective. Disorders were reduced to one third of the incidence seen in full-fed controls. Market weight was delayed by only 2 to 3 days due to subsequent accelerated growth following the period of feed restriction.

Su et al (1999) conducted two trials to investigate whether manipulation of feeding pattern or early feed intake affected prevalence of leg weakness. Fewer meals per day was associated with less TD, less hock burn, better walking ability, lower body weight and better feed conversion. The reason for the decrease in TD (even when body weight was controlled for) was unclear. Su et al (1999) suggested it could reflect the more organised behavioural patterns induced by meal feeding, resulting in more overall activity, or it could reflect changes in the pattern of circulating hormones such as insulin and growth hormone, which may result from the pulsate feed intake similar to those reported by Buyse et al (1997) for feed restricted broilers. Su et al (1999) also suggested that it is probable that the difference in food conversion between treatments was also reflected in differences in body composition with meal restricted birds being leaner (relative to more meals or being fed ad lib) as reported by Jones and Farrell (1992) and Cristofori (1997).

Earlier feed restriction, longer duration and a more severe level of restriction were all associated with lower prevalence of TD, better walking ability, lower body weight and better relative growth rates and feed efficiency (Su et al 1999). However, adjusting the observations for differences in body weight removed many of the significant differences; only birds that started feed

restriction earlier in life had less TD. Other feed restriction programmes which have also reduced growth rate have also consistently reduced the incidence of TD (Riddell, 1975b; Poulos et al., 1978; Edwards and Sorensen 1987; Lilburn et al., 1989; Su et al 1999) and VVD (Haye & Simons, 1978; Robinson et al., 1992) although there have been some contradictory findings (e. g. Poulos et al., 1978). Su et al (1999) concluded that meal feeding can beneficially affect the prevalence of leg weakness and that the major part of this effect is independent of changes in body weight. It was also concluded that early feed restriction decreased many aspects of leg weakness but that effects were mainly a result of a decrease in body weight. Both meal feeding and early feed restriction improved feed efficiency. Their trials indicated that leg weakness can be reduced by meal feeding or early feed restriction and because these manipulations can result in improved efficiency, they may be a feasible method for limiting leg weakness.

While short-term feed restriction may be a promising method for the reduction of leg disorders, a reduction in growth rate may not be the only mechanism at work. It has been suggested that fasting *per se* may be an important factor. Edwards & Sorensen (1987) fasted broilers for 8 hours a day, with the provision of *ad lib.* feed during the remainder of the day, during days 4 to 20 post hatching. The incidence of TD was consistently much lower than with normal feeding. A fasting period of at least 8 hours was needed to produce the maximal reduction of TD incidence (although fasting did not have to occur every day). They suggested that fasting may have a similar mechanism to reducing the photoperiod (discussed later), and that this mechanism may be the provision of intermittent periods of reduced growth during which the mobilisation of adequate nutrients to the faster growing growth plates could occur.

Qualitative feed restriction has also been used to reduce the incidence of leg disorders i.e. reducing the metabolizable energy density of the feed (e.g. Haye & Simons, 1978; Hulan & Proudfoot, 1987; Robinson et al., 1992). Several studies have found that broilers fed a low energy diet were able to catch up with full-fed controls before market-age (Griffiths et al., 1977; Leeson, 1990). While the effectiveness of reducing protein levels during the first 3 or 4 weeks of life remains unclear (e.g. Hulan et al., 1980; Cook et al., 1984), Plavnick & 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. Bokkers and Koene (2001) recently found that type of feed affected walking speed (activity) which is known to affect incidence of leg disorders (see exercise below).

Early feed restriction appears to be a promising management procedure which allows a decrease in leg disorders without substantial economic losses to the industry due to improved efficiency. However, there are welfare implications of feed restriction since hungry birds may suffer poor welfare.

(2) Lighting regimes

Continuous or near-continuous lighting is the norm for commercial broiler production, in order to maximise food intake and hence growth rate (Classen, 1992). It has been suggested that changing the photoperiod : scotoperiod from the commonly used 23 h light : 1 h dark to a more natural light : dark pattern may limit expression of skeletal deformities (FAWC 1992; Sorensen et al 1999). In particular, twisted leg (rotated tibia) will decrease (Buyse et al 1996), a topic reviewed by Hester (1994). Several alternative lighting regimes have therefore been designed in an attempt to reduce leg disorders without impairing growth rate.

There is some disagreement concerning the extent to which photoperiod affects growth rate and overall performance. Wilson et al. (1984) found that broiler productivity was superior under a restricted lighting regime compared with that occurring under continual light. Buyse et al (1996) indicated that final body weights of broilers reared under intermittent lighting schedules at market age were equal to or even higher than those of broilers reared under continuous lighting schedules. Renden et al (1996) found intermittent lighting decreased incidences of leg disorders while providing equivalent yield when compared with continual lighting. However, Sorensen et al (1999) recently found shorter photoperiods were associated with lower body weights.

Despite variation in the relative duration of light and dark phases between studies, intermittent lighting programmes have invariably reduced the incidence of leg disorders in broilers (e.g. Buckland et al., 1973, 1974b; Buckland, 1975; Classen and Riddell 1989; Simons, 1982, 1986; Wilson et al., 1984; Simons & Haye, 1985; Ketelaars et al., 1986; Renden et al., 1991, 1996), with some exceptions (e.g. Buckland et al., 1974b). Reviewing the literature Gordon (1994) noted many authors felt that increased activity levels contributed to a decrease in leg weakness (in birds reared under moderate photoperiods).

The effect of intermittent lighting programmes on early growth rate is inconsistent between studies, so other factors relating to lighting are implicated (Classen, 1992). Buyse et al (1996) outlined several hypotheses which seek to identify the mechanisms underlying reductions in leg disorders, particularly twisted leg. Firstly, it was suggested that the increased physical activity of birds under intermittent lighting may favour bone strength and development (Buyse et al 1996; Wilson et al., 1984; Ketelaars et al., 1986). Simons (1982) found that activity levels were higher in broilers reared with intermittent lighting than with continuous lighting while Newberry et al. (1988) found that high light intensity resulted in a reduction in the incidence of skeletal disorders. Prayitno et al (1997) found that activity levels were increased by high light intensity resulting in a reduction in leg disorders. In a more detailed analysis of activity levels, however, using radar equipment, Simons & Haye (1985) found that broilers were more active during the light periods, but less active over the entire day, compared with continuous lighting. Therefore, spurts of activity (and periods of rest) may have been beneficial in reducing leg disorders. Classen (1992) also suggested that circadian rhythms may play a more important role. A second hypothesis suggested that altering the growth curve by changing the lighting schedule induces a temporary reduction in muscle deposition without affecting skeletal development (Buyse et al 1996). In this case the skeleton is developed proportionally more before it is loaded with muscle tissues. Finally, a third hypothesis suggested the involvement of androgens in bone development may be implicated and affected by intermittent lighting (Buyse et al 1996).

Recent work by Sorensen et al (1999) expected to find, in a review of the literature, a beneficial effect on lameness with reduced photoperiod and that this would be independent of changes in body weight. However when data were corrected for differences in body weight they found the birds on shorter photoperiods had poorer walking ability (as measured by gait scores). This finding was consistent across four separate trials. The prevalence of TD decreased with decreasing photoperiod, independent of body weight. However, longer photoperiods were consistently associated with reductions in other indices of skeletal abnormalities, a result which is contrary to findings from others (Classen and Riddell 1989). This important study appears to indicate that moderate photoperiods, applied to broilers in the first three weeks, have little effect on the overall prevalence of leg weakness.

Step-up lighting programmes operate on the same principle as early feed restriction. Chicks are kept on a 23L : 1D schedule for the first 2 days of life, in order that they learn to find feed and water. Then a 6 hour photoperiod is employed until 21 days of age, to reduce feed intake and growth rate. This is then stepped up, abruptly or gradually, to a 23 hour photoperiod until slaughter. The increasing day length is thought to stimulate increased anabolic steroid production, causing an acceleration in growth rate (Classen, 1992). Classen & Riddell (1989), Classen et al. (1991) and Riddell & Classen (1992) found the step-up programme reduced the incidence of skeletal disorders in broilers by up to 50%, with no difference in body weight at 42 days compared with continuous lighting. The principle type of skeletal deformity in birds kept on a step up lighting programme was VVD, and this condition was the most positively influenced by the lighting programme. The incidence of rotated tibia and spondylolisthesis were also reduced, although there was no statistical analysis of these reductions. However, Riddell & Classen (1992) found no effect upon the incidence of TD. Newcombe, in a personal communication to Classen (1992), reported a significant reduction in TD incidence at 29 days of age, but this reduction was no longer apparent by 43 days. Renden et al. (1991) used a similar step-up programme with broilers, except that the 6 hour photoperiod was only applied during the first 2 weeks of life. There was only a tendency for the

step-up programme to reduce leg disorders at 42d and 57d, compared with continuous lighting. If the 6 hour photoperiod had been extended to 3 weeks of age, then this difference may have been significant (Hester, 1994).

Classen & Riddell (1989) observed that the activity level of broilers was increased in the step-up lighting programme. This could have been responsible for a reduction in leg disorders but early growth rate was considered a more likely mechanism (as also suggested by Buyse et al 1996). Classen & Riddell (1990) went on to establish that a reduction in early growth rate was not the only factor at work. They matched the growth rate of birds on a constant photoperiod of 24L : 0D to that of birds on a step-up lighting programme (by early feed restriction of the former group). The incidence of skeletal disorders in the group on constant lighting was reduced by the feed restriction, which the authors attributed to the effect of reduced growth rate. However, broilers on the step-up programme had a lower incidence of skeletal disorders than the birds on the constant lighting programme, despite the fact that their growth rates were equal. This suggested that other factors relating to lighting, such as circadian rhythms, must have a role in reducing skeletal disorders. A drawback with the use of the step-up lighting programme is that it has been shown to render broilers significantly more fearful and difficult to handle prior to slaughter than a constant photoperiod (Newberry & Blair, 1993b). Manser (1996) suggested that this may be a consequence of low levels of stimulation during the first weeks of life, being kept in darkness for 18 hours a day.

Conclusion

A reduction of growth rate by means of feed restriction is capable of reducing the incidence of non-infectious skeletal disorders in broilers probably without affecting body weight. Recent research has suggested that decreasing photoperiod early in life has little beneficial effect but previous research has suggested some beneficial effects. These manipulations can result in improved efficiency and may therefore be feasible methods for limiting leg weakness. There may be welfare implications associated with meal restrictions as the birds will be hungry (discussed later).

Food Conversion Efficiency and Body Conformation

As mentioned previously, Havenstein et al. (1994a) compared the performance traits of broiler-strains raised in 1991 with a strain raised in 1957. Genetic selection was found to be responsible for a 30% improvement in FCE. There was an additional 20% improvement attributed to the modern pelleted diet, although this was considered to be almost entirely due to a higher level of wastage of the 1957 mash diet. Havenstein et al. (1994b) assessed the changes that had occurred in carcass conformation as a result of improved food conversion efficiency. Breast meat yield, as a percentage of live body weight, was 3% greater for the 1991 strain, with an additional increase of 1.2% owing to the modern diet. The weight of breast muscle has therefore increased disproportionately to body weight. Unfortunately, the study did not examine the proportional development of the skeleton.

There have been few studies conducted to assess the changes in production traits and body conformation which have been brought about in the broiler industry (Havenstein et al. 1994a). Wise (1970a), in an attempt to establish how far skeletal disorders could be attributed to skeletal conformation and load-bearing, comparisons of the weight of body parts and the proportional growth of the leg bones were made between broiler and layer strains at various ages. When birds of two strains were compared at equal body weights, broilers had a slightly greater proportional trunk weight, and slightly smaller proportional leg, head and neck, and wing weights, but there was no difference in the proportional breast weight. There were also no differences in the ratio of leg bone lengths or other body dimensions to tibiotarsal bone length. It was concluded that the small differences could not account for the prevalence of observed skeletal disorders. In a subsequent study, Wise (1970b) found that at equal body weights, the skeleton of broilers was less mature than

that of layers, with poorer mineralization of the bones. This difference was particularly striking during the first weeks of life when the rate of skeletal growth and maturation was the highest. Thus a difference in bone strength during early life, as opposed to a difference in bone quantity, was implicated.

Since the 1970's growth rate and the proportional growth of breast muscle have increased greatly. Lilburn (1994) compared the findings of studies in 1970 (Deaton & Reece, 1970; Halvorson & Jacobson, 1970) which measured broiler performance traits with the findings of a more recent similar study (Acar et al., 1993). The growth rate was seen to have nearly doubled, while the growth rate of the *Pectoralis major* muscle had increased by a considerably greater extent. Havenstein et al. (1994a) also found that growth rate had nearly doubled when compared with findings with 1970's broiler strains (Sherwood, 1977; Chambers et al., 1981; Marks, 1979). In contrast to the findings of Wise (1970a), 1991 broiler strains had highly significant increases in breast and drumstick yield, and decreases in wing yield, compared with the less selected 1957 strain (Havenstein et al., 1994b). Conformation and load-bearing, in association with a rapid growth rate, have been suggested as factors involved in chondrodystrophy (Creek et al., 1960; Riddell, 1975c), dyschondroplasia (Wise & Jennings, 1972), rotated tibia (Thorp, 1988a), and VVD (Randall & Mills, 1981; Riddell et al., 1983).

Recently Williams et al (2000) investigated how the bone of meat chickens developed under typical commercial conditions and compared development with that in a genetic precursor flock. A modern fast growing strain and a slower-growing control strain were compared. A tibiotarsus from each bird was x-rayed and its dimensions and estimated resistance to bending were determined. Finally, cortical bone samples were ashed to measure total mineral, calcium and phosphorous content. The selected strain grew faster but both strains demonstrated similar periods of rapid bone formation (days 4 to 18) and mineralisation (days 4 to 11) and achieved similar estimates of resistance to bending. Cortical bone of the selected strain was less well mineralised and more porous than that of the control strain and showed a significant increase in molar Ca:P ratios above the expected range of values during the first 2 - 3 weeks of life. Williams et al (2000) concluded that despite production of bones with the correct dimensions for load support, the relatively poor density and mineral content of bone in the selected strain is likely to reduce effective breaking strength of the tibiotarsus. This could be due to rapid growth, genetics or an inadequate supply of Ca and P.

An even more recent study by Sandercock et al (2001) investigated muscle damage in meat chickens in relation to growth rate. They found that alterations in muscle membrane integrity in high growth-rate broilers could not be fully explained by live weight alone and that there was a greater degree of muscle damage (as measured by creatine kinase) in fast growing birds. This recent important study concluded that genetic selection for improved growth rate and muscle yield in modern broilers has detrimental affects upon muscle function and membrane integrity which may be attributable to genetically induced alterations in muscle fibre status.

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 very rapidly. The magnitude of biomechanical forces alone may not, however, be sufficient to produce skeletal disorders. Cook et al. (1984) were unable to increase the incidence of skeletal disorders by loading supplemental weight onto the bodies of broilers. Theories of the role of biomechanical forces in the development of dyschondroplasia, rotated tibia and VVD require that there be a pre-existing joint instability or angulation that causes pressure to be concentrated at a particular site of the bone. Minor defects of this sort are often seen in young animals, and in the absence of excessive biomechanical pressures, often rectify themselves as the animals mature (Fretz, 1980). If the theory that excessive biomechanical forces are exerted normally upon broiler bones is correct, then the incidence of disorders would be likely to depend on the incidence of minor congenital defects, and would not be

expected to increase when supplemental weight is loaded on to the animal (although the severity of the disorders might).

The mechanisms which have been proposed for biomechanical forces as factors in skeletal disorders will be considered in more detail when disorders are discussed individually, below. The issue of body conformation is also relevant to a discussion of subjective gait scoring which again is addressed later in the review.

Exercise (activity)

Increased exercise (activity) is believed to prevent leg problems in chickens, a topic most recently addressed by Leterrier et al (2001). Exercise on a treadmill increases bone density at several sites in the tibia (Rutten 2000) and improves vascular patterns of the proximal growth plate (Thorp and Duff 1988). A number of factors can affect activity. These include the genetic origin of the birds, manipulation of the environment, manipulation of different feeding strategies and lighting regimes.

Broilers housed in cages (which do not have the metabolic calcium deficiency of layers) whose exercise is restricted show a high incidence of skeletal disorders when compared with group-housed birds with a litter floor (Reece et al., 1971; Riddell, 1976, 1983; Rizk et al., 1980; Vitorovic et al 1995). The humerus and tibiotarsus are weakened by caging (Wabeck & Littlefield, 1972; Andrews & Goodwin, 1973; Merkle & Wabeck, 1975). In the humerus, bone strength appears to be closely related to cage height suggesting that exercise is an important factor (Travis et al., 1983). Lack of exercise may also cause weakness in the tibiotarsi (Classen, 1992). However, VVD is the major cause of leg problems in caged broilers (Haye & Simons, 1978; Riddell, 1983). Congenital abnormalities in appositional bone growth (Dämmrich & Rodenhoff, 1970) have been suggested as a factor predisposing broilers to VVD and these abnormalities have been reduced by increasing exercise (Rodenhoff & Dämmrich, 1973). It has been suggested by some authors that the reduced incidence of VVD resulting from feed restriction and shortened photoperiods in floor raised broilers may be due to increased activity levels (Riddell, 1983; Classen & Riddell, 1989; Riddell, 1992). Care must be taken here to distinguish between the effect of exercise upon VVD incidence and upon the incidence of skeletal disorders generally. Increased exercise does not appear to reduce the incidence of TD. Caged broilers actually have a lower incidence of TD than floor raised broilers (Veltmann & Jensen, 1980; Riddell et al 1983; Simons, 1986).

Reiter and Bessei (1998b) observed the locomotor activity of broilers of different growth rates in deep litter compartments. Fast growing strains showed lower activity levels while the slow growing strain maintained higher activity levels. Systematic training of fast growing broilers improved bone density and reduced bending and twisting of the leg bones. They concluded that increasing the distance between feeders and drinkers may be a method to increase locomotor activity under practical conditions and may therefore contribute to a reduction in leg disorders. Reiter and Bessei (2001) investigated the effect of reduced weight load on locomotor activity and leg disorders. This study is pertinent to sections on body conformation as well as activity. In their study they investigated whether a reduction in load bearing on the legs would reduce leg disorders by modifying activity. A special device was developed to alleviate 25% of the weight load of the legs. This suspension device consisted of a harness which suspended the birds alleviating 25% of the weight on the legs while allowing their continued mobility. Comparisons were made with birds which had no harness (and birds which had a harness but were not suspended). At 5 weeks of age birds were slaughtered. The mean daily distance travelled was higher in the suspended birds and torsional deformity was lower. They concluded that locomotor activity, bone building processes and leg deformities were positively affected by the weight load reduction (possibly due to a reduction in pain when the birds walked).

Weeks et al (2000) investigated the behaviour of six replicates of broilers obtained from commercial farms and made comparisons of behaviour between sound birds and lame birds. Significantly more lying was performed in lame birds (86 % vs. 76 %). Sound birds spent

significantly more time standing idle, standing preening and standing eating. Weeks et al (2000) concluded that lameness altered feeding activity and impaired welfare.

Recently, Mench et al (2001) investigated behavioural activity and its effects on leg problems. They housed birds in an enriched environment to stimulate activity and found that, while birds used the environment in a complex way, increased activity had the potential to improve welfare with little adverse effects on productivity. This enrichment included a tube with meal worms as manipulation of diet is known to affect activity. Diet composition can mainly be used to reduce growth rate (as outlined previously) but manipulation diet can also increase activity. For example, presentation of the diet (particle size, shape, hardness) seems to have significant consequences on behaviour (Picard et al 1999). Meal feeding can also have an effect (Su et al 1999) and sequential feeding has been shown to affect activity (Noirot et al 1998). Sequential feeding with a low-lysine diet increased activity without a reduction in body weight (Bizeray et al 2001). All these methods potentially allow for an increase in activity and lowering of leg problems.

Newberry et al. (1985) exposed broilers to periods of relatively high light intensity (around 12 lux), interspersed with low intensity levels (0.5 lux). Although there was increased activity during the periods of high light intensity, there was no effect on leg disorders. Newberry et al. (1986) found no effect of intensities varying from 0.1 to 100 lux on either activity or leg disorders. Newberry et al. (1988) found that broilers kept at 180 lux were more active than those at 6 lux, but there was no consistent effect on the incidence of leg disorders. Furthermore the housing system usually used for broilers does not seem enriched enough to stimulate a high level of exploratory activity (Newberry 1999) although perches have been shown to have no beneficial effect in reducing leg weakness (Sue and Kestin 2000).

Hulan & Proudfoot (1987) studied the effect of light quality on broilers. Pink fluorescent lighting reduced the incidence of skeletal disorders and VVD, although it increased the incidence of TD, when compared with incandescent lighting. Manser (1996) suggested that activity levels may have been higher with the pink fluorescent lighting, owing to the fact that chickens have better visual acuity in red light (Prayitno et al., 1993b), so that the intensity of this light source would have appeared greater to the birds. Classen (1992), on the other hand, suggested that differences in vitamin D metabolism may have been involved, as fluorescent light contains a higher level of ultraviolet radiation.

As mentioned in the section on lighting, Prayitno et al (1997) conducted two experiments to investigate whether manipulating the wavelength and intensity of light could be used to increase activity and reduce disorders. The first experiment examined the effects of wavelength and intensity of light on bird activity. Birds were reared on red and blue light at three intensities. In a second experiment, the effect on locomotion and leg disorders of stimulating bird activity with bright red light in the early or late stage of rearing were examined. Bright red light considerably increased walking, feeding and stretching particularly when applied early in the growth period. It was concluded that rearing broilers in bright red light increases activity, which reduces locomotion disorders in the late rearing period.

In interpreting a number of studies that found no difference in the levels of mortality for fluorescent or incandescent light (Wabeck & Skoglund, 1974; Wathes et al., 1982; Zimmermann, 1988), Classen (1992) argued that light quality is unlikely to be an important factor on leg disorders. Bizeray et al (2000) concluded that manipulation of activity by environmental means during the first week of life may be one way of preventing leg disorders.

To summarise, the evidence for effect of activity level indicates that leg disorders may be reduced by encouraging locomotion and increased activity early in the growing period. However, the exact relationship between activity and leg disorders remains unclear.

Circadian rhythms

Circadian rhythms are regular daily fluctuations in the levels of hormones, and hence in many metabolic processes such as the level of arousal in the sleep-waking cycle. The 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 (Cain & Wilson, 1974). If animals are exposed to a period of darkness, the onset of light has the effect of resetting the circadian rhythms, which would otherwise run on undisturbed under endogenous control. One effect of resetting the rhythms will be an increase in arousal and activity levels, at the onset of the light period. The photoperiod therefore has an important effect on activity levels and many other metabolic processes.

The findings of Classen & Riddell (1990) that the incidence of skeletal disorders was lower in broilers raised on a step-up lighting programme when compared with broilers matched with them for growth rate by the use of feed restriction, has led to some speculation about the role of circadian rhythms in broiler skeletal development. In mammalian species, it is known that bone growth and modelling follows a circadian rhythm (Simmons, 1962, 1968; Hansson et al., 1974), and there is no reason to believe that this is not also true for avian species (Classen, 1992). 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

Havenstein et al (1994a) reported that the typical 1991 broiler diet contained higher levels of protein, energy and fat, and lower levels of calcium and phosphorus than the typical 1957 diet. They found that feeding the 1991 diet increased the growth rate of both the 1957 and 1991 broiler strains by 18-26%. However, the increased growth rate of the 1991 strain on the modern diet was not maintained after 42 days. There was a rapid decrease in gain, especially in the male birds, which was attributed to a high incidence of leg disorders. This indicates that the increased growth rate induced by diet is sustainable up to broiler slaughter weight, but beyond normal slaughter weight, disorders have become so severe that the performance of most birds is severely compromised. There was no significant effect of diet on mortality, although the incidence of TD lesions in the 1991 birds at 42 days was almost doubled. The genetic strain of the birds had a much greater influence on both mortality and TD incidence than diet, indicating that while diet was a contributory factor to leg disorders, it was not the primary one.

Over the course of the last twenty years, reviews have summarised information relating to nutrition and skeletal disorders (Sauveur 1984; Leeson and Summers 1988; Edwards 1992, 2000; Leach and Lilburn 1992; Orth and Cook 1994; Whitehead 1998). The scientific literature contains evidence 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 (Edwards 2000). Vitamin A deficient birds have a staggering gait and exhibit ataxia but chemical analysis of the bones and histological studies clearly indicate poor calcification and bone development (Edwards 1992). Vitamin A toxicity causes an osteodystrophy characterised by abnormal thickening of the growth plate zone, depressed calcification and lameness (Baker et al 1967).

The use of cod liver oil to prevent rickets (caused by deficiency in vitamin D3) goes back to the nineteenth century (Edwards 1992) and since the 1950's vitamin D3 has been extensively used to supply the vitamin D needs of poultry. Despite this, field rickets has continued to occur (Edwards, 1992), and TD has become a major problem. However, a more thorough understanding of the role of nutritional deficiencies in disorders such as rickets and chondrodystrophy has reduced their incidence in modern broiler flocks (Pattison, 1992; Riddell, 1992) and studies indicate that the quantitative requirement for D3 is much greater than previously thought (Edwards 2000). However, despite this knowledge, rickets is commonly seen subclinically (Riddell & Pass, 1987), and outbreaks of field rickets still occur (Thorp, 1992).

In turkey poults, field rickets has been attributed to poor biopotency of D3 supplements (Yang et al., 1973), and to inadequate synthesis of 1,25(OH)2D3 in early life (Bar et al., 1987). Work on TD in broilers (Edwards, 1989, 1990) has established that when low calcium diets, which already contain many times the National Research Council (1984) requirement for D3, are

supplemented with 1,25(OH)₂D₃, the incidence of TD is dramatically reduced. The authors inferred that the rapidly growing broiler is unable to convert enough D₃ to 1,25(OH)₂D₃ to absorb calcium efficiently from a low calcium diet. In other words, the broiler's D₃ metabolism is incapable of meeting the calcium needs of the rapidly growing skeleton, at low levels of dietary calcium that may occur commercially. This problem may be resolved by 1,25(OH)₂D₃ supplementation of the diet (Rennie et al., 1994), or by genetic selection for an improved D₃ metabolism (Edwards, 1992). There is abundant evidence that the incidence of TD could be greatly reduced by dietary manipulation, especially by increasing dietary calcium levels, or by supplementation with 1,25(OH)₂D₃ (Edwards, 1992; Rennie et al., 1994; Edwards 2000). This issue will be discussed further in the section on TD.

In the modern broiler industry, the diet often contains 3 to 10 times the National Research Council (1984) requirement of D₃ (Edwards, 1992). These levels have actually been shown to cause increases in the incidence of VVD (Cruickshank & Sim, 1987) and in TD (Lofton & Soares, 1986).

Vitamin E affects bone calcification when fed in excess to either calcium deficient or vitamin D deficient birds (Murphy et al 1981). Nicotinic, folic acid and biotin deficiency may also cause severe problems such as perosis and death although this is not always the case (Edwards 1992). For example, Roland and Edwards (1971) produced a deficiency with a casein-gelatin purified diet and noted only dermal lesions mainly on the feet and changes in tissue fatty acid patterns. Pyridoxine and choline deficiency can cause problems and one report (Ferguson et al 1978) indicated that choline deficiency in broilers may cause tibial dyschondroplasia.

Feeding fast-growing broilers on experimental diets low in calcium or high in phosphorus produces a high incidence of both rickets and TD (Long et al., 1984; Riddell & Pass, 1987). In order to improve the efficiency of dietary fat utilisation, poultry feed has been formulated to contain relatively low levels of calcium. This has rendered broilers susceptible to the development of skeletal disorders, because there are many dietary and environmental variables that can impair the absorption or utilisation of the limited dietary calcium which is available (Edwards, 1992). In recent years TD has returned due to dietary factors having been previously controlled by genetic manipulation. Calcium metabolism is known to be affected by dietary levels of phosphorus (Edwards & Veltmann, 1981, 1983); fat (Edwards et al., 1960); sodium, potassium and chloride (Hulan et al., 1986, 1987a, b; Simons et al., 1987). Calcium absorption efficiency is affected by dietary D₃ and 1,25(OH)₂D₃ (Rennie et al., 1994), as well as by viral infection of the intestine, such as infectious stunting syndrome (Lilburn et al., 1982; Nelson et al., 1982).

Dietary protein levels are known to have no direct affect on the incidence of TD in broilers (Edwards, 1984b; Edwards et al., 1990). However, a review of early work done on the causes of perosis (chondrodystrophy) has indicated that high protein levels in conjunction with certain vitamin deficiencies produce a high incidence of TD (Edwards, 1992). Any apparent discrepancy in the literature where some investigators report perosis as a characteristic of a particular vitamin deficiency while others observe no perosis in birds deficient in the same vitamin may be explained by differences in dietary protein levels (Edwards 1992).

Stocking density

There is considerable evidence that high stocking density can increase the incidence and severity of leg disorders. In comparisons with birds housed at lower densities, individual birds that were provided only 500 cm² of walking area had reduced body weight and an increased frequency of breast blisters and ammonia burns of foot pad hocks (Proudfoot et al 1979; Thomsen 1992; Martrenchar et al 1997). It is now recognised that high stocking densities deleteriously affect bird growth and quality (Sorensen et al 2000).

Hall (2001) investigated the effect of stocking density on the incidence of leg problems. At higher stocking densities the incidence of leg problems (as measured by the number of leg-related culls), contact dermatitis and carcass bruising increased. This is in accordance with Grashorn and

Kutritz (1991) who found broilers suffered increased leg problems when kept to 50 kgm² (23.1 % of birds affected) as compared to 38 kgm² (4.3 % of birds affected). However, Reiter and Bessei (1999) concluded that stocking density (within the ranges they tested) had only a little influence on the performance and behaviour (but leg disorders were not measured).

Sorensen et al (2000) investigated the effects of stocking density on leg weakness in two trials. In trial 1, walking ability was assessed at three ages stocked at three different densities. In trial 2, birds were stocked at two densities and assessed for TD by radiographic examination and walking ability. The effect of stocking densities was consistent across both trials; higher stocking density were associated with poorer walking ability and reduced live weights. There was no effect of stocking density on prevalence of TD or angulation of the hock joint. The effect of high stocking densities on walking ability was apparent and, adjusting for differences in body weight, did not alter the findings. Sorensen et al (2000) concluded that lower stocking densities substantially reduced the prevalence of leg weakness.

Kestin et al. (1994) found that broiler flocks reared at 22.7 birds/m² had poorer gait scores at 7 weeks of age than flocks reared at 15.9 birds/m², which in turn had poorer scores than flocks reared at 12.2 birds/m². This may have been due to differences in activity level, although this was not measured. Reiter and Bessei (1995) reported that broilers given more exercise have less leg weakness. In these studies birds were exercised on a treadmill; those given more exercise had better walking ability. Thus it might be expected that a relationship would exist between stocking density, activity and leg weakness; birds at lower densities would move around more and thus limit the expression of leg disorders (Sorensen et al 2000).

Stocking density is unlikely to be a factor in disorders that primarily occur early in the broiler's life, because density is relatively low at this time (Pattison, 1992). Hence the reason why it could effect the prevalence of VVD, but is unlikely to be important in TD (Lilburn, 1994).

Apart from any effect upon the prevalence of leg disorders, Pattison (1992) has stated that stocking density is the main husbandry factor to increase the level of mortality resulting from leg disorders. It is clear that reduced stocking density may be a valuable method to limit leg weakness.

Overall conclusions

It is difficult to identify the underlying aetiology of non-infectious leg disorders and problems are frequently site or context specific. However, body conformation is important and the use of genotypes with slower growth rates is a long-term measure which should result in reduced incidence of leg disorders. Birds experimentally manipulated to reduce the weight of the body on the legs show greater levels of activity and lower incidences of leg disorders. Short-term husbandry techniques which reduce leg disorders by manipulating growth include meal feeding (Sue et al 1999), diet manipulation (Bizeray et al 2001), early feed restriction (Su et al 1999) and reduced stocking density (Sorensen et al 2000). Manipulation of photoperiod has recently been shown to be less effective than these techniques by Sorensen et al (1999) who found that moderate photoperiods applied up to three weeks of age had little overall effect on prevalence of leg weakness. The encouragement of locomotory activity may result in fewer leg-disorders (Bizaray et al 2000) and may be achieved by a number of techniques including environmental enrichment (Mench et al 2001), diet manipulation (Bizeray et al 2001) and lighting regimes but later in life increased activity may result in poor welfare (as existing disorders may be painful). Circadian rhythms mediated via photoperiod may also play some role in bone development and therefore skeletal disorders. Various nutrients play an essential role in the development of leg disorders such as TD, which can occur in response to deficiencies. Such conditions may be alleviated by supplementary feeding.

Specific leg-disorders in broilers: pathology and aetiology

The simple listing of the conditions which result in leg disorders is difficult because classification of conditions is often arbitrary (Thorp 1994) and authors disagree on a suitable

system (Bradshaw et al 2001). Summarised, they can be divided into two categories: those of infectious origin, which are largely responsible for very severe lameness and those that are caused by skeletal abnormality and result in less severe lameness (Lynch et al 1992; Kestin et al 1994). The relative importance of the two broad categories is not well established, but non-infectious causes are thought to be most likely responsible for the bulk of lameness seen (Kestin et al 1994).

The aetiology of many syndromes is poorly understood (Riddell 1992) and can be difficult to diagnose (McNamee et al 2000); this is particularly the reason why there is such little consistency between authors. This confusion sometimes makes comparisons between studies difficult. For example, some authors consider the terms femoral head necrosis or twisted legs to be misleading and inappropriate (e.g. Riddell 1992; Thorp 1992) while others continue to use them (Sorensen 1992; Pattison 1992). For a second example, some authors have sought to employ the term bacterial chondronecrosis with osteomyelitis to better describe femoral head necrosis (e.g. McNamee and Smyth 2000) but such a term can only be used when there is an infectious aetiology; others such as Thorp et al (1993) have adopted the term proximal femoral degeneration to cover infectious and non-infectious aetiology. Opinion differs as to the best term to use, but BCO as proposed by McNamee and Smyth (2000) seems the best descriptive term to describe the modern-day condition. However, FHN will continue to be used by many in the field.

In an attempt to clarify the situation, a number of researchers have classified disorders into distinct categories. For example, Morris (1993) listed the five primary causes of leg disorders by the factors which were thought to cause the pathology: nutritional disorders, infectious disorders, metabolic conditions, conformational problems and toxins. One of the problems with approach is that some conditions have a multifactorial aetiology and may fall into several categories. Riddell (1992) classified conditions into developmental, metabolic and degenerative disorders. Developmental disorders are abnormalities in cartilage or bone growth and have a genetic basis. Metabolic disorders consist of a failure in cartilage or bone synthesis, and have a nutritional basis. Degenerative disorders are characterised by tissue necrosis (cell death) and/or damage. Recently the EC (2000) classified disorders into three distinct categories: infectious, developmental and degenerative (the latter being of non-infectious aetiology). We adopt this EC (2000) system of classification as the basis upon which to base our review of specific pathology and aetiology.

It is important to realise from the outset that these disorders do not exclusively usually fall into these neat categories. Outbreaks of leg disorders can be site / context specific and it is important to stress the interactive nature of many disorders and the way in which they vary in their degree of severity from bird to bird. For example, mild rickets early in life may be viewed as TD later in life which, in turn, may lead to severe TD and mild or severe VVD. What is the aetiology of the VVD? Is it the rickets, the TD or the initial angulation from the growth plate caused (or perhaps not caused) by one of the lesions present (there may also be infection present to complicate matters further)? In addition, whether the condition matters in terms of welfare is a separate question (discussed further below). It is important to understand this complexity from the outset; specific disorders are discussed separately here only for reasons of clarity.

Infectious disorders

Bacterial Chondronecrosis with Osteomyelitis or Femoral Head Necrosis

The term femoral head necrosis (FHN) has frequently been used to describe a variety of gross lesions of the proximal femur. The conditions that this term includes have been listed by Reece (1992) in a review of infectious agents in leg abnormalities. Apart from epiphyseolysis (separation of the epiphysis from the bone, reviewed below in the section on degenerative disorders), there may be fracture of the femoral neck or disintegration of the femoral head, resulting from osteomyelitis, brittle bone disease or infectious stunting syndrome. Recent work by Thorp et al (1993), Thorp and Waddington (1997), McNamee (1998) and McNamee and Smyth (2000) suggest that bacterial infections of growing bone are the commonest cause of lameness in growing

fowl and that these conditions were previously reported in the proximal femur as FHN. They employ the term bacterial chondronecrosis with osteomyelitis (BCO) to describe these conditions, emphasising an infectious aetiology.

According to McNamee and Smyth (2000) BCO has been reported under a variety of names including osteomyelitis (a bacterial infection of the metaphysis), femoral head necrosis, long bone necrosis, proximal femoral degeneration, bacterial chondritis with osteomyelitis and bacterial chondronecrosis. This position is generally supported by Butterworth (1999) who uses a similar term implying an infectious aetiology: bacterial chondronecrosis (BCN). Butterworth (1999) stresses the term FHN fails to indicate that infectious bone necrosis is commonly seen in the tibia and that FHN may be an avascular and non-infectious necrosis of bone (Cisson's disease) whereas BCN and BCO have a bacterial causation (Butterworth 1999). The particular vulnerability of the femoral head to osteomyelitis may arise from a sluggish circulation in metaphyseal vessels contributing to localisation of bacteria. Pre-existing pathologies including disruption of the physal vasculature and small cracks and clefts commonly seen in broiler femoral cartilage (Thorp 1988) may then act as foci for bacterial invasion (EC 2000). Defects in cartilage mineralisation such as those arising from rickets and TD (see below) may further enhance the problem (EC 2000).

Thorp et al (1993) reported that FHN was not considered appropriate to describe observed pathologies and the term proximal femoral degeneration (PFD) was adopted. However, PFD does not take into account that lesions may occur in any bone (and cause leg disorders) and again include changes unrelated to bacterial infection (Duff and Randall 1987; Thorp 1994). Reece (1992) distinguished the terms bacterial osteomyelitis from FHN but pointed out the latter was poorly defined and inappropriately used for several conditions. In a review of non-infectious skeletal disorders, Riddell (1992) used the term epiphyseolysis of the femoral head and emphasised the confusion concerning the terminology applied to lesions of the femoral head in poultry.

The indiscriminate use of the term FHN in the literature appears to describe a number of conditions resulting from infectious or non-infectious aetiology. We have classified this condition under infectious aetiology because the most recent research (e.g. McNamee and Smyth 2000) suggests the term BCO best describes the current conditions. However, the situation remains confused and the term FHN often includes non-infectious conditions and is still frequently used in the field (and was used by EC 2000).

Incidence

Pattison (1992) suggested FHN was the most common cause of lameness in UK broilers. Thorp (1993) and Thorp and Waddington (1997) have cited BCO as an important cause of leg disorder. McNamee (1998) monitored 28 male flocks and 19 female flocks and found that the incidence of male birds culled was 0.52% and females was 0.38%. In a second study McNamee et al (1999a) found that 0.52% of all birds culled were due to leg problems. BCO was found in 17.3% of cases. This was the highest single category and BCO was present in many birds categorised as other culls (which suggested that BCO is not detected by stockman, McNamee and Smyth 2000). Thorp et al (1993b) examined the proximal femora of lame broilers that showed postural signs of proximal femoral degeneration, in order to establish which conditions were most prevalent. Out of 67 birds examined, 64% showed gross disintegration of the femoral head, mostly due to osteomyelitis and chondritis. 25% had epiphyseolysis while the remaining 11% had microscopic bacterial or osteochondritic lesions only. BCO (or FHN) is the most common cause of severe leg disorders in modern broilers.

Clinical signs and pathology

These conditions are severe degenerative disorders which frequently affect broilers towards the end of the growing period. EC (2000) indicates that birds with FHN may not show any outward sign of leg abnormality but are reluctant to walk, and when they do, they place their wing tips on

the ground to support themselves. Birds have a characteristic limping gait and in advanced stages, affected broilers are reluctant to move (Nairn and Watson 1972) and are unlikely to obtain food and water (McNamee and Smyth 2000). Thorp et al. (1993b) also reported that many of these birds vocalised loudly when the proximal femur was palpated, implying that the condition was painful. BCO (or FHN) is likely to have a severe impact on welfare and is likely to be painful and lead to birds being unable to feed and drink (although in the latter case they will tend to be culled out of the flock).

Clinical signs and pathology have most recently been described in detail (for BCO) by McNamee and Smyth (2000). The most commonly occurring sites are the proximal end of the femur and tibiotarsus (Nairn 1973). Emslie et al (1983) found lesions were most common in the proximal end of the tibiotarsus and distal end of the femur. Thorp and Waddington (1997) found a higher incidence in the proximal end of the tibiotarsus when compared with the femur of broilers with rickets.

Macroscopically, BCO may appear as focal areas of yellow caseous exudate or lytic areas, which cause affected bones to be fragile (Skeeles 1997). Lesions vary from small pale areas adjacent to the growth plate to large zones of yellow tissue extending from the growth plate to the medullary cavity (McNamee et al 1999a); only a fraction of lesions can be seen macroscopically (McNamee et al 1998). Histologically, there are usually clumps of basophilic bacteria in epiphyseal or physeal blood vessels and these are surrounded by poorly stained cartilaginous matrix containing necrotic chondrocytes (McNamee and Smyth 2000).

Aetiology

In BCO *S. aureus* is the most common bacterium recovered from egg and joint infections of poultry (Skeeles 1997). It has been frequently recovered from bone infections of commercial broilers (Nairn and Watson 1972; Randall and Reece 1996; Riddell 1997; McNamee 1998; McNamee et al 1998). Other bacteria have been recovered from infected bone include coagulase-positive *Staphylococci* e.g. *Staphylococcus xylosum* and *S. simulans* and other agents: *Escherichia coli*, *Mycobacterium avium*, *Salmonella* spp. and *Enterococcus* (Reece 1992; Thorp et al 1993; EC 2000; McNamee and Smyth 2000). When the term FHN is used non-infectious aetiology may be the cause (Butterworth 1999) and immunosuppression, nutritional and developmental factors may be implicated. EC (2000) suggests that birds may become infected due to pre-existing pathologies which may then act as foci for bacterial invasion.

Prevention

The use of anti-biotics is unlikely to provide long-term solutions due to the inherent resistance of the organisms and the concerns over increasing the development of antibiotic resistance (McNamee and Smyth 2000).

To date the use of vaccines has not been particularly successful. Jungherr and Plastridge (1939) had no success while Smith (1954) had little success. Current efforts to develop *S. aureus* vaccines for the prevention of bovine mastitis and human *S. aureus* infections may provide knowledge relevant to the broiler problem (McNamee and Smyth 2000). However, vaccines against *S. aureus* alone may be of little use because the disease is caused by other organisms.

Thompson et al (1980) and McCullagh et al (1998) suggested that appropriate disinfection and improved hatchery hygiene might reduce levels of infection. In recent surveys, 62 - 63 % of cases of BCO have been caused by *S. aureus* (McNamee 1998; McNamee et al 1998) and McNamee et al (1999b) showed that the younger the birds were exposed to *S. aureus*, the higher the incidence of BCO.

Bacterial interference has been used as a successful strategy to prevent *staphylococcal* infection in human nurseries (Kluytmans et al 1997) and turkeys (Jensen et al 1987). Preliminary studies of bacterial interference to control staphylococcal infections in chickens have been

successful (Nicoll and Jensen 1987). The mechanisms of interference is thought to be a function of both competition by the interfering bacterium for the same tissue receptor sites and the secretion of a bacteriocin that is bacteriocidal for *S. aureus* (Wilkinson and Jensen 1987). This approach is worthy of further investigation.

Conclusion

BCO is a common and severe disease which causes poor welfare. McNamee and Smyth (2000) and others have argued that the term FHN is inappropriate and that the term should be abandoned. The term BCO should be adopted. The most common cause of BCO is *S. aureus* which is a difficult pathogen to control. Development of vaccine is some way off and even if successful for *S. aureus*, other bacteria can cause BCO. The use of careful management practices and particularly strategies involving bacterial interference warrants further investigation.

Arthritis and tenosynovitis

Bacterial and viral infection

This term covers a range of problems characterised by arthritis and tenosynovitis in joints (EC 2000). Affected birds are lame and the tendons and joints swollen (Reece 1992). Butterworth (1999) indicates that these conditions result from the colonisation of synovia (sheaths) which results in inflammation of the hock (arthritis of the tibiotarsus - tarsometatarsus) and associated ligaments and tendons (inflammation of the gastrocnemius and digital flexor tendons - tenosynovitis). Affected birds are usually depressed and unable to walk (Jordon 1996). These conditions may be divided into arthritis / tenosynovitis caused by bacteria and viruses. Where these conditions occur it is likely to cause poor welfare due to pain and impaired locomotion.

The incidence of bacterial (staphylococcal) tenosynovitis is lower in broilers than in broiler breeders and accounts for 3 - 4 % of cases of lameness post-mortem (Pattison 1992; Reece 1992). In the commercial broiler harvested at 6 or 7 weeks of age, staphylococcal tenosynovitis is usually seen only in the acute phase but in broiler breeders at 7 - 12 weeks subsequent chronic fibrosis may be seen (Johnston 1972; Duff 1986; Duff and Hocking 1986). A variety of bacteria have been associated with this condition: *S. aureus*, *E. coli*, *Salmonella* spp., *Pasterrella multocida*, *Erysipelothrix rhusiopathiae* and *Pseudomonas* spp. (Reece 1992).

The viral condition has been reported as a significant problem in broiler chickens (Olson and Kerr 1966). Affected chickens at 4 - 6 weeks of age showed low mortality (< 5 %) but high morbidity (Heide 1977; Rosenberger and Olson 1991). Incidence in an affected flock has been found to vary from 1 - 10 %. The affected chickens are usually 3 - 8 weeks of age with a number of poorly grown chickens (Heide 1977; Kibenge and Wilcox 1983).

Treatment of birds clinically affected by bacterial infections, may be with a suitable chemotherapeutic agent but this often produces little clinical improvement. However, treatment of a flock decreased the number of new cases (Reece 1992). Attention should be given to quality of the litter, wire floors and slats since trauma to the feet allow bacteria on the skin to gain entry into joints.

In the case of viral infection there is no specific treatment and severely affected chickens have to be culled.

Mycoplasma infection.

The mycoplasma are the smallest self-replicating, infectious organisms which can multiply independently outside the cell, being only some 300 - 800 nm in diameter (Butterworth 1999). This condition has been described by Olson et al (1956) and Wyeth (1974) and it is clear that this disease has existed for many years. Infectious agents include *Mycoplasma gallisepticum*, *M. iowae* and *M.*

synoviae. The latter has the most direct association with leg pathologies which result in lameness. All commercial poultry species are susceptible to infection but strains vary in their pathogenicity (Kleven et al 1975).

Clinical signs and pathology include lameness and swollen tendons and joints (Reece 1992) due to oedema and thickening of periarticular tissue, particularly the synovial membranes, and the production of a viscous yellow to clear exudate in the tendon sheaths and joint cavities (Butterworth 1999). Morbidity in affected flocks was usually 5 - 15 % but varied from 1- 75 %.

Several chemotherapeutic agents have been successful in preventing the spread in infected flock, particularly tiamulin, linomycin-spectinomycin, tylosin and tetracyclines (Reece 1992). In the long run, effective control may be only successful by elimination of the disease by culling. Treatment of breeders with chemotherapeutic agents does not prevent vertical transmission but dipping eggs in antibiotics or incubation heat treatment is effective in limiting vertical transmission (Reece 1992).

Infectious stunting syndrome (ISS)

This condition was first observed in 1976 in the USA (Olsen 1977) and the Netherlands (Kouwenhoven et al 1978). Infectious stunting syndrome (ISS) has been referred to as 'the disease with many names and faces' (Reece 1996). Helicopter disease (Kouwenhoven et al 1978), brittle bone disease (Heide et al 1981), pale bird syndrome (Page et al 1980), malabsorption syndrome (Page et al 1982), runting and stunting syndrome (Kouwenhoven et al 1983) and other similar described conditions may all be manifestations of the same problem.

ISS has been reported in young chicks from most countries with intensive broiler production (EC 2000). Prevalence of severely stunted but active chickens is low, usually 1 - 5 %. Incidence varies for flocks infected from 5 - 20% (Reece 1992).

Clinical signs include stunted growth and swollen joints. One of the features is the presence of severely stunted chickens which remain small despite voracious appetites. The stunted nature of the chickens is noticeable as early as 4 - 8 days of age at which time they would tend to be 20 - 40 g lighter than their conspecifics. The problem is more noticeable after two weeks when the unaffected chickens grow rapidly. Histological examination reveal ricket-like changes in the tibiotarsi of 2 - 4 week old broiler chickens (Bergs and Goedegebuure 1982).

Aetiological factors include picornavirus-like particles, characterised by implication as a cause (Reece and Frazier 1990). Impaired digestion resulting in poor absorption of nutrients has also been suggested as a cause (Lilburn et al 1982; Nelson et al 1982; Goodwin et al 1985; Griffiths and Williams 1985) because evidence of enteritis has been found (Bracewell and Randall 1984; Reece and Frazier 1990). Some reports of ISS-disease may have been reports of poor growth due to other factors (Reece 1992).

Cold stress appears to increase the severity of stunting and adequate temperature should therefore be maintained (Reece 1996). Economic losses can be reduced by culling (up to 10% of the flock). Treatment with massive doses of vitamins (particularly A, D and E) has been known to ameliorate some effects (Reece 1992) and Salyi (1999) found that large doses of D-3 reduced rates of growth retardation. Recent studies have investigated bacterial and viral infection but their role remains unclear (Songserm et al 2000).

Viral induced neoplasia

Viral induced neoplasia include osteopetrosis, myelocytomatosis and Marek's disease which are not very prevalent. Osteopetrosis is any condition characterised by abnormal growth and modelling of bone (Beighton et al 1977). It is rare in chickens.

Myelocytomas involves overgrowth of the bone marrow and abnormal protruberances on the surface of various bones including the tibiotarsi and has been reported in broilers (Reece 1992).

Marek's disease is caused by infection of young susceptible chickens with herpes virus

(Reece 1992). It usually affects chickens from 6 weeks of age, but more usually after 7 weeks. For this reason the incidence of Marek's disease as a cause of neurological lameness in broilers is very low (Butterworth 1999). Vaccines are available which give effective protection under normal commercial conditions (Reece 1992).

Developmental disorders

Valgus and varus deformation of the intertarsal (hock) joint

Angular limb deformity of the valgus / varus (VVD) is the most common long bone distortion seen in broilers (EC 2000). Long bone distortion is a term used to describe distortions in bone shape and structure when ossification is normal (rotated tibia or twisted leg is also a long bone distortion - see below). Valgus deformity can be said to produce a characteristic bow-legged stance while varus deformity can be said to result in a knock-kneed stance. Osbaldiston and Wise (1967) first described twisted legs referring to a lateral angulation of tibiotarsal articulation. Later the term of 'twisted legs' or angular deformity was replaced by valgus-varus angulation or varus valgus deformation (VVD) by Randall and Mills (1981) and Julian (1984).

In VVD the distal tibiotarsus is angulated outwards or laterally (valgus), or inwards or medially (varus). On some occasions this includes the proximal tarsometatarsus. It is possible in some conditions to find one leg affected with valgus deformity and the other with varus deformity (Julian, 1984). Most birds affected are males and the right leg is more frequently affected (Riddell et al 1983; Riddell and Springer 1985; Riddell 1992). Peak incidences may occur at different ages (Riddell 1983; Riddell et al., 1983; Riddell & Springer, 1985). It is a disease of considerable commercial and welfare importance and can produce severe lameness with painful fractures and leg injuries if the birds are not culled (Julian, 1986).

Riddell & Springer (1985) raised the question of whether valgus and varus deformities were distinct conditions and Leterrier & Nys (1992) concluded that valgus and varus deformities have different pathogenesis. In their study every case of varus angulation was associated with displacement of the gastrocnemius tendon on the medial side of the distal tibiotarsus. Cutaneous erosions and septic arthritis were sometimes present in the intertarsal joints. Leterrier & Nys (1992) observed that varus deformity produced severe lameness at an early age, possibly from its onset, owing to displacement of the gastrocnemius tendon. In cases of valgus angulation, displacement of the gastrocnemius tendon only occurred in severe cases, there being no displacement in mild cases. The severity of valgus deformity becomes worse with age as does lameness (Randall & Mills, 1981; Leterrier & Nys, 1992), leading to an increasing number of culls throughout the growth period (Riddell & Springer, 1985). Measurement of ash content in both metatarsi of 7-week old chicks revealed poor mineralisation of bone of the broilers affected with varus or severe valgus deformity. Other studies have found the pathologies to be similar (Duff & Thorp, 1985b; Cruickshank & Sim, 1986). Most authors to date have classified valgus and varus deformities as a single disorder (Duff & Thorp, 1985b; Cruickshank & Sim, 1986). The present review will describe the conditions separately, to the degree that there is the information available to do so.

Incidence

VVD has been reported to be a common skeletal disorder in Canadian broilers (Riddell & Springer, 1985; Julian, 1986) and French broilers (Leterrier & Nys, 1992). The incidence in the UK has been reduced considerably by genetic selection (Pattison, 1992).

VVD lesions have been seen at hatching (Thorp, 1992), but the condition usually develops later, occurring throughout the growth period (Julian, 1984). 75% of cases occur when the birds are between 3 and 5 weeks of age (Randall & Mills, 1981). In Canadian flocks, valgus deformity is the commonest disorder in birds less than 7 weeks of age, while varus deformity becomes more

prevalent after this time in roasters and is often seen in broilers at processing (Riddell, 1983; Riddell et al., 1983; Riddell & Springer, 1985). In French flocks, varus deformity has been reported to occur between 5-15 days of age, while valgus developed after 2 weeks of age. Thereafter, valgus was by far the most prevalent disorder. The difference in incidence between Canadian and French flocks may be due to differences in the strains of the birds (Leterrier & Nys, 1992).

Clinical signs and pathology

Varus and valgus angulation of more than 20 degrees is considered to be a deformity (Duff & Thorp, 1985a). A degree of torsion in the tibiotarsus may accompany angulation in VVD (Riddell, 1992). In cases of varus deformity, Leterrier & Nys (1992) also found torsion in the femur.

Moderately affected chickens walk with a waddling or hobbling gate while severely affected birds are either unable to walk or walk on their hocks (Julian, 1984) which can cause bruising at the joint, ulceration and secondary infection (Randall & Mills, 1981). Extreme valgus angulation may lead to fracture of the distal tibiotarsus, separating the condyle from the shaft, and the broken end of the shaft eventually penetrates the skin (Randall & Mills, 1981). The metaphysis of the distal tibiotarsus may be weakened (Dämmrich & Rodenhoff, 1970), and following VVD, epiphyseolysis has been reported at this site (Julian, 1984). Alternatively, angulation and tendon displacement may lead to dislocation of the intertarsal joint (Duff, 1990). Leterrier & Nys (1992) observed this particularly in cases of varus deformity.

In some cases of VVD, angulation may be caused or exacerbated by displacement of ligaments at the intertarsal joint (Duff, 1990). Traumatic rupture of the ligaments at the intertarsal joint of broilers and broiler breeders has also been reported (Duff, 1986). However, while, in growing broilers, ruptured ligaments were always accompanied by angulation, it could not be ascertained whether ligament rupture or VVD had been the primary event. Riddell (1975c) demonstrated that when the gastrocnemius tendon was surgically severed, VVD occurred as a sequel. Spontaneous rupture of the gastrocnemius tendon or rupture following tenosynovitis has been reported (Riddell 1991), a phenomenon discussed further below.

Dyschondroplastic lesions are often seen in broilers with VVD. These may predispose to the disease as the cartilaginous lesions would weaken part of the proximal tibiotarsus (Randall and Mills 1981). However, dyschondroplastic lesions were not always found in VVD infected birds which suggests that it is not necessary for them to be present for VVD to develop. Leterrier & Nys (1992) found no difference between the incidence of dyschondroplasia in normal birds and those with VVD. Wise & Jennings (1972) suggested that the dyschondroplastic lesions may instead be secondary to VVD, due to uneven load-bearing on the proximal tibiotarsus. Duff & Thorp (1985b) proposed that existing dyschondroplastic lesions may be exacerbated by altered muscle pulling following displacement of the gastrocnemius.

Aetiology

The aetiology of VVD in chickens is poorly understood (Leterrier & Nys, 1992; Riddell, 1992; Hester, 1994). VVD has been investigated more thoroughly in mammals, especially the horse. In the foal, VVD is considered to be a biomechanical disorder caused by uneven weight-bearing in the rapidly growing young animal (Fretz, 1980; McLaughlin *et al.*, 1981; Doige, 1988). Factors that may predispose the foal to development of angular deformity include: joint instability at birth, owing to immaturity of the supporting tissues surrounding the joint; poor ossification of the leg bones at birth, which may be caused by defects in thyroid activity; poor conformation of the leg bones at birth, which may be a result of *in utero* malpositioning; and violent trauma to the leg after birth. These would all predispose the foal to VVD by rendering the leg joint slightly crooked. The tendency for VVD to develop is then exacerbated by factors that increase the pressure which is exerted on the leg. These include over-feeding (which increases growth rate), heavy muscling,

excessive activity and hard floor surfaces. Pressure on the growth plate interferes with the metaphyseal blood supply and inhibits chondrocyte maturation. When this pressure is unevenly distributed, it results in disproportionate growth on one side of the growth plate, and hence angulation. As the degree of angulation increases so the distribution of pressure across the joint becomes more uneven, thereby increasing the severity of the angulation further. The animal is eventually rendered lame.

In the broiler chicken, theories of the aetiology of VVD also focus upon biomechanical forces. Genetic defects, high growth rate and weight-bearing as well as lack of exercise have been implicated.

Incidence of VVD may be influenced by genotype since selection against VVD has been shown to considerably reduce its incidence (Leenstra et al., 1984). An autosomal recessive gene which was responsible for a major part of the variation in the occurrence of VVD was identified by Somes (1969). However, genetic selection alone is unlikely to be capable of preventing the occurrence of VVD as it is probable that multiple factors can produce an identical deformity (Riddell 1992). Sorensen (1992) suggested that breeding programmes could be made more effective by keeping the breeding stock in cages. The lack of exercise would increase VVD incidence, and so the expression of the genetic trait that needs to be identified would be enhanced. However, keeping broiler breeders in cages clearly would result in the creation of additional welfare problems.

There is some uncertainty concerning the importance of growth rate as a factor contributing to VVD. Both valgus and varus deformities occur more frequently in male birds than females (Haye & Simons, 1978; Riddell & Springer, 1985; Leterrier & Nys, 1992) and Riddell & Springer (1985) found that 74% of broilers with valgus deformity and 67% of birds with varus deformity were male. This suggested that growth rate is an important factor. The fact that VVD only occurs in broilers and turkeys also suggests that growth rate may be involved. However, investigations of the genetic correlation between VVD incidence and growth rate amongst different broiler strains have been inconclusive. Leenstra et al. (1984) and Mercer & Hill (1984) found that strains selected for a high growth rate had an increased VVD incidence but others have found that the variability in VVD incidence between broiler strains could not always be accounted for by differences in growth rate (Haye & Simons 1978; Dämmrich & Rodenhoff 1970 and Riddell & Howell 1972). Leterrier and Constantin (1996) found the reductions in growth and in VVD induced by a step up lighting schedule were small compared with those induced by diets containing high concentrations of fibre. They found that a low energy diet was most effective in reducing the growth rate and leg problems if presented in the form of fine particles.

It may be that congenital defects in bone structure or development are responsible for the predisposition of certain broiler strains to VVD. The role of growth rate may exacerbate the condition in fast-growing individuals. Dämmrich & Rodenhoff (1970) identified a delay in appositional bone growth as a possible factor predisposing broiler chicks to VVD. Cruickshank & Sim (1986) reported an abnormally shallow groove in the condyle of the distal tibiotarsus which may cause the early angulation in broiler chicks that then go on to develop VVD. Randall & Mills (1981) suggested that a minor defect in the stance of broilers would be sufficient to explain their predisposition to VVD but they were unable to detect any difference between the stance of broilers and layers.

Cruickshank & Sim (1986) put forward a theory of VVD pathogenesis and aetiology which made a distinction between congenital defects predisposing birds to the lesion and the mechanisms by which the deformity was exacerbated. They observed that both valgus and varus deformities developed from a slight deviation or torsion in the distal tibiotarsus. Birds that developed VVD consistently showed a more shallow groove in the condyle of the distal tibiotarsus. It was proposed that this congenital abnormality might produce a slight displacement of the gastrocnemius tendon, thereby putting uneven strain on the distal tibiotarsus. With time, this could produce angulation in the distal tibiotarsus and proximal tarsometatarsus as the bones grew. The angulation that was observed increased gradually with age and was considered to be a functional adaptation of the bone to the initial deformity.

Dämmrich & Rodenhoff (1970) developed a theory of VVD pathogenesis and aetiology from comparisons made between the internal structure of normal and deformed bones. They studied the internal structure of the femur, which is not directly relevant to the VVD condition. However, some of their findings have been confirmed in the tibiotarsus (Newbrey et al., 1988), and their results are widely cited in relation to VVD (Riddell, 1981, 1992; Leterrier & Nys, 1992; Hester, 1994; Lilburn, 1994; Thorp, 1994). In a comparison of two broiler strains, they found that the broiler strain with a higher incidence of VVD showed a delay in appositional bone modelling, and in particular a slower consolidation of bone with tangential lamellae. The authors argued that poor development of the tangential lamellae would render the bone more susceptible to angulation. Furthermore, the high growth rate of broilers would result in large pressures being exerted on the immature bone, exacerbating the problem. Hence, an imbalance between the rate of body growth and the rate of bone maturation was implicated as a cause of VVD. Itakura & Yamagiwa (1970, 1971) also found a delay in appositional modelling in broilers with VVD.

The growth of a broiler is fastest in the first weeks of life. Until the broiler is over one week of age, ash levels in the tibiotarsus and femur are low, indicating poor mineralization (Itoh & Hatano, 1964; Dilworth & Day, 1965; Wise, 1970b). It is during early life, when the appositional growth of poorly mineralised bone is increasing, that the bone is most susceptible to deformation by weight-bearing stresses. Later in life, the ash and mineral content of bone in broilers with VVD is normal (Rodenhoff & Bronsch, 1971), but if angulation has begun in the first week of life then it is likely to increase spontaneously thereafter (Thorp, 1992). Wise (1970b) compared the skeletal development of broilers and layers. He found that at equal body weights, the bones of broilers were less mature than those of layers, especially during the first 2 weeks of life. He concluded that the growth rate during the first weeks of life was critical for normal skeletal development.

Low activity levels have also been implicated as a factor interfering with the normal structural development of bone. Broilers kept in cages have a higher incidence of VVD than those which are floor-reared (Reece et al., 1971; Riddell, 1976, 1983; Hays & Simons, 1978), and broilers reared in outdoor runs have a lower incidence than those housed indoors (Rodenhoff & Dämmrich, 1971). Rodenhoff & Dämmrich (1973) found a higher proportion of femurs with the normal radial and tangential structure in broilers reared outside than in those reared in cages or on litter. Observed differences in bone structure were attributed to different levels of exercise. Newbrey et al. (1988) compared the structure of the tibiotarsus of broilers with VVD and those of normal broilers. All birds had been floor-housed indoors. They found the same types of lamellar arrangement as Rodenhoff & Dämmrich (1973) and confirmed the importance of internal bone structure in the pathogenesis of VVD. They considered the abnormal lamellar structure of the tibiotarsal diaphysis to indicate a failure of the appositional remodelling process to respond to the normal biomechanical forces that act upon the bone. This may have been due to a lack of exercise or it may have been a result of a congenital defect in prostaglandin metabolism. Prostaglandins are hormones that mediate between biomechanical forces and bone remodelling, via the regulation of 1,25(OH)₂D₃ synthesis. The authors found that plasma 1,25(OH)₂D₃ levels were reduced in birds with VVD, which supported the latter hypothesis but the theory remains tentative, because the levels of prostaglandins in broilers with VVD are unknown.

Prevention

Incidence of VVD has been reduced in recent years by genetic selection and by slowing the growth rate through early feed restriction (Riddell 1992), low energy diets (Leterrier and Constantin 1996) and shortened photoperiod (Classen and Riddell 1989). The incidence has been shown to be decreased in brds that are more active (Rodenhoff and Dammrich 1973). All of these methods should be seriously considered by the industry in order to tackle the problem of VVD.

Conclusion

To conclude, there are multiple and interdependent factors involved in the pathogenesis and aetiology of VVD. A congenital defect in bone structure or development may predispose broilers to the condition, while factors such as a high growth rate and low activity levels may act to exacerbate the deformity. Establishing aetiology is often complicated by the presence of other disorders such as TD. Whether varus deformation is produced by a similar mechanism to valgus deformation is a matter for debate. Valgus deformity is by far the commoner disorder in broiler flocks up to slaughter at six weeks of age. Methods which seek to prevent VVD should focus on genetics, decreasing growth rate and increasing activity.

Rotated tibia or twisted leg

Torsional deformity of the tibiotarsus is characterised by internal rotation of the bone shaft, or external rotation in excess of that which occurs normally during tibiotarsal growth and development (Duff & Thorp, 1985b). In broilers with rotated tibia, the tibiotarsus is often rotated externally through 90 degrees, so that the lower leg is abducted, and the bird is described as having spraddled legs. In severe cases, rotation may reach 180 degrees, so that the foot is directed backwards (Riddell, 1992). In the past it may sometimes have been included in disorders described as twisted and crooked legs (Riddell 1992) and is classified as a long bone distortion along with VVD (Sullivan 1994).

Incidence is generally less than 0.2 % but a higher incidence has been seen in some flocks. Sporadic outbreaks have been reported for leghorn chickens of between 10 - 15 % (cites Riddell 1992, pers com Julian).

Rotated tibia is distinct from VVD in terms of its pathology. In rotated tibia, there is no angulation of the distal tibiotarsus or proximal tarsometatarsus, and the gastrocnemius tendon does not become displaced (Laursen-Jones, 1968). It has been reported to be equally prevalent in both sexes, and in both left and right legs (Riddell & Springer, 1985), although Duff & Thorp (1985a) found that internal torsion was more common in the right leg. It usually occurs unilaterally. The peak incidence of rotated tibia in broiler flocks is usually at 3 weeks of age (Riddell & Springer, 1985).

The aetiology of rotated tibia is not fully understood (Riddell, 1991). As with VVD, biomechanical forces are believed to have a primary role in development. It may also be caused or exacerbated by the development of other disorders such as TD. Because it is seen in slower-growing breeds, it has been suggested that growth rate is unlikely to be an important factor (Riddell, 1992). However, Kestin et al. (1994) found that the severity of tibiotarsal torsion in degrees was closely correlated to the broiler's body weight. This indicates that growth rate may have a role to play, even if it is only exacerbates an existing problem. Feed restriction (Duff & Thorp, 1985b) and a step-up lighting programme (Classen et al., 1991) have been found to reduce the incidence of rotated tibia. It is not clear how far a reduction in growth rate was responsible, or whether the effect was due to an increased activity level.

Activity level and weight-bearing are both considered to be important factors in the aetiology of rotated tibia. Thorp (1988a) observed the bending of the columns of chondrocytes in the metaphysis of birds with torsional deformities, and suggested that this may have been caused by a high level of weight-bearing. Thus, rotated tibia may originate in a deformation of the growth plate by some alteration in weight-bearing. This abnormal pressure on the limb may be a result of low activity levels and a rapid early growth rate (Duff, 1990; Thorp, 1992). Once torsional deformity has begun to occur, there will be an alteration of compressive forces across the growth plate, which may further increase the severity of the deformity (Thorp, 1992). In addition to the biomechanical forces, nutritional deficiency may sometimes cause rotated tibia. Several studies have found that a biotin deficiency can produce twisting of the tibiotarsus (Watkins & Kratzer, 1987; Bain et al., 1988).

Tibial dyschondroplasia

Incidence

Tibial dyschondroplasia (TD) is the most common lesion seen in broiler leg bones (EC 2000). The lesion can lead to two types of problems. If the lesion is large, a fracture may develop through the growth plate. However, the more usual consequence of TD is the development of an abnormal tibial plateau angle (Lynch et al 1992) leading to VVD (EC 2000).

TD may be more common than clinically realised (Rowland 1998) because not all lesions result in gross deformities or detectable subclinical problems (Capps 1998). 26-60% of broilers may be affected (Riddell et al., 1971; Poulos et al., 1978; Riddell, 1981; Cruickshank & Sim, 1986). However, the lesion often becomes much more severe, and may produce lameness in anything from 1-40% of a broiler flock (Siller, 1970; Prasad et al., 1972; Friedman, 1977). It could be as high as 40 - 50% during summer months (Rowland 1988). Vestergaard and Sanotra (1999) cited a prevalence of 6 - 60 % (Cruickshank and Sim 1986; Sorensen 1992; Elliot et al 1995). TD is probably the most significant cause of leg problems in broilers (Yalcin et al 1995) and the most common (EC2000). It develops usually between 2 and 5 weeks of age (EC 2000).

Clinical signs

Subclinical problems are the most commonly observed results of TD; these problems include long bone deformities, lameness and downgrading during processing (Capps 1998). Severe lesions can result in deformity (Siller, 1970; Howlett, 1979) or bowing (Nairn & Watson, 1972; Wise, 1975; Poulos et al. 1978; Sauveur & Mongin, 1978; Rowland, 1988) of the tibiotarsus, or fracture of the fibula (Poulos et al., 1978). In some cases the tibiotarsus fractures below the lesion (Riddell et al., 1971; Meinecke et al., 1980). As in other types of osteochondrosis, TD lesions can sometimes act as foci for the development of osteomyelitis (Wyers et al., 1991). It develops usually between 2 and 5 weeks of age (Lynch et al 1992) before progressing (EC 2000). The lesion can lead to two types of problems: if large a fracture may develop in the growth plate but the more usual consequence is the development of an abnormal tibial plateau angle (Lynch et al 1992) leading to deformity.

Pattison (1992) stated that TD does not often produce lameness severe enough to impair a bird's performance. However, in a survey of leg disorders in commercial broilers, Kestin et al. (1994) using subjective gait scoring, found that 64% of birds with TD lesions had gait defects severe enough to impair walking ability. In 14% of birds, walking ability was considered to be very badly affected. Vestergaard and Sanotra (1999) investigated the presence of TD in 96 broiler chicks in groups of four. 34 birds were found to have TD and 54 had a detectable lameness problem as measured by gate score (Kestin et al 1992). Birds with TD had a higher lameness score.

Pathology and pathogenesis

The TD lesion was first described by Leach & Nesheim (1965). There is an accumulation of uncalcified chondrocytes in the metaphysis, producing an avascular cone of cartilage situated below the growth plate and extending into the metaphysis (Lowther et al., 1974; Gay & Leach, 1985; Farquharson and Jefferies 2000). It has been most commonly found in the proximal tibiotarsus but has been reported to occur in most long bones (Farquharson and Jefferies 2000). There is a failure to mineralise and a defect in bone formation (Leach & Nesheim 1965; Farquharson et al 1992; Lynch et al 1992; Rath et al 1994; Ling et al 2000; Reddi 2000). The rate of chondrocyte proliferation is normal (Hargest et al., 1985a; Farquharson et al., 1992), but there is a failure of hypertrophy (Poulos et al., 1978; Duff, 1984a; Hargest et al., 1985b; Praul et al 2000). Apparently, the chondrocytes and surrounding matrix of the TD lesion fail to provide the proper angiogenic signals to stimulate normal growth plate vascularisation (Praul et al 2000). Thus it is now accepted that TD is a consequence of an inability of the maturing chondrocytes to undergo terminal

differentiation, which normally leads to vascularisation and mineralisation (Farquharson and Jefferies 2000).

Theories differ concerning whether failure in vascular penetration or chondrocyte hypertrophy is the primary event. Failure of blood vessels to enter the growth plate has been suggested as a primary cause following the observation of extensive occlusion of vessels in the lesion (Duff, 1984a, 1989; Lynch et al., 1992) and the production of TD by experimental occlusion (Riddell, 1975a). It has been proposed that this may result from increased pressure on the growth plate vessels by high body weight (Wise & Jennings, 1972; Thorp, 1988a), or long periods of inactive sitting (Thorp, 1986). It has also been observed that birds with unilateral valgus-varus deformities tend to develop TD lesions in the weight-bearing limb (Duff & Thorp, 1985a; Thorp & Duff, 1988; Duff & Hocking, 1986, Leterrier & Nys, 1992), although experimental induction of unilateral weight bearing has failed to produce TD (Riddell, 1975c).

Failure of chondrocyte hypertrophy has been suggested as a primary cause of TD following observation that the number of functioning epiphyseal and metaphyseal vessels was normal in turkeys with TD (Poulos, 1978), while changes in their morphology suggested an inability to penetrate the lesion (Poulos, 1980). This was supported by in vitro work by Haynes & Walser (1982), who found that TD lesion cartilage was resistant to vascular penetration. Orth et al. (1991) found that the collagen in the pre-hypertrophic matrix of the lesion cartilage contained 10 times more non-reducible cross-links than normal, hypertrophic cartilage. These structural cross-links were considered to be responsible for the resistance of the lesion to vascular invasion.

Recently work has focused increasingly on the biochemistry of the TD lesion, and less on its morphology. There has been some uncertainty, however, concerning the importance of the role of 1,25(OH)₂D₃ in stimulating calcium absorption for the prevention of TD (Thorp et al., 1993a). While TD incidence is known to be increased by a low dietary calcium: phosphorus ratio (Edwards & Veltmann, 1983; Hulan et al., 1985), and 1,25(OH)₂D₃ supplementation increases the level of plasma calcium levels (Rennie, 1994), the growth plates of birds with TD have been found to contain normal levels of calcium and phosphorus (Hargest et al., 1985a; Farquharson et al., 1992). It has been suggested that high concentrations of plasma phosphorus relative to calcium, observed in broilers during the first 3 weeks of life (Shafey et al., 1990; Meluzzi et al., 1992; Rennie et al., 1993; Vaiano et al., 1994) may suppress the synthesis of 1,25(OH)₂D₃ (Baxter & Deluca, 1974; Sauveur et al., 1977; Frost & Roland, 1991). Hence, plasma calcium levels may only have an indirect effect upon TD incidence, by limiting the synthesis of 1,25(OH)₂D₃. Edwards & Veltmann (1983) proposed that the effect of plasma calcium: phosphorus ratio on renal vitamin D metabolism may be no different from that of any other cation: anion ratios implicated by Sauveur & Mongin (1978) in their theory of metabolic acidosis (described in more detail below).

It is now widely accepted that the primary role of 1,25(OH)₂D₃ in the production of TD is not in the facilitation of calcium absorption, but in the stimulation of chondrocyte hypertrophy (Thorp et al., 1993a). High 1,25(OH)₂D₃ levels in the growth plate have been associated with production of the hormone, transforming growth factor beta (TGF-β), which is synthesised by pre-hypertrophic chondrocytes, and is believed to regulate hypertrophy (Loveridge et al., 1993). Ling et al (2000) recently found a low concentration of TGF-β in the extracellular matrix adjacent to collapsed cartilage canals of high TD incidence chickens. They suggested this may be a factor in limiting vascular invasion of dyschondroplastic cartilage of TD lesions. Basic fibroblast growth factor (bFGF) is another hormone produced by growth plate chondrocytes. Like TGF-β, it is found at a lower concentration in TD lesion cartilage. It functions as a signal stimulating vascular invasion, by binding to receptors on metaphyseal capillaries (Twal et al., 1992). Hence it is still not known whether failure of hypertrophy or failure of vascular invasion is the primary event in the TD lesion (Leach & Lilburn, 1992).

The pathogenesis of the TD lesion produced by *Fusarium spp.* (Thorp, 1992) and copper deficiency are distinct (Orth & Cook, 1994) and principally produced as models in the laboratory which may not be comparable to lesions in the field (Orth & Cook, 1994).

Aetiology

TD is a condition of modern meat-type birds, which have been genetically and nutritionally manipulated to greatly exceed the growth rate of their random bred or wild counterparts (Orth & Cook, 1994). The rate of bone growth during the first few weeks of age greatly exceeds that seen at any other period of the broiler's life (Wise, 1970a; Marks, 1979; Bond et al., 1991). Additionally, the broiler chick is only capable of synthesising relatively small quantities of 1,25(OH)₂D₃ (a 1-hydroxylated metabolite of vitamin D - discussed further below) during the first week of life (Abbas et al., 1985; Vaiano et al., 1994). Therefore the broiler is likely to be particularly susceptible to TD during this time. Lilburn (1994) reported that the incidence of TD reaches a maximum around 3 weeks of age, and proposed that the very high rate of early growth was responsible.

Since its initial description (Leach and Nesheim 1965) TD has been studied extensively. These investigations have identified many factors that influence the occurrence of TD (most recently reviewed by Leach and Lilburn 1992; more recently by Whitehead 1998 for specific nutritional and metabolic factors). TD is generally recognised to have a multifactorial aetiology, where a similar lesion may be produced independently by various factors (Riddell, 1992; Thorp, 1994; Orth & Cook, 1994; Farquharson and Jefferies 2000). Recently Praul et al (2000) have proposed that mechanical stimulation in combination with genetic background and a variety of dietary factors disrupt the chain of events involved in chondrocyte differentiation.

The observation that the incidence of TD is strongly susceptible to genetic selection has subsequently been confirmed by a number of investigators (Riddell 1976; Sheridan et al 1978; Thorp et al 1993; Wong-vale et al 1993) using a hand-held fluoroscope (Lixiscope) to establish TD incidence (Wong-Valle et al 1993). However, the pattern of inheritance remains unclear (Praul et al 2000). TD has been prevented from occurring in broilers following genetic selection for three generations (Riddell, 1976; Sauveur & Mongin, 1978). Despite a considerable increase in the incidence of TD over the years, as broilers have been selected for ever higher growth rates (Leach & Lilburn, 1992), the incidence can still be reduced to 3.5% in 7 week old broilers after selection for four generations (Wong-Valle et al., 1993). In addition, Yalcin et al (2000a) practised divergent selection for seven generations to produce lines with high and low incidence of TD. In a second study Yalcin (2000b) found selection against TD did not reduce processing yield.

Genetic selection of broilers for a high growth rate increases the incidence and severity of TD (Kestin et al., 1994). Conversely, selection against TD has been shown by some studies to reduce growth rate (Sorensen & Ducro, 1991, cited in Sorensen, 1992). Growth is a major contributor to TD occurrence because restricted feeding virtually eliminates the condition (Su et al 1999; Praul et al 2000). Short-term early feed restriction (Robinson et al., 1992) and daily fasting at an early age (Edwards & Sorensen, 1987) reduce TD incidence with little or no effect on body weight at market age (Leach & Nesheim, 1972; Riddell, 1975b; Riddell, 1976). Unpublished findings by Ducro et al. have attributed this to reduced early growth followed by an acceleration in growth rate (Thorp et al., 1993a).

Despite the clear effects of feed restriction treatments upon both growth rate and TD incidence of broiler flocks, when afflicted and non-afflicted individuals are compared within a particular flock or treatment, a relationship between individual growth rate and lesion incidence and severity is not apparent (Leach & Nesheim, 1972; Riddell, 1975b; Elliot, 1992, cited in Elliot & Edwards, 1994). This led Riddell (1975b) and Elliot & Edwards (1994) to suggest that growth rate may not be the primary factor causing TD after all. It may simply exacerbate the effects of other, more fundamental factors. A possible alternative for the primary mechanisms of TD could be the changes in feeding patterns produced by feed restriction. When broilers are provided with ad lib. food on a continuous lighting programme, they do not show extended periods of rest (Lewis & Hurnik, 1990). This could place a high demand on the skeletal metabolism (Classen, 1992). Elliot & Edwards (1994) have speculated that restricted feeding programmes may alter the diurnal pattern of bone growth without necessarily affecting daily growth rate, in a fashion analogous to the effect

of daily fasting reported by Edwards & Sorensen (1987). Another possible fundamental mechanism for TD was suggested to be a genetic defect in vitamin D₃ metabolism, which will be discussed further below.

Leach & Lilburn (1992) have put forward a different interpretation of the lack of correlation between individual growth rate and TD incidence. They inferred that it was not the bird's absolute growth rate that affected TD incidence, rather it was the extent to which the bird was achieving its potential maximum growth rate. This proposition was not developed further, and so remains entirely hypothetical.

Nutritional factors that influence incidence of TD include calcium to phosphorous ratio, chloride, electrolyte balance, 1,25-dihydroxy vitamin D₃ (Praul et al 2000) and others, although an increasing number of them are being tied in with vitamin D₃ metabolism (Whitehead 1998).

Early studies such as that by Leach & Nesheim (1965), reported that calcium and phosphorus levels had no effect on the incidence of the condition. However, in the early 1980's a response to decreasing calcium or increasing phosphorus levels was found (Edwards & Veltmann, 1981, 1983). Leach & Lilburn (1992) have attributed this change to an increase in broiler growth rates. Levels of dietary calcium which are close to the recommended lower margin are capable of producing TD (Leach, 1982). The effect of calcium concentration is dependent on phosphorus levels (Edwards & Veltmann, 1983; Edwards, 1984a; Lilburn et al., 1989). While the calcium: phosphorus ratio has an important role in the aetiology of TD, the condition cannot be entirely prevented by manipulation of calcium and phosphorus levels (Edwards & Veltmann, 1983; Riddell & Pass, 1987). However, recent studies involving supplementary feeding with phytase resulted in lower incidences of TD (Scheideler and Ferket 2000).

High chloride levels have long been known to increase TD incidence (Leach & Nesheim, 1972; Sauveur & Mongin, 1978). Sauveur & Mongin (1974, 1978) established that it was the ratio of plasma chloride concentration to that of sodium and potassium that was important. They inferred that a high plasma anion: cation ratio was responsible for TD, probably by inhibiting 1,25(OH)₂D₃ production. The reduction in blood pH that would result from a high anion: cation ratio was termed metabolic acidosis. Despite the fact that researchers have failed to find any significant changes in blood pH (Leach & Nesheim, 1972; Sauveur & Mongin, 1978; Halley et al., 1987), a number of different anions and cations have the predicted effect on TD incidence (Orth & Cook, 1994), including sulphate (Halley et al., 1987) and magnesium (Edwards, 1984a; Halley et al., 1987). Some authors have suggested that the action of all anions and cations, including calcium and phosphorus, may be explained by this theory (Edwards & Veltmann, 1983; Leach & Lilburn, 1992). As has already been mentioned, the effect of dietary calcium levels is known to be dependent upon the dietary levels of sodium, potassium and chloride (Hulan et al., 1986, 1987a, b; Simons et al., 1987).

The most effective nutritional means of combating TD involves dietary supplementation with vitamin D metabolites (see also nutrition in section on general aetiological factors). Dietary supplementation with vitamin D itself is ineffective but adding 1-hydroxylated metabolites to diets markedly reduces TD incidence (EC 2000). The metabolite studied most extensively is 1,25-dihydroxyvitamin D₃ which has been shown to prevent TD (Edwards 1990; Rennie et al 1993). This compound is not commercially available but its metabolite precursor, 25-hydroxyvitamin D is available as a commercial feed additive (EC 2000). 25-hydroxyvitamin D is less effective than 1,25-dihydroxyvitamin D but has also been shown to decrease the occurrence of TD (Rennie and Whitehead 1996) though there has been variability in response (EC 2000). For example, recent trials conducted by Roberson (1999) investigated commercial broilers in brooder batteries and concluded that 25-(OH)D-3 did not prevent TD in commercial broiler chicks when levels as high as 250 µg/kg were added to diets adequate (0.95%) or low (0.65%) in calcium.

Sauveur & Mongin (1978) were the first to hypothesise that TD may be caused by a defect in vitamin D₃ metabolism. Edwards (1989) established that when broilers were fed a diet low in calcium and with adequate D₃, supplementation with 10 micro-g/kg 1,25(OH)₂D₃ was sufficient to greatly reduce the incidence of TD. 1,25(OH)₂D₃ supplementation produced a tremendous increase

in the efficiency of calcium utilisation (absorption) from the diet, compared with D3 alone (Edwards et al., 1990). At low calcium: phosphorus levels, it is believed that the broiler is unable to produce 1,25(OH)2D3 rapidly enough from D3 to meet the needs of the growing skeleton (Edwards, 1989, 1990; Elliot, 1992, cited in Elliot & Edwards, 1994; Elliot & Edwards, 1992). This defect in D3 metabolism may be seen as the inability of a normal metabolism to absorb dietary calcium efficiently enough on a commercial diet low in calcium: phosphorus availability to cope with the demand of a genetically increased rate of skeletal growth. As has been described earlier, a low plasma calcium: phosphorus ratio is thought to lead to impaired 1,25(OH)2D3 synthesis, and hence failure of growth plate chondrocyte hypertrophy.

Observations by Elliot & Edwards (1994), that a high plasma concentration of 1,25(OH)2D3 was sometimes associated with TD incidence, has led them to suggest an alternative hypothesis. It may be that there is a defect in the birds' ability to utilise 1,25(OH)2D3 at the receptor level (the chondrocytes). If this theory is correct, then TD may be regarded primarily as a consequence of a side effect in genetic selection. The effects of increased growth rate and reduced dietary calcium availability may be secondary factors. It may be possible to identify the genetic defect and eliminate it with no adverse effect on production.

The renal synthesis of 1,25(OH)2D3 is under the homeostatic control of plasma calcium levels, ensuring that the efficiency of calcium absorption is proportional to the broiler's calcium needs, albeit unable to meet them at low dietary calcium: phosphorus levels. The dietary supplementation of 1,25(OH)2D3 overrides this control mechanism (Rennie, 1994). At a concentration of 10 micro-g/kg, dietary 1,25(OH)2D3 produces hypercalcaemia (excess plasma calcium), which impairs growth rate (Rennie, 1994). At a concentration of 5 micro-g/kg, however, Edwards (1990) found that 1,25(OH)2D3 was capable of producing a significant reduction in TD incidence without a reduction in growth rate. Rennie (1994) has shown that the supplementation of ascorbic acid, which stimulates renal 1,25(OH)2D3 synthesis, acts synergistically with 1,25(OH)2D3 supplementation to reduce TD incidence. Dietary supplementation with 1,25(OH)2D3 and ascorbic acid is therefore a promising technique for commercial use. Given the complex association between calcium and D3 metabolism, it should be noted that if 1,25(OH)2D3 were used commercially, then the optimum levels of dietary calcium and all the elements with which it interacts would probably need to be re-evaluated (Edwards et al 1990).

Other dietary factors that have been reported to increase the incidence of TD include: copper deficiency (Leach & Gay, 1987); choline deficiency (Ferguson et al., 1978); excess of cysteine and homocysteine (Orth et al., 1992); hydrogenated soybean oil (Watkins et al., 1991); the seed fungicides thiram and antabuse (Vargas et al., 1987; Edwards, 1987); and feed contamination with *Fusarium* spp. (Lawler et al., 1987; Wu et al., 1991). There have been contradictory findings for the effect of vitamin A toxicity (Veltmann et al., 1986; Ballard & Edwards, 1988). Copper deficiency, excessive cysteine and homocysteine (Edwards, 1992), and *Fusarium* spp. contamination (Leach & Lilburn, 1992) are not considered to be a commercial problem.

Prevention

Notwithstanding the uncertainty about whether TD is a unitary syndrome or not, research has already indicated several strategies that could be used commercially to prevent or reduce the incidence of TD. Genetic selection, early feed restriction and dietary supplementation with 1,25(OH)2D3 are all methods that should be seriously considered. Genetic selection techniques have reduced its prevalence in recent years and 1,25(OH)2D3 supplementation may be further capable of reducing TD incidence without any detrimental effect on growth rate.

Conclusion

To conclude, despite extensive research into TD, its pathogenesis and aetiology are still not properly understood (Riddell, 1992; Leach & Lilburn, 1992, Thorp, 1994; Farquharson and

Jefferies 2000). There is still little information which clearly demonstrates the mechanism responsible for lesion formation (Praul et al 2000). This lack of clarity is partly due to a number of apparently disparate factors causing TD, possibly by several distinct mechanisms, but each resulting in the occurrence of a histologically similar lesion (Farquharson and Jefferies 2000). A number of authors have suggested that there may be various types of TD, sharing a similar pathology, but resulting from different aetiological factors (Duff, 1989; Riddell & Pass, 1987; Riddell, 1992; Thorp, 1994; Farquharson and Jefferies 2000). The focal nature of the lesion, the variety of nutritional factors that can influence lesion formation and the inability to map the genetic component of the disorder make TD research a significant challenge (Praul et al 2000). TD used to have a substantial genetic basis but following selection to decrease incidence of the condition, nutritional factors are now most important.

Rickets

Incidence

Subclinical cases of rickets are common in broilers, probably due to a marginal level of dietary calcium and excess dietary phosphorus (Riddell & Pass, 1987). A recent study of endochondral ossification defects in Australian commercial broiler flocks found that 50% of the birds in 3 out of 6 flocks had rachitic lesions in the tibiotarsus at 2 weeks of age. There was an incidence of 12-16% in the remaining flocks. These lesions were not sufficiently severe to cause lameness (Vaiano et al., 1994). Lesions of clinical severity are rare (Pattison, 1992). Rickets still occurs in broilers and turkeys, despite present knowledge about dietary requirements (Riddell, 1992).

Clinical signs and pathology

Birds affected with clinical rickets show reluctance to move, and remain sitting on their hocks even when startled. They may use their wings for balance when they do attempt to walk. The bones are soft, and break easily (Groth, 1962; Groth & Frey, 1966).

Although the gross pathology of rickets may be indistinguishable from dyschondroplasia (Thorp et al., 1991), there are differences at the cellular level and no aetiological link has been established (EC 2000). Broadly, there are two types of rickets (EC 2000) based on pathology: hypocalcaemic rickets is characterised by an accumulation of proliferating chondrocytes (Jande & Dickson, 1980), while in hypophosphataemic (phosphorus deficient) rickets the hypertrophic chondrocytes accumulate with normal metaphyseal vessel invasion (Lacey & Huffer, 1982). Vitamin D3 deficiency produces a similar lesion to calcium deficiency (Cheville & Horst, 1981). In the case of dyschondroplasia, however, there is an accumulation of pre-hypertrophic chondrocytes, with failure of metaphyseal invasion (Hargest et al., 1985a). In addition there is the term 'field rickets' which refers to an outbreak in the field where other factors appear to be involved.

Aetiology

Rickets is a metabolic disorder of young, growing poultry, characterised by thickened and poorly mineralised growth plates and poor mineralization of bone. The principle aetiological factors are dietary deficiencies of calcium, phosphorus or vitamin D3; a dietary imbalance between calcium and phosphorus; or interference with the utilisation of one of these nutrients (Riddell, 1992; Thorp, 1992). Although rickets is normally precipitated by nutritional deficiencies, susceptible strains can be selected (Austic et al., 1977), indicating that genetic factors are involved.

Outbreaks may be caused by a feed mixing error (Riddell, 1992), but in some cases nutrient deficiency of the feed cannot be implicated (Bar et al., 1987). These cases are known as field

rickets. Possible causes of field rickets include excess dietary magnesium (Lee et al., 1980), contamination of the feed with *Fusarium spp.* (Kohler et al., 1978) or high levels of dietary iron and aluminium which interfere with phosphorus utilisation (Edwards, 1992). A recent cause in turkeys was attributed to a toxic feed contaminant affecting bone development (Huff et al 1999). Infectious stunting syndrome can also produce rachitic lesions in broilers, owing to impairment of digestion or absorption of nutrients (Reece & Frazier, 1990).

Young broiler chicks may be predisposed to the development of subclinical rachitic lesions by their growth rate and the inadequacy of their D3 metabolism. During the first week of life, decreasing plasma calcium levels, and low but increasing levels of 1,25(OH)2D3 have been reported (Abbas et al., 1985; Vaiano et al., 1994). Calcium deriving from the eggshell is used up rapidly by the growing skeleton (Abbas et al., 1985). As has already been mentioned, the rate of skeletal growth during the first two weeks of age greatly exceeds that occurring at any other period of the bird's life. Absorption of calcium is poor at this time, because the chick is not yet able to synthesise 1,25(OH)2D3 in sufficient quantities (Vaiano et al., 1994). Vaiano et al. (1994) proposed that these factors may be responsible for the development of rachitic lesions. In support of this theory, Elliot & Edwards (1994) found that early feed restriction reduced the incidence of rickets seen at 16 days of age.

The role of vitamin D3 metabolism in rickets is believed to be the absorption of calcium and phosphorus from the intestine (Jande & Dickson, 1980) and the injection of 1,25(OH)2D3 has been shown to prevent the development of rickets in birds fed on a vitamin D3 deficient diet (Spencer et al., 1976; Cheville & Horst, 1981; Taylor & Dacke, 1984; Dickson et al., 1984). In the absence of fluorescent lighting and 1,25-(OH)2D3, 27.5 ug/kg cholecalciferol reduced the incidence and severity of rickets to levels equivalent to those produced by either fluorescent lighting or 1,25-(OH)2D3 alone (Elliot and Edwards 1996). However, even 50.0 ug/kg cholecalciferol was not as effective as fluorescent lights or 1,25-(OH)2D3 in reducing the incidence and severity of TD (Elliot and Edwards 1996).

Rachitic and TD lesions have been reported to occur under the same dietary conditions, and are often seen in the same bird (Leach & Burdette, 1987; Sooncharernying & Edwards, 1989). High phosphorus diets have been shown to cause rickets in broilers at two weeks of age, and later TD at four weeks of age (Long et al., 1984; Riddell & Pass, 1987; Parkinson et al., 1992). Long et al. (1984) suggested that TD may follow rickets as a result of decreased hypertrophy of chondrocytes as the bird attempts to adapt to a low calcium diet. If this is the case, then rachitic lesions may have a role in the development of TD.

The similarities between the incidence, lesion pathology and aetiological factors causing TD and rickets have led some researchers to conclude that there is an important connection between the two conditions (Edwards, 1992; Thorp, 1994). Thorp (1994) has gone as far as proposing that cases of TD with a nutritional aetiology should be classed as a form of rickets with a distinct histopathology, just as hypocalcaemic and hypophosphataemic forms of rickets are. He suggested that TD lesions that do not have a clear nutritional aetiology should be classed separately. Vaiano et al. (1994) has used the term "enchondral ossification defects" to include both rickets and TD.

It should be noted that not all authors agree with this view. Riddell (1992) classified TD and rickets quite separately, as types of developmental and metabolic disorder respectively.

Conclusion and prevention

It is clear that provision of adequate dietary calcium, phosphorus and vitamin D3 will prevent rickets (which is distinct from TD) under normal conditions, in the absence of malabsorption syndromes (EC 2000). Outbreaks of field rickets may be caused by additional unspecified factors and therefore more difficult to control. However, in some cases field rickets may occur due to easily rectifiable human error in feed mixing.

Spondylolisthesis

Spondylolisthesis, a deformation and displacement of the sixth thoracic vertebra, only occurs in broilers (Riddell, 1992). It is not present at hatching, but develops within the first few weeks of life (Wise, 1973). The appearance of clinical signs can occur at any time after the first week of life (Riddell & Howell, 1972), although the peak incidence is at 3 to 6 weeks (Wise, 1970c). A high incidence of subclinical spinal deformation, without damage to the spinal cord, has been reported, but the incidence of clinical spondylolisthesis is low (Wise, 1973). In the UK, the incidence of spondylolisthesis was reduced in the 1980's by genetic selection, to the point that by the mid-80's it had virtually disappeared. Since this time the incidence has increased again, although it is still considered to be infrequent (Pattison, 1992).

Broilers with spondylolisthesis are unable to stand. They have a hock-sitting posture, and tend to move backwards with the aid of their wings, if they move at all (Riddell & Howell, 1972). If not culled, the birds often die from dehydration, although some make a recovery from the paralysis (Riddell, 1973). It has a substantial impact on bird welfare but usually does not frequently occur.

Spondylolisthesis is characterised by deformation and displacement of the sixth thoracic vertebra (T6), which is the only thoracic vertebra in the chicken's skeleton that is not fused to other vertebrae. This leads to compression of the spinal cord between T6 and T7, and hence paralysis of the legs (Wise, 1973). An alternative pathology that produces the same clinical signs and is also known as spondylolisthesis consists of a step-like displacement of the vertebrae from T5 to T7 (Duff, 1990). Osteomyelitis of the thoracic vertebrae can also produce vertebral deformation and spinal cord compression. The clinical signs are indistinguishable from spondylolisthesis, but the aetiology and pathogenesis are completely different (Wise, 1970c).

The incidence of spondylolisthesis is different between different strains of broiler (Riddell & Howell, 1972), and can be increased by genetic selection (Riddell, 1973). Wise (1973) related the development of spondylolisthesis to the early and rapid development of lordosis that occurs in broiler chickens. Lordosis is a curvature of the thoracic region of the vertebral column, which is absent at hatching but begins to develop as soon as the chick starts to walk. It is a normal phenomenon in chickens, but its rate of development is related to the rate of early growth. Because avian bone is very immature during the first week of life (Wise, 1970b), Wise (1973) argued that a high degree of lordosis at this time would be liable to deform the vertebrae. Later in life, when the vertebrae are stronger and more mineralised, they would be better able to resist the deforming forces caused by lordosis. This was confirmed by the finding that a severe restriction in growth rate during the first 7 days of life completely prevented the development of spondylolisthesis, regardless of the subsequent rate of growth.

The use of a step-up lighting programme has been shown to reduce the incidence of spondylolisthesis (Classen et al., 1991). This may have been a result of reduced early growth rate. However, there is some evidence that activity levels may also be important, as broilers reared in cages developed a higher incidence than floor-reared birds in a study by Riddell (1983). Genetic selection has been successful in reducing the incidence of spondylolisthesis.

Chondrodystrophy

Chondrodystrophy is a disorder of the growth plates of long bones, where longitudinal growth is impaired while mineralization and appositional growth remain normal (Wise, 1975). The primary lesion occurs in the zone of proliferation, and consists of a lowered rate of cell division (Wise et al., 1973). Hypertrophy occurs as normal, but the zone of hypertrophy is thin as a result of reduced chondrocyte proliferation (Wise et al., 1973). The condition is characterised by shortened leg bones, enlarged hock joints, and often secondary valgus or varus deformity. In severe cases, displacement of the gastrocnemius tendon follows angulation (Wise, 1975). There has been confusion in the past between chondrodystrophy and VVD, both having been defined as perosis (Riddell, 1992), which was originally characterised by displacement of the gastrocnemius tendon,

enlargement of the hocks and bending of the leg bones (Thorp, 1992). The aetiology and pathogenesis of chondrodystrophy and VVD, however, are quite distinct.

Chondrodystrophy is a nutritional disorder of young, growing poultry. Dietary deficiencies of manganese (Kealy & Sullivan, 1966); choline (Jukes, 1940); niacin (Wise et al., 1973); vitamin E (Scott, 1953); folic acid (Danial et al., 1946); pyridoxine (Greis & Scott, 1972); and zinc (Young et al., 1958) have been shown to produce chondrodystrophy in broilers and turkeys. Early reports also implicated biotin (Jukes & Bird, 1942), although more recent studies have failed to confirm this (Edwards, 1992). Although these nutrients can all induce chondrodystrophy independently, there are many synergistic and antagonistic interactions that occur between them and with other nutrients. A high dietary protein level appears to have the general effect of increasing the incidence of chondrodystrophy when there is a nutritional deficiency in the diet (Edwards, 1992).

Chondrodystrophy has been regarded for a long time as a condition that is no longer of economic significance, as the dietary deficiencies that cause it have been corrected (Wise, 1975; Riddell, 1981, 1992; Thorp, 1992).

Degenerative disorders

Osteochondrosis

Osteochondrosis is a general term, which describes pathologies associated with disturbed endochondral (cartilage) ossification associated with degenerative and traumatic changes in growing cartilage of the growth plate and epiphysis (Riddell 1992). There is a great deal of confusion with respect to the use of the term, and its distinction from dyschondroplasia (Thorp, 1994). Osteochondrosis is characterised by focal (localised) thickening of the growth plate or epiphyseal cartilage due to the accumulation of pre-hypertrophic chondrocytes, clefts in the cartilage of the growth plate or epiphysis and areas of chondrocyte necrosis. Whether primary or secondary to the accumulation of chondrocytes, clefts in the cartilage are thought to be precipitated by repeated minor traumas (Duff, 1984b), although Pattison (1992) found it difficult to understand how this could occur in the relatively calm environment of a broiler house. Necrosis is a result of a poor blood supply to the chondrocytes (Duff 1984a), although whether the failure of blood supply causes the lesion or is the consequence of it, is uncertain (Riddell, 1992; Leach & Lilburn, 1992).

Osteochondritic lesions are common in broilers, turkeys and ducks, and can occur in many parts of the skeleton. Many of these lesions are microscopic and subclinical (Duff, 1990; Riddell, 1992; Thorp, 1992). In broiler chickens lesions are most commonly found in the growth plates and epiphysis on either side of the synovial joints adjacent to the sixth vertebral body (McCaskey et al 1982; Riddell et al 1983) and the proximal femur (Riddell et al 1983; Duff and Randall 1987) and less commonly in the growth plate and epiphysis of the distal tibiotarsus (Duff 1989).

Little work has been carried out on the pathogenesis of osteochondrosis of the proximal femur. Duff (1984a, b) found a higher incidence of this lesion in the contralateral limb of broilers afflicted with VVD unilaterally. This suggested that weight bearing trauma was likely to be involved. From the joints that were affected with similar lesions, Riddell et al. (1983) proposed that shear forces, as opposed to pressure forces were likely to be responsible. Riddell (1992) considered the condition to be analogous to osteochondrosis in mammals.

Osteochondrosis may be responsible for some cases of lameness in adult birds (Duff, 1984c) and growing broilers (Thorp et al., 1993b). Its main clinical importance in growing broilers, however, is its tendency to predispose to epiphyseolysis of the femoral head (Duff, 1984b; Duff & Randall, 1987).

Epiphyseolysis of the femoral head

Epiphyseolysis is characterised by separation of the epiphysis from the bone. Fracture occurs through the growth plate cartilage (Riddell, 1992). The femoral head is predisposed to

epiphyseolysis by osteochondritic lesions (Duff, 1984b; Duff & Randall, 1987). However, epiphyseolysis has a multiple aetiology, and osteochondrosis is not always involved (Thorp et al., 1993b). In the past the term femoral head necrosis (FHN) has been used indiscriminately and has included epiphyseolysis of the femoral head. Riddell (1992) recommended the term FHN be discontinued (see section on BCO or FHN under infectious disorders earlier in this review)

Traumatic epiphyseolysis is believed to be precipitated by severe abduction of the legs during catching (Mitchell & de Boon, 1986; Duff & Randall, 1987). The lesion is usually identified at the processing plant by haemorrhage of the surrounding muscles (Duff & Randall, 1987). It can be distinguished from epiphyseolysis occurring as an artefact of routine coxofemoral disarticulation at necropsy (Riddell, 1981) by the site of the fracture line in the growth plate (Riddell, 1992).

Spontaneous epiphyseolysis has also been identified in broilers prior to catching. Thorp et al. (1993b) carried out post-mortems on lame birds from commercial flocks. They attributed 50% of 17 cases of epiphyseolysis examined to severe osteochondritic lesions. The remainder were believed to be caused by osteomyelitis and chondritis.

Broilers with epiphyseolysis are lame, and frequently use their wings for support during locomotion and whilst sitting (Thorp et al., 1993b). Thorp et al. (1993b) also reported that many of these birds vocalised loudly when the proximal femur was palpated, implying that the condition was painful.

Degenerative joint disease

This is common in older birds (Riddell 1992) and is characterised by erosions and fibrillations in articular cartilage and, in some cases, by the formation of flaps of the same cartilage associated with formation of periarticular osteophytes and fibrosis of the joint capsule (Riddell 1992). Duff (1984) and Riddell (1991) have used the term osteoarthrosis which was supported by Riddell (1992).

Prevalence of DJD may be common in mature breeding birds but there have been no surveys (Duff 1984b; Duff and Hocking 1986). Birds which have been studied often have other defects such as tendon failure which makes it difficult to determine the significance of DJD (Duff and Hocking 1986). DJD causes chronic pain in turkeys (Duncan et al 1991) and is more common in males than in females (Duff 1984b).

DJD is considered to be due to repeated trauma, abnormal conformation or instability of joints (Doige 1988, Riddell 1992). In a study of adult turkeys food restriction appeared to alleviate the condition (Duff et al 1987) but in male broiler breeders no correlation was found between orthopaedic disease including DJD and dietary restriction (Duff and Hocking 1986).

Spontaneous rupture of the gastrocnemius tendon

Rupture of the gastrocnemius tendon has long been recognised as a sporadic cause of lameness in broilers and broiler breeders (Duff & Randall, 1986). Most outbreaks are seen in breeders over 12 weeks of age, where up to 20% of the flock can be affected. However, it has also been recognised in broilers as early as seven weeks of age. Rupture occurs just above the intertarsal joint. It produces lameness immediately and is thought to be painful and to impair welfare. The rupture can be unilateral or bilateral. When bilateral, it produces a characteristic posture in which the bird sits on its hocks with its toes flexed (Riddell, 1991).

Until recently, the condition has been attributed to tenosynovitis, a bacterial or viral infection of the tendon that produces inflammation and often leads to rupture (see section on tenosynovitis under infectious disorders). However, several studies have reported spontaneous rupture of the gastrocnemius tendon, with no signs of tenosynovitis. Riddell (1983) reported cases in roasters at 7 weeks of age. Duff & Randall (1986) reported cases in broilers at 8 and 9 weeks of age, as well as in older birds of 12 weeks and over.

Since rupture of the gastrocnemius tendon affects broilers more frequently than layers (Duff & Anderson, 1986), several studies have examined the normal development of broiler tendons in an attempt to identify differences in their physical structure that could predispose them to infection or rupture. Van Walsum (1975) established that the tendons of broilers have a lower tensile strength than those of layers. Van Walsum (1977) found that the gastrocnemius tendon of broilers had a less organised tissue structure, which could increase its susceptibility to infection and reduce its tensile strength. Duff & Anderson (1986) found chondrocyte plaques in the region of the gastrocnemius near to its attachment to the distal tibiotarsus, where rupture normally occurs. These plaques consisted of an accumulation of chondrocytes with poor vascular penetration, resulting in chondrocyte necrosis. The severity of the plaques increased with age, and were much more prevalent in broilers than in layers. It was considered that this abnormal tissue would reduce the tensile strength of the tendon where it occurred.

It is not known to what degree these structural defects in the tendons of broilers are congenital, and to what degree they are acquired by the action of biomechanical forces during life. However, biomechanical forces are believed to have an important role in precipitating the tendon rupture. Riddell (1983) found that rupture of the gastrocnemius tendon only occurred in broilers fed ad lib. being absent in birds on a restricted diet. Duff & Randall (1986) carried out post mortem examinations of broilers of various ages, and from flocks that had been restricted fed as well as flocks fed ad lib. They made no attempt to select a representative sample for examination, so their findings cannot be used to compare the incidence of ruptured tendons between flocks on different feeding regimes. Their findings indicated that some cases of spontaneous tendon rupture occurred in broilers fed a restricted diet, although cases seen in birds under 12 weeks of age all came from ad lib. fed flocks.

The rapid early growth rate of broilers fed ad lib. diets would be expected to put more strain on the tendons of the leg, and may thereby increase the incidence of spontaneous rupture. Riddell et al. (1983) compared the tensile strength of gastrocnemius tendons of birds fed an ad lib. diet and birds fed a restricted diet. While no absolute differences in tensile strength were found, there was a lower ratio of tendon tensile strength: body weight in birds fed ad lib. In other words, body weight gain was disproportionate to the rate of tendon development in fast-growing birds. Hence, growth rate was considered to be responsible for the incidence of spontaneous tendon rupture seen by Riddell (1983) in ad lib. fed roasters.

Duff & Randall (1986) suggested that biomechanical forces may have been responsible for the development of chondrocyte plaques in the gastrocnemius tendon, on the grounds that the site of the plaques and of tendon rupture was at the point where the tendon traversed the intertarsal joint. It would be exposed to a change in the direction of pull at this site. If weight-bearing associated with a rapid growth rate is responsible for structural changes in the tendon, then the predisposition of broilers to tendon rupture would be entirely due to their growth rate, and could not be considered to be congenital. A reduction in early growth rate may be the only means by which rupture of the gastrocnemius tendon can be avoided.

Contact dermatitis

Contact dermatitis is a disorder of the skin of the foot-pads, hocks and breast which has been reported in broiler chickens (Greene et al., 1985; Martland, 1985) and turkeys (Martland, 1984) when they are kept on litter flooring. The lesions have also been described as breast burns, hock burns, pododermatitis and scabby hip syndrome. They occur at body sites which are in contact with the litter for prolonged periods, and are believed to be caused primarily by corrosive substances or irritants deriving from the faeces (Nairn & Watson, 1972), such as ammonia (von Schmidt & Lüders, 1976; Hemminga & Vertommen, 1985) or micro-organisms (Jensen et al., 1970; Hester 1994). The lesions on the breast and hocks usually develop more slowly and are less frequent than lesions on the feet (Stephenson et al 1960). Foot-pad dermatitis also known as bumblefoot (Sullivan 1994) can produce lameness (Greene et al., 1985) and since the pathology of

the condition suggests that it is likely to be extremely painful, contact dermatitis is a considerable welfare concern.

Contact dermatitis to the foot and hock is the most relevant to this review in that these conditions will tend to lead directly to leg disorders. However, few studies separate leg related disorders from other conditions (e.g. breast blisters) and ameliorative strategies tend to approach the problem through a potential reduction in overall incidence in dermatitis through better management practice (regardless of the specific sight of the lesion).

Dermatitic lesions are not common if the management of litter is properly observed and there is no pre-existing leg disorder which leads to the bird sitting in the litter in damp conditions for long periods. However, it is important to review this condition for reasons of thoroughness as dermatitic lesions can, in some cases, cause lameness or from the point of view of animal welfare exacerbate existing conditions.

Incidence

Skin diseases in broilers are known to be the disorders which have increased the most over the last three decades, increasing from 1.4 % in 1969 to 34.5 % in 1988 (Hartung 1994). A survey of 1190 broiler flocks, in Northern Ireland, from 1986-1987, found the average incidence of hock and breast lesions to be 21% and 0.2% respectively (Bruce et al., 1990). In another study, also from Northern Ireland, the flock prevalence of contact dermatitis in general was reported to be up to 90 % (Greene et al 1985). A two-year survey of flocks owned by a large poultry organisation in the U.K. found that the average incidence of hock lesions amongst broilers was 20% (Pattison, 1987). These data were derived from grading information at the processing plant and will only include the more serious cases of hock and breast lesions. They omit cases of foot-pad dermatitis, which are the most frequent and severe lesions (Greene et al., 1985), because the legs are cut away below the hock at processing (Pattison, 1987). Swedish studies on foot-pad dermatitis have shown that the average flock prevalence of severe foot-pad lesions was 5 - 10% (Elwinger 1995; Berg 1998) with a range from 0 - 100% in different flocks (Ekstrand et al 1998).

The downgrading of carcasses with hock and breast lesions (Greene et al., 1985; Martland, 1985; McIlroy et al., 1987; Pattison, 1987) and the impairment of food conversion efficiency (McIlroy et al., 1987) and growth rate (Martland, 1985; Bray & Lynn, 1986; McIlroy et al., 1987) that has been associated with a high incidence of contact dermatitis, mean that the condition is of considerable economic importance to the broiler industry. The lesion may also act as a gateway for bacteria which may spread through the blood stream and cause joint inflammation and impaired product quality in other ways (Schulze Kersting 1996; EC 2000).

Clinical signs and pathology

It has been observed that birds afflicted with foot-pad dermatitis are reluctant to move (Greene et al., 1985), showing lower activity levels than broilers without such lesions (Martland, 1985). Martland (1985) has suggested that the reduction in growth rate associated with a high incidence of dermatitis may be a consequence of pain-induced inappetence. Birds with severe foot-pad lesions are lame (Greene et al., 1985).

The lesions have been described as 'burns', as they bear a resemblance to this sort of tissue damage. The histopathological changes observed in the skin are similar to those described in many other types of dermatitis and no lesions specific for the disease have been observed (Greene et al 1985; Martland 1985). The lesions begin as erosions of the superficial epidermis, and rapidly develop into ulcers characterised by subcutaneous inflammation, oedema and necrosis of the epidermis. Exudate from the ulcer dries to form a scab, often getting mixed with litter and faeces (Greene et al., 1985). Lesions appear first on the foot-pads (Greene et al., 1985; Hemminga & Vertommen, 1985), and it is here that they are the most frequent and the most severe (Greene et al., 1985). The frequency and severity of lesions on the foot-pads, hocks and breast increase with the

age of the birds (Greene et al., 1985; Hemminga & Vertommen, 1985; Martland, 1985; McIlroy et al., 1987). Greene et al. (1985) observed that in severe cases of foot-pad dermatitis, when the scabs covering the lesion were removed the majority of the foot-pad could be seen to have eroded away.

Aetiology

The lesions are thought to be caused by a combination of wet litter and unspecified chemical factors in the litter (Nairn and Watson 1972; Harms et al 1977; Greene et al 1985; Martland 1985; McIlroy et al 1987; Schulze Kersting 1996; EC 2000).

The most important factor in the aetiology of contact dermatitis is the moisture content of the litter. Experimental studies have established that the addition of the water to the litter greatly increases the incidence of contact dermatitis compared with dry litter conditions (Harms et al., 1977; Martland, 1985). Martland (1985) observed that broilers grown on wet litter rapidly became dirty, and developed foot-pad lesions within a week. At this time, hock and breast lesions were also beginning to develop. All lesions became increasingly severe with time. After 4 weeks all birds were afflicted with very severe lesions and growth rate was impaired. At 7 weeks of age, some birds were transferred from the wet litter condition to dry litter. Many of the lesions healed rapidly, and by 9 weeks of age foot-pad lesion scores were not significantly higher than those of birds which had been reared on dry litter only. Hock and breast lesion healed more slowly, but were significantly reduced compared to birds that remained on wet litter. Growth rate increased when the birds were transferred to dry litter, so that by 9 weeks their body weight was not significantly different to dry litter reared birds. Conversely, when birds were transferred at 7 weeks from dry to wet litter conditions, dematitic lesions developed rapidly, so that by 9 weeks lesion scores were not significantly different to those of birds reared on wet litter only. This was accompanied by a significant reduction in growth rate.

The condition of the litter will depend upon the relative rates of moisture input and loss, and upon the capacity of the litter material to absorb water. A wide variety of litter materials are in use, most of which have a high absorbency. While wood-shavings have the highest absorbency, it has been reported that other materials such as chopped straw, shredded paper, saw dust and rice hulls also produce a good quality substrate (Elson, 1993). Su et al (2000) recently concluded that wood shavings provide a better litter substrate than straw but McIlroy et al. (1987) found no difference in the effect of using straw or wood shavings upon the incidence of hock or breast lesions or the incidence of acute litter deterioration. The type of flooring beneath the litter is an important factor for litter moisture content. Where there is earth, or concrete without a damp-proof membrane, beneath the litter, moisture enters the litter by capilarity (Pattison, 1987). It has been shown that litter on an earth floor will contain as much as 10% more moisture on average than litter on a damp-proofed concrete floor (Sainsbury, 1993).

Water spillage at the drinkers can contribute significantly to the deterioration of litter quality. Bray & Lynn (1986) found that the nipple and cup system produced the least water spillage of twelve drinker systems examined. The traditional bell drinker produced considerably more spillage. Increased water spillage was associated with increased litter moisture and wet capping, as well as an increased incidence of carcass downgrading.

The view that contact dermatitis is caused by prolonged contact with corrosive substances in the faeces is supported by the finding that the incidence of folliculitis is considerably lower in broilers reared on wire floors than on litter or wooden slats (May et al., 1982; Simpson & Nakaue, 1987). The modern broiler may be particularly susceptible because it spends a great deal of its time inactive, sitting on the litter (Pattison, 1987). Jensen (1985) found that the incidence of foot-pad dermatitis was higher in adult broiler breeders kept on wooden slats than on wire, although Simpson & Nakaue (1987) found no consistent differences between the effects of these substrates on foot-pad dermatitis in broilers.

In considering the role of ammonia, the incidence of contact dermatitis is highest when litter conditions are most suitable for the activity of uric-acid digesting bacteria, which produce ammonia

from faecal matter. Wet litter conditions are associated with a high incidence of foot-pad, hock and breast lesions (Harms & Simpson, 1975; Harms et al., 1977; Martland, 1985; Bray & Lynn, 1986; McIlroy et al., 1987; Weaver & Meijerhof, 1991). High litter moisture levels result in 'wet capping' of the litter, where surface friability is lost. The pH of the litter increases and the litter temperature beneath the cap rises, creating an ideal environment for the bacterial synthesis of ammonia, which is trapped in the litter by its high water content (Bray, 1985).

High stocking density has been identified as a factor contributing to litter deterioration and contact dermatitis, owing to the quantity of faeces produced. Increased stocking density has been associated with deterioration in litter quality (Proudfoot et al., 1979; Hoej et al., 1983; Shanawany, 1988; Thomsen, 1992; Gordon & Tucker, 1993), increased incidence of acute outbreaks of litter deterioration (McIlroy et al., 1987), increased surface nitrogen levels (Gordon & Tucker, 1993), poor plumage condition owing to soiling (Proudfoot et al., 1979; Scholtyssek & Gschwindt-Ensinger, 1983; Shanawany, 1988; Gordon & Tucker, 1993) and increased incidence and severity of hock and breast lesions (Proudfoot et al., 1979; Hoej et al., 1983; McIlroy et al., 1987; Gordon & Tucker, 1993). Greene et al. (1985) observed that when a commercial broiler house was thinned at 40 days, half of the birds being removed, the litter dried out in a patchy fashion, the birds became cleaner, and evidence of healing was seen. However, despite this improvement, lesion severity had increased again by 62 days, suggesting that litter deterioration had resumed.

Certain dietary constituents can have an adverse effect on litter quality, either by causing an increased water intake which leads to wetter faeces, or by making the faeces sticky. Incidence of contact dermatitis has been reported to be increased when the litter has a sticky consistency, adhering to the feet and body. Sticky litter may result from the defecation of undigested dietary fat (Bray & Lynn, 1986; Pattison, 1987), or from feeding high levels of soybean meal (Jensen, 1985). High levels of dietary sodium, chloride or potassium all cause an increased water intake. Soybean meal, when fed in excessive quantities can increase water intake, because it is naturally high in potassium. Carbohydrate sources that are poorly digestible, such as tapioca when fed at high concentrations, or wheat flour in badly formed pellets, may also cause the birds to drink more water (Pattison, 1987). A diet high in protein and low in energy has also been associated with wet capping of the litter (Bray & Lynn, 1986). Pattison (1987) considered the poor digestibility of meat and bone meal and poultry offal meal to be a cause of diarrhoea when present in the finisher diet at concentrations higher than 5%.

High dietary fat levels, and especially fat sources of poor digestibility, make the faeces greasy with fat that has not been digested or absorbed. These faeces tend to adhere to the bird's body (Pattison, 1987). Bray & Lynn (1986) found that this led to hock lesions, but only when the moisture content of the litter exceeded 46%. This indicates that while the fat may cause the litter to stick to the skin, ammonia emission from the litter is responsible for producing the lesions.

High dietary protein levels may have a more direct effect upon the development of contact dermatitis, by increasing the nitrogen levels in the litter, and thereby providing the ammonia producing micro-organisms with additional substrate. Bray & Lynn (1986) found that severe hock lesions resulted from feeding broilers on a high protein diet. While the diet was observed to produce wet capping of the litter, a high nitrogen content in the litter appeared to exacerbate the problem.

In a two-year survey of the incidence of hock scabs among the broiler flocks of a large U.K. poultry organisation, Pattison (1987) noted that there appeared to be cycles of good and bad spells for the incidence of the condition. All the flocks were being fed the same ration from the same feed mill. The author suggested that the cycles may have related to changes in the source of dietary constituents, depending on what raw materials were available at the time. McIlroy et al. (1987), in their survey of broiler flocks in Northern Ireland, observed that despite all flocks being fed a diet made up to the same specifications, the incidence of hock and breast lesions were lower in flocks supplied by one of the three major feed manufacturers. This may support Pattison's (1987) hypothesis, although it should be noted that McIlroy et al. (1987) found many other factors, such as stocking density, weather conditions, flock sex and age, and the identity of the producer to be very

important. Bruce et al. (1990), in a subsequent survey, failed to find a relationship between the incidence of dermatitis and the feed manufacturer.

Litter deterioration may be accelerated by outbreaks of diarrhoea resulting from intestinal disorders, such as infectious stunting syndrome, coccidiosis and enteritis (Hemminga & Vertommen, 1985; Pattison, 1987). Although not primarily caused by any particular microbial agent the lesions often become infected by a variety of bacteria and fungi especially *Staphylococcus spp.* (Hester 1994). Greene et al. (1985) examined the bacterial and fungal flora of lesions, and found difference between the micro-organisms present on lesions and normal skin. Page et al. (1976) have demonstrated that the fungus, *Rhodotorula glutins* is capable of producing dermatitic lesions of the thigh and breast, however, and this could occasionally be responsible for outbreaks of dermatitis. Protozoan "coccidia" of the species *Eimeria* are known to cause enteritis and diarrhoea. While coccidiosis is generally under control by the widespread use of coccidiostats, Pattison (1987) has stated that subclinical coccidiosis can sometimes be complicated by secondary clostridial infection, leading to a necrotic enteritis. The presence of the bacterium, *Campylobacter jejuni* in the intestine has been shown to coincide with the sudden appearance of wet litter conditions (Neill et al., 1984). However, *Campylobacter spp.* has also been isolated from farms with normal litter, so as with other enteropathic organisms the particular strain of the organism may be important (Pattison, 1987). *Escherichia coli*, which causes septicaemia in poultry, may have an indirect effect upon litter condition. According to Pattison (1987), diarrhoea may occur as a result of the sick birds drinking more.

Litter conditions have also been associated with environmental conditions, such as humidity and temperature levels within the broiler house. Weaver & Meijerhof (1991) measured the effect of various levels of relative humidity upon litter condition and the incidence of contact dermatitis. They found that a high relative humidity increased litter moisture and caking, and produced an increase in breast and foot-pad lesions, along with a reduction in body weight. The reduction in growth was attributed in part to increased levels of atmospheric ammonia which accompanied litter deterioration. Musbah et al. (1977) and Musbah (1980) also found an increased incidence of dermatitis on the hip and thigh of broilers when humidity levels were high.

McIlroy et al. (1987) found that the incidence of hock and breast lesions, and that of acute outbreaks of litter deterioration in Northern Irish flocks were greatest in the winter months. They attributed this to the fact that producers normally reduced ventilation levels during the winter to conserve heat, which would lead to a humid atmosphere and wet litter conditions. Furthermore, in cold weather litter moisture is harder to remove by ventilation than at higher temperatures (Pattison, 1987). McIlroy et al. (1987) also found that the incidence of contact dermatitis and acute litter deterioration were correlated with the occurrence of high outdoor humidity levels. Page (1974) and Hemminga & Vertommen (1985) have both observed that the incidence of contact dermatitis is higher when the house environment is warm and humid. This may occur if ventilation levels are inadequate in the summer months. At high temperatures and moist conditions, the uric acid digesting bacteria in the litter will thrive, producing more ammonia (Bray, 1985).

Weaver & Meijerhof (1991) investigated the effect of increasing the rate of internal air circulation with the use of fans upon the quality of the litter. At air speeds of up to 24.5cm/s, the effect upon litter condition was equivocal, as different indices of moisture content were contradictory. There was a reduction in the incidence of breast and foot-pad lesions however. The authors suggested that higher levels of internal air circulation than 24.5cm/s might have a more marked effect on litter quality.

Biotin supplementation has been reported to reduce the incidence of feather breakage and contact dermatitis in turkey poults (Atwal et al., 1972; Misir & Blair, 1988), and to reduce dermatitis in broiler breeders (Harms & Winterfield, 1985). Harms et al. (1977) found that biotin deficiency exacerbated foot-pad lesions in broilers kept on wet litter, and produced lesions in broilers on dry litter. Supplementation corrected the dry litter lesions, and ameliorated the wet litter lesions to some extent. A dietary deficiency of sulphur-containing amino acids has also been shown

to increase the incidence of dermatitis (Musbah et al., 1977; Musbah, 1980), while sulphur amino acid supplementation improved feathering and reduced lesion incidence (Musbah et al., 1977).

Some authors have reported a higher incidence of contact dermatitis in cockerels than in hens (Page, 1974; Harms & Simpson, 1975; Musbah, 1980; McIlroy et al., 1987). This has been attributed both to poor feathering of cockerels and to their higher body weight, which may increase their tendency to spend long periods sitting (McIlroy et al., 1987). Several authors have reported that heavier birds showed a higher incidence of dermatitis (Harms & Simpson, 1975; Hemminga & Vertommen, 1985). However, Pattison (1987) and Martland (1985) reported no difference in the incidence of lesions between cockerels and hens. Harms et al. (1977) actually found hens to be more severely affected with foot-pad lesions. In the studies of Harms et al. (1977) and Martland (1985), the incidence of dermatitis was reported on litter that had been made wet experimentally by spraying water onto it. It may be that when litter moisture content is very high, its ammonia content is such that better feathering or reduced sitting make little difference to the incidence of dermatitic lesions.

Prevention

The incidence and severity of contact dermatitis can be maintained at extremely low levels throughout the growing period by good litter management. In order to keep the litter in a dry and friable state, the stockperson must continuously monitor its condition, and replace damp areas with fresh litter as soon as they appear (Martland, 1985). In Sweden, the litter has been kept dry very effectively by using only a thin layer of litter on the floor, in contrast to the traditional deep litter flooring. This means that the entire depth of the litter is disturbed by the birds' scratching behaviour, instead of only the surface layer (von Wachenfelt, 1993). The use of nipple and cup drinkers instead of bell drinkers is a simple and effective measure that can be taken to reduce litter moisture levels. Care must be taken in considering the constituents of the feed, and how they may affect water consumption and faecal consistency. If there is an acute outbreak of litter deterioration, then the birds must be removed from the house as soon as possible (McIlroy et al., 1987).

Effective control of humidity, temperature and air movement within the house is also essential for the maintenance of litter quality. A combination of insulation and good ventilation are needed to keep relative humidity levels low, to encourage the evaporation of litter moisture, and to prevent the condensation on indoor surfaces which occurs at relative humidities greater than 80% (Sainsbury, 1983). It is particularly important that good ventilation is maintained in the winter, when low temperatures reduce the rate of evaporation from the litter. According to Pattison (1987) the consequences of reduced ventilation upon litter deterioration and dermatitis are sufficiently adverse that it makes economic sense to maintain good winter ventilation despite the cost of supplemental heating. A high rate of internal air circulation may increase the evaporation of water from the litter to some extent, but it cannot be considered as a substitute for ventilation (Weaver & Meijerhof, 1991).

Dobrzanski & Bialas (1993) have reported the experimental use of a floor heating system for the environmental control of broiler housing. It consisted of polyethylene pipes positioned on the litter surface, with water flowing through them at 65°C. It was tested in the winter. Compared with the use of wall radiators, it produced dryer, warmer litter, and the surface ammonia content was halved. It also produced a higher house temperature, lower humidity levels and lower atmospheric ammonia levels. Growth was improved, while feed conversion efficiency and mortality were reduced.

An alternative strategy that has been proposed is the use of raised perforated floors or cages to eliminate the use of litter altogether, and to separate the birds from their faeces. A number of authors have found the incidence of contact dermatitis to be reduced on raised or cage floors compared to litter (May et al., 1982; Simpson & Nakaue, 1987; Bolder et al., 1992). Where alternative flooring types produced a high incidence of dermatitis, they were frequently unsuitable for the maintenance of good hygiene, for example plastic mats (Bayer et al., 1976; May et al., 1987)

and wooden slats (Bayer et al., 1976; Simpson & Nakaue, 1987). In some cases, however, well-perforated plastic or metal floors have been associated with a higher incidence of dermatitis than litter (Andrews et al., 1975; 1990). A problem with the use of wire floors is the high incidence of breast blisters which they produce (Reed et al., 1966; Yates & Brunson, 1971; Andrews, 1972; Andrews & Goodwin, 1973; Andrews et al., 1974; Elson et al., 1976; May et al., 1982; Simpson & Nakaue, 1987).

Neither raised flooring nor caging are to be recommended on welfare grounds. Caging is a particularly unsuitable form of housing, as the severe restriction it imposes on movement results in an increased incidence of skeletal disorders (Classen, 1992) and prevents the birds from engaging in normal behavioural activities. Both raised flooring and caging are likely to be highly unsatisfactory for broiler welfare as they deny the birds a suitable substrate for the performance of scratching and dust-bathing behaviours, and restrict the performance of foraging behaviour to the feed troughs or pans. A study by Bessei (1992a) indicated that the incidence of floor-scratching behaviour was drastically reduced in broilers housed on Florana compared with litter.

Work in Holland by Bolder et al. (1992) has established that a raised, air-permeable floor, aerated from below and with a litter covering, produced an incidence of dermatitis which was similar to the Florana system, and considerably lower than a conventional littered floor.

Conclusions

Contact dermatitis is a widespread problem for European broiler production systems and causes leg disorders either via foot-pad or hock lesions, or subsequent invasion of affected sites by micro-organisms. The condition can not be easily controlled through genetic selection or through changes in age or weight at slaughter within commercial ranges (EC 2000). Management practices seem to be the most important factor in preventing the occurrence of wet litter (EC 2000) which is believed to be the main factor underlying the prevalence of disease outbreak. The maintenance of dry litter, in turn, involves the careful management of food composition and levels of humidity, temperature and air movement. The provision of nipple drinkers which prevent spillage are also important. Thus, in summary, the provision and maintenance of dry litter through careful management are the key to reducing incidences of contact dermatitis.

Overall conclusions

In our review of specific disorders it is clear that genetics, growth rate and management are very important in the prevention of specific leg disorders. These factors will also necessarily affect activity which may in turn also affect the incidence and severity of leg problems. In addition certain aspects of the diet are also important. Causes of infectious origin are largely responsible for severe lameness (e.g. BCO) while those caused by illness of a non-infectious aetiology are more common but cause less severe lameness (e.g. TD).

Among the disorders classified as infectious, BCO (or FHN) is the most common condition of modern birds and causes the greatest impact on welfare. It is often severely painful and birds can not reach food and water or perform other normal behaviours. Recent work has suggested it has a bacterial aetiology, the most common cause being *S. aureus* (but other bacteria also cause the condition). The use of bacterial interference in preventing infection looks promising. Other infectious conditions are not as prevalent. In the case of tenosynovitis attention should be given to quality of litter (which is also very important in the case of dermatitis as outlined further below), wire floor and slats as trauma to the feet allow bacteria or viruses to gain entry into joints. When these conditions do occur they are likely to impair welfare by causing pain and preventing normal behaviour. Tiamulin, linomycin-spectinomycin, tylosin and tetracyclines can prevent the spread of mycoplasma infection. Stunting (ISS) is difficult to diagnose, is known to be exacerbated by cold stress and can be treated with some degree of success with high doses of vitamins (particularly A, D

and E). While the birds are small and stunted, the impact on welfare is less severe than in other conditions.

Among the diseases classified as developmental, VVD is a reasonably common condition (though not as common as TD, see below). Its incidence may be (and has been) reduced by genetic selection and the slowing of the growth rate by early feed restriction, low energy diets and shortened photoperiod. Birds which are more active have been shown to have a decreased incidence. In the case of rotated tibia (which is distinct from VVD) activity levels and weight-bearing are both considered to be important causative factors. Several studies have found that a biotin deficiency can produce twisting of the tibiotarsus. In the case of both VVD and rotated tibia, aetiology is particularly complicated due to interaction with other conditions. For example, a bird which develops VVD may also be carrying TD lesions and have suffered from rickets. TD is probably the most common condition but tends to occur sub-clinically and its aetiology is not well understood. In many cases it does not cause particularly poor welfare; only in hens it is severe. Apparently disparate factors may cause TD, possibly by several distinct mechanisms, but each results in the occurrence of an histologically similar lesion. This makes treatment difficult but genetics and diet can affect prevalence. Rickets is distinct from TD, tends to precede it and may be prevented through the provision of adequate dietary calcium, phosphorus and vitamin D3. Outbreaks of field rickets may be caused by additional unspecified factors and are more difficult to control. Spondylolisthesis may be reduced by reducing growth rate in early development although activity levels and genetics are also important contributing factors. This condition is not very common but when it occurs it is likely to cause pain and impair welfare.

Amongst the disorders classified as degenerative, osteochondrosis is important because of its tendency to predispose to epiphyseolysis of the femoral head. DJD is considered to be due to repeated trauma, abnormal conformation or instability of joints. Spontaneous rupture of the gastrocnemius tendon appears to be due to a high growth rate and a reduction in growth rate in early development may be the only means by which rupture of the gastrocnemius tendon can be avoided. It is likely to cause severe pain and impair normal behaviour such as feeding and drinking. Contact dermatitis is a very widespread problem for European broiler production systems and may cause leg disorders either via foot-pad or hock lesions, or subsequent invasion of affected sites by micro-organisms. The condition can not be easily controlled through genetic selection or through changes in age or weight at slaughter (within commercial ranges). Management practices seem to be the most important factor in preventing the occurrence of wet litter which is believed to be the main factor underlying the prevalence of disease outbreak.

In summary, the most common leg disorders are BCO (or FHN) and TD. BCO (or FHN) is the most common modern disorder and can severely impact on welfare if the birds are not culled. TD is very common, mainly due to the effect of diet or absorption problems, but occurs most frequently sub-clinically and is only of welfare concern in its most severe forms. The situation is complicated by the suggestion that many veterinarians report outbreaks of leg disorders to be site / context specific.

While acknowledging the complexity of these issues, it is necessary to simplify conclusions in order to allow access to a wide audience. We therefore conclude that genotype should be manipulated over the long term in order to reduce the birds' growth rates. Short term measures to reduce leg disorders include meal feeding, early feed restriction and reduced stocking density later in life. BCO may be controlled by bacterial interference while dermatitis must be controlled by careful management of the litter. TD appears to be sensitive to factors in the diet. All other lesions are less prevalent but may impact on welfare.

Order of frequency of occurrence of leg disorders

It is impossible to provide a simple listing of the order of frequency of occurrence of conditions. Many have simply not been surveyed. In addition, in some cases it is difficult to establish the precise nature of the disorder which is being reported. The issue of a disorder's impact

on welfare is a separate issue and addressed further in the next section. In many cases birds with one condition may have another e.g. VVD with TD.

The most common modern conditions are BCO (or FHN incl. epiphyseolosis and BCN); TD (incl. osteochondrosis); contact dermatitis and VVD (which is much less common due to genetic selection). BCO is a major welfare problem while TD is less of a problem but is very common. Rotated tibia, gastrocnemius tendon, spondylolisthesis; tenosynovitis and arthritis are less common than these conditions. Viral induced neoplasia, DJD (which affects older birds), ISS (which includes numerous diseases) and chondrodystrophy are not very common at all.

This listing should be treated with considerable caution. A fully independent commissioned survey (with full pathological back-up) has yet to be conducted to establish overall prevalence of specific disorders.

Gait scoring studies and animal welfare

Introduction

So far we have focused on general aetiological factors and the specific pathology of disorders (Riddell 1992) and their prevention. However, in many recent studies of lameness in relation to welfare, leg disorders have been diagnosed by the application of a subjective gait scoring method (Kestin et al 1992) without an attempt to identify underlying pathology. Most recent studies have employed subjective gait analysis in relation to welfare (e.g. McGeown et al 1999; Danbury et al 2000; Mench et al 2001).

In reviewing the literature on gait scoring and the welfare of broilers with lameness, a number of issues merit specific attention. Firstly, we need to assess the validity of subjective gait scoring analysis and other methods of gait analysis. Secondly, in any specific assessment of welfare, we must take into consideration the fact that welfare may be impaired by a number of factors which include:

(a) the occurrence of pain when the birds are walking or moving (we must attempt to distinguish between lameness that can result from pain in the limb and lameness which can result from pathological changes which could alter mechanical function without necessarily being painful).

(b) the failure of birds to feed and drink due to impaired locomotion (the possibility that birds with severe lameness may starve and be culled or die and thus will be underrepresented in live-bird data sets must also be considered).

(c) the failure of birds to perform normal behaviours which may lead to poor body condition or frustration. This may include the inability of birds to stand up which impairs their ability to perform normal behaviour and may result in contact dermatitis. Thus this also relates to poor physical condition and health.

In the latter two cases lameness may not be directly painful. Thus even if some conditions are not painful they may still be welfare problems.

Finally, the degree to which birds experience different degrees of severity of a particular disorder must also be taken into account. It is not a case of the bird either having the condition or not, but a question of how severely (i.e. to what degree) the bird is affected. When assessing welfare it is important to assess the welfare of individual birds (regardless of the frequency of individual occurrences of the disorder). Animal welfare is essentially individual centred: it is irrelevant to one individual in pain that twenty or two thousand of its conspecifics experience the same level of pain.

Analysis of gait

Leg weakness is considered to be characterised by an abnormal gait or impaired walking ability (Kestin et al 1992). To develop a measure of the severity of lameness that could be made by

observation and without recourse to clinical or post-mortem examination, a number of studies have given subjective severity values to lameness in commercial birds. Indeed, it is not always possible to identify specific skeletal abnormality. Walking ability may therefore be directly measured using a subjective gait score, a system which has its advantages in the field. Such a system allows the direct assessment of gait regardless of underlying pathology. This has the benefit of allowing the assessment of a large number of birds relatively speedily. Nestor (1984) described a rating for turkeys in which 16-week old male experimental birds were given subjective scores from 1 to 5. Emmerson et al (1991) used the scheme described by Nestor (1984) to compare the walking ability of different strains of turkey. Mench (pers. com.) has indicated that the subjective gait scoring system has recently been modified further, creating a system which allows for greater consistency of measurement (Garner et al, submitted cited in Mench et al 2001).

Kestin et al (1992) employed a subjective gait score to measure the prevalence and severity of lameness in four flocks of broilers. Scores range from 0 to 5; birds with 0 have normal and agile walking ability. Birds scoring 1 and 2 have slight defects of varying degree that result in abnormal gait but defects do not seriously compromise the ability to move. In birds with score 3 the gait defect impairs walking ability to the extent that the bird has a limp, with a jerky or unsteady strut and loss of manoeuvrability, acceleration and speed. These individuals often preferred to squat when not forced to move. Birds with scores 4 and 5 are incapable of sustained walking on their feet. Kestin et al (1992) specifically tested the repeatability of measuring subjective gait scores on 300 hundred birds and found that the re-assessment of the same birds was generally highly consistent.

This system of subjective gait scoring has several advantages. A large number of birds may be investigated and ranked in different environments which should allow an overall estimate of the walking ability of every bird regardless of the pathological condition of the birds. In addition Kestin et al (1992) claimed that with suitable divisions between the categories, such a system can provide a useful indication of the welfare of the birds.

Surveys have been conducted to look at leg disorders through gait scoring in broiler houses. As Kestin et al (1992) developed their gait scoring method they studied the distribution of scores in flocks of broilers in the UK. Four flocks of healthy, intensively reared commercial broilers, housed at normal stocking densities were assessed at slaughter; a total of 1127 birds. In addition, two flocks of commercial free range broilers were assessed (200 and 176 birds for each flock respectively). Finally, a total of 645 birds from the slughter generation of four breeds of broilers were assessed at seven weeks of age. The overall prevalence of poor gait in the intensive flocks was high. Kestin et al (1992) reported only 10 % of commercial birds were identified as normal (scoring 0) and 26 % were given a gait score of 3 or higher (severe gait problem). The results showed that leg weakness was a significant problem and that there was little variation between the flocks observed. Ninety percent of broilers reared under intensive condition had a detectable abnormality of the gait. Results of the investigation of four breeds implied that there was a genetic basis to the problem of leg weakness with huge variation of gait scores between different breeds. Kestin et al (1992, 1994) described the application of this method to the gait analysis of commercial birds scored twice on the same day. Kestin et al (1999) investigated four crosses of commercial broiler lines and concluded that there were large differences in some important traits associated with leg weakness among the commercial line crosses. Kestin et al (1992) concluded that this suggested that genotype makes a major contribution, a position supported by many others (e.g. Duff and Thorp 1985; Sorensen 1989; Nestor 1984 for turkeys). Kestin et al (1992) also makes the useful suggestion that walking ability should be one of the criteria included in the selection index when breeding birds. The free range reared birds, provided with a good opportunity for exercise, and either on a low plain of nutrition or on intensive broiler rates of feed showed a lower rate of leg weakness (although not as low as might be expected). Thus, while husbandry practices can reduce leg incidence (through light levels, stocking density, levels of protein in the diet and growth rate), levels of leg weakness are also controlled by other underlying causes. Their study confirmed the finding that higher growth rate resulted in increased leg weakness. In these estimates, numbers of birds with high gait scores would

be underestimates because under commercial conditions they would tend to be culled before data were collected.

These techniques have now been extensively applied by a number of researchers investigating pain responses (McGeown 1999; Danbury et al 2000), behaviour (Weeks et al 2000; Mench et al 2001), strain differences (Kestin 1999; 2001), photoperiod (Sorensen et al 1999) and feed restriction (Su et al 1999). The latter two studies have been extensively discussed in the section on growth (see 'aetiology'). A recent survey in Denmark which included 28 broiler flocks (8% of the total number of flocks in Denmark) showed that the mean prevalence of chicks with gait scores > 0 and > 2 was 75 % and 30.1 % respectively (Sanotra et al 2001). Few of these recent studies have attempted to assess the specific cause of the gait impairment recorded. In Sorensen et al (1999), Su et al (1999) and Kestin et al (1999) only TD was additionally recorded alongside gait (using an x-ray device described by Bartels et al 1989). In Sanotra et al (2001) the prevalence of TD, VVD, crooked toes and foot pad burns was also recorded; this study was a survey of commercial flocks rather than an experimental trial.

There are a number of key elements to subjective gait scoring which may be problematic.

(1) The degree of gait deformity deemed acceptable before the welfare of a bird is seriously compromised may be difficult to establish. Kestin et al (1992) suggested analogy to other species e.g. dairy cows and pigs to try and answer this question. They concluded that a score of between 2 and 3 would be likely to impair welfare in poultry.

(2) The relationship between specific leg disorders and subjective gait scoring remains unclear. Recently EC (2000) suggested that a high proportion of birds with scores 4 and 5 are generally affected by FHN (while Vestergaard and Sanotra (1999) indicated there is an association between TD and lameness at the end of the growing period). More specifically, the gait of an individual bird may be impaired due to a problem of conformity and/or a problem associated with pathology. The 'normal' gait of modern broilers may be ungainly but this does not mean the birds welfare is necessarily compromised. It may be difficult to establish how a bird with a particular gait score should walk and 0 may therefore signify a waddling gait or a completely non-waddling gait. In the former case 90 % of birds may be classified as having some gait disorder while in the latter case it may be only 25 %. To date, no specific study has been carried out to investigate the relationship between gait scoring methods and underlying specific pathology. However, Kestin's gait score makes no claim to identify pathology and should be seen as a valuable and useful tool in the field. In addition Kestin has produced a video to assist in deciding specific gait scores and train researchers.

(3) Some potentially conflicting findings of the research scientists (who may report high incidences of severe lameness) and the industry (who tend to give low incidences of severe lameness) may call the method into question. However, this may also be the consequence of one of the parties inaccurately measuring the birds' gait rather than a failing of the method per se. It may also reflect differences in the flocks studied.

Most of these problems may be overcome. The most serious difficulty is the conflation of conformation with pathology. However, as discussed further below, birds with a poor gait which do not experience pain may also suffer poor welfare due to an inability to perform behaviours and / or feeding / drinking which may result in poor health and body condition. It may also be argued that subjective gait scoring provides a useful practical tool precisely because of the difficulty in identifying underlying pathology (which often may be complex and difficult to identify).

Reiter and Bessei (1997) quantified and analysed gait in laying hens and broilers. The gait pattern was recorded by videotracking. Three points of the body - the cloacal region and both feet joints - were marked by small patches of reflecting foils. The vertical and horizontal movements of the marked points were recorded by a camera in posterior position, while the birds walked on a treadmill. Kinetograms showed a clear difference in the walking pattern of laying hens and broilers. They concluded that limping in broilers with leg problems can be measured by differences in the lateral and vertical movements of the right and left leg.

Finally, Corr et al (1998) have investigated a pedobarograph as a novel objective method of gait analysis in poultry. Pressure patterns were established for various part of the foot, the highest pressure being the medial and back toes. The metatarsal region which is often associated with lesions was subject to a lower pressure. While this system has not yet been applied in the field and is less convenient to use than subjective scores, it does provide information on plantar pressure, allowing comparisons to be made of pressure patterns and peak pressures between birds, feet and areas of the foot. This system may have potential applications in early screening in poultry breeding programmes (Corr et al 1998) and other applications, providing more objective methods of analysis.

Effects of leg disorders and animal welfare

Broadly, there are two main factors which may impair welfare in broilers with leg disorders: the condition may be directly painful; the condition may impair the performance of essential behaviours. In the most severe cases these behaviours may be feeding and drinking although other important consequences may result from the inability to perform essential maintenance and other behaviours.

(a) Pain and leg disorders

The suggestion that leg disorders may be painful or discomforting to the affected birds is indicated by the strutting walk of many birds (Sorensen 1989) and in severe cases birds may be unable to reach food and water (Wong-Valle et al 1993) due to impaired locomotion (EC 2000). Manipulations to encourage increased activity should be limited to early in the growing phase when developmental effects on the early development of leg disorders are possible; later in the growing phases increased activity may be uncomfortable or painful due to existing leg problems. Reiter and Bessei (2001) suggested increased activity due to decreased weight bearing on the legs of broilers which had been experimentally manipulated may have been due to the alleviation of pain. Abnormalities resulting in degeneration or inflammation are likely to be directly painful. Cartilage does not have nerves so conditions affecting the growth plate are probably not directly painful, though pain may arise because of the presence of receptors in the synovial membrane (EC 2000). Birds with severe femoral head necrosis show an overt pain response (EC 2000).

Birds with high lameness negotiated an obstacle course faster after administration of a non-steroidal anti-inflammatory drug (McGeown et al 1999) and lame birds self-administered drug feed (Danbury et al 2000). The administration of an analgesic to identify lameness has commonly been used for several years for rats, horses and sheep (De Castro Costa et al 1981; Nolan et al 1987; Chambers et al 1995). In turkeys Duncan et al (1991) showed that administration of betamethazone resulted in more speedy and spontaneous activity in birds with degenerative disease of the hip joint.

McGeown (1999) used carprofen (6-chloro-alpha-methylcarbazole-2-acetic acid) which is a non-steroidal anti-inflammatory drug of the aryl propionic acid class with analgesic, anti-inflammatory and anti-pyretic properties (Randall and Baruth 1976). It has been shown to be analgesic in horses (Nolan and Reid 1993) and laboratory animals (Welsh and Nolan 1994). McGeown (1999) administered carprofen to lame and normal slaughter-age broilers to investigate whether it improved agility and walking ability. Thirty six lame (score 3) and 36 sound broilers were selected from a commercial flock at four weeks of age. Birds were assigned twelve each for each condition (lame or not lame) to one of three groups). In the first group carprofen was injected subcutaneously. In the second group saline was injected. The third group was handled but no injection was given. A half an hour later each bird was guided through an obstacle course to obtain food and conspecifics. The lame birds treated with carprofen took a significantly shorter time to complete the course than the other lame birds. Taken in isolation the results do not show that broilers with gait score 3 suffer pain but McGeown (1999) contends that when the results are taken with behavioural evidence (Weeks and Kestin 1997) and the results of self-selection studies

(Danbury et al 1997), they do provide a good evidence that moderately lame birds (> gait score 3) suffer pain when they walk.

Danbury et al (2000) investigated the self-selection of carprofen by lame broiler chickens. Lame and sound broilers were trained to discriminate between different coloured feeds, one of which contained carprofen. The two feeds were then offered simultaneously and the birds could choose which feed they wished to ingest. In the first study, the plasma concentration of carprofen was linearly related to the birds dietary intake. The walking ability of lame birds improved in a dose dependent manner and lame birds consumed more carprofen-food. In a second study with a fixed amount of carprofen in the food birds which were lame consumed more food with carprofen than sound bird. As the severity of lameness increased lame birds consumed a higher proportion of the carprofen-feed. Danbury et al (2000) concluded that carprofen is an effective analgesic and/or anti-inflammatory in chickens. The results further support others that lameness is painful to broilers. In addition, Hocking (1994) found the administration of an anti-inflammatory drug did not improve the walking ability of broiler breeder males with mild cartilage degeneration in the hip-joint.

While this evidence is compelling, some caution should be exercised as the drug may have had some other non-specific effect on the birds which resulted in greater activity. Further research is therefore required. However, it may be concluded that severe lameness is likely to be painful to broilers.

(b) Inability to perform behaviours

Birds may be inhibited from performing essential behaviour patterns (Vestergaard and Sanotra 1999) due to leg disorders. These problems have been observed in behavioural studies which have shown reductions in feeding, drinking, walking, scratching, pecking and dustbathing, and an increase in the time lying, resting and sleeping (Blokhuys and van der Haar 1990; Bessei 1992).

Birds which are unable to walk clearly have poor welfare. As previously stated behavioural studies show lame birds spend more time lying and sleeping and less time on activities such as standing, running or dust bathing (Vestertgaard and Sanotra 1999). Weeks et al (2000) investigated the behaviour of six replicates of broilers obtained from commercial farms. Comparisons were made between sound birds and those of varying degrees of lameness between 30 and 49 days of age. Sound birds averaged 76 % of their time lying which increased significantly to 86 % in lame birds (> gait score 3). Lying increased with age. Birds spent significantly more time standing idle, standing preening and standing eating when not lame. Walking declined with age. Walking occupied 3.3 % of time which was reduced to 1.5 % in the severely affected birds. Sound birds fed over 50 times in 24 h whereas this figure was on average 30 times for lame broilers. Meal time was adjusted by the birds so there was no overall difference in time spent feeding. Weeks et al (2000) concluded that the alterations of the time budget, in particular the reduction in activities performed whilst standing, and the different feeding strategies adopted, are consistent with lameness imposing a cost on the affected broilers to the detriment of their welfare.

Vestergaard and Sanotra (1999) found that leg disorders, as assessed using gait scores and incidence of TD, reduced dustbathing and lengthened the period of tonic immobility (although there is some controversy over using duration of tonic immobility as a measure of fear because it may be associated with factors that are not related to fear, Campo and Carnicer 1994). Infrequent dustbathing has been shown to be associated with frustration (Vestergaard 1994), physiological stress (Vestergaard et al 1997) and chronic fear (Vestergaard et al 1993). Others have pointed out that lameness can be expected to reduce the bird's ability to compete for food and water, leading to mortality, especially when stocking density is high (Kestin et al 1992; Pattison 1992) and predispose the birds to the development of contact dermatitis on the breast and hocks (Harms and Simpson 1975; Martland 1985). There is good evidence that welfare is poor in birds with gait scores of 3 or higher (Kestin et al 1992; McGeown et al 1999).

Another problem of welfare may be created when birds are feed restricted. These birds may be hungry and deprived of the ability to perform their feeding behaviours. It would be better from the point of view of welfare to alter genetics to decrease growth rate rather than restrict feed intake for these reasons.

Order of impact on welfare of leg disorders

It is very difficult to assess the impact on welfare of specific disorders because, in most cases, they have simply not been studied. There has been a tendency to use gait scores rather than assess specific underlying pathology (only TD is routinely recorded in some cases using lixoscope methodologies).

The problems in assessing the impact on welfare are as follows: (1) The disorders vary by degree; (2) The disorders interact; (3) Most disorders have not been studied in relation to welfare impact; (4) Impact on welfare may be due to direct pain or due to the inability to feed, drink or perform other important behaviours.

BCO (or FHN and BCN) is common and often severe in form. TD (incl. epiphyseolysis) and rickets is common, often sub-clinical but, when severe, has a considerable impact on welfare. Contact dermatitis is common and causes poor welfare when severe. Gastrocnemius tendon slippage, tenosynovitis, DJD and spondylolisthesis is not common but causes poor welfare (i.e. pain and debilitation when it does occur along with difficulty walking). VVD and rotated tibia can be common but tend not to be directly painful unless another condition is present which is directly painful. However, they can cause impaired welfare if the birds are not culled and are unable to locomote, feed, drink and perform other behaviours. ISS, viral induced neoplasia and chondrodystrophy have only a small impact on welfare, the latter because it is no longer encountered.

Conclusions

Welfare may be impaired due to pain associated with leg disorders and the prevention of behaviours due to the inability to walk (and other related problems).

It is difficult to establish through gait scoring at which point the welfare of a bird is impaired. However, it is clear that at higher gait scores (> 3) the welfare of the birds is likely to be compromised. Gait scoring as a technique is still being developed with the aim of improving the technique (Garner et al submitted cited Mench et al 2001).

The pain pathways of broilers is little understood. There is little doubt that in certain circumstances (e.g. acute FHN or BCO and spondylolisthesis) leg disorders are very painful to birds. In studies which have administered analgesics to investigate pain, it is clear that the administration of pain-reducing drugs results in an increased ability to walk (e.g. increased activity). Further work is needed.

Birds which are lame are unable to perform their usual activities and their welfare is clearly impaired as a consequence. This is the case if the birds are unable to feed and drink but also if they are unable to perform other key behaviours.

Finally, it is concluded that gait scoring is a practical method for assessing broiler lameness in the field (i.e. commercial flocks). The method provides a useful tool to employ in the field without recourse to pathological investigation and, while the method conflates conformity with pathology, it is a helpful and constructive additional method to assist in welfare studies. For birds with scores greater than 3, lameness may be viewed as severe enough to potentially impair welfare.

The relationship between gait scoring and pathology is unclear and merits further investigation (see further below).

Overall conclusions and recommendations for review

Aetiology is often complex and difficult to identify. Problems encountered by veterinarians are often site or context specific. However, genetics, growth rate and management are very important in the prevention of specific leg disorders. In addition certain aspects of the diet are also important. Generally, causes of infectious origin are largely responsible for severe lameness (e.g. BCO); those caused by non-infectious aetiology are probably more common but are less severe. A definitive independent survey of the prevalence of leg disorders has yet to be carried out.

The frequency of occurrence of leg problems which result in poor welfare has increased during recent years as growth rates have increased although the specific disorders have changed as genetics have changed and management altered.

Body conformation is important and the use of genotypes with slower growth rates is a long-term measure which should result in reduced incidence of leg disorders. Increased activity early on tends to decrease leg disorders. Birds experimentally manipulated to reduce the weight of the body on the legs show greater levels of activity and lower incidences of problems (Reiter and Bessei 2001). Short-term husbandry techniques which reduce leg disorders by manipulating growth include meal feeding (Sue et al 1999), diet manipulation (Bizeray et al 2001), early feed restriction (Su et al 1999) and reduced stocking density (Sorensen et al 2000). However, feed restriction of fast growing genotypes may in itself cause further welfare problems as the birds are hungry. Manipulation of photoperiod has recently been shown to be less effective than these techniques by Sorensen et al (1999) who found that moderate photoperiods applied up to three weeks of age had little overall effect on prevalence of leg weakness. The encouragement of locomotory activity may result in fewer leg-disorders (Bizaray et al 2000) and may be achieved by a number of techniques including environmental enrichment (Mench et al 2001), diet manipulation (Bizeray et al 2001) to reduce growth rate and lighting regimes (but increased activity later in the growing cycle should be avoided as it may exacerbate poor welfare because of existing leg problems). Circadian rhythms mediated via photoperiod may also play some role in bone development and therefore skeletal disorders. Various nutrients play an essential role in the development of leg disorders such as TD, which can occur in response to deficiencies. Such conditions may be alleviated by supplementary feeding.

BCO (or FHN) is a common and severe disease which causes poor welfare. The most common cause of BCO is *S. aureus*. Development of a vaccine is distant but the use of bacterial interference in preventing infection looks promising. Other infectious conditions are not as prevalent. In the case of tenosynovitis attention should be given to the quality of litter (which is also very important in the case of dermatitis as outlined further below). Tiamulin, linomycin-spectinomycin, tylosin and tetracyclines can prevent the spread of mycoplasma infection. Stunting is difficult to diagnose, is known to be exacerbated by cold stress and can be treated with some degree of success with high doses of vitamins (particularly A, D and E).

Among the diseases classified as developmental, VVD is a reasonably common condition. There is uncertainty about the pathogenesis and aetiology of VVD. Its incidence may be (and has been) reduced by genetic selection and the slowing of the growth rate (by early feed restriction in order to reduce growth rate, low energy diets and shortened photoperiod). Birds which are more active have been shown to have a decreased incidence. A congenital defect in bone structure or development may predispose broilers to the condition. Valgus deformity is by far the commoner disorder in broiler flocks up to slaughter at six weeks of age. In the case of rotated tibia (which is distinct from VVD) activity levels and weight-bearing are both considered to be important factors. Several studies have found that a biotin deficiency can produce twisting of the tibiotarsus. TD is probably the most common condition, tends to be sub-clinical but its aetiology is not well understood. In the past the cause seems to have been genetic whereas modern day broilers are afflicted by diet-based forms of TD. Apparently disparate factors may cause TD, possibly by several distinct mechanisms, but each results in the occurrence of an histologically similar lesion. Rickets is distinct from TD, tends to precede it, and may be prevented through the provision of adequate dietary calcium, phosphorus and vitamin D₃ (which may also be true of TD). Rickets may

result from a general calcium deficiency rather than a bone lesion. Spondylolisthesis may be reduced by reducing early growth rate although activity levels and genetics are also important.

Amongst the disorders classified as degenerative, osteochondrosis is important in its tendency to predispose to epiphyseolysis of the femoral head. DJD is considered to be due to repeated trauma, abnormal conformation or instability of joints. Spontaneous rupture of the gastrocnemius tendon appears to be due to a high growth rate and a reduction in early growth rate may be the only means by which rupture of the gastrocnemius tendon can be avoided. Contact dermatitis is a very widespread problem for European broiler production systems and may cause leg disorders either via foot-pad or hock lesions, or subsequent invasion of affected sites by micro-organisms. It is most likely to affect birds already suffering from another condition which prevents the bird from moving about. Management practices seem to be the most important factor in preventing the occurrence of wet litter which is believed to be the main factor underlying the prevalence of disease outbreak.

The most common modern conditions are BCO (or FHN incl. epiphyseolysis and BCN); TD; contact dermatitis and VVD. BCO is a major welfare problem while TD is less of a welfare problem as it tends to be mild but is very common and when severe causing very poor welfare. Rotated tibia, gastrocnemius tendon, spondylolisthesis, tenosynovitis and arthritis are less common than these conditions. Viral induced neoplasia, DJD (which affects older birds), ISS (which includes numerous diseases) and chondrodystrophy are not very common at all.

Welfare may be impaired due to pain associated with leg disorders and the prevention of behaviours due to the inability to walk and related problems.

Most welfare studies employ subjective gait scoring methods. It is difficult to establish through gait scoring at which point the welfare of a bird is impaired. However, it is clear that at higher gait scores (> 3) bird welfare is likely to be compromised. Subjective gait scoring is a very useful technique to allow the simple auditing of flocks and is still being developed as a technique to improve consistency of measurement and utility (Garner et al submitted cited Mench et al 2001).

The pain pathways of broilers is little understood. There is little doubt that in certain circumstances (e.g. acute FHN or BCO and spondylolisthesis) leg disorders are very painful to birds. In studies which have administered analgesics to investigate pain, it is clear that the administration of pain-reducing drugs results in an increased ability to walk (e.g. increased activity). Further work is needed.

Birds which are lame are unable to perform their usual activities and their welfare is clearly impaired as a consequence. This is the case if the birds are unable to feed and drink but also if they are unable to perform other key behaviours.

It is concluded that gait scoring is a practical method for assessing broiler lameness in the field (i.e. commercial flocks). The method provides a useful tool to employ in the field without recourse to pathological investigation and, while the method conflates conformity with pathology, it is a helpful and constructive additional method to assist in welfare studies. For birds with scores greater than 3, lameness may be viewed as severe enough to potentially impair welfare.

It is very difficult to assess the impact on welfare of specific disorders because, in most cases, they have simply not been studied. There has been a tendency to use subject gait scores. The problems in assessing the welfare impact are also inherently difficult because the disorders vary by degree, they interact, they have not been studied in relation to welfare impact and welfare impact may be due to pain or due to the inability to perform certain behaviours. The following may be tentatively concluded:

BCO (or FHN and BCN) is the most common condition and is often severe in form. TD (incl. epiphyseolysis) and rickets is common, often sub-clinical but when severe is a considerable impact on welfare. Pododermatitis is common and causes poor welfare when severe. Gastrocnemius tendon slippage, tenosynovitis, DJD and spondylolisthesis is not common but causes poor welfare when it occurs (i.e. pain when it does occur and an inability to move). VVD and rotated tibia can be common but tend not to be directly painful unless another condition is present which is directly painful. However, they can cause impaired welfare if not culled and an inability to walk, feed, drink

and perform other behaviours. ISS, viral induced neoplasia and chondrodystrophy have only a small impact on welfare, the latter because it is no longer encountered.

Final conclusion

Leg disorders in poultry substantially impair welfare when they occur in severe forms. Serious leg pain and severe restriction of behaviour caused by impaired walking ability are usually the most important causes of poor welfare in broiler chickens. The implementation of a commercial programme to reduce their incidence should be a high priority. Infectious disorders may be controlled by careful management and treatment. A reduction of growth rate by means of genetics (to reduce growth rate), meal feeding, feed restriction (to encourage a more balanced and sustainable growth rate) and reduced stocking density is capable of reducing the incidence of skeletal disorders in broilers probably without affecting body weight. However, feed restriction of fast growing genotypes may in itself cause further welfare problems as the birds would be hungry. Increased activity also decreases leg disorders and may be achieved by manipulation of genetics, environment, feed and lighting regimes (to allow for periods of activity and rest). This should be carried out early in the growing cycle as later on the birds may experience pain when walking. There needs to be careful management of litter to prevent dermatitis and attention must be paid to important factors in the diet (such as Ca, P, phytase and D3). A number of areas which urgently need addressing for further research may be outlined below.

Areas urgently requiring attention: suggestions for further targeted research

A number of areas which require specific urgent attention may be identified as follows:

1. An *independent* survey of commercial flocks with a pathological investigation should be carried out on a large scale to establish the prevalence of specific disorders within specific commercial conditions (incidence of leg problems differs between systems).

2. Studies should be carried out to further develop methods of gait scoring. These should include subjective gait scoring. The relationship between pathology and conformity should be investigated. Gait scoring is very useful for the industry to 'audit' large numbers of flocks (but clearly must be conducted by independent assessors). In addition any study should involve attempts to assess the welfare impact of the specific disorders. While some argue that subjective gait scoring reveals little of meaning relating to the overall welfare of birds due to the conflation of conformity and pathology in its measurements, others argue that since it is very difficult to establish pathology and many factors may interact, it is a useful measure in allowing the study and audit of large flocks. We generally agree with the latter point of view.

3. A study of pain and analgesics in broilers should help to or assist in establishing which leg disorders are painful to broilers. However, regardless of how painful leg disorders may be, welfare may be impaired due to the inability to perform behaviours.

4. Specific controlled-trials in a commercial context need to be carried out. These should further focus upon: husbandry, feed restriction, meal feeding, nutritional factors, disease and stocking density as short-term measures which could be manipulated to decrease leg disorders. The relationship between these manipulations, activity levels and leg problems should be further investigated. Increased activity early in life appears to be an important factor in limiting the prevalence of leg disorders. However, manipulation of husbandry later in life to increase activity may be detrimental to birds with already established problems. One problem may be that results could be found to be site-specific (veterinarians often appear to find this to be the case).

5. An open and transparent forum representing all interested parties needs to be established to allow discussion. There are a number of different approaches and researchers and others would benefit from an exchange of ideas and a sharing of knowledge.

6. There needs to be a coherent and long-term funding strategy in order to conduct this research. The present system pitches one group of researchers against another in competition and

leaves groups with disjointed funding and an inability to retain staff, expertise and laboratories with expensive specialised equipment . It would be more efficient to direct funding to one or two centres and provide a long-term coherent strategy for funding.

Acknowledgements

We thank DEFRA (formerly MAFF), UK for funding this work. We also thank the following who were either visited in person or kindly offered opinion relating to specific issues: Dr M. Gentle (Roslin Institute), Mr K. Gooderham (Lakeside Veterinary Centre, Hemingford Grey), Dr P. Hocking (Roslin Institute), Professor R. J. Julian (University of Guelph), Mr S. Lister (Attleborough, Norfolk), Dr G. S. Sanotra (Royal Veterinary and Agricultural University, Denmark), Professor C. Whitehead (Roslin Institute), Dr S. Wilson (Roslin Institute), Dr D. Wise (University of Cambridge). We also thank the following for their views during their attendance at the 6th European Symposium on Poultry Welfare, Zollikofen, Switzerland: Dr K. Reiter (University of Hohenheim), Dr W. Bessei (University of Hohenheim), Professor J. Mench (University of California, Davis), Dr B. H. Thorp (Aviagen Ltd) and Dr P. M. Hocking (Roslin).

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