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## **LEG AND HEART PROBLEMS IN BROILER CHICKENS**

### **Introduction**

The term “broiler chickens” is used to refer to chickens reared for their meat, as opposed to hens kept for egg laying. The conditions in which broilers on the one hand and egg-layers on the other are kept are completely different, as are the health and welfare problems affecting these birds.

The terms “leg problems”, “leg weakness”, “lameness” and “leg disorders” are used fairly interchangeably when discussing broilers.

More than 4.4 billion broilers are produced each year in the EU, of which over 820 million a year are reared in the UK.

Nearly all UK broilers are factory farmed. They are kept in huge, windowless sheds which can hold up to 30-40,000 birds. The sheds are so overcrowded that, as the birds grow bigger, one can barely see the floor, so thickly is it ‘carpeted’ with chickens.

### **LEG PROBLEMS**

It is generally accepted that the principal factor detrimentally affecting broiler health and welfare are leg problems. In their March 2000 report, the European Commission’s Scientific Committee on Animal Health and Animal Welfare (SCAHAW) said that “Leg disorders are a major cause of poor welfare in broilers” (Conclusion 6 on p.104).

It is also generally accepted that the main cause of leg problems is that modern broilers have been selectively bred (often referred to as “genetic selection”) to grow extremely quickly. Today’s broilers reach their slaughter weight in around 41 days, which is twice as fast as around 30 years ago. These accelerated growth rates have

been achieved primarily by selective breeding, but also through the use of rich diets and, until recently, growth-promoting antibiotics.

What grows quickly is the muscle (meat), but the supporting structure of legs, heart and lungs fail to keep pace with the rapid body growth, and can buckle under the strain of supporting the overgrown body. As a result, each year in the UK millions, probably tens of millions, of broilers suffer from painful, sometimes crippling leg disorders. In addition, millions die each year of heart failure (heart failure will be discussed later in this paper).

### **Significant increase in growth rates**

The fact that there has been a major increase in growth rates will not, I think, be disputed.

Figures from The Cobb Breeding Company Limited show that in 2000 it took a broiler on average 34 days to reach 1.82 kg., whereas in 1966, 60 days were on average needed to reach the same weight. The figure for 1956 was 84 days.

McKay et al (2000) of Ross Breeders Limited state that broiler liveweights at 42 days have more than doubled in the last 23 years (from 1050g. to 2600g.). The authors added that the time to achieve 2 kg. (a common killing weight) has reduced from 63 days in 1976 to 36 days in 1999. Moreover, a paper by van der Sluis (2000) in *World Poultry* states that over the past 25 years, broiler liveweights at 42 days have increased by 130% (1375 g.). (Ross & Cobb supply the majority of broiler chicks in the UK; Ross have recently been renamed as Aviagen).

The SCAHAW report states that selection for growth rate has been very efficient and between 1925 and 1998, age at 1,500 g. body weight decreased from 120 to 33 days (p.21, and Figure 1 on p.21 which illustrates how age at 1,500 g. body weight has changed between 1925 and 1998).

A still to be published new paper states that in the UK, broilers are normally slaughtered at about 6 weeks of age when they weigh approximately 2.2 kg. which contrasts with the growth of an 'unimproved' traditional strain of bird such as a White Sussex, which would weigh about 800g. at the same age.

### **Gait-scoring system**

It is helpful at the outset to refer to a major study in this area published in 1992 by Kestin et al. In this study, the authors described a method for measuring the prevalence of leg weakness by assessing the walking ability of broilers; this is often referred to as the "gait scoring" system. Walking ability is scored in one of six

categories ranging from 0 (completely normal) to 5 (immobile). Birds scoring 1 have slight defects. Birds with gait score 2 have a definite and identifiable defect in their gait. Birds with score 3 have an obvious gait defect. For example, the defect could take the form of a limp, or a jerky or unsteady strut or severe splaying of one leg as it moved. The birds often prefer to squat when not coerced to move and their manoeuvrability, acceleration and speed are affected. Kestin et al (1992) stated that there is likely to be chronic pain and discomfort for birds with gait score 3, 4 or 5.

Broilers with score 4 have a severe gait defect; they can only walk with difficulty and when driven or strongly motivated. Score 5 birds are incapable of sustained walking on their feet; they can only move with the help of their wings or by crawling on their shanks.

In their study, Kestin et al found 26% of the birds had gait scores (GS) 3, 4 or 5. The authors stated that “there is likely to be chronic pain and discomfort associated with the immobility of birds with gait scores of 3, 4 or 5”. In their report, the SCAHAW stated (p.36) that birds with GS 4 and 5 “obviously have very poor welfare” and (p. 36) “There is good evidence that welfare is poor in birds with gait score 3 or higher”. The SCAHAW report added that “Gait scoring surveys have found that large numbers of broilers have impaired walking abilities and there is evidence that birds with score 3 or higher experience pain or discomfort” (p.36).

Kestin et al’s 1992 paper first introduced the gait scoring system for broilers, which is often referred to in the scientific literature thereafter.

### **Growth rate as a major factor in causing skeletal disorders in broilers**

Later in this paper I will refer to more recent gait scoring surveys of the incidence of leg problems in broilers. However, first it is important to establish to what degree genotype is significant in causing leg problems.

One of the studies comprised in Kestin et al’s 1992 paper looked at the prevalence of leg weakness in four breeds of broilers. The birds were grown at commercial stocking densities and on commercial rations. Breeds a, b and c were commercial breeds which, at least at that time, were available worldwide and accounted for most of the broilers killed in the US and the UK. The fourth (d) was a control line from the same original stock as one of the UK commercial breeds, but randomly bred for eleven generations.

Table 3 of the 1992 paper shows that none of the breed d birds had GSs of 3, 4 or 5, whereas 11% of the breed a birds, 27% of breed b and 2% of breed c had GSs of 3, 4 or 5. The breed d birds (i.e. the control line flock) had not been subjected to selection for growth rate and feed efficiency but were kept under husbandry conditions

identical to those for flocks a, b and c. Commenting on the difference in GSs 3, 4 and 5 between d on the one hand and the other 3 breeds on the other, Kestin et al stated (p.193; 1st column) “This suggests that genotype makes a major contribution to the prevalence of leg weakness.” The authors added (p.193; 2nd column) that “the overzealous selection of birds for increased liveweight gain could encourage the development of strains with a higher prevalence of individuals with impaired gait.”

On a separate point, Kestin et al pointed out (p.193; 2nd column) that “The small numbers of birds given scores 4 and 5 could be an underestimate of the prevalence of very poor gait and the true number is likely to be higher, because birds with very poor gaits could already have been culled [by the stockman] or have died.” Indeed, a good stockman should cull birds with very poor gait as they are likely to be in pain and could well have difficulty in reaching the food and water points. Kestin et al’s comment that the number of birds with GS 4 and 5 may underestimate the problem could, in principle, apply to any gait scoring study.

I should point out that rapid growth rates are not the only cause of leg disorders in broilers. The SCAHAW report sets out (ps.31-34) the causes of leg disorders which it sub-divides into 3 categories: infectious disorders; developmental disorders; and degenerative disorders (which latter category mainly affects birds grown to greater ages and weights for breeding purposes).

It is generally accepted, and was specifically stated (p.336) by Sørensen et al (1999), that the factors responsible for leg weakness can be divided into two main categories: those of infectious origin, which are largely responsible for very severe lameness [in effect GSs 4 and 5], and those caused by skeletal abnormality, which results in less severe lameness [in effect, GS 3]. The authors added that preliminary estimates indicate that the non-infectious causes of leg weakness are probably responsible for the bulk of lameness seen in commercial flocks (p.336). One expert I have spoken to said that very roughly around 50% of GSs 4 and 5 are caused by infections and 50% arise from skeletal disorders.

There is broad agreement that the non-infectious causes of lameness probably result from the intensive genetic selection for liveweight gain and breast meat yield to which broilers have been subjected. For example, Kestin et al (1999) pointed out that a large proportion of the leg weakness seen in commercial broilers “is related to the rapid juvenile growth rate” and that “there is a strong correlation between leg weakness and growth rate” (p.1085; 2nd column).

In their 2000 report, the SCAHAW concluded that “Most of the welfare issues that relate specifically to commercial broiler production are a *direct consequence* of genetic selection for faster and more efficient production of chicken meat, and associated changes in biology and behaviour” (my emphasis), (Conclusion 2, p.104).

In the first paragraph (p.110) of their report's Recommendations, the SCAHAW stated that "It is clear that the major welfare problems in broilers are those which can be regarded as side effects of the intense selection mainly for growth and feed conversion. These include leg disorders, ascites, sudden death syndrome in growing birds and welfare problems in breeding birds such as severe food restriction [ascites and sudden death syndrome will be referred to in more detail later in this paper]. It is apparent that the fast growth rate of current broiler strains is not accompanied by a satisfactory level of welfare including health."

In their report, the SCAHAW stated (p.9) that "Accelerated skeletal growth has led to an increased incidence of bone disorders, most resulting from growth plate pathologies." The SCAHAW also stated (p.9) that "Increasing breast muscle yield has caused broilers' centre of gravity to move forward and breasts to be broader. These changes have implications for walking ability, gait and mechanical stresses on legs and hip joints."

The SCAHAW stated (ps.26 & 27) that "Heavy birds show a reduced capacity for antibody production .... This can partly explain an increased mortality due to reduced resistance to infectious agents but most of the health problems encountered in broilers are either cardiac (ascites and sudden death) or leg (tibial dyschondroplasia) problems ... These two types of metabolic diseases are far more frequent in heavy lines ... than in control or light lines of chicken but can be very variable from line to line."

In their Conclusion 4 (p.104), the SCAHAW stated that "A wide range of metabolic and behavioural traits in broilers have been changed by selection practices. Major concerns for animal welfare are the metabolic disorders resulting in leg problems, ascites and sudden death syndrome and other health problems."

The association between genotype and leg problems is illustrated by contrasting broilers and egg-layers. Layers have very few leg problems and grow at a much slower rate than broilers. A graph in the SCAHAW report (Figure 3 on p.25) shows that at 6 weeks of age (the normal broiler slaughter age) layers weigh around 476g., whereas at the same age female broilers weigh around 2,190g. and male broilers weigh around 2,570g. In short, at the age of 6 weeks, a female broiler is over 4 times heavier than an egg-laying hen. As indicated earlier, broilers are much more susceptible to leg problems than laying hens.

## **Which has greater influence on leg problems: husbandry methods or genotype?**

The influence of genetics on leg problems can be seen by contrasting certain husbandry methods (varying lighting and feeding regimes) used to try and address leg problems with the susceptibility of different genotypes to leg weakness.

One husbandry approach has been to change the light/dark pattern provided from the commonly used 23 hours light : 1 hours dark to a more natural light : dark pattern in the hope that this would limit skeletal deformities. A recent paper, however, has concluded that moderate photoperiods (i.e. a more natural light : dark pattern) applied during the first three weeks after hatch “have little beneficial effect on the overall prevalence of leg weakness” (Sørensen et al, 1999, ps.341-2).

Two methods of manipulating the birds’ feeding patterns have also been tried. Normally broilers are given feed *ad libitum*. One alternative approach is to restrict the amount of feed given in the birds’ early days of life. (Whilst we would welcome a reduction in the level of lameness in broilers, CIWF is concerned that significant restrictions of feed are likely to be stressful in themselves.)

Su et al (1999) found that early feed restriction led to better walking ability, but that adjusting the observations for differences in body weight removed many of the significant differences. In fact, the authors concluded that only limited improvements in walking ability can be achieved by early feed restriction. They pointed out that making the feed restriction more severe (i.e. giving the birds even less feed) or applying it for longer, did not lead to improved walking ability. They concluded that the limited improvement that can be achieved by early feed restriction “can be achieved by the least severe and shortest period of restriction applied in this study” (i.e. feed restriction lasting 5 days and calculated to achieve 75% of the predicted growth of *ad libitum* birds).

Another approach is to give the birds two, three or four meals per day rather than feed them *ad libitum*. Su et al (1999) found that birds fed fewer meals per day had better walking ability, less hock burn and less tibial dyschondroplasia than those fed more meals or *ad libitum*. These findings remained the same even when the data were adjusted for body weight; in other words, the major part of the improvement in leg weakness is independent of changes in body weight.

Birds fed fewer meals also had improvements in feed conversion ratio (FCR). The authors stress that “the improvements in FCR and leg weakness achieved in this trial were substantial ..... If the improvements in FCR are maintained until slaughter, and there is no reason to believe they would not be, meal feeding may provide a viable method for improving production efficiency and limiting leg weakness”.

Kestin et al (1999) stated that the studies referred to above showed that alterations to husbandry practices (lighting and feeding) lead to only relatively minor improvements in leg weakness, but “that progress can be made in reducing and eliminating leg disorders by genetic means” (p.1085; 2nd column).

In their 1999 paper, Kestin et al reported on a trial which was conducted to investigate the susceptibility of different genotypes of broilers to leg weakness. Four crosses of commercial broiler lines were assessed, most of which are commonly used in the UK and are available world-wide. One of the crosses was found to have substantially worse walking ability than the others.

Crosses 1 and 2 (both these categories being Ross birds) were similar and tended to have the lowest scores for indices of leg weakness, whereas Cross 3 (Cobb birds) tended to have the highest scores and Cross 4 tended to have more misshapen bones and legs (p.1088; 2nd column).

There were large and significant differences in gait scores and other indices of leg weakness with Crosses 1 and 2 having the best gait scores and Cross 3 the worst. Cross 3 also had the highest foot pad burn scores and the highest hock burn scores.

In previous studies looking at the effect of husbandry changes on walking ability, the largest difference in gait scores achieved was what Kestin et al (1999) refer to (p.1088; 2nd column) as “a highly significant 0.240 gait score units”. The body weight penalty for this improvement was 109 grammes. In Kestin et al’s own study on the impact of genotype on walking ability, there was a difference of 0.509 gait score units between two of the Crosses (1 and 3), with there being no difference in body weight between the two Crosses. In short, a much bigger difference in walking ability was found between genotypes than was achieved by any changes in husbandry methods.

In conclusion, it is clear that genotype has a major influence on lameness, a much greater influence than any husbandry changes that have been studied to date.

### **Recent study examining a range of genotypes**

Recently, a major study by Kestin et al (2001) concluded that the study’s results **“support the hypothesis that the lameness which develops in modern genotypes of broiler is a result of their selection for high liveweights and rapid growth rates, resulting in abnormally high loads being placed on relatively immature bones and joints”** (p.197; 2nd column).

This study examined lameness in 13 genotypes of broiler. They ranged from modern intensive hybrids to traditional “dual purpose” breeds, and had a very wide range of growth profiles. Two different feeding programmes were used, with some birds

being fed on a commercial non-limiting ration and others being fed on a 'Label rouge' diet. The birds' degree of lameness and liveweight were measured at 54 and 81 days of age.

The study found that the more traditional and slower growing genotypes tended to be less lame than the modern genotypes reared on the same feeding regimen. All the birds, irrespective of their genotype or diet, were less lame after 54 days than after 81 days. However, when liveweight was included in the analysis as a covariable, many (but by no means all) of the differences disappeared.

In summarising their findings, the authors stated that "the modern genotypes of broiler were less able to walk than the slower growing genotypes, but when corrected for differences in liveweight, it was found that the differences were due to their higher liveweight" (p.197; 2nd column). In short, very broadly speaking, all broilers of a particular weight will suffer from approximately the same degree of lameness. However, crucially, fast growing breeds will reach a particular liveweight and thus a particular level of lameness at a much lower age than slower growing genotypes.

Having emphasised the importance of the relationship between liveweight and lameness, the authors went on to also stress the importance of growth rate. As indicated above, fast-growing genotypes will of course reach a particular liveweight at a younger age than slower growing genotypes. However, in addition, the authors found that the age at which a particular liveweight is reached significantly affected the walking ability of the birds, with the birds being approximately 0.6 units of gait score worse if they reach a particular liveweight at 54 days of age than if they reach that liveweight at 81 days. In other words, although I said earlier that this study shows that at a particular liveweight all broilers will have approximately the same degree of lameness, in fact this is not the complete picture. A broiler that reaches a particular liveweight at 54 days will be approximately 0.6 units of gait score worse than a broiler that reaches the same liveweight at 81 days. This led the authors to conclude "that, in addition to liveweight, the growth rate of the birds was also important in determining lameness. The birds which, for example, reached 2.70 kg. (the average liveweight of all the birds in the study) in 54 days were almost 1 gait score unit worse than the birds which reached 2.70 kg. in 81 days" (p.197; 1st column).

### **High prevalence of leg disorders**

As indicated earlier, in their 1992 study, Kestin et al found 26% of the birds had gait scores 3, 4 or 5.



In their 1992 Report, the Farm Animal Welfare Council (FAWC) stated that their Working Group found leg problems of varying degrees of severity on nearly every farm visited (FAWC, 1992). The Report stressed that in the worst cases birds were only able to move with great difficulty. Such birds, added the Report, were obviously distressed and had problems in reaching food and water. (The FAWC is an independent body, appointed to advise the Government on farm animal welfare).

A 1999 Danish study assessed the prevalence of lameness in a large and representative sample of commercial flocks. This study found that 30.1% of the birds had gait scores of 3, 4 or 5, scores which indicate that they are suffering from chronic pain (Sanotra, 1999).

It has been alleged that the type of bird used in the Danish study – the Ross 208 – is not commonly used in the UK. CIWF believes, however, that it is in fact used by several UK producers. The crucial point, however, is that in the study referred to earlier by Kestin et al (1999) the Ross 208 had the **lowest** level of leg weakness of all the genotypes examined, which included all those used in the UK. Cobb 500 broilers, which are commonly used in the UK, were found to be substantially *more* susceptible to lameness than Ross broilers. Thus, if Cobb 500 broilers had been used in the Danish study instead of Ross 208, the incidence of lameness would probably have been even higher.

Moreover, a recent Swedish pilot study surveyed eight flocks from four different farms. It found that 14.8% of the birds had gait scores of 3 or above (Berg and Sanotra, 2001). A more recent detailed Swedish study examined 15 flocks of Ross 208 broilers and 16 flocks of Cobb broilers. The study found that 14.1% of the Ross 208 broilers and 26.1% of the Cobb broilers had gait scores of 3 or above (Sanotra et al, 2002).

I have been told that the Farm Animal Welfare Council visited three farms as part of a recent study on broilers. I gather that at one farm they visited three houses in which the proportion of broilers with gait scores of 3 or above averaged above 20%.

In a letter dated 16 April 2002 to the Parliamentary Secretary at the Department for Environment, Food and Rural Affairs, the Chairwoman of the Farm Animal Welfare Council, referring to the findings in the British industry's own survey (completed in 2000), stated that on some broiler production units lameness prevalence was clearly unacceptable. She pointed out that the industry report notes instances where as many as 28% of birds in one shed were lame and others where, within a production unit (i.e. across multiple flocks), the incidence was over 11%.

Earlier in this paper I stated that very approximately 50% of GSs 4 and 5 result from infections, with the other around 50% being caused by skeletal disorders. Most GS 3s

(which are much more prevalent than GSs 4 and 5) result from developmental disorders. Two of the main forms of developmental disorders are angular limb deformities and growth plate defects including tibial dyschondroplasia (TD). There are two forms of angular limb deformity, i.e. either outward (valgus) or inward (varus) angulation of the limb at the intertarsal joint. (See ps. 32-4 of the SCAHAW report for more detail).

The 1999 Danish study referred to above found that 36.9% of the birds had angular limb deformities (varus/valgus) and 57.1% had TD. The detailed 2002 Swedish study referred to above found that 46.4% of the Ross 208 broilers had angular limb deformities (varus/valgus) and 45.2% had TD. The Swedish study found angular limb deformities (varus/valgus) in 52.67% of the Cobb broilers and TD in 56.3% of the Cobb broilers.

### **Strong evidence that lameness is painful**

There is strong evidence that lameness in broilers is painful. Weeks & Kestin (1997) showed that lame birds spend less time in activities requiring them to stand, behaviour which is consistent with them suffering chronic pain when they do stand. Moreover, Danbury et al (1997) have shown that lame broilers self-select more feed containing an analgesic agent than sound birds.

McGeown et al (1999) tested the ability of lame and normal broilers to traverse an obstacle course. Some of the lame birds were treated with carprofen, which is a non-steroidal drug with analgesic and anti-inflammatory properties. The authors commented: "Carprofen increased the speed and walking ability of the lame birds so much that there was no significant difference between the time they took to complete the course and the time taken by the sound birds". They concluded that the results of the study "provide good evidence that moderately lame birds, as defined by a gait score of 3, suffer pain when they walk".

In another study by Danbury et al (2000), chickens were allowed to choose between feed that contained carprofen and their normal feed. The lame birds chose to eat more of the feed containing carprofen and the amount of carprofen consumed increased with the severity of the lameness. The authors concluded that their results support the view that: "lame broiler chickens are in pain and that this pain causes them distress from which they seek relief".

In their 2000 report the SCAHAW concluded that "there is evidence that birds with score 3 or higher experience pain or discomfort" (p.36).

The SCAHAW report (p.35) stated that "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. This is likely to be the case in TD ... However, regardless of the inherent painfulness of lesions, if growth plate or other conditions result in a deformity causing pronounced lameness, this is likely to produce abnormal strains on tendons and muscles leading to inflammation in these structures and consequent pain.”

### **The industry’s selection policies**

The SCAHAW report concluded that broilers are “mostly selected for growth rate and food conversion ratio. Other traits such as low frequency of leg disorders or resistance to pathogens are likely to be also included in the selection index by most breeders but the importance given to such traits is often low and **up to now has not improved welfare**” (Conclusion 3; p.104) (my emphasis).

### **Incidence of leg problems is likely to increase**

CIWF fears that, in the absence of legislation to prohibit the use of fast-growing broiler genotypes, the incidence of leg problems is likely to substantially increase.

An article in *World Poultry* in 2000 stated that liveweight at 42 days is projected to increase by a further 575g. (24%) to the year 2005 (van der Sluis, 2000). McKay et al (2000) stated that liveweight at 42 days is projected to reach 3 kg. by the year 2007 (as opposed to 2.6 kg. at the time of the paper).

Such increased growth rates could well impose further stress on broilers’ legs and lead to an increase in the proportion of broilers with gait scores of 3 and above. Indeed, when Hardiman (1996) indicated that breeders predicted an increase in growth rate of 600g. to 40 days of age over the next ten years, Kestin et al (1999) predicted that an increase in growth rate of this size would lead to a deterioration in walking ability of approximately 1.1 units of gait score, assuming that no active selection to improve walking ability is undertaken.

The industry asserts that it is taking leg health seriously in its selection policies. This claim is undermined by the fact that the industry is continuing to select for faster growth rates despite widespread agreement that these are a major contributor to leg problems. Indeed, as indicated earlier, in its report the SCAHAW concluded that breeders often give low importance in their selection index to reducing the incidence of leg problems and that up to now their attempts have “not improved welfare”.

## **Muscle abnormalities**

In their report, the SCAHAW stated (p.37) that “Focal and stress induced myopathies are also seen in poultry and may have a strong genetic component, with elevated levels of muscle damage being associated with genetic selection for high growth rate.” The SCAHAW added (p.37) “There are also greater signs of muscle damage in modern lines selected for rapid growth than in slower growing lines. Moreover, faster growing birds show signs of more severe muscle damage when exposed to acute heat stress ... The evidence thus suggests that genetic selection for production traits predisposes birds to spontaneous or stress induced muscle damage ....”

## **Mortality**

Crucially, the SCAHAW pointed out (p.27) that all these problems (referring to cardiac and leg problems) induce a weekly mortality in fast-growing broilers that is 7 times higher than in pullets (egg-laying hens) and 4 times higher than in slow growing meat type birds, such as “Label rouge” strains. The SCAHAW stated (p.24) that mortality in standard broilers is 1% per week, whereas it is just 0.25% per week in “Label rouge” chickens and 0.14% per week in pullets. In their Conclusion 5 (p.104), the SCAHAW stated that “Mortality in older birds is often related to metabolic disorders caused by rapid growth.”

## **Other welfare implications of leg disorders**

No only do birds affected by musculoskeletal disorders suffer pain or discomfort, they also experience other consequences of impaired locomotion. As the SCAHAW pointed out (p.35) “welfare is obviously compromised in birds that are unable to reach food and water and die from starvation and dehydration.”

Broiler chickens are in general very inactive. As will be seen, this inactivity leads to

- ?? a higher level of leg problems; the lack of exercise/activity partly caused by leg problems in turn exacerbates the incidence of leg problems.
- ?? breast blisters, hock burns and foot pad burns due to prolonged periods sitting or lying on the litter, which in some cases is wet.

In some cases, the birds’ degree of lameness will discourage activity. However, it is likely that inactivity also partly results from their selective breeding for rapid growth and feed conversion and their heavy weight which leave them no spare energy for exercise.

Broilers' inactivity will be compounded by the crowded conditions in the broiler sheds, giving them too little room to move around easily. Whatever the reason, there is a striking difference between the activity level of standard broilers and other chickens.

Several studies have shown that broilers do less walking/running or pecking/scratching of litter than laying hens and that they spend more time sitting/resting as they get older. Unlike laying hens, they do very little dust-bathing, wing-flapping and wing-stretching. Over the whole growing period they spend over 75% of their time sitting or resting compared to less than 30% spent sitting or resting by laying hens of the same age. This inactivity increases broilers' susceptibility to leg weakness and to hock burns or breast blisters (these are forms of contact dermatitis) from prolonged sitting or lying in their litter material (the floor of broiler sheds is covered with litter, often woodshavings). (This paragraph is largely drawn from p.10 of the SCAHAW report).

Skin diseases such as those referred to above are usually characterised by conditions or lesions of the skin on the breast, hocks or feet of the birds. The lesions are commonly referred to as breast blisters, hock burns and foot pad burns respectively. They are thought to be caused by a combination of wet litter and various chemical factors in the litter. Inactive birds which spend prolonged periods with their shanks or breasts in contact with wet litter are more susceptible to developing breast blisters, hock burns or foot-pad dermatitis. As well as being painful, these lesions can, said the SCAHAW report (p.38) "be a gateway for bacteria, which may spread through the blood stream and cause joint inflammations."

A recent study by Weeks et al (2000) found that between 39 and 49 days of age, non-lame broilers averaged 76% of their time lying and this increased significantly to 86% in lame birds (GS 3). Although non-lame broilers spent only a minor part of the day on their feet, they spent significantly more time standing idle (7%), standing preening (3.5%) and standing eating (4.7%) than lame birds.

Walking declined with age, but occupied an average 3.3% of the time of a slaughter-weight broiler. Lameness, however, significantly reduced this to a minimal 1.5% in the worst affected birds.

Non-lame birds predominantly chose the usual standing posture for eating, whereas lame birds lay down to eat for almost half their feeding time.

Lameness also altered the feeding strategy of broilers. Whereas non-lame birds fed over 50 times in 24 hours, the number of visits to the feeder was reduced with increasing lameness to an average of around 30 in the lamest broilers. However, meal duration was adjusted to give no overall differences in time spent feeding per

day. The authors stated “The behaviour of broilers in our studies is indicative of a cost to the lame bird in getting up and walking over to the feeder” (p.123).

The authors pointed out that the level of time spent ground-pecking by broilers of under 3% is dramatically less than the 60% seen in their genetic ancestors, Red Jungle fowl.

In considering the causes of broilers’ inactivity, the authors stated (ps.121-2) “The work reported here gives evidence that lameness, with its associated disability and probable pain, significantly increases the time spent lying. Selection for high breast meat yield has moved broilers’ centre of gravity forward, which may unbalance them and predispose them to sitting behaviours. It is likely that sternal recumbency (here termed lying) is the most comfortable, and least physically demanding, posture for a bird with leg problems”.

The authors concluded that “Overall, these results characterise broilers as an extremely inactive strain of chicken, quite distinct from their Jungle fowl ancestors” (p.123). They also concluded that “Lameness significantly changes the time budgets of many behaviours and dramatically alters feeding strategy” (p.123).

The authors also stressed (p.111) 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”. They concluded that “The selection pressures that have resulted in altered time budgets and apparently abnormal behaviours are continuing and may result in further health and behavioural changes to the detriment of broiler chicken welfare” (p.123).

## **HEART PROBLEMS**

It is not only broilers’ legs which fail to keep pace with the rapidly growing body, but also their heart and lungs. In their report, the SCAHAW stated (ps.9 and 10) that “Stocks in which rapid growth is combined with low FCR [food conversion ratio] typically show an increased disposition to low thyroid hormone concentrations, low metabolic rate, hypertrophy [overgrowth] of the right ventricle of the heart and ascites. These pathologies can be attributed to an insufficient oxygen supply in metabolism, **due to genetically (and environmentally) induced mismatches between energy-supplying and energy-consuming organs.**” (My emphasis).

Broilers suffer from two forms of heart failure: ascites and sudden death syndrome (SDS). One study quoted by the SCAHAW (p.27) states that “a primary reason for the increased incidence in ascites is the focus in selection on growth, weight and feed conversion, which has led to some neglect of the maintenance needs of the birds”.

Ascites and SDS are relatively common and are likely to be due to the fact that the broilers' fast growth requires high levels of oxygen to support metabolic demands. All their energy is spent on growth and efficient feed conversion, leaving them short of oxygen for their other bodily needs so that their hearts have to work much harder. As indicated earlier, the SCAHAW stated that broilers selectively bred and managed for very fast growth have a genetically induced mismatch between their energy-supplying organs and their energy-consuming organs. The SCAHAW concluded that:

“Fast growth rates increase the risk of ascites and SDS by increased oxygen demand of the broilers, which intensifies the activity of the cardio-pulmonary system. Since growth rate and oxygen demand coincides with other physiological challenges in the young chick (e.g. change in the thermoregulation) this may lead to failure of cardiac function” (Conclusion 10, p.105).

In a recent Communication from the European Commission to the EU Council and the European Parliament, the Commission stated (para. 32 on p.9) “There are currently various scientific initiatives focussing on animal welfare as an active constituent of animal health or, more accurately, on extreme farming conditions (associated with more intensive production) as a source of animal illness. One of the most extreme cases is that of poultry: the faster growth of broiler chickens means a high metabolic rate and higher oxygen requirement, seemingly in excess of the birds' respiratory and circulatory capacity. The result is increased mortality from ascites (fluid in the abdomen) and other related problems” (European Commission, 2002).

The incidence of mortality from ascites was reported by UK broiler producers to be 1.4% in 1993 (Maxwell and Robertson, 1998). UK broiler producers reported an incidence of SDS of 0.8% in 1993 (Maxwell and Robertson, 1998). This means that around 2% of UK broilers die of heart failure, which amounts to over 16 million birds a year.

### Ascites

Ascites affects fast growing chickens when the right side of their hearts becomes enlarged in response to increased workload during the bird's rapid growth. The bird has to breathe more rapidly and its lungs become congested. The liver function is affected, the abdomen becomes swollen with fluid increasing the risk of heart failure.

The SCAHAW stated (Conclusion 9, p. 105) that ascites “has a serious negative effect on broiler welfare. The problem has increased in recent years.” One clinical study

has shown that “birds affected by ascites are severely distressed. In advanced stages the birds are unable to reach the drinkers and become dehydrated. Death is usually preceded by prolonged agony and is likely a result of dehydration, starvation, respiratory failure and heart failure” (Olkowski and Classen, 1998).

Ascites is one of the major causes of death in broilers; an estimated 4.7% of broilers worldwide have this disease (p.40 of SCAHAW report).

Fast growth rates are a major factor in the causation of ascites. The SCAHAW report stated that “There are direct correlations between high growth rate, hypoxia and ascites” (Conclusion 9, p.105). As indicated above, they also stated in Conclusion 10 that “fast growth rates increase the risk of ascites”.

### Sudden death syndrome

SDS is an acute heart failure disease that affects mainly male fast-growing chickens which seem to be in good condition. The SCAHAW stated that SDS “is characterised by sudden vigorous wing-flapping, muscle contractions and obvious loss of balance. Vocalisation can be heard in some cases. In the final phase the birds fall on their back or to the side, and die. The duration from the first unrest until death ... [ranges] from 37 to 69 seconds” (p.41). The SCAHAW concluded that “Even though the apparent time from onset of the syndrome until death occurs is only a matter of minutes, it may still have an important impact on bird welfare” (Conclusion 10, p. 105).

The SCAHAW stated (p.41) that SDS is a major cause of mortality in broilers, and in Europe its reported incidence is between 0.1 and 3%.

The authors of a recent paper on cardiovascular disease in poultry stated “... this phenomenal growth rate, as a consequence of continued improvements to intensive genetic selection and farming practices over the past four decades, has only been achieved at the expense of many casualties along the way. Considerable shortcomings in the adaptive metabolic status of the species have endangered its ability to survive. (My underlining). (Maxwell and Robertson, 2000).

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### REFERENCES:

Berg C. and Sanotra G.S., 2001. A survey of the prevalence of leg weakness in Swedish broiler chickens – A pilot study. Svensk-Veterinertidning 53 (1): 5-13.



Danbury T.C., Chambers J.P., Weeks C.A., Waterman A.E. and Kestin S.C., 1997. Self selection of analgesics by broiler chickens. *Animal Choices*. Occasional Publication No. 20. Eds. Forbes J.M., Lawrence T.L., Rodway R.G. and Varley M.A. British Society of Animal Science.

Danbury T.C., Weeks C.A., Chambers J.P., Waterman-Pearson A.E. and Kestin S.C., 2000. Self selection of the analgesic drug carprofen by lame broiler chickens. *Veterinary Record* 146: 307-311.

European Commission, 2002. Communication from the Commission to the Council and the European Parliament on Animal Welfare Legislation on farmed animals in Third Countries and the Implications for the EU. Brussels, 18.11.2002. COM (2002) 626 final.

FAWC, 1992. Report on the Welfare of Broiler Chickens. Farm Animal Welfare Council.

Hardiman, J.W., 1996. Broiler breeding by the year 2006. Ps.461-467 in: Proceedings of the XX World's Poultry Congress. Vol. 1. New Delhi, India.

Kestin S.C., Knowles T.G., Tinch A.E. and Gregory N.G., 1992. Prevalence of leg weakness in broiler chickens and its relationship with genotype. *Veterinary Record* 131: 190-194.

Kestin, S.C., Su, G. and Sørensen, P., 1999. Different commercial broiler crosses have different susceptibilities to leg weakness. *Poultry Science* 78: 1085-1090.

Kestin S.C., Gordon S., Su G. and Sørensen P., 2001. Relationships in broiler chickens between lameness, liveweight, growth rate and age. *Veterinary Record* 148: 195-197.

McGeown D., Danbury T.C., Waterman-Pearson A.E. and Kestin S.C., 1999. Effect of carprofen on lameness in broiler chickens. *Veterinary Record* 144: 668-671.

Maxwell M. and Robertson G., 1998. UK survey of broiler ascites and sudden death syndrome in 1993. *British Poultry Science* 39: 203-215.

Maxwell M. and Robertson G., 2000. Cardiovascular disease in poultry: epidemiology – current trends and correlates. Proceedings of the XXI World's Poultry Congress, Montreal, Canada.

McKay J.C., Barton N.F., Koerhuis A.N.M. and McAdam J., 2000. The challenge of genetic change in the broiler chicken. British Society of Animal Science. Occasional Publication; No. 27.

Olkowski A.A. and Classen H.L., 1998. Ascites in broiler chickens from a welfare point of view. Poultry Science, 77: 1842. Abstracts of poster presentations from the Poultry Welfare Symposium.

Sanotra G.S., Berg C. and Lund J.D., 2002. A comparison between leg problems in Danish and Swedish broiler production. Unpublished poster presentation at Assessment of Animal Welfare at Farm and Group Level, 2nd International Workshop. School of Veterinary Science, Bristol University. 4-6 September 2002.

Sanotra G.S., 1999. Registrering af aktuel benstyrke hos slagtekyllinger. (Velfaerdsmoniteringsprojekt). Pub: Dyrenes Beskyttelse.

SCAHAW, 2000. The welfare of chickens kept for meat production (broilers). Report of the Scientific Committee on Animal Health and Animal Welfare. Adopted 21 March 2000.

Sørensen, P., Su, G. and Kestin, S.C., 1999. The effect of photoperiod : scotoperiod on leg weakness in broiler chickens. Poultry Science 78: 336-342.

Su, G., Sørensen, P. and Kestin, S.C., 1999. Meal feeding is more effective than early feed restriction at reducing the prevalence of leg weakness in broiler chickens. Poultry Science 78: 949-955.

van der Sluis W., 2000. New challenges in broiler breeding. World Poultry-Elsevier Vol. 16, No. 8.

Weeks C.A. and Kestin S.C., 1997. Effect of leg weakness on the behaviour of broilers. Proceedings of the 5th Poultry Welfare Symposium, Wageningen. The Netherlands. Eds: Koene P. and Blokhuis H.J. p.117.

Weeks C.A., Danbury T.D., Davies H.C., Hunt P. and Kestin S.C., 2000. The behaviour of broiler chickens and its modification by lameness. Applied Animal Behaviour Science 67: 111-125.