SHORT COMMUNICATION

DEHYDRATION AND LAMENESS IN A BROILER FLOCK

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Abstract

Animal Welfare 2002, 11: 89-94

A case study is described in which a high incidence of lameness in broiler chickens (Gallus gallus domesticus) is associated with rearing to 84 days of age; broilers are normally slaughtered at less than 50 days of age. A sample of 185 birds was scored for walking ability, and 24 birds from this group were assessed for dehydration by measuring plasma osmolality. The proportion of the group having gait abnormalities was compared with that recorded during a recent survey of Danish flocks and was found to be higher. A relationship is established between the degree of gait impairment and the level of dehydration. The degree of dehydration is related to the findings for plasma osmolality from previous research, and the results indicate that some of the birds in this case may have been deprived of access to water for periods in excess of 60 hours.

Keywords: animal welfare, broiler chicken, leg weakness, lameness, dehydration, slaughter age

Introduction

In the UK, broiler chickens are normally slaughtered at about six weeks of age, when they weigh approximately 2.2 kg. In contrast, an 'unimproved' traditional strain of bird, such as a White Sussex, weighs about 800 g at the same age. The rapid growth of broilers has been achieved through careful genetic selection and the development of nutritional programmes.

Lameness, characterised by an abnormal gait and impaired walking ability, is prevalent in these rapidly growing birds (Sorensen 1992) and has been highlighted as a major welfare concern (European Commission 2000; FAWC 1992). The principal causes of broiler lameness can be divided into two main categories: those of infectious origin (Butterworth 1999), which cause small numbers of birds to become profoundly lame; and those that are caused by skeletal abnormalities (Williams *et al* 2000), which cause larger numbers of birds to become moderately disabled. Recent studies have indicated a strong genetic influence on the incidence of lameness in broilers (Kestin *et al* 1999).

The incidence of lameness can be minimised through attention to husbandry and management (Hester 1994; Sorensen *et al* 2000). Under certain circumstances, however, lameness and associated skeletal deformities may affect a significant proportion of the birds, constituting a serious welfare problem. This case study outlines such a circumstance.

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Case study

The authors were asked by the State Veterinary Service to attend a broiler rearing unit where a flock of 18,000 Cobb broilers had been reared to 84 days of age in two houses, the majority of birds having a live weight of over 4 kg. The flock had been stocked with 24,000 chicks at the start of production, but a combination of thinning, culling, and death of birds had reduced the number, and the stocking density at the time of the visit was calculated to be 36 kg m⁻². On the day of the study described, the ambient temperature peaked at 24°C and the house temperature and relative humidity averaged 28°C and 78 per cent, respectively. The birds had originally been intended for marketing at 42 days but the producer had been unable to find a market for them. The birds were conventionally reared on chopped straw litter, with commercially compounded feed available *ad libitum* from chain feeders and water from bell drinkers. On inspection, the environmental conditions in the houses appeared consistent with normal broiler house conditions, with friable litter and acceptable air quality.

Large numbers of birds were observed to be disabled by lameness. The walking ability of 185 birds randomly selected from the two houses was assessed using a gait scoring method (Kestin *et al* 1992). In this scoring system, a bird with a score of 0 is normal, whilst a bird with a gait score of 5 is unable to walk. Scores of 1 to 4 are subjective scores indicating incremental stages in the 'worsening' of walking ability.

Many of the lame birds appeared unable to reach the bell drinkers (400 mm from the litter) and drank avidly when the drinkers were lowered. To determine the extent of dehydration, blood samples were taken from a sample of 18 high-gait-score birds (gait scores 4 and 5) and six medium-gait-score birds (gait scores 2 and 3) picked from the population at random. It was not possible to find any birds that did not have some gait impairment. The 24 sample birds were humanely euthanased, the appendicular skeleton aseptically dissected and bacteriological swabs taken from bone and cartilage of the proximal femur, proximal tibia and tibiotarsus using an established protocol (McNamee *et al* 2000). After aseptic sampling, the birds were subjected to a *post mortem* examination. Plasma osmolality was determined using an automated micro-osmometer (Camlab Micro-Osmometer, Cambridge, UK) and an independent-samples Student's *t*-test carried out in order to compare the mean values.

Results

There was no difference in the mean gait score between the two houses; therefore, the results were combined. The distribution of walking abilities in the flock as a whole is shown in Figure 1. The average flock gait score was 3.8, with a median of 4.0. For comparative purposes, the average distribution of gait scores from a large study of Danish commercial flocks assessed at normal slaughter age (Singh-Sanotra 1999) is also given in Figure 1.

The average plasma osmolality of the high-gait-score group was 375 mOsmol kg⁻¹ (standard error of mean = 8.67), and that of the medium-gait-score group was 339 mOsmol kg⁻¹ (standard error of mean = 4.39). The independent-samples *t*-test for these two groups was significantly different (P = 0.005). The mean values are plotted onto the line x-y in Figure 2. This line, defined by the equation y = 312.36 + 0.479x, $r^2 = 0.794$, has been derived by fitting a best fit line to the data provided by four studies on dehydration in broilers (Arad *et al* 1985; Knowles *et al* 1994; Robinson *et al* 1990; Stallone & Braun 1986). The value of plasma osmolality for the high-gait-score birds in this case study exceeds the values seen in previous experimental work; the line has, therefore, been extrapolated beyond x-y so that the value of plasma osmolality for the high-gait-score group can be used to predict the

length of time of water deprivation. Assuming a linear relationship between time and progression of dehydration (the relationship is not known for the effects of prolonged dehydration), it would appear from these data that the birds in the high-gait-score group may have been unable to drink for more than 100 hours.

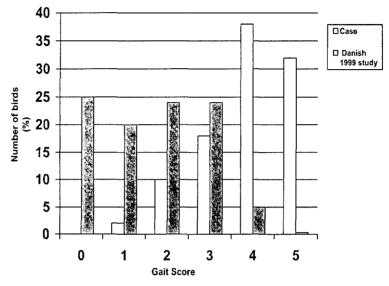
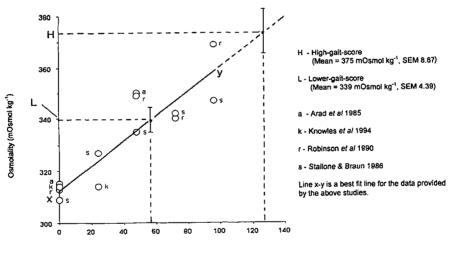


Figure 1 A comparison of gait score frequency seen in this case study and in a study of Danish flocks at normal slaughter age (Singh-Sanotra 1999).



Time of water deprivation (h)

Figure 2 The effect of length of time of dehydration on plasma osmolality in domestic fowl. A best fit line x-y has been drawn for the data available from previous work (Arad *et al* 1985; Knowles *et al* 1994; Robinson *et al* 1990; Stallone & Braun 1986). Mean plasma osmolality values for high-gait-score (H) and lower-gait-score (L) birds in this case study are also indicated, as are error bars for the standard error of the mean.

Of the 24 birds examined *post mortem*, isolations of *Staphylococcus aureus* indicative of osteomyelitis were made from two birds in the high-gait-score group. Hock burn of moderate severity was also recorded in two birds from the high-gait-score group. No gross pathologies were revealed during the *post mortem* to suggest that flock health was compromised by generalised disease.

Discussion

The birds examined in this case study had been managed according to accepted broiler practice, but they had been grown for several weeks longer than is normal for this genotype; the effect of growing the birds for longer was that they were substantially heavier than the usual marketing weight for broilers (COBB 1995). The prevalence of lameness in this flock was much higher than has been reported in previous studies (Singh-Sanotra 1999; Singh-Sanotra *et al* 2001; Sorensen *et al* 1999; Su *et al* 1999). The range of pathologies identified during the *post mortems* in the birds examined was typical of commercial broiler flocks (Pattison 1992), but the lameness profile was not typical. More than 65 per cent of the flock had a gait score of 4 or 5, meaning that a large proportion of the flock was severely compromised in the ability to walk.

It is interesting to consider how such a serious welfare incident could have developed. The husbandry methods and environmental conditions conformed to accepted agricultural practice, and the birds were not suffering from a flock-wide infectious condition that could account for this level of lameness. Although the calculated stocking density of 36 kg m^{-2} slightly exceeds MAFF recommendations of 34 kg m⁻² (MAFF 1987), commercial experience shows that many producers routinely grow birds at 40 kg m⁻² for reasons of economic expediency. However, the birds were much heavier than is normal and there is an established relationship between lameness and body weight. The correlation between live weight and lameness has been reported to be as high as 0.5 in a study covering a wide range of genotypes grown on several planes of nutrition (Kestin et al 2001). In other studies in which more restricted genotypes have been examined, the correlation between live weight and lameness has been reported to be 0.35-0.43 (Kestin et al 1999). These studies made no attempt to separate phenotypic and genetic influences. Other studies calculate the genetic correlation between live weight and specific leg disorders thought to contribute to lameness to be 0.25 (Sorensen 1992). The abnormally high body weight in the broilers in this study is thus likely to have been the major factor contributing to the lameness seen in this flock.

Serum osmolality can be affected by a number of factors, including the disease status of the animal. In this case, two of the high-gait-score birds were identified to be suffering from *S. aureus* osteomyelitis; however, no *post mortem* pathology that could explain the level of dehydration was found in the remaining birds. Comparing the data for osmolality with previous studies (Figure 2), the implication is that some of the high-gait-score birds from this case study may have been unable to drink for several days. It is not known how long broilers can survive without water, but dehydration could have been responsible for much of the daily mortality in this flock, reported by the stockman to be approximately five per cent per day.

The birds on this farm had been managed according to established broiler rearing practice. In particular, they had been grown at a stocking density typical of commercial practice, under normal lighting patterns, and had been fed commercial broiler rations. However, by growing the birds beyond their normal marketing weight, serious lameness had developed, resulting in distress, dehydration and mortality. It is possible to limit expression of lameness in broilers grown to increased weight or age by restricting their food intake through meal feeding

(Su *et al* 1999), or by use of specialised lighting programmes (Sorensen *et al* 1999) or lowerdensity stocking (Sorensen *et al* 2000). However, all of these husbandry manipulations must be initiated during the early stages of the birds' growth if they are to be effective.

Animal welfare implications

The findings of this case study support the hypothesis that the rapid growth of modern highly selected broiler genotypes, if unchecked by modified husbandry practices, can outstrip the ability of the developing skeleton to support the animal. In commercial flocks, birds are slaughtered just before this growth imbalance starts to affect productivity, although welfare may already be affected (Singh-Sanotra 1999; see also Singh-Sanotra *et al* 2001). If the birds are allowed to grow for a comparatively short period beyond normal slaughter age — as in this case — the imbalance becomes dominant and can seriously impinge on productivity and welfare.

Because of the poor welfare of the birds in this case, the attending State Veterinary Officers, after discussion with the stockman, requested that birds with a gait score of 4 or 5 (10,000 birds) be destroyed on-farm. The owner of these birds was subsequently prosecuted by MAFF legal branch for causing unnecessary pain and unnecessary distress under the Agriculture (Miscellaneous Provisions) Act 1968.

Acknowledgements

The authors would like to acknowledge Dr Toby Knowles, Mr Andy Phillips and the staff at the Veterinary Laboratories Agency, Langford, for their assistance in this study.

References

- Arad Z, Arnason S S, Chadwick A and Skadhauge E 1985 Osmotic responses to heat and dehydration in the fowl. Journal of Comparative Physiology 155: 227-234
- Butterworth A 1999 Infectious components of broiler lameness: a review. World's Poultry Science Journal 55: 327-352
- COBB 1995 COBB 500 Broiler Management Guide: East Hanningfield, Chelmsford, Essex CM3 8BY, UK
- **European Commission** 2000 The welfare of chickens kept for meat production (broilers). Report of the Scientific Committee on animal health and animal welfare. European Commission Report B3, R15, 2000. Produced by Unit B3, Directorate B of the European Commission
- Farm Animal Welfare Council (FAWC) 1992 Report on the welfare of broiler chickens. FAWC Secretariat: 5th Floor, 1a Page Street, London SW1P 4PQ, UK
- Hester P Y 1994 The role of environment and management on leg abnormalities in meat-type fowl. *Poultry* Science 73: 904-915
- Kestin S C, Adams S J M and Gregory N G 1992 Leg weakness in broiler chickens: a review of studies using gait scoring. Proceedings of the 9th European Poultry Conference, Glasgow UK, Vol II: 203-206
- Kestin S C, Gordon S, Su G and Sorensen P 2001 Relationship in broiler chickens between lameness, liveweight, growth rate and age. *Veterinary Record 148*: 195-197
- Kestin S C, Su G and Sorensen P 1999 Different commercial broiler crosses have different susceptibilities to leg weakness. *Poultry Science* 78: 1085-1090
- Knowles T G, Warriss P D, Brown S N, Edwards J E and Mitchell M A 1994 Responses of broilers to deprivation of food and water for 24 hours. *British Veterinary Journal 151*: 197-202
- MAFF 1987 Codes of recommendations for the welfare of livestock domestic fowls. PB0076, DEFRA Publications: Admail 6000, London SW1A 2XX, UK
- McNamee P T, King D C, Spratt-Davidson S, Ball H and Smyth J 2000 Guidelines for the investigation of lameness in commercial broiler fowl. Irish Veterinary Journal 53(4): 191-194

- Pattison M 1992 Impacts of bone problems on the poultry meat industry. In: Whitehead C C (ed) Bone Biology and Skeletal Disorders in Poultry pp 329-338. Carfax: Oxford, UK
- Robinson B, Koike T I, Kinzler S L and Nekdon H L 1990 Arginine vasotocin and mesotocin in the anterior hypothalamus, neurohypophysis, proventriculus and plasma of leghorn cockerels during dehydration. *British Poultry Science 31*: 651-659
- Singh-Sanotra G S 1999 Registrering af aktuel benstyrke hos slatekyllinger (Velfaerdsmoniteringsprojekt). Dyrenes Beskyttelse, Alhambravej 15, 1826 Frederiksberg C, Denmark [Title translation: Recording of broiler leg problems seen at slaughter age]
- Singh-Sanotra G S, Lund J D, Ersboll A K, Petersen J S and Vestergaard K S 2001 Monitoring leg problems in broilers: a survey of commercial broiler production in Denmark. *World's Poultry Science Journal 57*: 55-69
- Sorensen P 1992 The genetics of leg disorders. In: Whitehead C C (ed) Bone Biology and Skeletal Disorders in Poultry pp 213-229. Carfax: Oxford, UK
- Sorensen P, Su G and Kestin S C 1999 The effect of photoperiod: scotoperiod on leg weakness in broiler chickens. *Poultry Science* 78: 336-342
- Sorensen P, Su G and Kestin S C 2000 Effects of age and stocking density on leg weakness in broiler chickens. *Poultry Science* 79: 864-870
- Stallone J N and Braun E J 1986 Regulation of plasma arginine vasotocin in conscious water-deprived domestic fowl. *American Journal of Physiology 250*: 644-685
- Su G, Sorensen 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
- Williams B, Solomon S, Waddington D, Thorp B and Farquharson C 2000 Skeletal development in the meat-type chicken. *British Poultry Science 41*: 141-149