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Peter Stevenson  
Claimant  
First  
PS 1 & 2  
August 2003

**IN THE HIGH COURT OF JUSTICE**  
**QUEEN'S BENCH DIVISION**  
**ADMINISTRATIVE COURT**

**Claim No:CO/1779/2003**

**B E T W E E N :**

**COMPASSION IN  
WORLD FARMING LIMITED**

Claimant

**and**

**THE SECRETARY OF STATE FOR THE  
ENVIRONMENT, FOOD AND RURAL AFFAIRS**

Defendant

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**STATEMENT OF PETER STEVENSON**

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I, PETER STEVENSON, the Political and Legal Director of the Claimant **WILL SAY** as follows:

1. I make this statement in response to the Witness Statement of David Pritchard. It would not be appropriate to place large amounts of scientific evidence before the Court in a claim for judicial review; however, Compassion in World Farming (CIWF) considers certain aspects of that Witness Statement cannot be left uncorrected.
2. In paragraph 14 of his Witness Statement, David Pritchard states that the papers and quotes cited by CIWF do not provide a balanced view of the underlying science. In paragraph 18, he adds that the information presented is based on selective and unobjective use of the available literature. I dispute both those statements. I consider that the scientific material justifies the key points made by CIWF in its Claim Form and the two papers annexed to that Form. CIWF asserts that there is a large and

growing body of scientific evidence on the health problems which are prevalent among fast-growing broiler genotypes, which supports the following propositions:

- a) the very substantial increase in broiler growth rates over the last around 25-40 years is mainly due to the use of fast-growing genotypes;
  - b) a substantial number of UK broilers suffer from leg disorders;
  - c) the principal cause of most leg disorders is the use of fast-growing genotypes;
  - d) most leg disorders are painful;
  - e) rapid growth rates lead to increased mortality from two forms of heart failure:
    - f) ascites and sudden-death-syndrome;
    - g) broiler breeders are subjected to very restricted feeding regimes which result in chronic hunger.
3. Mr Pritchard's statement largely ignores the evidence which CIWF has deployed. However, at no point does he suggest that the studies relied upon are flawed, have been disproved or are otherwise open to doubt. That evidence therefore stands. It is CIWF's case that unless it can be substantially dismissed, the evidence strongly suggests that it cannot reasonably be expected that fast-growing genotypes of broiler can be kept without detrimental effect on their health or welfare. In the following paragraphs of this statement, I seek to demonstrate that the material relied on by Mr Pritchard does not undermine the evidence presented by CIWF.
4. In order to illustrate the matters to which I refer in this statement, I exhibit ("PS 1") six photographs. Photographs 1 – 3 illustrate the environment in which broiler chickens are usually kept. Numbers 4 – 6 are pictures of broiler chickens with leg disorders.

### **Role of genotype in causing rapid growth rates**

5. In paragraph 19 of his Witness Statement, Mr Pritchard accepts that there has been a significant increase in broiler growth rates over the last 30 years and that this is "mainly" due to selective breeding for improved growth rate. While he argues that other

factors have played major roles, there is no doubt that genetic selection has been by far and away the main factor in this substantial increase in growth rates.

6. The importance attached by the breeding companies to genetic selection for achieving high growth rates can be seen from an article in the July 2003 edition of 'Poultry World' about Cobb, an international broiler breeding company. The article states that Cobb 500 breeding stock from the USA is being increasingly used in the UK and that the accent with these birds is very much on maximising meat yield. The article adds that the Cobb 500 shows increases in bodyweight of 45-50 g. a year, equivalent to reaching market weight one day earlier each year. Tables accompanying the article show daily liveweight gain for the Cobb 500 of 61.66 g. compared with 59.02 g. for an unnamed competitor, and a weight at 41 days of age of 2.528 kg. compared with 2.420 kg. for an unnamed competitor.
7. The key role of genotype in causing rapid growth is highlighted by Havenstein et al (1994) who compared a 1991 broiler strain with a 1957 strain. They found, in the 1991 strain, a 420% increase in bodyweight at 42 days of age, about 85-90% of which was attributable to genetic selection.
8. The importance of genetic selection in achieving rapid growth is also stressed by Savory, 2002. He extracted data from a series of management manuals from a leading broiler breeding company dating from 1972 to 1999. Savory reports that, judging from these, the time taken to reach 2 kg. has declined from 57 to 37 days and that body weight gain per day has increased from 34 to 53 g., which represents a 54% increase. Savory writes that "some of this change reflects improved nutrition and management but **most is due to genetic selection**" (my emphasis).
9. The role of genotype in causing rapid growth is also highlighted by Corr et al (2003a) who compared two strains of broilers, selected and 'relaxed' (i.e. not selected). The authors found that the selected birds grew to 2.4 kg. in approximately half of the time taken by the non-selected birds.

### **Validity of gait-scoring system in assessing prevalence of lameness**

10. In paragraph 21, Mr Pritchard accurately states that CIWF places much reliance on the gait-scoring system as evidence of lameness. CIWF is justified in so doing. In a paper published in 2002 in British Poultry Science, Garner et al stated that the gait-scoring system has enjoyed widespread popularity and has been

widely used to evaluate leg problems in both industry and research. The authors pointed out that the gait-scoring system has been validated in a number of studies and that the many factors and measures associated with this scale have empirically established its external (biological) validity.

11. Garner et al point out that birds with high gait scores also rate highly as regards a number of other factors which are indicative of lameness. The authors cite a range of scientific papers which show that high gait score birds show a higher rate of self-selection of analgesics in their feed than sound birds; take longer to reach food and traverse obstacles; have a higher incidence of hock burn; and tend to perform behaviours such as feeding while sitting where possible. The fact that the gait-scoring system relates consistently to other biologically important variables and behavioural differences associated with lameness demonstrates its validity as an evaluation of the incidence of lameness.
12. Moreover, subjective methods of assessing lameness are widely used in veterinary practice for many other animals, for example, cattle, dogs, horses and pigs.

### **Use of fast-growing genotypes is one of main causes of lameness**

13. Mr Pritchard states in paragraphs 24 - 28 that lameness is multi-factorial in its causation. CIWF accepts that husbandry, management and nutritional factors play a part in causing broiler lameness. However, there is now a very substantial body of evidence to show that by far the most important factors are the use of fast-growing genotypes and infections. Mr Pritchard appears to ignore, or at least does not refer to, this body of evidence.
14. The gait scores (GSs) which are likely to cause chronic pain are GS 3, 4 & 5 (Kestin et al, 1992 and SCAHAW, 2000). GSs 4 & 5 reflect the most severe categories of lameness. Substantially fewer birds have GSs 4 & 5 than have GS 3. Kestin et al (1994) reviewed the main causes of lameness in birds with different degrees of lameness taken from 10 commercial flocks. Infections in joints and bones were found only in birds with a GS of 4 and 5. No birds with a GS of 3 or lower had infections. In birds that did not suffer from infections in bones and joints (i.e. the majority of lame birds), skeletal abnormalities closely linked to live weight was found to be the main cause of lameness.

15. I have recently discussed the principal causes of GSs 3, 4 & 5 with Dr. S.C. Kestin. His estimate is that, whilst the pattern of causes of lameness varies from flock to flock, currently:

?? most birds with a GS of 3 are lame because they have skeletal abnormalities primarily caused by rapid growth;

?? around 70% of GSs 4 are lame because they have skeletal abnormalities primarily caused by rapid growth, with the remainder being mainly caused by infections;

?? around 70% of GSs 5 are primarily caused by infections, with the remainder being mainly caused by rapid growth.

Dr Kestin also considered that lameness caused by poor nutrition such as rickets was a rare event.

16. The scientific evidence indicates that other factors such as feeding programmes and lighting programmes have a substantially lesser impact in reducing the prevalence of lameness than genotype (Sørensen et al, 1999; Su et al, 1999; Kestin et al, 1999; Kestin et al, 2001). In other words, changing the genotype being used is much more effective in reducing the incidence of lameness than changing various management and husbandry factors.

17. Changing factors such as feeding and lighting programmes can properly be seen as ways of alleviating the high incidence of leg disorders resulting from the use of fast-growing genotypes rather than as primary factors involved in the causation of leg disorders. Modifying such management factors is much less effective in reducing the prevalence of leg disorders than using slower-growing genotypes.

18. This thinking can be seen in Bradshaw et al (2002) who stated "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".

19. Sanotra et al (2001) found 30.1% of the Danish broilers that they surveyed to have GSs 3, 4 or 5 whereas Sanotra & Berg (2003) reported that in flocks investigated in Sweden, 20.4% had GSs 3, 4 or 5. The mean GS in the Danish study was 1.656, while that in the Swedish study was 1.426, a difference of 0.230 GS units.

20. The latter authors commented that the better leg health results in Sweden appear to be due to relatively low stocking density (34 – 36 kg/m<sup>2</sup>) in combination with a diet moderate in protein and a

lower age at slaughter (35 days) in Sweden as compared with Denmark.

21. In conclusion, differences in certain husbandry practices as between Denmark and Sweden appear to have led to a reduction in lameness from 30.1% to 20.4% (even the latter figure is far too high from a welfare viewpoint). However, Kestin et al (2001) show that genotype has a much greater impact on the incidence of lameness. This study examined thirteen different genotypes. These were divided into three groups: Group 1 contained the faster-growing genotypes; Group 2 contained the genotypes with moderate growth characteristics; and Group 3 contained the traditional slower-growing genotypes.
22. Kestin et al found that at 54 days of age, with the birds being fed on a normal commercial diet, the Group 1 birds had a mean GS of 2.92, the Group 2 birds had a mean GS of 1.53 and the Group 3 birds' mean GS was just 0.07.
23. The authors concluded that their 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".
24. Studies have investigated the impact on lameness of changing the light/darkness pattern, early feed restriction and giving the birds two, three or four meals per day rather than feeding them *ad libitum* (Sørensen et al, 1999 and Su et al, 1999).
25. Kestin et al (1999) pointed out that the largest difference in GSs achieved by the changes in lighting or feeding programmes reported in the above two papers was 0.240 GS units. Similarly, as indicated in paragraph 19, differences in husbandry practices in Denmark and Sweden led to a difference of 0.230 GS units between the two countries. In contrast, Kestin et al's (1999) own study on the impact of genotype found a difference of 0.509 GS units between two modern, fast-growing genotypes. Moreover, Kestin et al (2001) found a difference of 2.85 GS units between faster- and slower-growing genotypes.
26. In conclusion, it can be seen from a range of studies, that changes in genotype have a much greater impact on the incidence of lameness than changes in husbandry and management.
27. Mr Pritchard entirely ignores the study by Kestin et al (2001) which investigated thirteen different genotypes and which demonstrates the crucial importance of genotype as a

determinant of lameness. Also ignored is the report on broilers published in 2000 by the European Commission's Scientific Committee on Animal Health and Animal Welfare (SCAHAW). This report is the only recent comprehensive review of the scientific literature on broilers.

28. The SCAHAW made it clear that leg disorders are one of the main welfare problems facing broilers and that they result primarily from selection for rapid growth rates. The SCAHAW stated "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." 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" (my emphasis).
29. A number of recent papers acknowledge that the main factor in causing lameness in broilers is the use of genotypes selected for high liveweights and rapid growth. Garner et al (2002) state that "selection for rapid growth and increased meat and breast weights in broiler chickens has also resulted in a tendency for these strains to develop severe skeletal problems".
30. A recent major review of the scientific literature on leg disorders stressed that "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" (Bradshaw et al, 2002).
31. In their investigation of lameness in Swedish broilers, Sanotra & Berg (2003) concluded that the prevalence of leg weakness in broiler chickens was related to the genotype, most likely mainly as an effect of bodyweight (the rapid growth rates of many modern genotypes mean that they achieve high bodyweights at a relatively early age).
32. Corr et al (2003a) state that "In the modern broiler, heavy selection pressure has been directed toward obtaining rapid growth rates to high bodyweights ... It is recognised that increased bodyweight puts greater demands on the skeleton.... A strong correlation has been demonstrated between liveweight, growth rate and "leg weakness" or lameness in poultry".
33. The authors concluded that **"the rapid growth rate to high end-bodyweights that is demanded of these birds creates**

**high loads on bones that are still immature. The potential through selective breeding to manipulate the rate at which soft tissues such as muscle develop is not matched in bone, the growth rate of which is inherently limited”** (my emphasis).

34. In their study of leg problems in Denmark, Sanotra et al (2001) concluded that “one of the main factors responsible for leg problems in broilers is rapid growth which results in the achievement of high bodyweights within short time periods.”
35. I do not understand Mr Pritchard to say that this evidence – of which DEFRA are of course aware – is flawed or is otherwise to be disregarded in determining issues of policy in relation to the welfare of broilers.
36. Mr Pritchard exhibits with his Witness Statement Table 1 (DP2) and Figure 1 (DP3) which, he asserts, show the causes of lameness. However, both Table 1 and Figure 1 are of limited value as they make no attempt to give any weighting or ranking to the causes of lameness. They simply list the causes, but do not attempt an assessment as to which are the principal causes and which are of relatively minor importance.

### **Prevalence of leg disorders in broilers**

37. In paragraph 29 of his Witness Statement, David Pritchard relies on the British industry’s own survey of leg disorders which found only 2.5% of broilers to have GSs 3, 4 or 5.
38. However, the validity and reliability of that survey have been discredited by a report prepared for CIWF by Professor Donald Broom, who is Colleen MacLeod Professor of Animal Welfare at the University of Cambridge. Professor Broom is one of the UK’s leading scientists in the field of farm animal welfare. The industry asked Professor Pfeiffer and Ms Dall’Aqua to analyse their data and it is that analysis which Professor Broom has examined. Professor Broom’s report is at Exhibit “PS 2”.
39. Professor Broom states that the analysis by Pfeiffer and Dall’Aqua of the data presented is good “but that the data themselves are flawed to such an extent that the conclusions are not meaningful”.
40. Given the view of Professor Broom that meaningful conclusions cannot be drawn from the industry’s own survey, it is important to re-examine other recent surveys, to which Mr Pritchard has not referred, but of which DEFRA is no doubt aware.

41. As indicated above, a recent Danish study found that 30.1% of the broilers had GSs of 3, 4 or 5 (Sanotra et al, 2001).
42. Moreover, the recent Swedish study referred to above found that 14.1% of the Ross 208 broilers studied and 26.1% of the Cobb 500 broilers studied had GSs of 3, 4 or 5. Taking the two groups together, 20.4% of the birds had GSs 3, 4 or 5 (Sanotra & Berg, 2003).
43. In a letter dated 16 April 2002 to the Parliamentary Secretary at DEFRA, the Chairwoman of the Farm Animal Welfare Council pointed out that the industry report notes incidences 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%.

**Strong evidence that leg disorders are painful and compromise welfare**

44. In paragraph 32 of his Witness Statement, Mr Pritchard accepts that there is good evidence that some types of lameness are painful. But he contends that some lameness neither is, nor appears to be, related to pain. However Mr Pritchard has no sound basis on which to make such a confident assertion. There is good scientific evidence that most lameness is painful and that where it is not painful, it compromises welfare in other ways, for example, by leaving the birds less capable of reaching the feeders or drinkers or by increasing the time spent lying which, if the litter quality is poor, can lead to an increased incidence of hock burns, foot pad burns and breast blisters.
45. The paper on leg and heart problems annexed to CIWF's Claim Form (hereafter referred to as "CIWF's leg and heart paper") cites studies which provide strong evidence that lameness in broilers is painful. The paper also cited a study which shows that, compared with non-lame birds, lame birds spend significantly more time lying and less time walking. Moreover, whereas non-lame birds predominantly chose the usual standing posture for eating, lame birds lie down to eat for almost half their feeding time (Weeks et al, 2000). The authors pointed out that inhibition of activity is often noted in animals experiencing chronic pain. The authors concluded that the increased time spent lying results from lameness with its associated disability and "probable pain".
46. There are other papers, in addition to those referred to in CIWF's leg and heart paper, which strongly indicate that leg disorders are associated with pain. Vestergaard & Sanotra (1999) hypothesised that leg problems would reduce the frequency of dust-bathing.

Dust-bathing, especially the vertical wing-shaking component, involves forceful movements with the legs and wings. Thus, experience of pain associated with these movements may be expected to reduce the performance of dust-bathing behaviour. The authors found that leg disorders do indeed reduce dust-bathing in broilers.

47. In another study, Sanotra et al found that chickens with leg weakness problems perform fewer of the vertical wing-shakes involved in dust-bathing than normal (i.e. non-lame) broilers, and that the reduction in frequency of vertical wing-shakes increases with increasing severity of the leg problems. They also found, when treated with an analgesic, broilers with leg problems perform as many vertical wing-shakes (or more) as normal broilers. They concluded that this strongly indicates that leg problems are associated with pain.
48. In two 2003 papers, Corr et al (2003a & b), compared two strains of broilers, selected and 'relaxed' (i.e. not selected), raised under two feeding regimes, ad-libitum fed and restricted-fed. The first paper (2003a) concluded that

"The ad-libitum-fed selected birds reached heavier bodyweights at younger ages, had wider girths, and developed large amounts of breast muscle which probably displaced their centre of gravity cranially. At cull weight, they had shorter legs than birds in the other groups and greater thigh-muscle masses; therefore, greater forces would have to be exerted by shorter lever arms in order to move the body. The tarsometatarsi were broader, providing increased resistance to greater loads, but the bones had a lower calcium and phosphorus content, which would theoretically make them weaker. Many of these morphological changes are likely to have detrimental effects on the musculoskeletal system and therefore compromise the walking ability and welfare of the birds".
49. Corr et al (2003b) pointed out that along with the rapid growth rate to high bodyweight, the modern broiler has been selected to produce more breast muscle, resulting in a change of conformation. Both of these factors, the authors state, can affect locomotion: the rapidly increasing bodyweight will place greater demands on the immature skeleton, and the change in shape can alter the forces produced during walking.
50. The authors found that the ad-libitum-fed selected birds walked more slowly, with lower cadences and took shorter steps. The steps were wider, and the toes were pointed outwards, resulting

in a wider walking base. They kept their feet in contact with the ground for longer periods, having longer percentage stance times, shorter percentage swing times and increased double-contact times compared to the relaxed birds. These changes serve to increase stability during walking and are a likely consequence of the morphological changes in the selected broiler – in particular, the rapid growth of breast muscle moving the centre of gravity forward and the relatively short legs compared to their bodyweight.

51. The authors state that this altered gait would be very inefficient and would rapidly tire the birds and could help to explain the low level of activity seen in the modern broiler. They conclude that even if the abnormal gait pattern results from bio-mechanical limitations rather than from pain, welfare will be compromised if, for example, the birds are less capable of reaching the feeders or drinkers.
52. Leg disorders have several consequences for welfare, in addition to pain:
  - a) in severe cases it may be difficult for birds to get to food and water.
  - b) Vestergaard & Sanotra (1999) pointed out birds may be inhibited from performing essential behaviour patterns. The authors added that such inhibitions have been observed in behavioural studies which revealed reductions in feeding, drinking, walking, scratching, pecking and dust-bathing, and an increase in the time spent lying down, resting and sleeping.

Weeks et al (2000) also found that lame birds spent significantly more time lying down than sound birds (this paper is referred to in detail in CIWF's leg and heart paper).

- c) As lame birds spend more time lying on the litter than sound birds, they are more likely to develop hock burns, foot pad burns and breast blisters if the litter quality is poor.

### **Likely increase in future prevalence of leg disorders**

53. In paragraphs 33-35 of his Witness Statement, Mr Pritchard disagrees with CIWF's statement (in its leg and heart paper) that, as the industry aims to substantially increase broilers' bodyweight at 41 days of age over the next few years, there is a real danger that the incidence of leg disorders will increase.

54. CIWF recognises that future improvements in, for example, nutrition management, could well help prevent certain leg disorders. However, any such benefits will probably be outweighed by the industry's drive to increase growth rates. The resultant higher bodyweights at any given age are likely to lead to an increased prevalence of leg disorders. In its leg and heart paper, CIWF referred to Kestin et al (1999), who predicted that an increase in growth rate of 600 g. to 40 days over a ten year period 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.
55. In paragraph 34 of his Witness Statement, Mr Pritchard relies on the analysis by Pfeiffer and Dall'Aqua of the industry's own data. However, as indicated above, Professor Broom has concluded that the industry's data are flawed to such an extent that the conclusions are not meaningful.

### **Broiler breeders**

56. In paragraphs 44 and 45 of his Witness Statement, Mr Pritchard challenges CIWF's statements that broiler breeders are given very much less to eat than if they were fed "normally" or were fed the "quantity they want to eat". In fact it is the practice of the scientific literature in this field to compare the quantity broiler breeders are given to eat with what they would eat normally or if fed *ad-libitum*, as the following extracts show:
- a. Whitehead (2002) – who is quoted by Mr Pritchard in paragraph 57 of his Witness Statement – writes that: "More severe and prolonged feed restriction is routinely applied to broiler breeders. Intakes of 50% of **normal** *ad-libitum* intake are applied for most of the rearing period with proportionately less restriction during lay" (my emphasis).
  - b. Mench (2002), who concluded that restricted-fed broiler breeders are "chronically hungry", writes that "feed allocations during rearing are 60-80% less than birds would consume *ad-libitum*, and are 25-50% less during the laying period".
  - c. Savory et al (1993), who concluded that restricted-fed broiler breeders are "chronically hungry, frustrated and stressed", stated that the recommended commercial restricted feeding programme, for growing female broiler breeders, suppressed food intake between 2 and 20 weeks of age by some 70%, compared with food intake of birds "that were allowed to eat as much as they wanted".

55. The severe degree of feed restriction imposed on broiler breeders is highlighted by the data in this and the following two paragraphs. These data are drawn from booklets setting out performance objectives published by Aviagen Limited in respect of the Ross 308 broiler. This is the most common broiler used in Britain, with an estimated market share of over 70%. At 42 days of age, an ordinary female broiler weighs 2,272g., whereas a feed-restricted female broiler breeder weighs just 660g. Similarly, at 42 days of age, an ordinary male broiler weighs 2,676g., whereas a feed-restricted male broiler breeder weighs just 1080g. (In this and the following two paragraphs, when referring to "ordinary" broilers, I am referring to the broilers reared for their meat as opposed to the breeding flock).
56. The extreme nature of restricted feeding regimes for broiler breeders can also be seen by the fact that at 42 days of age, an ordinary female broiler is given 178g. of feed per day, whereas a female broiler breeder is given just 49g. per day. Similarly, at 42 days of age, an ordinary male broiler is given 200g. of feed per day, whereas a male broiler breeder is given just 68g.
57. A similar pattern emerges if one looks at the amount of feed given at 28 days of age. An ordinary female broiler is given 128g. of feed per day, whereas a female broiler breeder is given just 41g. Similarly, an ordinary male broiler is given 143g. of feed per day, whereas a male broiler breeder is given just 57g. of feed per day.
58. In paragraph 52 of Mr Pritchard's Witness Statement, DEFRA acknowledges that restricted feeding of broiler breeders does result in hunger. Similarly, in paragraph 61, Mr Pritchard accepts that hunger is associated with restricted feeding. In paragraph 57, Mr Pritchard quotes Whitehead (2002) who acknowledges that restricted feeding may be associated with some welfare disadvantages "not least in relation to one of the 'five freedoms' (freedom from hunger)".
59. In the paper on restricted feeding annexed to CIWF's Claim Form, we referred to several studies which conclude that restricted-fed broiler breeders suffer from hunger.
60. Savory (2002) points out that "another consequence of selection for faster growth in broiler progeny is that the parent stock (broiler breeders), which have a similar genetic potential for growth, need to be subjected to increasingly severe food restriction during rearing in order to constrain their body weight within desired limits at sexual maturity.... Female broiler breeders reared according to a recommended feeding programme

typically eat their daily ration in <15 minutes, eat a third as much and grow a third as fast as do ad libitum-fed control birds, and are highly motivated to feed at all times. They are much more active than unrestricted birds, and (unlike the latter) show behaviours characteristic of frustration of feeding. There is also evidence that blood indices of stress are raised in restricted-fed birds. Taken together, these facts indicate that the welfare of growing broiler breeders is compromised by current commercial food restriction."

61. The essence of Mr Pritchard's position appears to be that broiler breeders need to be fed on restricted regimes in order to reduce the incidence of heart and leg disorders and of mortality.
62. Hocking (2003) – a paper referred to several times by Mr Pritchard – implicitly acknowledges that modern broiler breeder genotypes are in essence unsustainable and unviable creatures, as nearly half would be dead before 60 weeks of age unless their feed intake was restricted to the point of chronic hunger. Table 1 in Hocking (2003) shows that the mortality rate at 60 weeks of age of broiler breeders fed ad-libitum is 46%. A bird which, as a result of its poor health, is susceptible to such high mortality rates, is an inherently unsustainable genotype.
63. CIWF considers that the proper (and lawful) approach is not to use a genotype which, to keep much over 50% of the birds alive, must be restricted-fed to such a degree that it experiences chronic hunger; instead, slower-growing genotypes should be used.
64. As indicated earlier, Kestin et al (2001) examined thirteen genotypes, which they divided into three Groups: Group 1 contained the faster-growing genotypes; Group 2 contained the genotypes with moderate growth characteristics; and Group 3 contained the traditional slower-growing genotypes.
65. One batch of each Group was fed a normal commercial diet. All the feeds were available ad-libitum. At 54 days of age there were major differences in liveweight between the three Groups. The Group 1 birds had a mean liveweight of 3.67 kg., the Group 2 birds had a mean liveweight of 2.39 kg., and the Group 3 birds' mean liveweight was 1.24 kg. The mean liveweight difference at 54 days between faster- and slower-growing genotypes, when fed ad-libitum on exactly the same diet, was 2.43 kg.
66. **This demonstrates that it is possible to achieve low liveweights very effectively by using slower-growing genotypes and that to do this it is not necessary to impose**

**hunger through restricted feeding on broiler breeders; the slower-growing birds were fed ad-libitum.**

- 67. Moreover, the slower-growing genotypes with their low liveweights had very low levels of lameness. The mean GS of the slower-growing genotypes at 54 days was just 0.07 as compared with the mean GS of 2.92 of the faster-growing genotypes.
- 68. In the same study, when the faster-growing genotypes were fed on a diet less rich in protein and energy than the normal commercial diet, their weight at 54 days dropped to 2.52 kg. and their mean GS to 1.73. However, this weight and GS were still much higher than those of slower-growing genotypes fed on a richer diet. This shows that weight and lameness can both be more effectively reduced by the use of slower-growing genotypes than by changes in diet.

I believe that the matters stated in this statement are true.

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Dated.....

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