DO OSTEOPOROSIS AND SKELETAL UNDERMINERALISATION LIMIT EGG PRODUCTION?

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Summary

Problems of weight loss in the pre- and post-peak phases of egg production have been identified in many commercial and experimental flocks in Australia. These periods of weight loss have been associated with loss of egg production, muscle paralysis in some birds and the development of skeletal abnormalities characteristic of osteoporosis. The weight loss and sub-optimal production do not appear to respond to conventional dietary supplementation with energy and/or protein.

These phenomena may be consequences of environmental stress. Further research is needed to identify the metabolic changes that lead to osteoporosis and the relationship of these changes to the depletion of skeletal mineral reserves, the decline in structural bone volume, and the break in the continuity of egg production.

I. INTRODUCTION

(a) Historical descriptions of cage layer fatigue

In his review of skeletal diseases of poultry, Riddell (1981) suggested that cage layer osteoporosis underlies both cage layer fatigue and the bone breakage which occurs when hens are culled at the end of lay. Recent research on induced osteoporosis in the laying hen is compatible with this hypothesis, and the possibility remains that modern laying strains still experience sub-clinical cage layer fatigue in association with osteoporosis.

The clinical signs of cage layer fatigue include muscle paralysis, sternal deformation, sigmoidal shaped ribs, and infolding of the ribs caused by small fractures at the costochondral junctions. Cortical bone is thin and medullary bone mass is decreased. The paralysis is believed to be due to compression fractures of the fourth and fifth vertebrae (Bell and Siller, 1962) and in some birds that recover, these vertebral fractures are thought to have healed. In some cases, cage layer fatigue appears to be associated with hypocalcaemia (Whitehead, 2001).

Cage layer fatigue is more frequently found in high producing leghorns near peak production (Couch, 1955; Riddell, 1981), and can be accentuated in underweight pullets coming into lay in summer (Grumbles, 1959). The disease appears to have been in decline since the 1980's when Riddell (1981) recorded a prevalence as high as 0.5% per month in some case studies of commercial layers.

Clinical signs of cage layer fatigue will clearly be evident in single bird cage selection of elite breeders, and should also be reflected in lower annual hen housed egg production. Hence there should be some selection pressure against cage layer fatigue, particularly if the breeding companies have been able to select full sisters in both a single bird cage environment and under commercial conditions. Bell and Siller (1962) have suggested that the highly productive laying hen may be on the threshold of its minimum endogenous calcium requirements, and may lack a mechanism to decrease egg production when calcium supply is insufficient.

Through ongoing genetic selection processes, modern strains, in contrast to standard layer breeds, may have a greater resistance to cage layer fatigue by being able to balance

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continuous egg production with skeletal integrity. If this is so, the modern birds may still use their skeletal mineral reserves to maintain egg production, but may have the ability to pause egg production to prevent the development of cage layer fatigue.

(b) Induction of osteoporosis in laying hens

Recent research on induction of osteoporosis in the laying hen has found that there is a loss of structural bone in early lay and that this precedes the accumulation of medullary bone under the influence of oestrogen. It is believed that laying hens cannot produce structural bone during egg production (Rennie, 1997), but in hens that are out of lay with lower oestrogen levels, the medullary bone is resorbed and structural bone formation can recommence.

Experimental studies indicate that the erosion of structural bone begins by about 20 weeks of age and then stabilises between 30-40 weeks of age (Wilson and Whitehead, 1992; Thorpe et al., 1993). Within a flock there appears to be almost no further loss of structural bone between 40 weeks of age and 68-72 weeks of age and the bone density at 40 weeks of age is the same as that at the end of the laying cycle (Fleming et al., 2000; Whitehead, 2001).

It has been postulated that the development of osteoporosis in highly productive strains is associated with the length of time that eggs are continuously produced rather than with the actual number of eggs produced. Birds that cease production or have a pause in egg production may be able to regenerate structural bone (Rennie et al., 1997). The time of continuous egg production required to erode structural bone, and the length of time of a pause in production needed to restore structural bone remain to be defined.

Clearly clinical cage layer fatigue compromises egg production and shell quality. The questions still needing answers are firstly whether cage layer fatigue is still occurring in commercial flocks, and secondly, whether moderate erosion of the skeleton in modern layer strains can trigger a pause in egg production or induce a lower rate of egg production as protective mechanisms to maintain skeletal strength.

(c) Weight loss and emaciation in layers

Summers (1983) has suggested that in pullets with low reserves of energy at sexual maturity, protein and calcium are unable to meet the demand for egg production when the intake of these nutrients is low. Furthermore, Leeson (1990) has described problems at peak production of low appetite, weight loss and a subsequent marked slump in egg production in the post-peak period. It seems likely therefore that the appetite of birds in commercial flocks may be inadequate during early egg laying, so that with the drain of production, weight loss and eventually diminished production are inevitable. The relationship of these phenomena to skeletal development and osteoporosis has been poorly described.

Gregory and Devine (1999) have reported that many commercial flocks contain emaciated birds by the end of the production cycle. They speculate that the metabolic demand on birds during egg production induces tissue catabolism. At present we have little knowledge about either the physiological consequences of this emaciation, or the ages at which it occurs in the modern bird.

Australian research has identified problems of sub-optimal growth in early egg production that appears to be correlated with defects in skeletal structure and also with muscular paralysis.
II. BODY WEIGHT LOSS IN EARLY EGG PRODUCTION

Experimental studies in Australia under controlled environmental conditions, with birds fed on commercial diets, have found periods of depressed growth in layers between 20 to 26 weeks of age (Figure 1), similar to the observations of Leeson (1990). Groups of birds have been identified with significant weight losses over a 1-2 week period. Approximately 14% of birds in the flock lost 100 grams or more live weight, whereas the breed standards predict that birds should gain, on average, 80 to 150 grams live weight throughout the same period. Clearly a significant proportion of the flock is undergoing marked tissue catabolism in the early lay period and this is associated with lower egg production performance (Figures 1 and 2).

With tissue catabolism of this extent, it is possible that the processes of bone growth and development are also disrupted. The normal pattern is for medullary bone reserves to be laid down at the same time as structural bone is being resorbed. Significant tissue catabolism would be likely to affect both these processes. Furthermore, these problems of early weight loss may be contributing to the emaciation observed by Gregory (1999) in older birds because the decreased body weight can become a chronic problem (Figure 1).

![Figure 1](image_url)  
**Figure 1.** Average flock body weights in two groups of birds (■ - high egg production (> 96%), ● - low egg production (<88%)).
Figure 2. Percentage of days that eggs were produced versus age, in two groups of birds (■ - high egg production (>96%), ● - low egg production (<88%)).

III. TRANSITORY PARALYSIS IN HIGH PRODUCING BROWN EGG-LAYERS

Birds of a brown-feathered egg-laying strain, aged between 17 and 45 weeks, were fed a commercial laying diet under controlled environmental conditions. The flock achieved both body weight and egg weight standards and had above average egg production performance (99% peak egg production) (Figures 3 and 4). The ratio of egg mass output to body weight was 3.0 grams egg weight/kg live weight for the commercial brown egg-layer at 45 weeks of age.

Between 26 to 30 weeks of age the flock growth rate declined against the breed standards and there was almost no growth between 29-30 weeks of age. At 28 weeks of age, 4% of the birds showed muscle paralysis similar to that seen in cage layer fatigue. A period of weight loss of between 50 to 150 grams over 1 to 2 weeks preceded the paralysis and eventually the affected birds ceased to produce eggs. After a further 1 to 2 weeks, these birds regained both muscle function and body weight and resumed laying at their previous production rates.
Figure 3. Percentage of days that eggs were produced versus age in a flock of commercial brown egg-layers between 21-45 weeks of age (● - breed standard).

Figure 4. Average body weight in commercial brown egg layer flock between 17 to 45 weeks of age (● - breed standard).

IV. SKELETAL ABNORMALITIES IN HIGH PRODUCING BROWN EGG LAYERS

Assessment of deformity and swelling of the costochondral junction of the rib cage was done at 45 weeks of age in the highly productive commercial brown egg-laying flock described in figures 3 and 4. The scoring scale ranged from 0 to 5, with 5 indicating very severe lesions and 0 indicating no lesions. Across the whole flock, 57% of birds had a rib abnormality score of 1-5, whilst 29% had a score of 3-5. A retrospective analysis of the flock growth patterns of two groups (those not affected or only mildly affected (score 0-2) against those which were severely affected (score 3-5)) revealed that the birds developing the severe rib abnormalities had a loss of body weight between 29 to 31 weeks of age, which corresponded to the period of transitory paralysis in 4% of birds. A partial recovery from the loss of body weight eventually occurred between 31 and 34 weeks of age, but these birds
remained about 100 grams lighter than the unaffected birds. The loss of body weight was associated with a 15% decrease in egg production, however production eventually recovered as the birds began to gain weight.

Figure 5. Average body weights of groups severely effected (■) and those not or mildly effected (●) with rib abnormalities

Figure 6. Percentage of days that eggs were produced versus age of groups severely affected (■) and those not affected or only mildly affected (●) with rib abnormalities

V. MOULTING AND RECOVERY IN STRUCTURAL BONE MASS IN BROWN EGG-LAYERS

Research to date indicates a poor correlation between egg production and structural bone volume in a modern commercial strain (Rennie, 1997), but it is apparent that standard egg-laying breeds, with lower egg production performance, have higher structural bone volumes at the end of the egg production cycle than those strains with higher egg yields. Within the modern strains, a high egg production rate and erosion of structural bone may
interact with an ability of birds to regenerate structural bone by ceasing or pausing egg production. Research in moultng hens indicates that trabecular bone volume can increase from 5.7% to 21.5% over an 8-week period of recovery (Table 1).

Table 1. Changes in the volumes of proximal tarsometatarsal trabecular bone (TBV%) and medullary bone (MBV%) in brown egg-layers during the tissue recovery process following a moult ending at 72 weeks of age (mean (SE))

<table>
<thead>
<tr>
<th>Age (weeks)</th>
<th>TBV%</th>
<th>MBV%</th>
</tr>
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<tbody>
<tr>
<td>72</td>
<td>5.7 (1.0)</td>
<td>5.9 (1.3)</td>
</tr>
<tr>
<td>80</td>
<td>21.5 (1.2)</td>
<td>1.5 (0.2)</td>
</tr>
<tr>
<td>107</td>
<td>13.7 (1.1)</td>
<td>2.9 (0.6)</td>
</tr>
</tbody>
</table>

VI. CONCLUSIONS

An important question to be resolved is whether the body weight loss, muscular paralysis and skeletal abnormalities described in this paper, are linked to the mechanisms that induce osteoporosis in commercial layers. The induction of osteoporosis in individual birds may occur independently of, but nevertheless interact with, the depletion of reserves of energy, protein and calcium from high rates of egg production.

If the bone density and bone strength at 40 weeks of age, are strongly correlated with the bone density and strength at the end of lay, then an examination of environmental interactions which occur between the onset of lay and the peak of egg production will help in devising management strategies to prevent the problem of osteoporosis.

Experiments with individual birds should also be done to investigate whether small decreases in body weight and feed intake are predisposing factors in the excessive erosion of skeletal mineral reserves. Because the ability of birds to pause in egg production allows structural bone mass to regenerate, the importance of skeletal mineral reserves in sustaining egg production and shell quality should not be underrated.

REFERENCES