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FOOD INTAKE, WEIGHT CHANGES AND EGG PRODUCTION IN CAPTIVE RED GROUSE BEFORE AND DURING LAYING: EFFECTS OF THE PARASITIC NEMATODE *TRICHOSTRONGYLUS TENUIS*[1]

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Abstract. We investigated variations in egg production of captive hen Red Grouse *Lagopus lagopus scoticus* in relation to food intake, weight change, and parasitism by the nematode *Trichostrongylus tenuis*. Experimental infections of *T. tenuis* were used to mimic the relative effects on laying grouse of an adult worm burden, the resumed development of over-wintered worm larvae or no infection. Variations in egg production were significantly related to hen food intake and weight loss during laying but not to food intake or weight gained in the five weeks before laying. This supports the Thomas (1988) hypothesis that grouse produce eggs from 'income' rather than 'capital' reserves. There were no significant differences in the total weight, number or rate of eggs produced by hens from different treatment groups. However, hens infected with worms developing at the onset of laying lost significantly more weight while laying and, at the end of egg laying, were 13% lighter than other birds. In wild birds this could affect the quality of incubation and maternal care of chicks. The precise timing of the resumed synchronous development of previously arrested *T. tenuis* larvae may be a critical determinant of how this parasite affects egg production by wild Red Grouse.

Key words: egg production; resources for laying; Red Grouse; Lagopus lagopus scoticus; *parasitism;* Trichostrongylus tenuis.

INTRODUCTION

The relative importance of reserves acquired before and during egg-laying varies widely amongst bird species (Owen and Reinecke 1979). Drent and Daan (1980) classified birds with little reliance upon stored reserves for egg formation as 'income' breeders, and those depending on accumulated reserves as 'capital' breeders. Thomas and Popko (1981) and Thomas (1982) showed that Rock Ptarmigan (*Lagopus mutus*) and Willow Ptarmigan (*Lagopus lagopus*) depended on food supplies during laying for egg production. Thomas (1988) subsequently described the Tetraoninae as extreme 'income' breeders. Savory (1975), however, found that the total weight of eggs laid by captive hen Red Grouse was significantly related to food intake during the five weeks preceding laying but not to food intake during laying, suggesting that Red Grouse depend more on reserves for laying than Thomas's generalisation would suggest. The present study re-examines this apparent contradiction.

The process of acquiring resources for egg production in birds may be influenced by many factors, such as food shortage and parasitism. It is not clear whether such stresses are likely to have their biggest effect on egg production during the accumulation of reserves before laying, or during laying itself. Nor is it clear whether the consequences of the timing of such stresses differ between 'income' and 'capital' breeders.

One natural stress that reduces the number of eggs laid by Red Grouse, both in the wild and in captivity, is infection with the parasitic nematode *Trichostrongylus tenuis* (Jenkins et al. 1963, Wilson 1979, Potts et al. 1984, Hudson 1986a, Shaw and Moss 1990, Hudson et al. 1992). For most of the year *T. tenuis* eggs voided in the faeces of infected grouse hatch and develop into infective larvae which migrate onto adjacent heather tips where they may be ingested by feeding birds (Leiper 1910, McGladdery 1984). Larvae that successfully reach the caeca develop into sexually mature adult worms within 16 days (Shaw 1988b) and may live for years (Shaw and Moss 1989). However, larvae ingested in late autumn and winter may arrest their development in the host until the following spring (Shaw 1988a,

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Moss et al. 1993, Delahay 1995). Developing larvae are more pathogenic than adult worms (Shaw and Moss 1990, Delahay et al. 1995) and the synchronous resumed development of larvae arrested over winter may explain why outbreaks of trichostrongylosis in wild grouse occur in spring and early summer (Committee of Inquiry 1911, Macintyre 1918, Jenkins et al. 1963, Hudson et al. 1992).

Shaw and Moss (1990) found that infecting captive grouse hens with *T. tenuis* five weeks before laying began had a bigger effect on egg production than infecting them during laying. The effect of developing *T. tenuis* on grouse egg production was greatest at the 'capital' stage of reserve acquisition and had little impact at the 'income' stage.

It follows that the precise timing of resumed development of larvae arrested over winter is likely to be an important determinant of the parasites' impact. Moss et al. (1993) recorded large increases in the number of worm eggs in the caecal faeces of wild birds in north-east Scotland in mid-April in some years and attributed these to the maturation of previously arrested larvae. Wild grouse in north-east Scotland generally begin laying in mid to late April and so larvae previously arrested over winter seem to develop at or shortly before the onset of laying.

If larvae were to develop during laying in wild grouse, hens might meet the cost of maintaining a normal rate of egg production by eating more food or by using more reserves. The present study tested these possibilities by measuring food intake and weight changes in hens challenged with developing larvae during laying, and in controls.

The experiment used a single-dose infection of *T. tenuis* larvae, just before the onset of egg-laying, to simulate the effects of the resumed development of previously arrested larvae on grouse egg production. Hens with an established adult worm burden were also included in the experiment to check the previous observation (Shaw and Moss 1990, Delahay et al. 1995) that developing larvae are more pathogenic than adult worms.

MATERIALS AND METHODS

HOSTS AND PARASITES

Captive-bred Red Grouse of one to four years old were checked for *T. tenuis* infection by examining faeces for eggs. Experimental birds were individually housed in wire-floored cages in the open air, as described by Moss (1969). Fresh water, pelleted food (Dalgety Agriculture Ltd, Aberdeen) and grit were provided ad libitum. Bundles of freshly cut heather were also supplied as this has been found to increase egg laying by captive grouse (Moss et al. 1971).

T. tenuis larvae were obtained from cultures of worm eggs obtained from infected captive birds, and incubated for 8–10 days at 20° C (Shaw 1988b, Delahay 1995). Infective larvae (L3) were recovered using a Baermann apparatus and concentrated on a 125 μm sieve. Birds in the infected groups were given an oral dose of approximately 6,000 larvae suspended in 1 ml distilled water. Previous experimental work (Delahay 1995, Delahay et al. 1995) showed that this was sufficient to establish a sub-lethal but energetically costly worm burden. Worm eggs in grouse caecal faeces were monitored weekly.

TREATMENT GROUPS

Seven birds were infected between October 1992 and February 1993, so that by the onset of laying in April they had long-established adult worm burdens (group A). Six birds were infected in mid-April 1993 when the first egg of the season was laid, so that at the start of laying they had burdens of developing worms (group D). Uninfected birds (group C) were subjected to the same handling and dosed with an equivalent volume of water when group D were infected.

FOOD INTAKE, BODY WEIGHT AND CONDITION

Once weekly at 10:00 heather bundles were removed from the cages and a known weight of pelleted food was provided in feeding troughs. Uneaten and spilled food was weighed 48 hrs later and its moisture content determined by oven drying samples to constant weight at 80 °C. The dry weight (DW) of food eaten was calculated and daily food intake was corrected for body weight according to isometry (W^1; Delahay et al. 1995).

At the end of each feeding trial, birds were weighed and their condition assessed by palpating sternum musculature and assigning a score on the scale 1–15 (emaciated to plump condition) according to muscle mass (Moss et al. 1985).

EGG LAYING

From the beginning of March, a cock was put with each hen for 1.5–2 hours daily. This was

reduced to alternate days once laying began and was discontinued in late May.

Eggs were removed from cages each day, individually weighed and their volume estimated as the volume of water displaced by immersion. The rate of laying is defined as the number of eggs laid divided by the duration of laying in days.

STATISTICAL ANALYSIS

All measures of egg production were approximately normally distributed (Shapiro-Wilk tests, P always > 0.1) except for proportions (p) which were transformed (arcsine $\sqrt{p}$) before analysis. Differences in egg production between treatment groups were tested for significance at the 5% level. Since the likely direction of differences between treatment groups was known (Shaw and Moss 1990), one-tailed probabilities were used for comparisons among treatment groups. Tests were performed using SAS (1989).

Daily mean food intake and net changes in body weight and condition score of individual hens were measured for five weeks before laying and during laying. These summary statistics were used to explain egg production in generalised linear models which also controlled for effects of hen age (models specified in Appendix). Correlations were also performed so that the strength of individual relationships could be seen clearly (Fig. 1). Differences in rates of change in weight and condition of hens between treatment groups were tested for significance using one-way t-tests on regression coefficients.

As birds in treatment group D were not infected until the onset of laying, groups C and D were pooled for analysis of effects before laying. One bird in group C was obese at the start of the experiment. Although the inclusion of this bird in the analyses did not alter the conclusions, its food intake, body weight and condition were deemed unrepresentative and were excluded from calculations involving these variables.

RESULTS

FOOD INTAKE, BODY WEIGHT, CONDITION AND EGG PRODUCTION

Generalised linear models controlling for grouse age, and correlations (Figs. 1A and B), revealed similar associations among hen food intake, body weight and egg production.

The weight of hens at the start of the experiment was positively but not significantly related to the number and total weight of eggs produced. After controlling for hen age, however, hen weight at the start was related to the number and weight of eggs produced (GLM model 1, $F_{1,8} = 8.98$, $P = 0.02$; $F_{1,8} = 7.06$, $P = 0.03$ respectively). However, neither hen food intake nor weight gain before laying was significantly related to clutch size, total weight of eggs or the rate of laying (GLM models 1 and 2, P always > 0.05).

In contrast, food intake and weight loss during laying were significantly related to egg production after controlling for effects of hen age and initial body weight. Grouse that consumed more food produced a greater total weight and number of eggs, at a faster rate (GLM model 2, $F_{1,8} = 8.24$, $P = 0.02$; $F_{1,8} = 8.48$, $P = 0.02$; $F_{1,8} = 6.7$, $P = 0.03$ respectively). Birds that lost less net weight (weight at onset of laying − weight at the end of laying) during laying also produced a greater total weight of eggs (GLM model 1, $F_{1,8} = 6.18$, $P = 0.04$) although this relationship was less significant when corrected for food intake (GLM model 3, $F_{1,6} = 5.62$, $P = 0.06$). Changes in the condition score of hens before or during laying were not significantly related to egg production (GLM model 4, P always > 0.05). Although young hens (< 1 year old) laid more eggs than older birds, these differences were not significant (GLM models 1 and 2, P always > 0.05).

PARASITE ESTABLISHMENT RATES

Counts of worm eggs per gram (epg) of grouse caecal faeces reflect numbers of adult *T. tenuis* present in the hosts (Shaw 1988b, Moss et al. 1993, Delahay 1995). There was no significant difference in worm egg counts (mean of six weekly counts for each bird) between treatment groups A and D ($t_{11} = -0.68$, $P > 0.05$; see Fig. 2), and no evidence of infection was detected in the control birds. A regression equation relating epg to numbers of adult worms at necropsy ($\log_{10}(\text{worms} + 1) = 0.81 + (0.57(\log_{10}\text{epg} + 1))$; Delahay 1995) was used to estimate mean adult worm burdens of 3170 (95% confidence limits: 1963–5117) and 3512 (95% confidence limits: 2104–5860) for treatment groups A and D respectively. These are consistent with establishment rates in previous experimental work (Shaw 1988b, Delahay et al. 1995) and levels of infection in wild Red Grouse (see Wilson 1979, Hudson 1986b, Shaw 1988b, Delahay 1995).

Within treatment groups, worm egg counts were

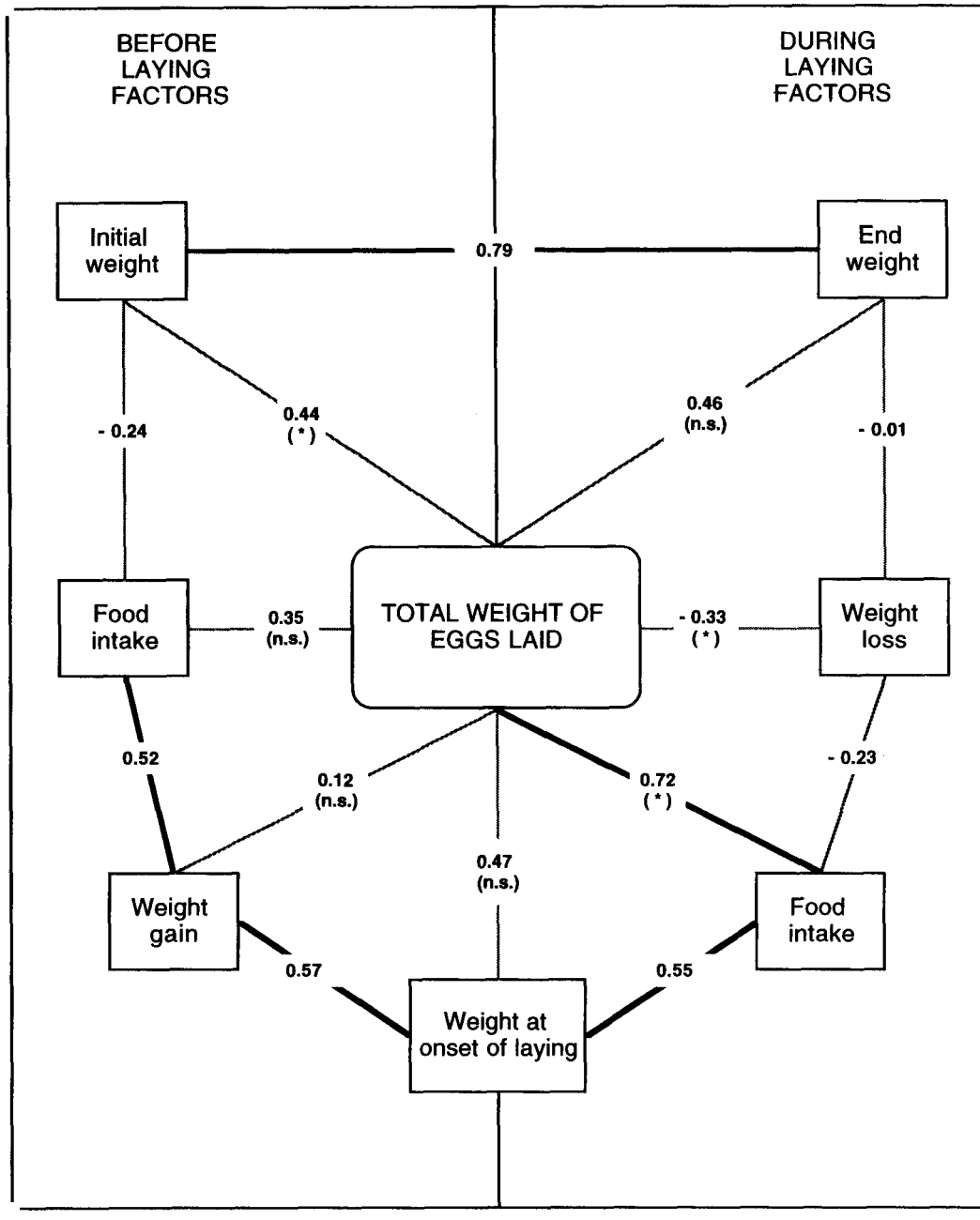

FIGURE 1. Associations between hen food intake, body weight, and the total weight (A) and number (B) of eggs produced. Numbers are Pearson correlation coefficients, bold lines indicate significant correlations ($P < 0.05$) and shaded lines non-significant correlations ($P > 0.05$). Parentheses show the significance of associations between hen egg production and weight or food intake tested by generalised linear models which corrected for hen age (ns = $P > 0.05$, * = $P < 0.05$).

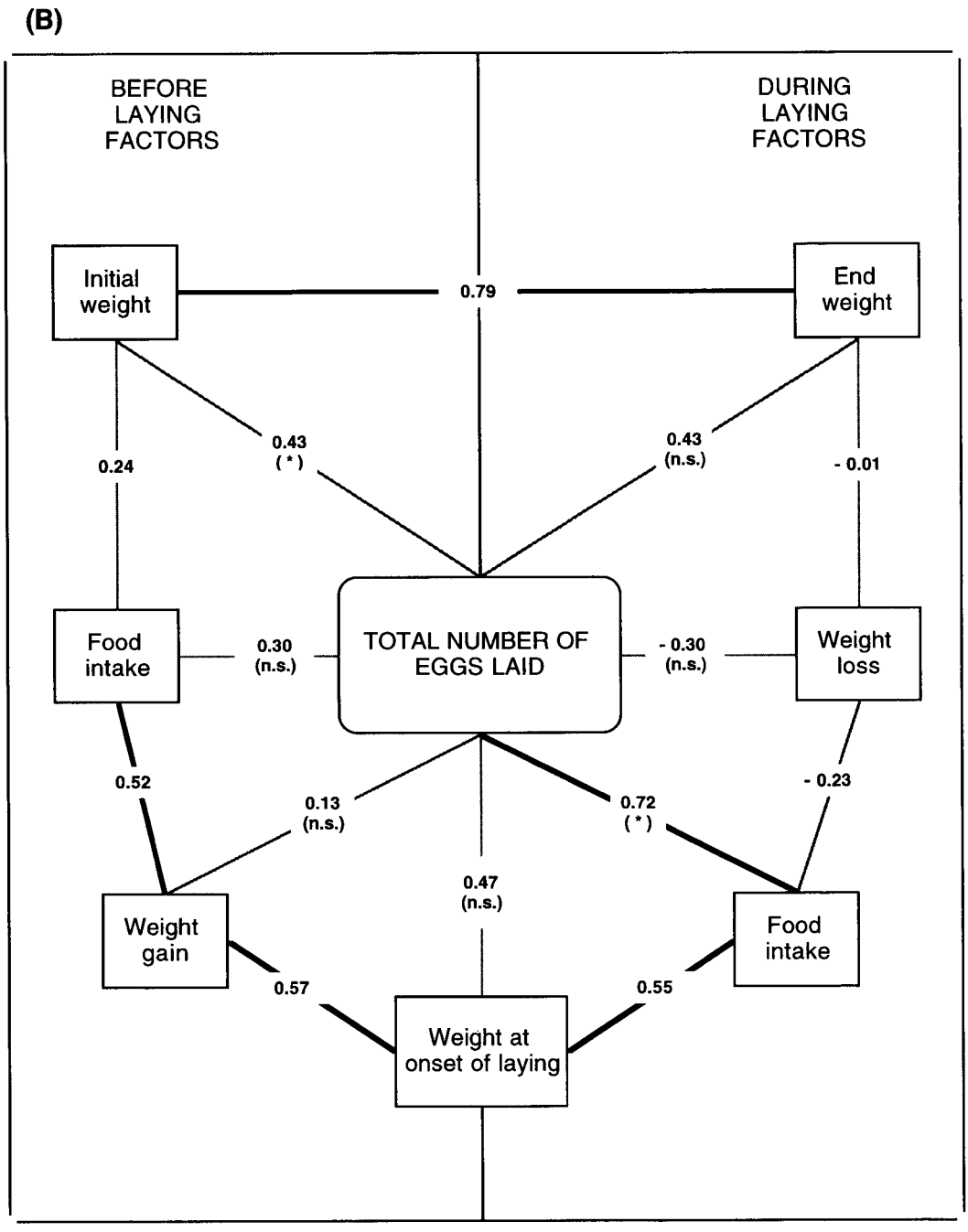

FIGURE 1. Continued.

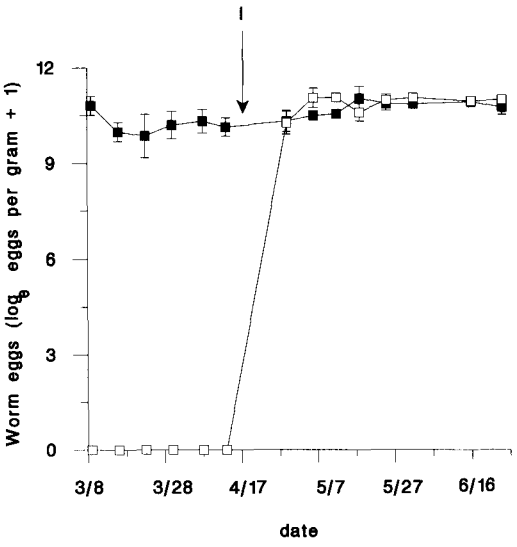

FIGURE 2. Mean counts of parasite eggs (log_e(*T. tenuis* eggs per gram of caecal faeces + 1)) from hen grouse in treatment groups A (■) and D (□). The arrow indicates the time of infection (I) for treatment group D.

not significantly correlated with measurements of hen food intake, body weight, condition or egg production (Pearson correlations, P always > 0.05). This agrees with evidence from other studies (Shaw and Moss 1990, Delahay et al. 1995) which show wide variation in resilience to *T. tenuis* infection among individual hosts.

EFFECTS OF PARASITES ON EGG PRODUCTION

Grouse egg laying began on 13 April and ceased on 22 June. The onset of laying for each bird was the number of days after the first egg of the season. The daily removal of eggs caused grouse to lay more eggs than they would in the wild.

Mean egg weight, egg volume and the proportion of deformed eggs per hen did not differ according to treatment group (t-tests, P always > 0.05).

Mean measures of egg production (Table 1) show that uninfected birds (group C) laid more eggs and produced a greater total weight of eggs than both groups of infected birds (groups A and D). However, these differences were not statistically significant (t-tests, P always > 0.05). After controlling for the effects of grouse age there was no significant relationship between treatment group and any of the measures of egg production listed in Table 1 (GLM Model 5, P always > 0.05).

EFFECTS OF PARASITES ON HEN FOOD INTAKE, BODY WEIGHT AND CONDITION

There were no significant differences in food intake, net weight change and condition scores of hens from different treatment groups, during either the pre-laying or laying periods (Table 2). Although group A birds already carried patent worm burdens at the start of the experiment, initial body weight and condition did not differ significantly from controls (t-tests, P always > 0.1). The mean food intake and body weight of infected and control birds increased before the onset of laying (Figs. 3A and B). Group A birds consumed similar amounts of food to uninfected birds (groups C and D) during the pre-laying period, but gained less weight. However, differences in weight gain and the efficiency of food conversion (food intake/weight gain) between treatment groups were not significant (t-tests, P always > 0.05). Also, comparison of regression coefficients showed no significant differences in the rates of weight gain or condition change be-

TABLE 1. Mean (± 1 SE) measurements of egg production by uninfected hen grouse (C), hens infected with 6,000 *T. tenuis* L3 in winter 1992–1993 (A) and in mid April-1993 (D). The onset of laying is the number of days after the first egg of the season.

	Treatment group		
	C	A	D
Onset of laying (days)	18.3 ± 8.4	18.2 ± 4.9	11.8 ± 1.5
Mean egg weight (g)	21.6 ± 1.3	21.4 ± 0.6	21.6 ± 0.7
Mean egg volume (cm^3)	21.1 ± 1.2	20.8 ± 0.5	21.2 ± 0.6
Number of eggs laid	15.8 ± 6.3	12.1 ± 3.3	13.8 ± 4.5
Rate of laying (eggs/day)	0.32 ± 0.1	0.30 ± 0.1	0.33 ± 0.1
Interval between eggs (days)	2.5 ± 0.4	3.7 ± 0.6	3.8 ± 0.7
Total weight of eggs (g)	330 ± 134	259 ± 70	313 ± 112

TABLE 2. Mean (± 1 SE) food intake, body weight and condition of hen grouse, for the five weeks before laying, and for the duration of laying. Control birds for means before laying comprised groups C and D. For measurements of weight change and peak weight during laying, values in the same rows with subscript letters all different are significantly different (one-way t-tests, $P < 0.05$).

Before laying	Infected birds (group A)	Control birds (groups C and D)
Initial body weight (g)	613 ± 28	598 ± 19
Food intake (g/Kg/48 hrs)	115.4 ± 5.7	117.3 ± 8.0
Weight gain (g/Kg)	58.3 ± 38.1	125.5 ± 32.1
Change in condition score	−1.0 ± 0.6	−1.1 ± 0.9
Body weight at onset of laying (g)	647 ± 32	671 ± 29

	Treatment groups		
During laying	A	D	C
Food intake (g/Kg/48 hrs)	130.3 ± 6.5	127.7 ± 9.5	116.5 ± 9.9
Weight loss (g/Kg/d)	2.7 ± 0.3	2.6 ± 0.5	3.4 ± 1.1
Change in condition score	−0.6 ± 0.8	−0.2 ± 1.2	−1.3 ± 1.7
Body weight at end of laying (g)	621 ± 43	552 ± 32	663 ± 11
Weight change during early laying (g/d)	1.3 ± 0.9[a]	−0.9 ± 0.4[b]	1.4 ± 0.8[a]
Peak weight (g)	666 ± 41[ab]	600 ± 25[b]	737 ± 32[a]
Weight change during late laying (g/d)	−1.7 ± 0.5[ab]	−1.3 ± 0.7[b]	−3.0 ± 0.7[a]

tween infected and control birds before laying (t-tests, P always > 0.1).

During laying both groups of infected birds consumed more food and lost less net body weight and condition than uninfected ones (see Table 2), although differences between treatment groups were again not significant (t-tests, P always > 0.05). There were also no significant differences between treatment groups in the conversion of food or body weight to egg production (t-tests, P always > 0.05). Controlling for the effects of hen age and initial body weight yielded similar results (GLM model 6, P always > 0.05).

There were, however, significant differences amongst treatment groups in the patterns of hen weight change during laying. After the onset of laying in most birds there was a phase of increasing hen body weight followed by a decline (Fig. 3). Regression coefficients for the rates of change in hen body weight and condition were calculated for each phase (see Table 2). The rate of weight gain in groups A and C was significantly faster than that of group D birds which actually lost weight during early laying (one-way t-tests; for A vs. D, $t_8 = -2.12$, $P = 0.03$; for C vs. D, $t_4 = -2.4$, $P = 0.04$). Control birds gained weight fastest and were significantly heavier than group D birds at their peak (t-test; $t_6 = -3.4$, $P = 0.02$). During the decline phase control birds lost weight at a faster rate than group D birds (one-way t-test; $t_4 = 2.22$, $P = 0.04$). After producing their final egg, hens from group D were significantly lighter than all other birds (one-way t-test; $t_{13} = 1.86$, $P = 0.04$).

DISCUSSION

THE RELATIVE IMPORTANCE OF CAPITAL AND INCOME TO EGG PRODUCTION

In the present study, grouse egg production was not significantly related to food intake or to weight gain during the five weeks before laying but was related to food intake during laying. These results differ from those of Savory (1975) and are consistent with Thomas's (1988) hypothesis that grouse egg production is heavily reliant on nutrients acquired as 'income' during laying rather than as 'capital' before laying. Nonetheless, egg

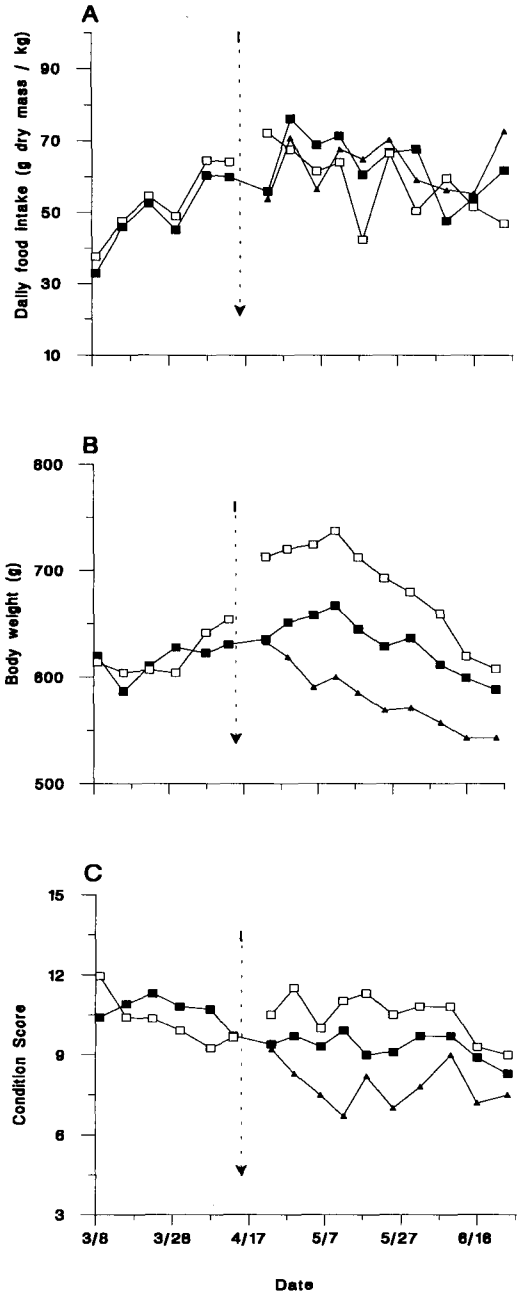

FIGURE 3. Mean food intake (A), body weight (B) and condition (C) of hen grouse infected with 6,000 *T. tenuis* L3 during the previous winter (■), in mid April (▲) and uninfected controls (□). The arrow denotes the mid April infection and the first egg of the season.

production was related to the hens' body weight at the onset of laying and, almost as strongly, to the weight of the hen several weeks before laying. This observation is confirmed by other work (R. Moss unpubl. data).

The weight gained by hens in the five weeks before laying may not entirely reflect the accumulation of reserves for egg production. Other contributions to pre-laying weight gain in Galliformes include development of the ovary and oviduct (Thomas 1982), hypertrophy of the liver (Thomas 1982, Pulliainen and Tunkarri 1984) and intestines, and water retention (Brisbin 1969).

The observation that hens consuming more food and losing less net weight between the onset and the end of laying produced a greater weight of eggs suggests that the acquisition of resources by hens during laying may limit egg production. If so, we might expect parasite-induced pathology occurring at this time to be more detrimental to egg production than such pathology occurring before laying. In fact, the opposite is the case (Shaw and Moss 1990).

EFFECTS OF PARASITES ON GROUSE EGG PRODUCTION, BODY WEIGHT AND CONDITION

Shaw and Moss (1990) showed that the effects of *T. tenuis* on egg production in captive Red Grouse varied according to the timing of infection. Hens with burdens of adult or developing worms during the pre-laying period consumed less food, gained less weight and subsequently laid fewer eggs than controls. The largest reductions in egg production occurred in hens that were infected about five weeks before laying began, whereas infection during the early stages of laying resulted in smaller reductions in egg production.

There were, however, no significant reductions in the number or total weight of eggs laid by infected birds in the present study. The difference between the two studies may be because Shaw and Moss (1990) infected grouse with 10,000 L3, while only 6,000 were used in the present study.

Grouse infected with *T. tenuis* L3 at the onset of laying in the present study showed very different patterns of weight change to those with adult worm burdens and the controls. Developing worms depressed weight gain and reduced the body condition of hens during early laying. By 29 days post infection, hens infected at the onset of laying were in poorer condition, on av-

erage about 19% lighter than uninfected birds and 10% lighter than birds with adult infections. Despite these differences, and despite the fact that their mean weight at the end of laying was 13 % less than that of other birds, the production of eggs by birds infected at the onset of laying was scarcely affected.

It seems paradoxical that egg production in grouse is so resilient to parasitic infection during a critical period of nutrient acquisition, especially since acute host energy imbalance can occur during the synchronous development of many *T. tenuis* larvae (Delahay et al. 1995). To compensate for this energetic drain without reducing egg production, laying hens would have to increase food intake, to mobilize 'capital' reserves or reduce the rate of egg laying. Although infected birds ate more food during laying than control birds, perhaps compensating for the effects of parasites, the difference was not significant. There was also no significant evidence that infected hens were reducing their daily energy requirement for egg formation by increasing the interval between consecutive eggs. Relatively rapid reductions in the body weight of infected hens during laying suggests that capital reserves were being used. Thomas (1983) found that Lesser Snow Geese (*Anser caerulescens*) lost substantial amounts of fat and carcass protein during egg laying. Grouse, however, generally have little depot fat (Thomas 1982) and so their weight loss was probably largely protein. In short, it seems that infected grouse hens maintained egg production by sacrificing body weight during laying.

One should be careful before extrapolating this result to wild birds. Captive grouse have unlimited access to a relatively digestible food with high concentrations of nutrients and are therefore not bulk (Moss 1989) or nutrient limited (Moss and Hanssen 1980). The impact of parasites on wild birds eating largely heather, a low-protein, high-fiber food, might be greater.

Retention of the ability to lay during periods of poor food may be a successful avian strategy (Ricklefs 1974). However, hens that begin incubation with lower body weight may be more likely to desert the nest (Steen and Unander 1985). Infected hen grouse that retain the ability to lay but finish in poorer condition may therefore be trading investment in current egg production for future incubation costs. The consequences of *T. tenuis* infection for incubation and maternal care of chicks have yet to be investigated.

CONCLUSION

This study confirms the relatively low impact of developing *T. tenuis* on egg production in laying captive grouse observed by Shaw and Moss (1990). This seems paradoxical since grouse appear to be 'income' breeders. However, the pathological effects of parasites in wild grouse existing on a lower plane of nutrition may be more severe.

In wild grouse the precise timing of the resumed development of previously arrested worm larvae may be critical in determining how much they affect egg production. If larvae develop synchronously during the pre-laying period (usually from early March to April) then relatively large parasite-induced reductions in clutch size may occur. If however, larvae develop at the onset of, or during, laying (usually from mid-April onwards) then they may have less effect upon hen egg production, but may threaten the quality of incubation and maternal care. The factors determining the magnitude and timing of the resumed development of overwintered worm larvae are therefore likely to be important determinants of how *T. tenuis* affects grouse egg production.

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APPENDIX. Generalized linear models (GLMs) tested for significant relationships between the dependent and independent variables. As the independent variables effected their influence sequentially, type I sums of squares were used, although conclusions did not differ for type III sums of squares. Models initially included all first order interactions, but for clarity they were dropped if not significant at the 5% level. Grouse food intake and weight change were correlated, and so to avoid confounding them they were generally not included in the same models.

Model	Dependent variables	Independent variables
1	N_e **or** W_e	a, bw_1, bw_2, bw_3, bw_4, t
2	N_e or W_e or R_e	a, bw_1, f_1, bw_2, f_2, t
3	W_e	a, bw_1, f_1, bw_2, bw_3, f_2, bw_4, t
4	N_e or W_e or R_e	a, bw_1, c_1, bw_3, c_2, t
5	N_e or W_e or R_e	a, t
6	W_c or F_c	a, bw_1, t

Where dependent variables, N_e = number of eggs laid, W_e = total weight of eggs laid, R_e = rate of egg laying, W_c = hen weight conversion to egg production, F_c = hen food conversion to egg production; and independent variables, a = hen age, bw_1 = hen weight at start of experiment, bw_2 = hen weight change before laying, bw_3 = hen weight at onset of laying, bw_4 = hen weight change during laying, f_1 = hen food intake before laying, f_2 = hen food intake during laying, c_1 = change in hen condition score before laying, c_2 = change in hen condition score during laying, t = parasite treatment group.