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SECOND AUTUMN WORKSHOP ON MATHEMATICAL ECOLOGY

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"Regulation and Stability of a Free-Living Host-Parasite System: *Trichostrongylus tenuis* in Red Grouse 1. Monitoring and Parasite Reduction Experiments"

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Regulation and stability of a free-living host–parasite system: *Trichostrongylus tenuis* in red grouse. I. Monitoring and parasite reduction experiments

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Summary

1. Intensive population studies were conducted for 10 years on red grouse (*Lagopus lagopus scoticus*) and the parasitic nematode, *Trichostrongylus tenuis*, in northern England. Winter loss was the key factor determining changes in grouse numbers, although breeding losses were also important.
2. *T. tenuis* had an aggregated distribution within the adult grouse population, even though the degree of aggregation was relatively low compared with other parasite systems. Recruitment of parasites into the adult worm population was dependent on grouse density.
3. Both winter loss and breeding losses were correlated with the intensity of parasite infection.
4. Experimental reduction in parasite burdens consistently increased breeding production and winter survival of grouse thus demonstrating that parasites cause increased winter and breeding losses.
5. The red grouse–*T. tenuis* system exhibits three conditions that will generate population cycles: (i) parasite-induced reduction in grouse breeding production, (ii) a low degree of parasite aggregation within the grouse population, and (iii) time delays in parasite recruitment.

Key-words: population cycles, parasite–host dynamics, *Trichostrongylus tenuis*, red grouse, parasite-induced effects.

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Introduction

The regular fluctuations in animal numbers known as population cycles have been described and studied in a range of species and are widely believed to be caused by density-dependent regulatory effects acting with a time delay (May 1981). Competition, predation, parasitism and dispersal have all been proposed as possible density-dependent factors although no mechanism has been clearly demonstrated in a natural animal population.

A number of grouse species are known to exhibit cyclic fluctuations in numbers. For example, the red grouse *Lagopus lagopus scoticus* (Lath.) exhibits cyclic changes in the number harvested each autumn on upland estates in England and Scotland (Potts, Tapper & Hudson 1984; Williams 1985), although this does not occur on all estates (Hudson, Dobson & Newborn 1985; Hudson & Dobson 1990). Predation, food, cover and shooting mortality are not believed to be the causative agents in the cycles (reviewed by Moss & Watson 1985, Lawton 1990). Two mechanisms have been proposed: firstly, intrinsic mechanisms acting through spacing behaviour (Watson *et al.* 1984) and, second, the influence of the parasitic nematode, *Trichostrongylus tenuis* (Eberth.), on breeding production and survival (Potts *et al.* 1984; Hudson 1986a,b; Hudson & Dobson 1990, 1991).

This study concentrates on the effects of *T. tenuis* on the population dynamics of red grouse in northern England. The objects of this paper are threefold: (i) to describe the dynamics of the grouse–*T. tenuis* system during a 10-year study and obtain population parameters for both host and parasite; (ii) to identify the key losses from the grouse population and examine whether these were related to intensity of parasite infection; and (iii) to test experimentally the effects of the parasite on the grouse. The companion paper (Dobson & Hudson 1992) uses these details to develop a population model that examines

whether the effects of the parasites are sufficient to cause oscillatory behaviour in red grouse numbers.

Materials and methods

LIFE CYCLE OF *TRICHOSTRONGYLUS TENUIS*

Adult *T. tenuis* inhabit the relatively large caeca (70 cm) of red grouse and their eggs pass from the host in the bird's caecal faeces. Embryos develop when the temperature exceeds 5 °C and the yield of infective larvae is dependent on temperature when moisture is adequate (Hudson 1986b; Watson 1988; Shaw, Moss & Pike 1989). Under optimal conditions development from egg to the third stage infective larvae is 7 days, although eggs may remain unhatched for several months. Third stage larvae migrate from the caecal faeces to the growing tips of heather (Wilson & Leslie 1911; McGladdery 1984) and grouse most likely become infected when they feed on their main food plant, heather.

PARASITE PREVALENCE, INTENSITY AND EGG PRODUCTION

Intensity of adult worm infection was estimated from gut samples collected from shot grouse. Worms were extracted and total number of worms per bird estimated by flushing the caeca with water, collecting the contents over a 210 μm gauze, diluting into 300 ml of water and subsampling three times in 10 ml (Wilson 1983; Hudson 1986a). To conform with previous studies (Wilson 1983; Hudson *et al.* 1985; Hudson 1986b; Hudson & Dobson 1988) mean intensity of parasite infection was determined as the geometric mean ($\log_{10} + 1$) worms per bird. Intensity of infection in immature grouse was determined in 1982 every 2 weeks from early July to mid December; samples were collected on shooting days and when young birds were accidentally killed by dogs. Dogs caught young grouse when the weather was hot and muggy, the sample was not considered to be biased.

Egg concentration in caecal contents were estimated from 422 grouse shot during August and September. From one of the two caeca, worm eggs were sampled by collecting approximately 1 g of caecal contents from the proximal end, this being the material most likely to be defaecated next. Eggs were counted using the McMaster egg counting technique described by Gordon & Whitlock (1939). Intensity of worm infection was determined from the second caeca and egg concentration expressed as eggs per worm g^{-1} caecal contents (this assumes there is no difference in worm numbers between caeca (Wilson & Leslie 1911)). The quantity of caecal faeces produced per day was determined by weighing faeces collected from night roosts. Additional caecal faeces were rarely found away from night roost sites.

To determine whether environmental conditions influenced year-to-year differences in the recruitment rate of parasites, details of daily rainfall for Gunnerside village (SD 951983; 3 km from the study area) and minimum and maximum temperature for Malham Tarn (SD 895672; 33 km from the study area) were obtained through the Meteorological Office at Newcastle Weather Centre.

POPULATION BIOLOGY OF RED GROUSE

Intensive population studies were conducted from July 1979 to July 1989 on an area of 0·8 km^2 of managed grouse moor, west of Gunnerside Ghyll, Swaledale, North Yorkshire. Serial correlations have been conducted on 107 years of bag record data from the estate and produced a damped correlogram with a significant negative coefficient at half the cycle period of 4·7 years (Potts *et al.* 1984; Hudson & Dobson 1990). Nisbet & Gurney (1982) classified this cyclic pattern as 'phase forgetting quasi-cycles' although we use the term 'cycle' within this paper to describe populations with a significant tendency to fluctuate in a regular manner.

With the aid of trained pointing dogs, total counts of the study area were conducted in April to estimate breeding density and again in July, when chicks were 7 weeks of age, to estimate breeding production (Jenkins, Watson & Miller 1963). In May of each year, nests of grouse were found using the dogs and clutch size and subsequent number hatched determined.

As with previous studies (e.g. Dempster 1975; Hudson 1986b), five periods of female loss were identified: (i) hunting mortality = k_0; (ii) overwinter loss = k_1; (iii) reduction in clutch size through an inability to lay a maximum clutch (12) = k_2; (iv) egg mortality = k_3; (v) chick loss = k_4. Overwinter loss included losses through natural mortality and also net loss through emigration and immigration. Losses are expressed as k-values:

$$k_i = \log_{10}(N_i/N_{i+1})$$

where N_i and N_{i+1} are the number of females entering and the number surviving the ith period. Overall loss, K_{tot} was calculated as the sum of k_0, k_1, k_2, k_3, k_4. The key factor was identified by plotting each loss as the dependent variable against total loss and calculating regression coefficients (b); the submortality with the regression coefficient closest to unity is considered the key factor (Podolor & Rogers 1975).

PARASITE REDUCTION EXPERIMENTS

Hudson (1986a) describes the experimental procedure used to reduce the intensity of infection

in wild grouse by catching, tagging and treating grouse in spring with the anthelmintic levamisole hydrochloride (Nilverm). Experiments were conducted 0·5 km south of the main study area. Relative survival of treated and untreated birds was measured as the proportion of tagged birds that survived from the time of capture in early spring (February–April) through the breeding season to the shooting season. Most grouse found dead on the study area during field work carried high parasite burdens and died in spring (further details in Hudson & Dobson 1988, 1989, 1990; Hudson, Dobson & Newborn 1992); these would be classified as overwinter loss (k_1). Worm burdens for treated and untreated grouse were estimated when grouse were shot, after 12 August and usually during August and September.

Mean brood size of treated and untreated females were compared when chicks were 7–8 weeks of age.

Results

DISTRIBUTION OF PARASITE NUMBERS PER HOST

All adult grouse inspected (2739 birds) and 99·2% (2723 birds) of immature grouse were infected with *T. tenuis*. The 21 uninfected birds were less than 2 months old. The distribution of nematodes per host was aggregated with the variance greater than the mean. No significant difference was found between the distribution of parasites in adult male and female grouse (contingency test, with classes of 1000 worms and classes greater than 9000 worms combined: $\chi^2 = 7{\cdot}18$; df = 9; $P > 0{\cdot}05$).

The pattern of nematode distribution in the adult grouse population varied between years with variance to mean ratios of 0·81–2·50 (10-year mean = 1·854, SE = 0·219); the variance to mean ratio was less than unity in 2 of the 10 years. Within the 8 years when the distributions were aggregated, the parameter k of the negative binomial distribution ranged from 1·2 to 5·8 (mean = 2·85, SE = 0·65). In comparison with other parasite distributions the degree of aggregation is relatively low, partly a consequence of high prevalence and intensity of infection (Anderson 1978).

ADULT WORM MORTALITY

Wilson (1979) presents data showing the decline of egg production by worms from captive grouse over 72 weeks following artificial infection with infective larvae. Maximum egg production was reached within 6 weeks of initial infection and then fell exponentially. If egg production per worm is constant, the decline in egg production would indicate a worm survival of 34% year^{-1}. Shaw & Moss (1989) also recorded a decrease in egg production with age of infection in captive grouse but also found that individual worm fecundity decreased with age so adult worm survival would be an underestimate. However, they also found differences in the fecundity of worms between captive and wild grouse making extrapolation between captive and wild birds difficult; this difference may arise as a consequence of captive grouse having shorter caeca than wild grouse (Moss 1972).

WORM FECUNDITY AND EGG PRODUCTION

Concentration of eggs in caecal contents was 6·05 eggs worm^{-1} g^{-1} (SE = ±1·109) for the 422 individuals sampled. There is evidence of a slight fall in egg production with intensity of worm infection in old grouse but this is addressed in more detail by Hudson & Dobson (unpublished). The quantity of caecal faeces produced at night roosts averaged 18·84 g bird^{-1} (SE = 1·06, $n = 52$), so overall worm fecundity can be calculated as 4×10^4 eggs worm^{-1} year^{-1}. Shaw & Moss (1989) estimate fecundity from *in utero* egg counts as 356 eggs female worm^{-1} day^{-1}, with a sex ratio of 1·35 males:females this gives a comparable figure of $5{\cdot}5 \times 10^4$ eggs worm^{-1} year^{-1}.

RECRUITMENT INTO ADULT WORM POPULATION

Serial sampling of grouse indicates that there are two periods of recruitment into the worm population (Hudson & Dobson 1990), the first in late summer, the second in late winter.

Summer recruitment

The uptake of worms during the summer (Fig. 1) is associated with the availability of infective larvae recovered from heather (Hudson & Dobson 1990). Summer recruitment was defined as the geometric mean number of worms in immature grouse (W_i) shot during September.

Variations between year in the size of this summer infection were positively correlated with minimum July temperature ($r = 0{\cdot}83$, $n = 10$, $P < 0{\cdot}01$) but not with rainfall or either maximum or minimum temperature in any one month. Variations between year were also positively correlated with density of grouse (adults and immatures) in the July of the previous year ($r = 0{\cdot}844$, $n = 10$, $P < 0{\cdot}01$) and intensity of parasite infection in adult grouse ($r = 0{\cdot}828$, $n = 10$, $P < 0{\cdot}01$). Partial correlations found that density in the previous year was more closely associated with level of summer infection when the effects of parasite intensity in adults and July minimum temperature were removed, although this relationship (Fig. 2) relies on a single outlying point (partial correlation with density, $r = 0{\cdot}78$, $P < 0{\cdot}02$; parasites in adult grouse, $r = 0{\cdot}68$, $P > 0{\cdot}5$; July minimum temperature $r = 0{\cdot}70$, $P < 0{\cdot}05$).

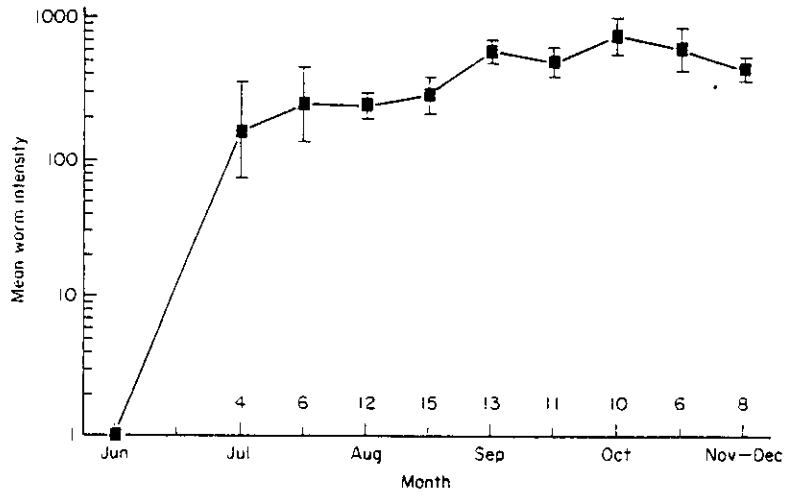

Fig. 1. Age–intensity curve of *T. tenuis* in immature red grouse for the period of summer infection in 1982 showing mean worm burden ($\log_{e+1}$) ± 1 SE: sample sizes are shown at the base of the figure and worm burden at hatching in June taken as zero. Summer recruitment was defined as the mean number of worms in immature birds in September.

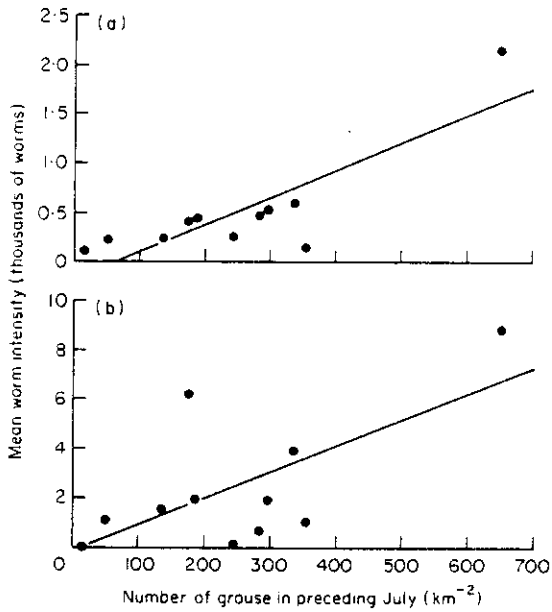

Fig. 2. Increase in parasite burden during (a) summer and (b) winter infection periods in relation to density of grouse in the preceding July.

Winter recruitment

Serial sampling of grouse reveals an increase in intensity of infection in late winter, when infective larvae cannot be recovered from vegetation (Watson & Hudson 1988; Shaw, Moss & Pike 1989; Hudson & Dobson 1990). The most likely explanation for this recruitment is that larvae that infected hosts during the autumn enter a period of hypobiosis (Shaw 1988), a period of temporary delayed development (analogous to diapause in insects) which is resumed during the following February or March.

The net winter recruitment (R_w) could not be assessed directly but was estimated indirectly from consecutive worm counts of adult and immature grouse. This was the difference between mean worm burdens in adult grouse in the autumn of year $t+1$ ($W_{a(t+1)}$) and mean worm burdens in all grouse in autumn of year t (W_t) and the level of summer infection measured in immature grouse in year $t-1$ ($W_{i(t+1)}$):

$$R_w = W_{a(t+1)} - W_{i(t+1)} - W_{(t)}$$

This assumes that infection rate in adult and immature grouse is similar, a reasonable assumption as there appears to be little resistance to infection (Watson, Lee & Hudson 1988; Shaw & Moss 1989; Hudson & Dobson, unpublished).

In all but one year, levels of winter recruitment were greater than summer recruitment. Year-to-year variations in winter recruitment were neither correlated with weather conditions nor previous levels of infection, but were correlated with density of grouse in the previous July (Fig. 2, $r = 0{\cdot}751$, $n = 10$, $P < 0{\cdot}01$), although this relationship is dependent on one outlier.

KEY FACTOR ANALYSIS

Changes in the size of the breeding population of hen grouse and the overall breeding success, measured as young per hen in July, are shown in Fig. 3.

Regression analysis identified overwinter loss (k_1) as the key factor because this explained most of the variation in annual loss. This is perhaps not surprising as k_1 covered the greatest time period (Table 1, Fig. 4). However, with the exception of shooting loss (k_0), regression coefficients between total loss and other losses (k_1-k_4) were also significant (Table 1). There were no significant trends in the k values during the period of study. Only k_1, overwinter loss, was correlated with grouse density immediately prior to the time the loss occurred ($n = 10$, $r = 0{\cdot}651$, $P < 0{\cdot}05$).

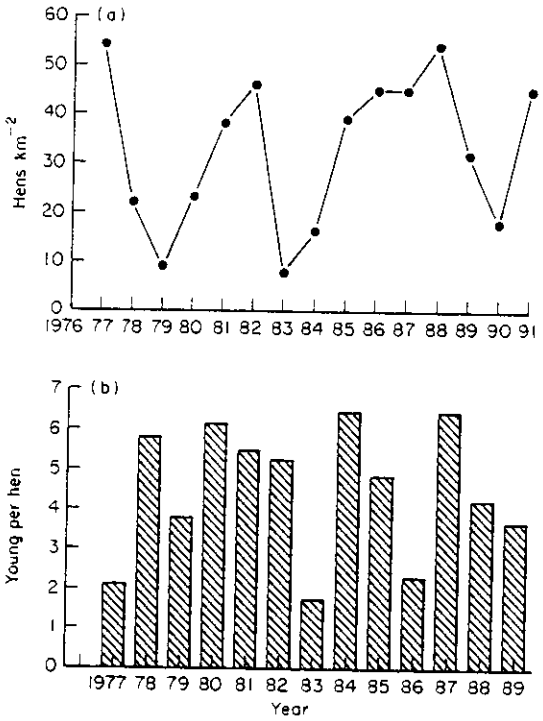

Fig. 3. Changes in the size of (a) the breeding population, and (b) breeding success of red grouse on the main study area.

Table 1. Correlation (r) and regression coefficients (b) between individual losses and total losses from the Gunnerside grouse population over 10 years. Significant coefficients ($P < 0{\cdot}05$) are indicated with an asterisk and k_1 considered the key factor with b closest to unity

			Losses				
	Value		k_0	k_1	k_2	k_3	k_4
Shooting	k_0	r	–				
Winter	k_1	r	0·11	–			
Clutch	k_2	r	0·37	0·70*	–		
Egg	k_3	r	0·13	0·65*	0·12	–	
Chick	k_4	r	0·38	0·78*	0·63*	0·43	–
Total	K_{tot}	r	0·38	0·93*	0·75*	0·68*	0·82*
	K_{tot}	b	0·07	0·59	0·05	0·09	0·20

Several of the k-values were correlated with each other. Overwinter loss (k_1) was positively correlated with each of the breeding losses; failure to lay a maximum clutch (k_2) was positively correlated with chick loss (k_4) (Table 1). These associations between k-values may reflect some common factor affecting all losses.

POPULATION LOSSES AND PARASITE INTENSITY

Overwinter loss (k_1) was significantly correlated with both grouse density and the intensity of worm infection in adult grouse (determined during the subsequent shooting season and thus including the period of winter infection) ($r = 0{\cdot}883$, $n = 10$, $P < 0{\cdot}001$, Fig. 5). Partial correlation analysis revealed that overwinter loss was more closely associated with parasite intensity (P) when the effects of density were removed (partial correlation k_1 versus intensity of infection with the effects of density removed $r = 0{\cdot}90$, $P < 0{\cdot}01$, k_1 versus $\log_{10}$ female density with the effects of intensity of infection removed, $r = 0{\cdot}71$, $P < 0{\cdot}05$).

Both egg mortality (k_3) and chick loss (k_4) were positively correlated with mean intensity of parasite infection (Fig. 5, k_3 and intensity of infection in breeding grouse estimated during the subsequent shooting season, $r = 0{\cdot}63$, $n = 10$, $P < 0{\cdot}05$; chick loss and intensity of infection $r = 0{\cdot}74$, $n = 10$, $P < 0{\cdot}02$). Failure to lay a maximum clutch (k_2) showed only a weak association with intensity of infection ($r = 0{\cdot}60$, $0{\cdot}1 > P > 0{\cdot}05$, $n = 10$).

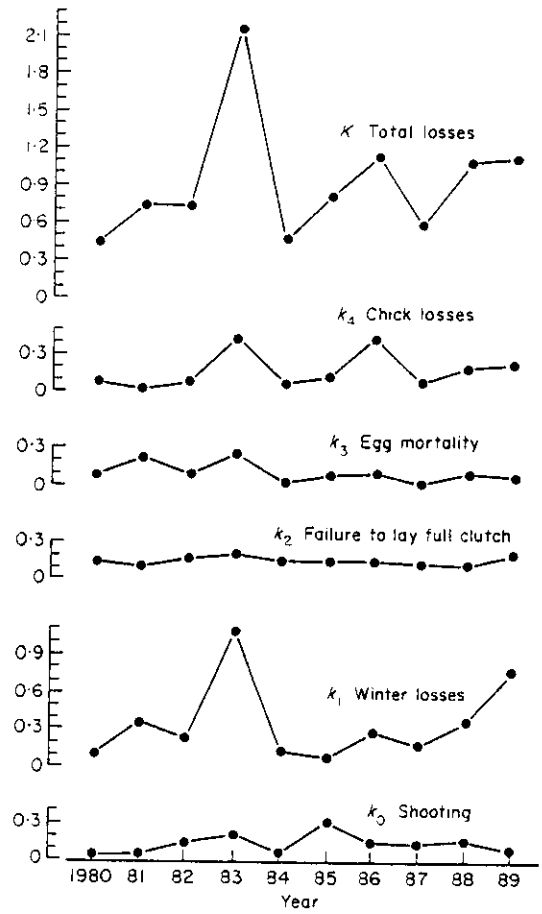

Fig. 4. Year-to-year changes in mortality and losses from the Gunnerside study population expressed as k values. While winter loss (k_1) was the factor, breeding losses (k_2, k_3 & k_4) were also associated with total loss (see Table 1).

PARASITE REDUCTION EXPERIMENTS AND GROUSE SURVIVAL

The relative survival of treated birds (from time of treatment to the shooting season) was significantly greater for grouse with reduced worm burdens (Table 2).

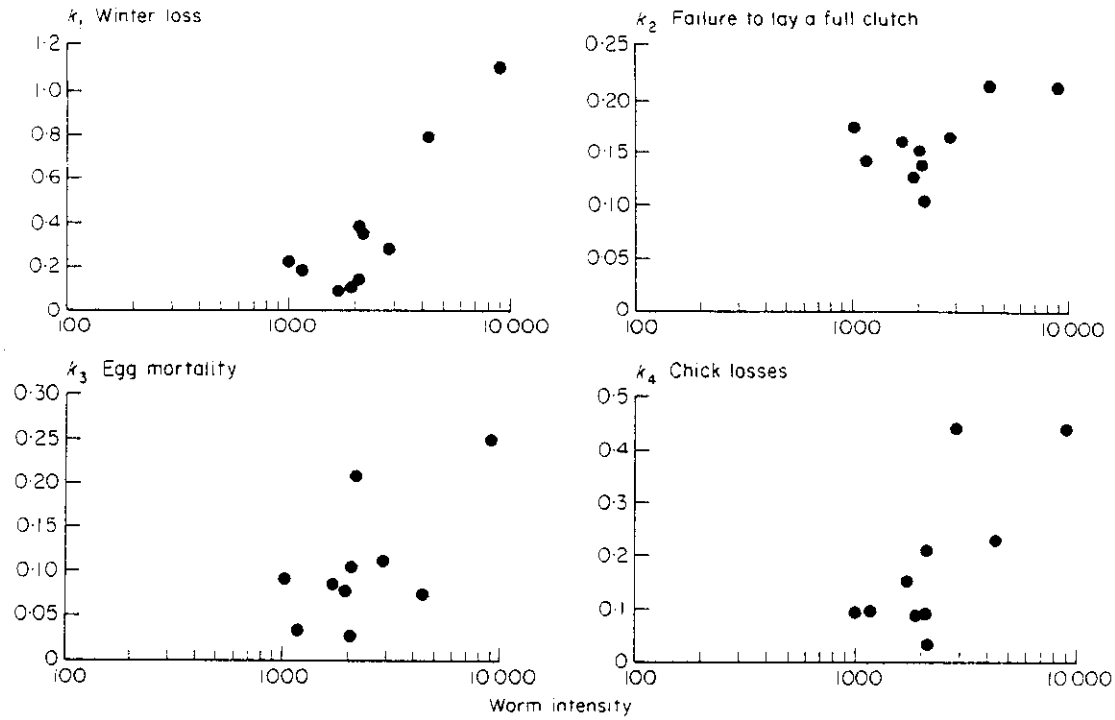

Fig. 5. The relationship between losses from the Gunnerside population and intensity of worm infection in breeding adult grouse. Winter loss, egg mortality and chick losses all show a positive correlation ($P < 0{\cdot}05$) while failure to lay a full clutch shows a weak positive association ($0{\cdot}1 > P > 0{\cdot}05$).

Table 2. Recovery of grouse either treated or untreated with an anthelmintic and subsequently shot in the following autumn. Overall, treated grouse with reduced worm burdens survived better than grouse with natural levels of infection

Year	Treatment	Number	Number shot	Number not shot	P*
1982–83	Treated	15	7	8	0·107
	Not Treated	86	23	63	
1983–84	Treated	93	38	55	0·048
	Not Treated	14	2	12	
1988–89	Treated	65	5	60	0·104
	Not Treated	36	0	36	
			Combined P value		<0·05†

* P estimated from Fisher Exact Test.
† Individual probabilities combined by $\chi^2 = -\Sigma\ 2\ \log_e$

Instantaneous mortality rates induced by the parasite ($\log_e(N_i/N_{i+1})$, after Krebs 1989) were estimated from the data presented in Fig. 5. The slope of the regression of female loss on worm burden provides an estimate of the instantaneous rate of 3×10^{-4} grouse worm^{-1} year^{-1} (SE $= 5 \times 10^{-5}$).

PARASITE REDUCTION EXPERIMENTS AND GROUSE BREEDING PRODUCTION

Female grouse with experimentally reduced parasite burdens consistently produced larger clutches than untreated females; this increase was significant in 4 of 8 years (Table 3a). Two-way analysis of variance found both a year ($F = 3{\cdot}80$, $P < 0{\cdot}05$, df = 7) and a treatment effect ($F = 21{\cdot}16$, $P < 0{\cdot}01$, df = 1) but no interaction between the two ($F = 0{\cdot}82$, $P = 0{\cdot}54$, df = 5). When an analysis of covariance was conducted with mean annual worm intensity as the covariate, then the effects of treatment and year were insignificant. However, worms were significant when considered alone ($F = 8{\cdot}474$, $P < 0{\cdot}02$, df = 14), suggesting that worms were a major factor reducing the breeding production of red grouse.

Hatching success of treated females was significantly greater than controls in 3 of 4 years (Table 3b). As with clutch size, two-way analysis of variance found both a year ($F = 4{\cdot}72$, df = 7, $P < 0{\cdot}05$) and treatment effect ($F = 36.8$, df = 1, $P < 0{\cdot}001$) but with no significant effect with worms as a covariate while overall worms were significant ($F = 12{\cdot}28$, df = 14, $P < 0{\cdot}02$).

Brood size was greater in each year of treatment. Once again, two-way analysis of variance found

Table 3. Clutch size and hatching success of anthelmintic treated and untreated female grouse. In every year treated birds consistently produced larger clutches with a greater hatching success. Values show mean ± SE and sample sizes in parentheses

Year	Treated female	Control females	Significance
(a) Clutch size			
1982	8·25 ± 0·62 (2)	7·93 ± 0·36 (14)	NS
1983	8·00 ± 0·34 (13)	5·28 ± 0·65 (11)	***
1984	8·80 ± 0·22 (15)	7·59 ± 0·47 (22)	*
1985	9·00 (2)	8·00 ± 0·49 (10)	NS
1986	8·67 ± 0·33 (3)	7·61 ± 0·42 (23)	NS
1987	8·40 ± 0·68 (5)	7·90 ± 0·38 (10)	NS
1988	10·4 ± 0·36 (11)	8·31 ± 0·51 (16)	**
1989	8·25 ± 0·25 (4)	6·86 ± 0·34 (7)	***
(b) Hatching success			
1982	0·97 ± 0·02 (8)	0·77 ± 0·09 (14)	NS
1983	0·75 ± 0·11 (13)	0·38 ± 0·13 (11)	**
1984	0·96 ± 0·02 (15)	0·92 ± 0·24 (22)	NS
1985	0·94 ± 0·06 (2)	0·76 ± 0·12 (10)	NS
1986	0·96 ± 0·04 (3)	0·75 ± 0·08 (23)	NS
1987	0·95 ± 0·05 (5)	0·91 ± 0·03 (10)	NS
1988	0·92 ± 0·05 (11)	0·68 ± 0·09 (16)	**
1989	1·00 ± 0·00 (4)	0·90 ± 0·04 (7)	*

* $P < 0·05$; ** $P < 0·01$; *** $P < 0·001$ using t-test.

both a year ($F = 6·13$, df = 7, $P < 0·001$) and treatment effect ($F = 85·8$, df = 1, $P < 0·001$) but with no significant effect with worms as a covariate while overall worms were significant ($F = 20·92$, df = 14, $P < 0·001$). These results demonstrate that parasites reduce the breeding production of female red grouse and that differences are consistent between years (Fig. 6). The experimental data provide a series of replicates from which the instantaneous rate of parasite-induced reduction on grouse productivity can be estimated as 5×10^{-4} grouse worm^{-1} year^{-1} (SE = 2×10^{-5}).

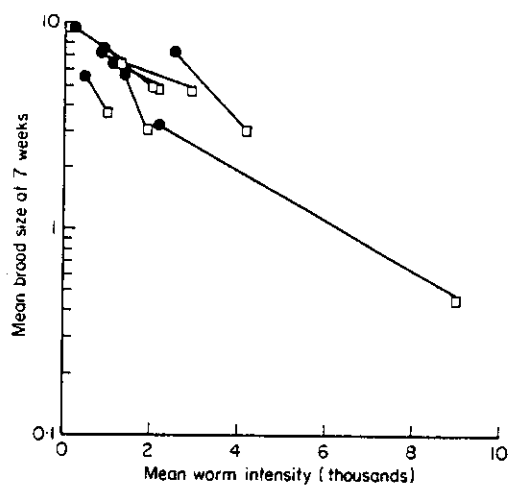

Fig. 6. Brood size of treated (•) and untreated (□) female grouse when chicks were 7 weeks of age in relation to mean worm intensity estimated 5 months after treatment showing an increase in breeding production following treatment. Standard errors omitted for clarity.

Discussion

The intensive monitoring and parasite reduction experiments described in this paper suggest that *T. tenuis* has a profound effect on the population dynamics of red grouse. Losses from the population were associated with intensity of parasite infection and experimental evidence demonstrates that these associations were one of cause and effect. These data support earlier models which have proposed that *T. tenuis* is an important agent influencing population changes in red grouse (Potts *et al.* 1984; Hudson *et al.* 1985). Before considering the dynamical consequences of these effects on the grouse population, the data from the monitoring programme and manipulation experiments are discussed.

KEY LOSSES AND INTENSITY OF PARASITE INFECTION

As with previous analyses of grouse populations (Dempster 1975; Watson *et al.* 1984; Hudson 1986b), overwinter loss was the key factor that accounted for changes in the total loss from the population. Breeding losses, failure to lay a complete clutch, egg mortality and chick loss were also associated with total loss and not surprisingly were intercorrelated, suggesting a common cause may be influencing the various population processess. Overwinter loss, egg mortality and chick loss were all correlated with intensity of parasite infection in breeding grouse. As correlations are not proof of cause and effect, they were tested through parasite reduction experiments.

7

PARASITE REDUCTION EXPERIMENTS

Previous population studies on grouse have confirmed that parasitism is an important cause of death (Jenkins, Watson & Miller 1963; Watson 1985; Hudson 1986a,b) and can be expected to be important throughout much of the range of grouse (Hudson & Dobson 1990). The experiments demonstrate that parasites are a factor influencing survival and productivity of red grouse, furthermore the consistent relationship between grouse productivity and parasitism in the experimental data illustrates that parasitism is a principal cause of reduced breeding production.

Earlier grouse studies in Glen Esk (Jenkins, *et al.* 1963) recorded average levels of infection and breeding production in grouse. Although they did not look for a relationship, the slope of the regression lines of parasites and breeding production was not significantly different to that recorded in this study ($F = 0{\cdot}516$, $P = 0{\cdot}48$). The hypothesis that parasites reduced breeding production and influenced population cycles on Glen Esk cannot be rejected.

Experiments on wild animals are frequently limited by logistic problems, and during the course of this study only parasite-removal experiments were conducted. Ideally, the experiments should have included a group of birds that had been artificially infected. Experiments with captive grouse have confirmed that addition of parasites will reduce breeding production (Shaw & Moss 1990). While results on captive grouse are interesting they cannot be applied directly to conditions in the wild because caecal development and size is significantly different from wild birds (Moss 1972).

The anthelmintic used in these experiments to reduce intensity of parasite infection (levamisole hydrochloride) has a broad spectrum and consequently would kill gut parasites other than *T. tenuis*. It is not a systemic anthelmintic, so would not have killed external parasites such as the sheep tick, *Ixodes ricinus* (Hudson 1986b,c). Red grouse are frequently infected with a number of cestode species although analysis of samples have shown these tend to be prevalent principally in late summer (P.J. Hudson, unpublished) and hence are not likely to make an impact on the breeding production or winter survival of grouse.

POPULATION DYNAMICS OF THE GROUSE–*T. TENUIS* SYSTEM

In a series of mathematical models, Anderson & May (1978) and May & Anderson (1978) consider the conditions under which parasites will regulate or destabilize a helminth–host system. For a directly transmitted nematode parasite such as *T. tenuis*, three conditions may destabilize the system and generate population cycles: (i) parasite-induced reduction in host breeding production; (ii) parasites distributed with a low degree of aggregation within the host population; and (iii) time delays in recruitment into the adult parasite population. All three of these processes were found to operate in this grouse–*T. tenuis* system.

The pattern of parasite distribution has interesting consequences for the dynamics of the host–parasite system in relation to the parasite-induced reduction in host fecundity. For a host population to have stable regulation by a parasite, May & Anderson (1978, Model D) show that the parasites' effects on host reproduction must be small relative to their effects on host survival and/or the parasite should be highly aggregated.

Alternatively, with regular and random distributions such a system has no stable equilibrium. In the grouse–*T. tenuis* system the pattern of parasite distribution exhibits a low degree of aggregation, in most years the variance to mean ratio of parasite distribution was low and in 2 years fell below unity. Even without developmental time delays, the significant impact of the parasites on host breeding production will tend to make the system intrinsically unstable and slight perturbations may lead to damped oscillations. Ultimately, the pattern of fluctuations observed will reflect the balance between the destabilizing effects of parasite-induced reduction in host fecundity and the effects of regulatory factors such as the parasite-induced host mortality, a feature explored in more detail in the accompanying paper (Dobson & Hudson 1992).

Within all nematode systems, time delays occur between production of the egg stage and recruitment of infective larvae into the host. If the larvae enter a period of arrested development, or hypobiosis, there may be further delay between recruitment and impact on the host. May & Anderson (1978 Model F) show that the destabilizing effects of the time delay depend on the pattern of parasite distribution within the host population. When this is close to a random distribution, as observed in this system, then even short time delays can generate stable limit cycles. While there will be time delays in the period of recruitment during the period of summer recruitment, the longest time delay will occur during the winter period of recruitment following larval arrestment. Observations and correlations indicate that arrested larval development effectively provides a time delay in the region of 3–6 months. Worm eggs deposited in autumn may develop into larvae which are subsequently ingested by grouse but remain in an arrested form until developing into adult worms in the following spring. This is supported by the experimental studies of Shaw (1988) and Shaw & Moss (1989). The interesting dynamical consequences of arrestment on the grouse–*T. tenuis*

system are investigated in more detail in Dobson & Hudson (1992).

CONCLUSIONS

Taking the three components of reduced host breeding production, a low degree of aggregation of parasites within the host population and the developmental time delays due to larval arrestment, it would seem that *T. tenuis* produces the population cycles observed in this grouse population. In the accompanying paper (Dobson & Hudson 1992) the basic Anderson and May models are developed to explore in detail the aspects of the system that could generate sustained population cycles.

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