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TRANSMISSION DYNAMICS AND HOST–PARASITE INTERACTIONS OF *TRICHOSTRONGYLUS TENUIS* IN RED GROUSE (*LAGOPUS LAGOPUS SCOTICUS*)

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ABSTRACT: Two components of the transmission dynamics of *Trichostrongylus tenuis* in red grouse are examined and quantified, namely parasite transmission rate and density-dependent reductions in egg production. Age-intensity data for birds of known age suggest that the rate of parasite uptake increases during the first 6 mo of a bird's life and this increase reflects an increase in feeding rate with age and exhibits no signs of self-cure. Analysis of these age-intensity curves permits us to estimate the transmission rate of the free-living infective stages. Reinfection rates of adults treated to reduce parasite intensities were not significantly different from infection rates of naive immature grouse. Secondary infections continued to rise over a period of 18 mo and this suggests that there is no strong host-mediated response against the parasite. Any density-dependent reduction in parasite fecundity is probably very weak and would act through interspecific competition between parasites. Initial analysis of worm egg production in relation to the intensity of worm infection found weak evidence of density-dependent suppression of egg production at high worm intensities. However, a more rigorous analysis found that such a relationship suffered from Type I errors and was a consequence of the aggregated distribution of the parasites. Any density-dependent suppression of parasite egg production is too weak to be detected and would only occur at high worm intensities. The potential density-dependent reductions in fecundity on the population dynamics of *T. tenuis* and red grouse are examined using a mathematical model. The model suggests that the presence of density-dependent reductions in worm fecundity could produce significant reductions in the propensity of the grouse–nematode system to exhibit population cycles. The sustained cycles observed in the long-term dynamics of the grouse populations in the study area suggest that density-dependent reductions in worm fecundity and establishment are either absent or only operating at levels that are not detectable in field studies.

In comparison with free-living helminths, parasitic helminths are highly fecund; in many species, the females may produce thousands of viable eggs per day. One apparent consequence of this is that parasite burdens in some host populations can reach remarkably high intensities; for example, in some red grouse populations the parasitic nematode *Trichostrongylus tenuis* can attain levels in excess of 30,000 worms per bird (Hudson, 1986b). However, the quantification of rates of parasite transmission from 1 host to the next is generally considered the most difficult aspect of any epidemiological study (Anderson and May, 1991).

In this paper, we describe a quantitative analysis of the main components of the transmission of the nematode *T. tenuis* in free-living red grouse populations. We examine the factors that involve direct interactions between the parasite and the host; specifically, we quantify the uptake of infective larvae by the hosts and the influence of worm burden on the rate at which eggs are produced. The influence of temperature and other variables on the development time and survival of the free-living larval stages has been described by Shaw et al. (1989), whereas factors affecting the establishment in the host have been examined by Shaw and Moss (1989).

Rates of egg production by parasitic worms are known to be highly variable (Anderson and Schad, 1985; Shostak and Scott, 1993). Such variation could be influenced by a number of factors, including differences between worms, although in general 2 mechanisms are believed to be dominant. First, density-dependent suppression of egg production through competition between worms for resources, and second, the immunological or nonspecific response of the host to the infection. These potential regulatory mechanisms can have profound effects upon the population dynamics of host and helminth parasite and consequently have important implications for control (Keymer, 1982; Smith, 1985).

Levels of intraspecific competition for space, food, or other resources will depend predominantly on current helminth intensity; in contrast, the host's immune response to the parasite will be a more complex function of the hosts' cumulative exposure to infection (Anderson and May, 1985; Woolhouse, 1992). Identifying the difference between these 2 mechanisms within a helminth host system is not easy and cannot be readily determined from patterns of primary infection because intensity and exposure will be correlated, irrespective of the underlying mechanisms.

To test for host-mediated reductions in worm fecundity and establishment, we utilize comparative data on primary and secondary infections. In a system where competition for resources is the regulatory mechanism, reinfection rates should be a simple function of current parasite intensity and will be similar in both naive and treated individuals. In contrast, in a system where a host-mediated immune response is primarily responsible for parasite regulation, reinfection rates will be a function of past exposure. If parasite burdens were previously high, reinfection rates will be significantly lower in treated compared to naive individuals.

Unfortunately, detecting any density dependence in the fecundity of parasite populations is a problem fraught with statistical biases; these are mainly produced by the aggregated statistical distribution of parasites in the host population (Keymer and Slater, 1987; Shostak and Scott, 1993). However, it is also possible that density-dependent reductions in host fecundity or survival operate at a subtle intensity, which makes their influence profound but their detection difficult. It is also possible that analyses will imply strong evidence of density-dependent reductions in parasite fecundity, but the regulation of the parasite population operates in another part of the parasite's life cycle.

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The influence of differences in transmission rate and density-dependent reductions in parasite fecundity on the dynamics of the system are explored using a modification of a mathematical model, previously applied to the dynamics of the grouse–*T. tenuis* system (Hudson et al., 1985; Dobson and Hudson, 1992). This paper is part of a series of papers that describe the long-term population dynamics of the parasite and its host. Hudson, Newborn, and Dobson (1992) investigated the interaction between the adult stages of the parasite and red grouse survival, whereas Dobson and Hudson (1992) examined the dynamical consequences for the population.

MATERIALS AND METHODS

Study sites

The birds used in this study were shot on grouse moors in the north of England between 1982 and 1990. The data for the study were principally collected from Bleasdale (Lancashire), Bolton Abbey (North Yorkshire), Conistone (North Yorkshire), Dallowgill (North Yorkshire), Gunnerside (North Yorkshire), Ramsgill (North Yorkshire), and Woodhead (South Yorkshire).

Age-intensity infection

Broods of grouse were located on the main study area at Gunnerside, North Yorkshire, using trained pointing dogs. The dogs pointed individual chicks hiding in the heather when the chicks were 2–15 days of age; each chick was individually tagged using small numbered ptagial wing tags. When tagged grouse were shot during subsequent shooting seasons, the age of the grouse at death was determined from when the grouse had hatched, the viscera removed, and intensity of worm infection estimated.

Analysis of worm intensity and egg counts

Worm eggs were sampled by taking approximately 1 g of cecal material from the proximal end of the ceca (this being the cecal material most likely to be defecated next by the bird). All samples came from grouse shot during August and September; they were aged as either immature birds hatched that year (<4-mo-old) or adult birds (>15-mo-old). Eggs were counted using the McMaster egg counting technique described by Gordon and Whitlock (1939) and used in previous studies by Hudson (1986a, 1986b).

In each case the intensity of worm infection was determined from the second ceca, there being no significant difference in worm intensities between ceca (Wilson, 1911; Watson, 1988). Worms were extracted by removing ceca and washing the contents over a 210-μm sieve. Material collected from the sieve was diluted with 300 ml water, mixed, and subsampled into 3 × 10-ml samples or until a minimum of 100 individuals had been counted from subsamples. The total number of worms per bird was then estimated from these counts. The techniques are described in full by Wilson and Wilson (1978) and Hudson (1986a). In accordance with previous studies, worm intensity is presented as the geometric mean worms per bird.

Primary and secondary infections

Grouse were captured during March and early April by dazzling the roosting birds at night with a strong quartz–halogen lamp; they were caught in a net and then treated orally with Levamisole hydrochloride (method described in Hudson [1986a] and Hudson, Newborn, and Dobson [1992]). Each bird was individually marked with a numbered ptagial Quadtag. This procedure was replicated 15 times on grouse populations in North Yorkshire, Lancashire, Highland, and Borders regions. On 1 estate in Invernessshire, a sample of several hundred grouse were treated in consecutive years from 1988 to 1990 and samples taken of shot birds in autumn that had been treated either in the previous spring, or the spring before this one, to determine whether treated individuals show a continued increase in parasite infection.

During the subsequent shooting season (particularly 12 August to 30 September), a sample of immature (3–4-mo-old), adult (>15 mo with a primary infection) and treated adult grouse (>15 mo, secondary infection) were collected and intensity of worm infection determined. The sample of immature birds represented a primary summer infection that had occurred in naive individuals over the preceding 3–4 mo. Treated individuals represent a sample of adult birds treated in spring and then exposed to infection between April and the following August/September and are referred to as a secondary infection. The period of exposure was some 6–10 wk longer than for the immature grouse that hatched in the June of that year, but it includes the period of time when the primary infection is occurring and the main period when larvae were active on the vegetation (Watson, 1988; Hudson and Dobson, 1990). If a bird possesses an immunological memory, then the previous presence of worms should reduce subsequent rates of establishment; secondary infections should exhibit lower levels of infection than primary infections. Alternatively, if there is no immunological memory and density dependence is mediated only through parasite competition for resources, then uptake rates in primary and secondary infections will be similar. Furthermore, secondary infections will continue to rise and a year later, i.e., 18 mo after treatment, parasite burdens should be significantly greater than they were in the previous year.

Individual egg production by grouse

Twenty-seven grouse were caught in the spring, radio-tagged, and followed during June in 1982 and 1983. Roost sites of radio-tagged grouse were pinpointed at night and cecal material collected the following morning. Whereas cocks may roost close to the female and brood, the use of radio tracking allowed accurate pinpointing of the female's roost pile. Worm eggs per gram of cecal material were estimated for each bird roost pile and geometric means and variance calculated for individual birds.

Regulation of parasite fecundity

The most straightforward way to determine initially if increased worm burdens lead to reductions in rates of parasite fecundity is to consider the relationship between worm eggs in feces and intensity of infection. If net egg output is directly proportional to intensity of worm infection, then a linear model passing through the origin would be expected (Anderson and Schad, 1985). However, if increased parasite burdens lead to reductions in parasite fecundity, then a power function with an exponent significantly less than unity will provide the best description of the data.

There are 2 ways this analysis may be undertaken. In the simplest case, a power function can be fitted to all the data and confidence limits calculated for its exponent. Keymer and Slater (1987) have shown that attempts to identify density-dependent effects in parasites may often be confounded by the statistical distributions of parasites in their host populations and this technique will tend to artificially reduce the slope below unity. The aggregated distributions of the parasite population may cause pronounced Type I errors in studies that ignore the statistical distributions of the parasites when attempting to examine density dependence.

Shostak and Scott (1993) have recently suggested an alternative method for estimating density-dependent reductions in parasite fecundity. This method takes into consideration variations in fecundity between worms and calculates the confidence limits on parasite fecundity based on the observed variation in hosts with low parasite burdens. We used the data for hosts with less than 200 worms to calculate the 95% confidence limits on eggs per worm per gram of feces. These confidence limits were then used to calculate the variability in worm fecundity over the complete range of observed worm burdens.

Population dynamics of *T. tenuis*

The dynamics of a host–parasite interaction that includes both free-living infective larvae and arrested or hypobiotic larvae may be described by 4 coupled differential equations. The model framework we have used is based on the Anderson and May (1978) and May and Anderson (1978) models for parasitic helminths and their hosts. A full derivation of the models discussed here is given in Dobson and Hudson (1992). The model describes changes in the numbers of the hosts, H, free-living parasite eggs and larvae, W, arrested or hypobiotic larvae, A, and adult worms, P. The parameters of the model are described in Table I.

TABLE I. Population parameters for *T. tenuis* and red grouse.

Parameter*	Symbol	Estimated value (range)
Grouse fecundity	a	1.8 (0–2)/yr
Grouse mortality	b	1.05/yr
Parasite fecundity	λ	11 (9.2–11.5)/yr
Adult worm mortality	μ_P	1.0 (0.8–1.2)/yr
Arrested larval mortality	μ_A	0.5/yr
Mortality of free-living stages	γ	6.5–13/yr
Duration of arrestment	$1/\theta$	0.19–0.25/yr
Proportion of larvae that enter arrestment	σ	0–1
Parasite pathogenicity	α	3×10^{-4}
Parasite reduction in host fecundity	δ	5×10^{-4}
Aggregation of parasites in host	κ	1.0 (0.5–1.8)
Transmission rate	β	0.11 (0.06–0.15)/larvae/host/yr
Transmission constant	H_0	60 – 120 (γ/β)

* The derivation of the parameter estimates for the parasite and grouse are discussed in Hudson et al. (1992) and Dobson and Hudson (1992).

$$dH/dt = (a - b)H - (\alpha + \delta)P \quad (1)$$

$$dW/dt = \lambda P - \gamma W - \beta WH \quad (2)$$

$$dA/dt = \sigma\beta WH - (\mu_A + b + \theta + \alpha P)A \quad (3)$$

$$dP/dt = \theta A + (1 - \sigma)\beta WH - (\mu_P + b + \alpha)P - \alpha(P^2/H)[(\kappa + 1)/\kappa] \quad (4)$$

A modification of this model may be used to examine the transient dynamics of infection in immature grouse over their first 6 mo of life. We assume that the cohort of young grouse that hatch in the first week of June of each year act as "tracer birds" that sample the available larvae in the habitat. Given a constant establishment rate, the rate at which worm burdens accrue in the young grouse will depend on the feeding rate of the young grouse, and the availability of infective larvae on the vegetation; this in turn is a function of the density of the adult birds at the beginning of the season and their worm burdens.

Two alternative assumptions may be made about the feeding rate of young grouse. Either the feeding rate of grouse is constant and unchanged with age, or alternatively, we can assume that feeding rates are a function of body size. Both of these possibilities are explored here. Body mass measurements of young grouse were obtained from the principal study site at Gunnerside (Hudson, 1986b). A standard von Bertalanffy growth curve was fitted to each of these sets of data. The function describes the weight at age, t, in terms of the asymptotic adult weight, Wght_∞, and 2 parameters a and b. Standard nonlinear curve-fitting techniques were used to estimate a and b in the following equation:

$$\text{Wght}_t = \text{Wght}_\infty (1 - e^{-at})^{1/b} \quad (5)$$

The birds achieve their final adult body size by the age of 4–5 mo. If we assume that ingestion rate is directly proportional to body size, then we can rescale the transmission rate in Equations 2, 3, and 4, to be a variable determined by the feeding rate of young birds. The rate which β changes with age is then the integrand of Equation 5:

$$d\beta_t/dt = a/b[(\beta_\infty/\beta_t)^{1/b}]\beta_t \quad (6)$$

Here β_∞ is the transmission rate to fully grown grouse and β_t is the transmission rate to a chick of age t. This relationship was used to estimate the rate at which grouse of different ages ingest the infective stages of the parasite.

RESULTS

Estimation of transmission rates

The data obtained on worm burdens in young grouse were used to estimate the transmission rate β. In each case, the model of the grouse and parasite populations was seeded with the known density of adult birds per km^2, the parasite intensity in adult birds, and the number of chicks per km^2 on the moor at the beginning of June (just after hatching). Estimates of transmission rate were then made assuming β to be constant, β_c, or dependent upon the age of the chicks, β_t. We determined the value of β that minimized the squared difference between observed and expected values of worm burdens and the values of β that produced the lowest and highest estimates, i.e., the value that produced a curve that passed through the extreme data values for each of 4 yr (1982, 1984, 1985, 1987).

The estimates of age-dependent transmission, β_t, were consistently higher than the estimates for constant transmission, β_c; they provided a better description of the observed data in 3 of the 4 yr (Fig. 1; Table II). Furthermore, the age-dependent estimate of transmission, β_t, is closer to the independent estimate ($\beta = 0.1$) obtained by determining the threshold density of grouse required to continuously sustain an infection by the parasite population (Table II; Dobson and Hudson, 1992). This suggests that estimates of transmission rates need to consider age-dependent feeding rates of young grouse.

Primary infection, age-intensity curve

The estimate of transmission rate can be used to produce an expected age-intensity curve for *T. tenuis* infection in grouse. The mean parameter estimates provide expected age-intensity curves that are similar to the observed age-intensity curve for the cohort of grouse that hatched on Gunnerside in 1982 (Fig. 2b). The intensity of infection increases continuously with age and there are no signs of the spontaneous "self-cure" reduction of worms, characteristic of helminth systems in mammalian hosts (Wakelin, 1984; Anderson and Medley, 1985; Anderson and May, 1991; Hudson and Dobson, 1994).

Secondary and primary infections compared

The intensity of secondary infections in adult grouse that had been treated to reduce their worm burdens was correlated with the intensity of primary infections in immature grouse during the same year (Fig. 3) and was not significantly different from a 1:1 relationship ($F = 15.31$, $P > 0.10$). Obtaining levels of exposure from wild birds of known age was not possible within this system although if a host-mediated response was important we could expect an association between levels of secondary infection and parasite intensity in untreated adult grouse. No such association was found ($r = 0.228$, $P = 0.43$, NS), suggesting that rates of reinfection were not influenced by previous levels of infection.

These results show that reinfection rates of treated individuals were not different from naive, primary infections. Parasite intensity in treated grouse continued to rise after treatment, grouse collected 18 mo after treatment carried significantly greater worm burdens than the same cohort of treated grouse carried 6 mo after treatment (Table III; $t = 3.01$, $P < 0.001$). These data are not consistent with the hypothesis that a host-mediated response is limiting establishment of worms in infected hosts.

Variation in cecal egg counts from individual hosts

Fecal samples were collected from radio-tagged birds to examine the variability in worm-egg production rates from the

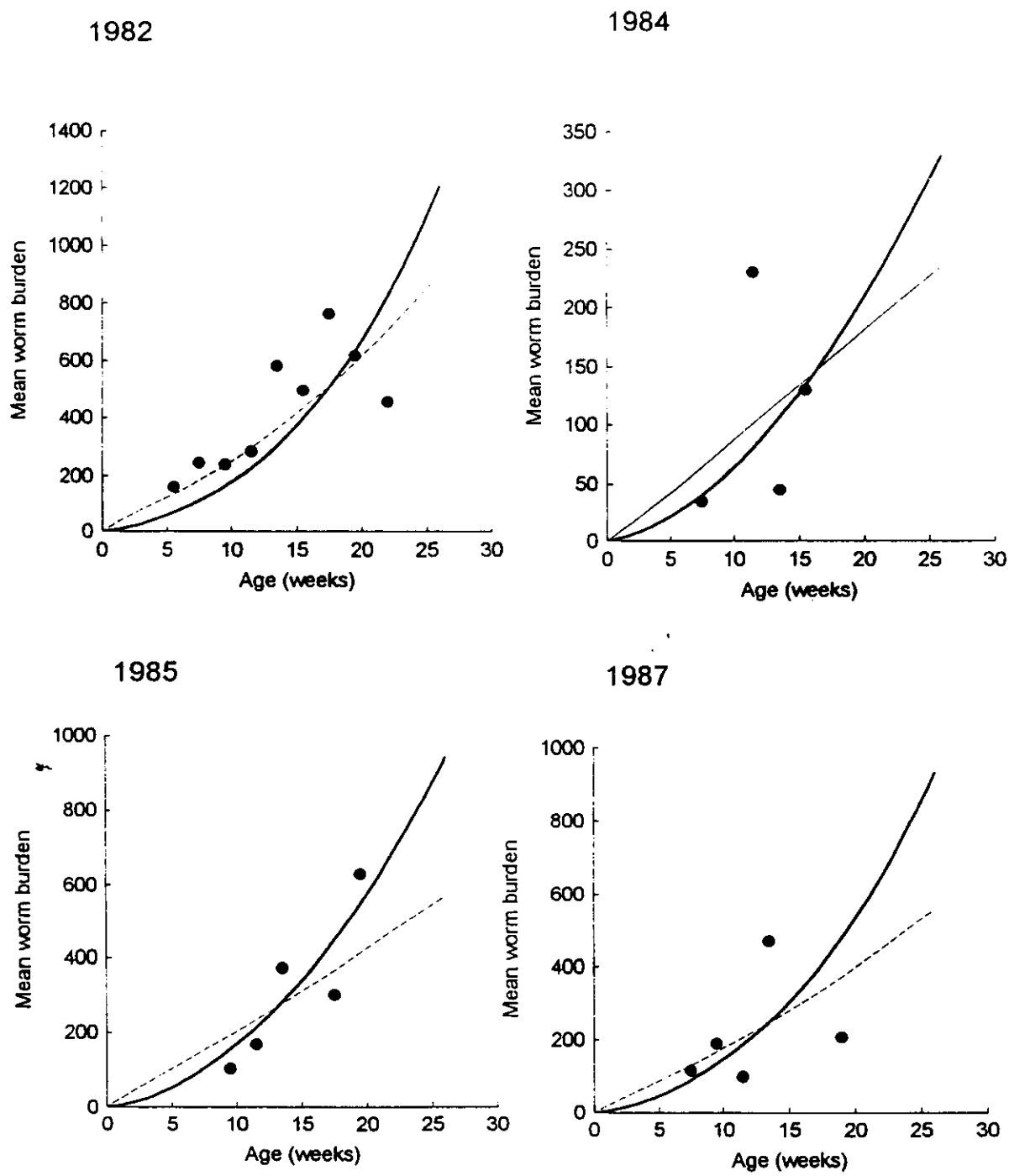

FIGURE 1. The fit of the 2 transmission models to the observed age-intensity curves for *T. tenuis* in young red grouse. The data are for each of 4 different years and in each case the dotted line illustrates the fit of a model that assumes transmission is constant and independent of feeding rate, whereas the thick line assumes the worm intake is an age-dependent function of feeding rate. Parameter estimates are in Table II.

same host. Consecutive daily collection of cecal material from individual grouse during June showed inherent variability in egg production. The relationship between $\log_{10}$ variances and $\log_{10}$ means of egg per g cecal material from the 27 individuals was linear, with a slope of 1.76 ($r = 0.98$, $n = 27$, $P < 0.001$, Fig. 4) and was significantly different from a slope of unity ($t = 2.833$, $P < 0.01$). If the day-to-day counts were randomly distributed, this relationship would have a slope of unity; the

TABLE II. Estimates of the transmission rate, β, from the data according to a constant transmission model and an age-dependent transmission.

	Constant β*			Age-dependent β		
Year	Min.	Max.	Best	Min.	Max.	Best
1982	0.010	0.024	0.015	0.025	0.065	0.035
1984	0.003	0.016	0.007	0.010	0.100	0.020
1985	0.004	0.013	0.008	0.016	0.032	0.022
1987	0.004	0.018	0.009	0.012	0.050	0.025
Mean			0.009			0.025

* In each case, we give the value of β that provides the best fit to the observed data and the 2 extreme estimates that bound all the observed data points for each year. The mean values fall within the extreme values estimated for all years.

slope of 1.76 indicated high variability in egg production and in practical terms makes accurate measures of worm intensity from cecal samples difficult.

Worm fecundity and egg production

The intensity of infection and worm fecundity, measured as number of eggs per gram of cecal material (epg), were determined for a total of 422 grouse shot in August and September. Initially, we examined the data using standard nonlinear curve-fitting techniques. Overall, egg production per worm was not dependent upon worm intensity ($r = 0.003$, $P > 0.05$, $n = 422$). When only data from adult grouse are considered, there was a significant decrease in per capita egg production with intensity of infection ($r = 0.157$, $P < 0.05$, $n = 299$), but no such relationship was found for immature grouse ($r = 0.114$, $P > 0.05$, $n = 112$). This is perhaps not surprising because few immature grouse carry a heavy parasite infection, whereas 16 (5%) of the mature grouse carried worm infections greater than 10,000 worms per bird. When all the data from this survey are combined, the relationship is best described by a power function whose exponent is less than unity. This suggests there is a tendency for parasites in hosts with high worm burdens to produce fewer eggs.

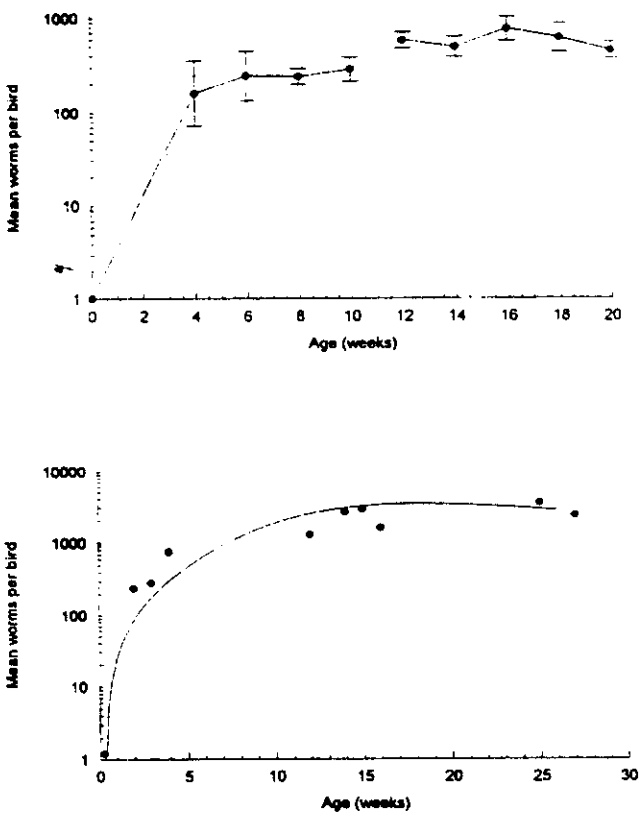

FIGURE 2. Observed and predicted age-intensity curves for young grouse: (a) observed age-intensity curve with mean and standard error shown for known aged birds collected from Gunnerside in 1982 after Hudson, Newborn, and Dobson (1992); (b) expected age-intensity curves for model of constant rate of ingestion (dotted) or age-dependent rate of ingestion (solid line) with points indicating observed data. The age-dependent curve begins to decline at 18 mo when heavily infected birds begin to die and cause the overall mean to decline.

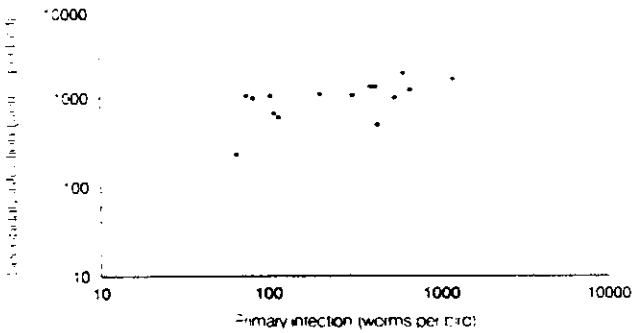

FIGURE 3. Relationship between geometric mean secondary worm burden (adult grouse treated 6 mo earlier) and geometric mean primary infections (immature grouse in first year of life, not treated). The relationship is significant ($r = 0.584$, $P = 0.022$) but the slope is not significantly different from a 1:1 line. Points represent the mean for each case, 1 population in 1 yr.

Shostak and Scott (1993) have suggested a more rigorous technique for detecting density dependence in data collected from parasitic helminths. The confidence limits on the expected distribution of eggs per gram of feces were calculated using their method. This analysis implies that none of the data sets provide significant evidence for the presence of density-dependent reductions in worm fecundity.

The population dynamics of reductions in egg production

All of the empirical evidence indicates that density-dependent reductions in egg production by *T. tenuis* in red grouse is

TABLE III. Geometric mean worm intensities in grouse sampled 18 mo after treatment were significantly greater than grouse sampled 6 mo after treatment, showing that reinfections continue to increase, a prediction consistent with the hypothesis of a low immunological response.

	Period	Mean	SD	n
Treated spring 1989 Sampled autumn 1989	6 mo	790	2.41	151
Treated spring 1989 Sampled autumn 1990	18 mo	1,425	1.96	22
Treated spring 1990 Sampled autumn 1990	6 mo	291	2.93	27

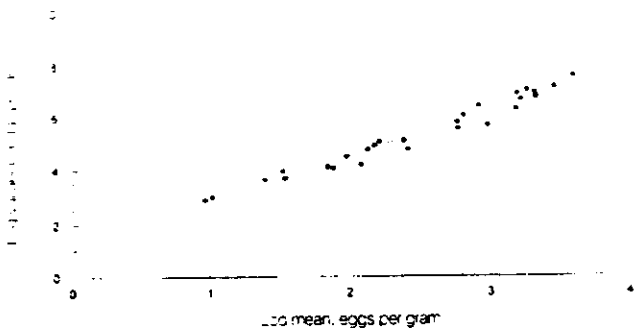

FIGURE 4. Day-to-day variability in egg production from individual grouse. The relationship between log variance and log mean egg production has a slope of 1.76 (best-fit linear model), indicating a high degree of variability that increases with increasing worm burden.

either very weak or nonexistent. Nevertheless, because density-dependent effects are often hard to detect in ecological data, we developed a mathematical model to examine the sensitivity of helminth population dynamics to different levels of density dependence. The impact of density-mediated reductions in worm fecundity may be explored by including the effect into a mathematical model for the population dynamics of red grouse and *T. tenuis* (Dobson and Hudson, 1992; Hudson et al., 1992). The simplest function that provides a description of the observed data is a power function. The reductions in egg production as worm burden increases may be described by the following function:

$$\lambda = \lambda_0\{1 + (P/H)\,[(\kappa + 1)/\kappa]^{c-1}\} \qquad (7)$$

Here c is the exponent of the power function and the expression may be included into the model by simple modification of Equation 2:

$$dW/dt = \lambda_0 P\,\{1 + P/H\,[(\kappa + 1)/\kappa]\}^{c-1} - \gamma W - \beta WH \quad (8)$$

The effect of density-dependent reductions in fecundity on the transient dynamics of the model are illustrated in Figure 5 for 3 different values of c in Equation 8. These simulations suggest that low levels of density dependence have their biggest effect in situations where interactions between the parasite and its host would otherwise lead to divergent cycles of abundance. Further discussions of the robustness of the model are discussed in Dobson and Hudson (1992).

The system can display 3 different patterns of dynamic behavior; when transmission rates are low, the parasite fails to establish, and the host population either grows exponentially, or rises to a density determined by other regulatory factors, such as food, predators, or cover. If the parasite establishes in a host population where other regulatory forces act, the populations of parasites and host either settle to a stable point or exhibit damped or diverging cycles that eventually lead to extinction of 1 of the 2 species. The dynamics of host–parasite systems are very sensitive to the presence of density-dependent worm fecundity. In particular, small changes in aggregation can produce dramatic differences in the behavior exhibited by the parasite and host system. All of the parameter estimates obtained from the empirical data for *T. tenuis* in red grouse suggest that regulation is either very weak or undetectable with logistically realistic sample sizes. Furthermore, the pronounced

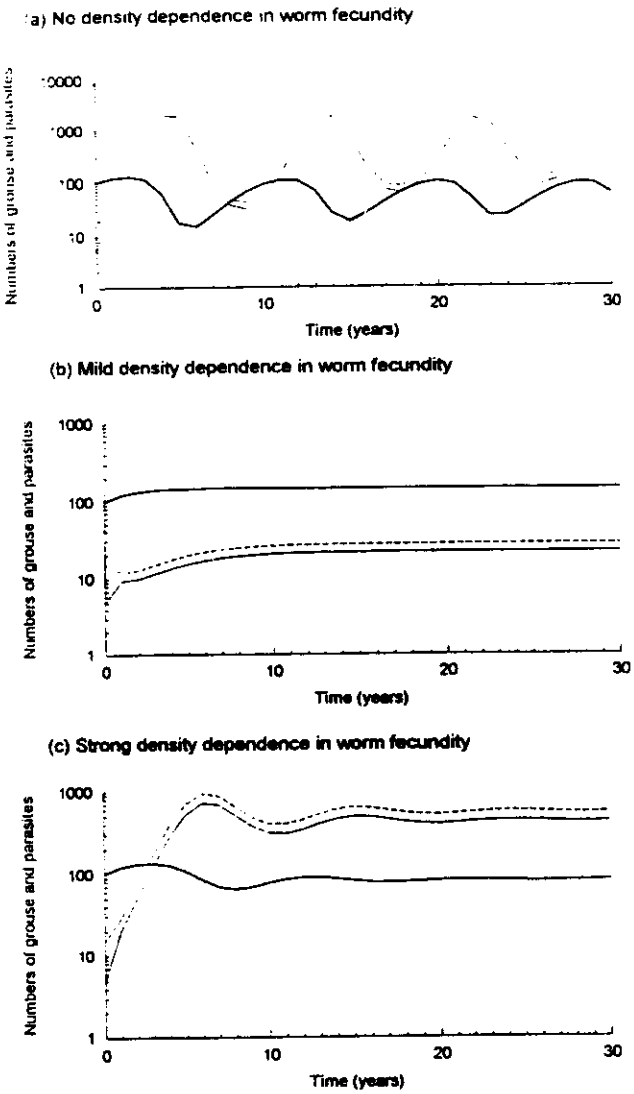

FIGURE 5. The effect of density-dependent reductions in worm fecundity on the dynamics of the grouse–*T. tenuis* system in a model designed to mimic grouse populations. The parameters used in each simulation are given in Table I and assume that fecundity is a power function of worm burden: (a) shows the case for no density-dependent effects, b = 1; (b) for mild density-dependence where b = 0.95; and (c) for strong density-dependence where b = 0.8. In each figure the thick line shows changes in host density, the thin line changes in adult worm burden, and the broken line mean adult worms and arrested larvae.

cycles of host and parasite abundance seen in long-term studies of *T. tenuis* and red grouse imply that significant levels of density-dependent reductions in worm fecundity are unlikely to operate in the system.

DISCUSSION

The experiments described in this paper have attempted to quantify the 3 major components of parasite transmission that involve an interaction between the parasite and its host, e.g.,

egg production rates, infection rates, and establishment of larvae in a new host. In each of these processes, the host's prior experience of infection may lead to a reduction in parasite transmission rates. All of the experiments suggest that such density-dependent effects are either absent or operate at very low intensities. Moreover, a mathematical model that examines the population dynamics of the grouse–*T. tenuis* system suggests that if significant density-dependent effects diminished parasite fecundity, then the host–parasite system would not show the propensity to cycle observed in most field studies (Potts et al., 1984; Hudson, 1992; Hudson, Newborn, and Dobson, 1992). This mathematical evidence confirms that density-dependent processes that directly restrict parasite fecundity and establishment are unlikely to be important in the dynamics of *T. tenuis* and red grouse.

The data on worm burdens of grouse chicks collected over their first year of life allowed us to obtain estimates of the transmission rate for *T. tenuis*. We know of no other study of a parasitic helminth in a wild population where this has been undertaken. Indeed, there are only a few examples of estimates of transmission rates from microparasite studies (McCallum, 1982; Hone et al., 1992). The analysis suggests that estimates of force of infection need to consider the worm burdens present in the other hosts present in the habitat and the age-dependency in the ingestion rates of young birds.

The age-intensity curve for *T. tenuis* in red grouse shows no evidence of a spontaneous cure to infections unlike *T. tenuis* infections in other host species, such as domestic chickens (Watson et al., 1988), grey partridge (R. Connan and D. Wise, pers. comm.), and quail (Moore et al., 1986). The evidence from reinfection rates is consistent with the hypothesis that the hosts produce a relatively poor immune response to this infection. The difference between grouse and other hosts is interesting and may be related to the relatively poor protein content of their diet compared with other hosts (V. Apanius, pers. comm.). Experimental studies found an increase in globulins following *T. tenuis* infection in captive, well-nourished grouse (Wilson and Wilson, 1978), although the authors cautioned about applying such results to wild populations. The field experiments described in this paper suggest that any response is either short-lived or of limited efficacy in preventing reinfection.

The experiments that compared infection rates in immature birds, adult birds with reduced worm burdens, and control adult birds with natural infections suggested that any immunological response in grouse to *T. tenuis* is of limited capacity and has no detectable effect on the establishment of larvae into previously infected hosts. A clearer understanding of this system could be obtained if worm intensity was known prior to treatment and could be compared with rate of secondary infection. Replication between individuals would provide an alternative view of the system which could be compared to the replicates taken between populations in this study. However, this approach would not be simple within this system because levels of infection based on egg counts in cecal feces are inaccurate and there are technical problems of catching and recatching wild individuals. Laboratory studies on many systems could improve our understanding, although there are clear differences in the gut structure of captive and wild hosts (Moss, 1972) that would make the application of laboratory results to wild hosts difficult. Even so, Shaw and Moss (1989) showed that captive grouse acquired little effective immunity to reinfection after a challenge with a single dose of infective *T. tenuis* larvae. An alternative approach would be to remove animals from the field, treat and subsequently challenge them with an infection, and determine the nature of the response in seminatural conditions, a technique used by Keymer and Tarlton (1991) to study *Heligmosomoides polygyrus* infections in mice.

Identifying the presence of density dependence within any biological system is not simple and whereas a number of studies have suggested that density dependence is infrequent (Dempster, 1983; Strong, 1986; Stiling, 1988), others have shown that density dependence can only be detected when time delays and spatial scales are taken into consideration, or when sufficient data are available (Hassell, 1985, 1987; Hassell et al., 1989; Turchin, 1990). Within parasite–host systems, variations between hosts and parasites together with methodological errors make the detection of density dependence difficult. Previous studies on hookworms (Anderson and Schad, 1985) have shown that such depression can occur at low levels of infection, perhaps as a result of the hosts' response to infection rather than competition between helminths. Other studies have used reinfection rates to determine the presence of an immunological response, particularly in schistosomes (Hagan, 1987), although evidence in other groups is not always clear (Anderson, 1986; Bundy, 1988). The lack of density dependence within this system is interesting and confirms earlier work by Shaw and Moss (1989) on *T. tenuis*, who counted the number of eggs in utero in females and found no decrease in the number of eggs per female with intensity of worm infection.

To reveal the nature of underlying density-dependent mechanism requires an experimental approach in which parasite intensity, resources (nutrients and space), or the host's immune response are manipulated. Furthermore, different mechanisms may be operating to influence rates of establishment, arrestment, mortality, and fecundity. Because laboratory studies may not readily extrapolate to field conditions, manipulations are frequently limited to a series of infection experiments. With such information and an understanding of the dynamics of each life history stage Grenfell et al. (1987) estimated the relative importance of different regulatory mechanisms in the cattle nematode *Ostertagia ostertagia* and found that only fecundity was dependent on intensity; mortality was dependent on exposure, whereas establishment was time dependent.

As with many helminth systems, the high variability in egg production suggests that egg counts are poor measures of intensity of worm infections. Egg count data could allow the qualitative division of individuals into those with high and low levels of infection, but quantitative assessment should be considered with great care. For example, Moss et al. (1990) produced a significant relationship between estimates of *T. tenuis* burdens and egg counts; they then used this relationship as a basis for estimating worm burdens in birds where only egg count data were available. Whereas this relationship was statistically significant, it showed a high degree of variability at low counts, failed to pass through the origin, and was based on a relatively low sample size ($n = 44$) with only a single individual exceeding 4,000 worms per bird. Whereas the relationship may hold true for individuals with low to intermediate worm burdens, results based on egg counts should be treated with caution par-

ticularly for individuals with worm burdens above 6,000 worms per bird.

Density-dependent egg suppression of *T. tenuis* was not demonstrated in adult grouse and comparisons of rates of infection in treated grouse provided no evidence for a pronounced immune response to *T. tenuis*. The data simply indicate that if any host-mediated response is present, it operates without any memory of past infection. Modeling of the system and examination of various density-dependent mechanisms show that a low level of density-dependent egg suppression can have a significant effect on the dynamics of the grouse–*T. tenuis* system and could be sufficient to reduce the oscillatory behavior of this system. As such, the primary regulatory factor in the parasite population is probably parasite-induced host mortality, and heavily infected hosts are likely to die before there is any significant reduction in egg production.

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