Regulation of nematode fecundity in the ring-necked pheasant (*Phasianus colchicus*): not just density dependence

D. M. TOMPKINS* *and* P. J. HUDSON

Institute of Biological Sciences, University of Stirling, Stirling FK9 4LA, UK

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SUMMARY

Patterns of nematode fecundity were investigated for infections of the caecal worm *Heterakis gallinarum* in the ring-necked pheasant (*Phasianus colchicus*). Worm length was a good predictor of parasite fecundity. After controlling for worm length no other factors, including parasite intensity, were related to worm fecundity. Density dependence in worm size was detected in natural infections at parasite intensities above a threshold of 96 worms (worm size decreased with increasing parasite intensity). However, below this threshold, worm size actually decreased with decreasing parasite intensity (inverse density dependence). The interaction between density dependence and inverse density dependence in regulating the development and subsequent fecundity of *H. gallinarum* worms in ring-necked pheasants was demonstrated in an infection experiment. Density dependence was observed in the stunted growth of worms in heavily infected hosts, relative to worms in lightly infected hosts. Inverse density dependence in worm size was the common pattern across hosts by the end of the experiment, when parasite intensities were below the density dependence threshold. This is the first study to document both density dependence and inverse density dependence in parasite fecundity in the same host–helminth system.

Key words: *Heterakis gallinarum*, inverse density dependence, parasite, threshold.

INTRODUCTION

A general characteristic of parasitic helminth populations is their relative degree of temporal stability in undisturbed host populations, implying some form of regulation (Hudson & Dobson, 1995). While density dependence can operate at any stage in the parasite life-cycle, and so regulate abundance, density-dependent reductions in worm fecundity are thought to be the primary regulatory mechanisms responsible for such stability (Keymer, 1982; Anderson & May, 1985). Supporting evidence comes from a wide range of host–helminth systems (e.g. Michael & Bundy, 1989; Goater, 1992; Gulland, 1992; Stear, Park & Bishop, 1996), although there have been several studies which failed to detect such density dependence (e.g. Shaw & Moss, 1989; Coyne, Smith & Johnstone, 1991; Hudson & Dobson, 1997; Marcogliese, 1997), and at least one which identified inverse density dependence in helminth fecundity (Quinnell, 1992). Such variation, however, is not surprising given the complex nature of host resistance to parasite infections (Woolhouse, 1992). Indeed, 2 conflicting mechanisms may operate to influence helminth fecundity. First, induced host responses may reduce worm fecundity at high parasite intensities while, second, variation among individuals in predisposition to infection, affecting both parasite survival and parasite fecundity, may result in lower worm fecundity in the lighter infections of more resistant hosts than in the heavier infections of more susceptible hosts (Keymer & Slater, 1987).

The aim of this study was to investigate the relationship between parasite intensity and the fecundity of the caecal nematode *Heterakis gallinarum* (Schrank, 1788) in ring-necked pheasants (*Phasianus colchicus*), and to determine if fecundity patterns emerge during the course of an infection. Nematode fecundity is often related to worm size; reduced fecundity in smaller worms has recently been recorded in several host–nematode systems: *Trichuris muris* infections of mice (Michael & Bundy, 1989), *Rhabdias bufonis* infections of toads (Goater, 1992), and *Ostertagia circumcincta* infections of sheep (Stear *et al.* 1996). Thus, our first step was to determine whether worm size is a good predictor of *H. gallinarum* fecundity. We then investigated the potential density dependence in *H. gallinarum* fecundity by determining whether negative correlations between worm fecundity and parasite intensity are apparent in natural infections, and by exploring these relationships by creating, through experimental infections, groups of lightly and heavily infected hosts.

Estimates of *per capita* fecundity for parasitic helminths have traditionally been obtained by quantifying parasite egg production in host faecal samples, and then counting expelled worms following anthelmintic treatment. Estimates, however, are frequently inaccurate since the variance in faecal

* Corresponding author: Tel: +01786 467808. Fax: +01786 464994. E-mail: dmt1@stir.ac.uk

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egg production increases faster than the parasite intensity (Anderson & Schad, 1985; Keymer & Slater, 1987). Furthermore, these sampling biases coupled with the aggregated distribution of helminths in host populations will tend to suffer from Type I statistical errors and give the illusion of density dependence (Keymer & Slater, 1987; Shostak & Scott, 1993; Hudson & Dobson, 1997). These problems were avoided to some extent in this study by using the direct method of counting *in-utero* eggs as our measure of parasite fecundity, and by using parasite size as an index of fecundity when analysing for density-dependent patterns.

MATERIALS AND METHODS

Estimation of the intensity of Heterakis gallinarum *infection*

Viscera were removed from 171 pheasants shot from 2 populations in Scotland during November and December 1996. The first population was at Drumdruills estate, in Stirlingshire, and the second population was at Dumnaglas estate, in Invernesshire. All birds were approximately 6 months of age and were sexed by plumage characteristics. *H. gallinarum* worms were removed from one of the two caeca of each bird by sequentially washing the caecal contents through a coarse sieve (1·4 mm), to remove host tissue, and a fine sieve (0·2 mm), to collect the worms. Sieving is a standard method for the collection of intestinal worms (Doster & Goater, 1997). Worms recovered were counted under 25 × magnification of a binocular microscope, and identified as either larval, male, or female by the presence/absence of genital structures (Clapham, 1933). Parasite intensity was estimated as twice the single caeca worm count. To test the accuracy of this estimate, all of the worms were removed from the entire intestinal tract of 20 of the 171 birds.

Estimation of parasite fecundity

The number of viable eggs contained within female *H. gallinarum* worms was used as our index of nematode fecundity. An egg was assumed to be viable if the embryo developed through to the infective stage after being maintained for 21 days in 0·5 % formalin solution at 21 °C; a standard protocol for embryonating *H. gallinarum* eggs (Lund & Chute, 1972). Worm fecundity was estimated per host from 10 randomly chosen female worms. Eggs were counted by breaking open the worms and liberating their genital tubes under 100 × magnification. Fecundity estimates were log-transformed to conform to the assumptions of parametric statistics.

Estimation of parasite size

The body lengths of all the female worms collected were measured to the nearest 0·025 mm using an ocular micrometer under 40 × magnification.

Infection experiment

To identify worm development rates and investigate temporal patterns of worm fecundity in heavy versus light infections, pheasants were experimentally infected with *H. gallinarum*. Forty-two hen pheasants were caught in February 1997 (9 months of age) and maintained on wire mesh in individual cages. The birds were supplied with ad-lib food (gamebird maintenance pellets), water, and grit (medium flint grit). During the first week after capture the birds were treated with an anthelmintic feed supplement, Flubenvet Intermediate™, to eliminate all gut parasites prior to the experiment. To test the efficacy of the anthelmintic 2 birds were culled after treatment. They were both worm-free.

Twenty of the hen pheasants were each given a single infection of approximately 500 embryonated *H. gallinarum* eggs, whilst the remaining 20 were each given a single infection of approximately 100 embryonated *H. gallinarum* eggs. Nematode eggs, suspended in 1 ml of saline for each dose, were administered orally via a tube into the birds crop. The oral dosing procedure was carried out 3 weeks after the anthelmintic treatment when there should have been no trace of the anthelmintic drug. Worm development was monitored over the following 50 days by culling 4 birds from each treatment group at 10 day intervals. All worms present in the caeca were counted and measured.

Statistical analyses

For the naturally infected hosts from which more than 2 female worms were collected, female worm length was analysed with respect to log-transformed parasite intensity using the nonlinear regression module on Statistica™ v5.1, 1997. All other statistical procedures were carried out using S-PLUS™ v4.0, 1997. Unless stated otherwise, analyses were conducted using generalized linear models (glm) with gaussian error distributions. Significance levels were calculated, using χ^2 tests, from the deviance explained by each factor following stepwise deletion (Crawley, 1993). Parasite intensity was calculated as the mean across all individuals (including uninfected birds), and host location was controlled for by including it as a two-level factor in all relevant analyses.

RESULTS

Intensity and distribution of Heterakis gallinarum *infection*

Single caeca from the guts of 171 male and female ring-necked pheasants were examined for nematode worms. From these, a total of 4385 *H. gallinarum*

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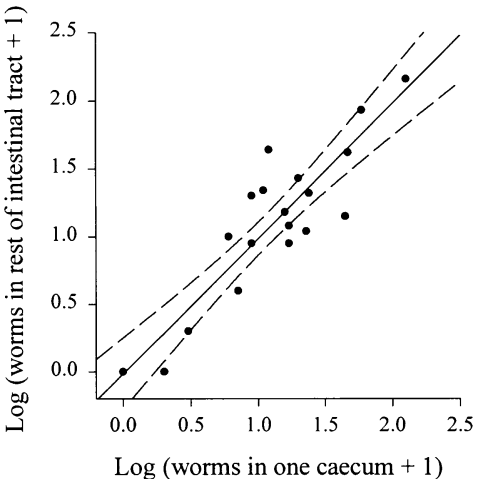

Fig. 1. Linear regression between the log-transformed number of *Heterakis gallinarum* worms counted in 1 caecum and the log-transformed number counted in the rest of the intestinal tract of ring-necked pheasants (n = 20 birds). Dashed lines are 95 % confidence intervals.

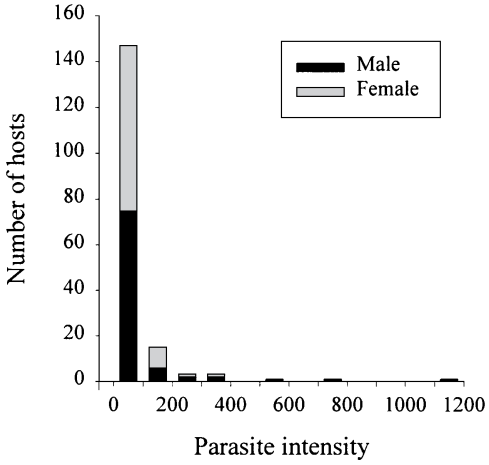

Fig. 2. Frequency distribution of *Heterakis gallinarum* intensity in male (n = 88) and female (n = 83) ring-necked pheasants.

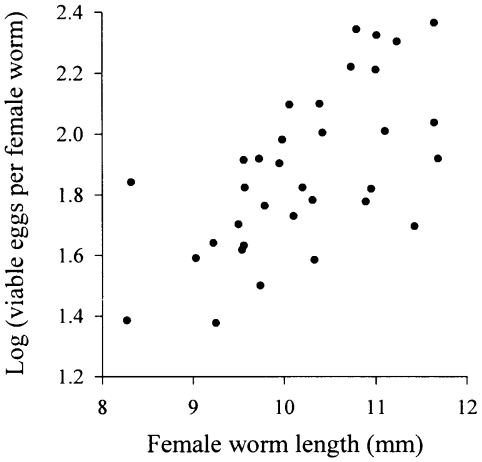

Fig. 3. Relationship between the log-transformed mean number of viable *in utero* eggs per female *Heterakis gallinarum* worm and mean female worm length in ring-necked pheasants (n = 34 birds).

worms were removed, of which 93 % were adults. Of the adult worms, 53 % were female and 47 % were male. No correlations between either parasite age structure (larvae versus adults) and worm intensity or parasite sex ratio and worm intensity were apparent.

Worm counts were undertaken on the entire intestinal tract of 20 pheasants. For these hosts, the single caeca worm counts were highly correlated with the number of worms found in the rest of the intestinal tract (linear regression; $n = 20$, $r^2 = 0{\cdot}81$, $P < 0{\cdot}001$). The slope of this relationship was not significantly different from 1, showing that the sample counts accounted for half of the total worm burden of the pheasants examined (Fig. 1). This confirms that *H. gallinarum* generally only occurs in the two caeca of infected hosts.

Parasite intensity followed an aggregated distribution (Fig. 2) that was not significantly different from a negative binomial with an arithmetic mean of 51·29 and a positive exponent of 0·30 (chi-squared test; $\chi^2 = 1{\cdot}39$, D.F. = 2, $P = 0{\cdot}25$). This value of k is similar to that reported previously for *H. gallinarum* in chickens (0·21; Shaw & Dobson, 1995). As a consequence, we analysed these data using a glm with an explicitly defined negative binomial error distribution (Wilson, Grenfell & Shaw, 1996; Wilson & Grenfell, 1997). This demonstrated that the intensity of *H. gallinarum* infections was significantly higher in male than in female pheasants (deviance = 9·72, D.F. = 1, $P = 0{\cdot}002$).

Parasite fecundity

In-utero egg counts, conducted on 10 mature female *H. gallinarum* worms removed from each of 34 pheasant hosts, produced a mean (±S.E.) of 90·17 (±10·14) viable eggs/worm. There was a positive correlation between the log-transformed mean number of viable eggs/female worm and mean female worm length (Fig. 3; deviance = 5·07, D.F. = 1, $P = 0{\cdot}02$). Worm length was the strongest predictor of worm fecundity (explaining the most deviance in the statistical analysis) and, after controlling for worm length, neither parasite intensity (deviance = 0·38, D.F. = 1, $P = 0{\cdot}54$) nor host sex (deviance = 0·48, D.F. = 1, $P = 0{\cdot}49$) were related to worm fecundity. However, a non-linear relationship between mean female worm length and parasite intensity was identified (see below).

Parasite size

A total of 2161 female *H. gallinarum* worms, with a mean (±S.E.) length of 9·64 mm (±0·11), were removed from the single caeca of 120 of the 171 ring-necked pheasants examined. Visual examination of the worm length data suggested that, at low parasite

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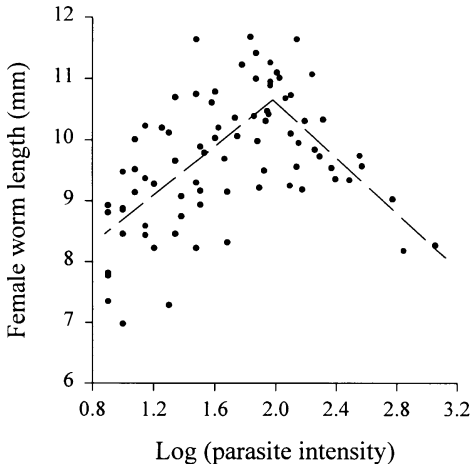

Fig. 4. Relationship between the mean length of female *Heterakis gallinarum* worms and log-transformed parasite intensity in ring-necked pheasants from which more than 2 female worms were collected ($n = 82$ birds). The dashed line indicates the least-squares breakpoint regression that best fits the data ($r^2 = 0{\cdot}40$). The breakpoint in the body length data is at a parasite intensity of $10^{1{\cdot}984}$ (96) worms.

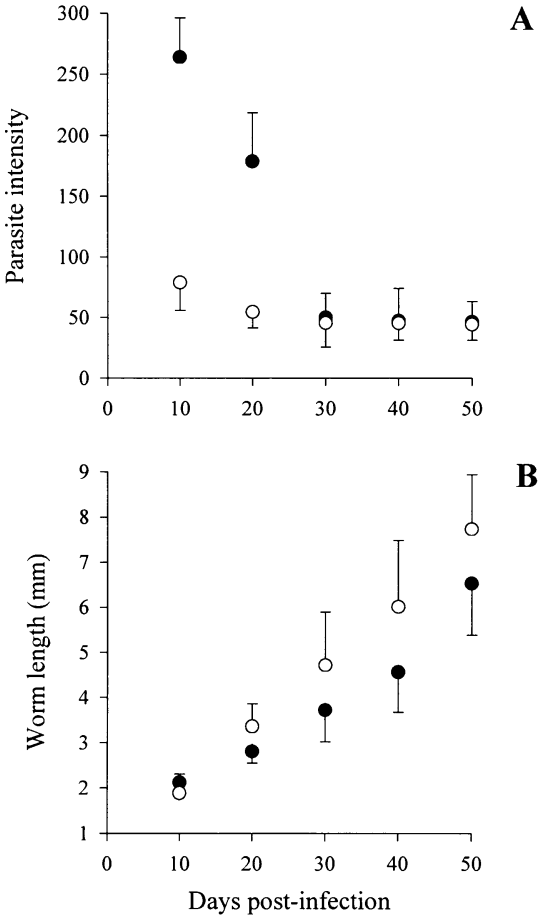

Fig. 5. Relationship between (A) parasite intensity and time since infection, and (B) mean worm length and time since infection, for 20 ring-necked pheasant hens orally infected with approximately 100 embryonated *Heterakis gallinarum* eggs (○) and 20 hens orally infected with approximately 500 embryonated *H. gallinarum* eggs (●). Means and standard errors are shown for 4 birds from each treatment group culled at 10 day intervals post-infection.

intensities, female worm length tended to increase with increasing worm burden whilst, at high parasite intensities, female worm length tended to decrease with increasing worm burden (Fig. 4). The significance of this pattern was confirmed by the fitting of a least-squares breakpoint regression of the form: $y = (m_1 x + c)(x \leqslant b) + (m_2 x + m_1 b + c)(x > b)$, where $(x \leqslant b)$ and $(x > b)$ are logical functions denoting either side of the breakpoint (b). Both the inverse density dependence of female worm length observed for the lower worm burdens ($t = 6{\cdot}15$, D.F. = 78, $P < 0{\cdot}001$), and the density dependence of female worm length observed for the higher worm burdens ($t = 4{\cdot}15$, D.F. = 78, $P < 0{\cdot}001$) were statistically significant. The breakpoint in the data was identified as a parasite intensity of 96 worms ($t = 32{\cdot}48$, D.F. = 78, $P < 0{\cdot}001$).

Infection experiment

Although time since infection was the primary determinant of *H. gallinarum* burden (deviance = 83 270, D.F. = 1, $P < 0{\cdot}001$), pheasants infected with 500 embryonated *H. gallinarum* eggs did contain more worms than those infected with only 100 embryonated *H. gallinarum* eggs (Fig. 5A; deviance = 40 323, D.F. = 1, $P < 0{\cdot}001$) up until 30 days post-infection (interaction deviance = 47 775, D.F. = 1, $P < 0{\cdot}001$). This interaction suggests that *H. gallinarum* establishment is density dependent, i.e. the success rate of larvae developing to adult worms decreases with increasing parasite intensity.

After controlling for time since infection (deviance = 123·73, D.F. = 1, $P < 0{\cdot}001$), worms were significantly larger in the lightly infected birds than in the heavily infected birds (Fig. 5B; deviance = 5·93, D.F. = 1, $P = 0{\cdot}01$). Within the lightly infected birds, after controlling for time since infection (deviance = 105·93, D.F. = 1, $P < 0{\cdot}001$), there was a positive correlation between worm length and parasite intensity (Fig. 6; deviance = 25·08, D.F. = 1, $P < 0{\cdot}001$). Furthermore, as time since infection increased, the positive slope of the correlation increased (interaction deviance = 18·10, D.F. = 1, $P < 0{\cdot}001$). Within the heavily infected birds, after controlling for time since infection (deviance = 25·10, D.F. = 1, $P < 0{\cdot}001$), there was no significant correlation between worm length and parasite intensity (Figure 6; deviance = 2·89, D.F. = 1, $P = 0{\cdot}09$). This was due to the relationship between worm length and parasite intensity being negative at days 10 and 20 post-infection, and positive thereafter (interaction deviance = 5·29, D.F. = 1, $P = 0{\cdot}02$).

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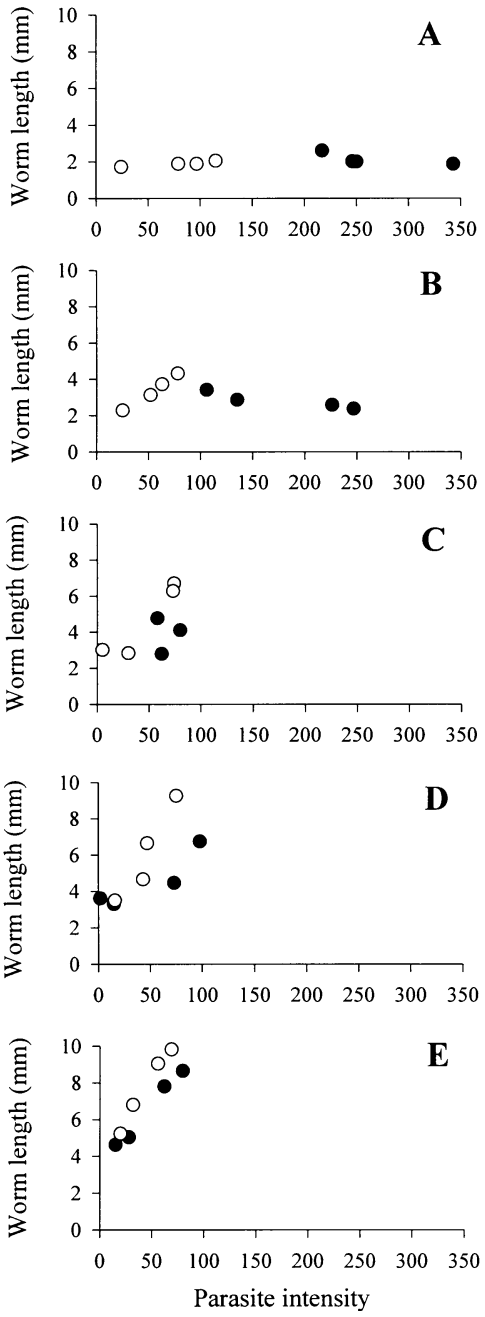

Fig. 6. Relationship between mean worm length and parasite intensity for *Heterakis gallinarum* worms removed from 4 ring-necked pheasant hens orally infected with approximately 100 embryonated *H. gallinarum* eggs (○) and 4 pheasant hens orally infected with approximately 500 embryonated *H. gallinarum* eggs (●) at (A) 10 days post-infection, (B) 20 days post-infection, (C) 30 days post-infection, (D) 40 days post-infection, and (E) 50 days post-infection. There are only 3 data points for the heavily infected group in graph C since one bird was free of infection.

DISCUSSION

These data identified a clear negative relationship between nematode size, a good predictor of worm fecundity, and parasite intensity for natural infections of *Heterakis gallinarum* in the ring-necked pheasant. The density-dependent reduction in fecundity was only apparent at high parasite intensities, but this is not unusual since a threshold in density-dependent fecundity is observed in many host–helminth systems. For example, in infections of *Trichostrongylus colubriformis* in sheep, worm fecundity is only reduced when densities reach 3000 worms per host (Dobson, Waller & Donald, 1990). What is of particular interest is the identification of a positive relationship between nematode size and parasite intensity below the density-dependence threshold. This inverse density dependence may confound the pattern of helminth fecundity observed in this system, but was insufficient to mask the reduction in worm fecundity that occurs at high parasite intensities.

The occurrence of both density dependence and inverse density dependence in parasite fecundity has not previously been documented in the same host–helminth system. Here, we not only illustrate their co-occurrence in natural infections, but we also conduct experimental infections which demonstrate their dynamic interaction in regulating the development and subsequent fecundity of *H. gallinarum* worms in ring-necked pheasants. Density dependence can be seen in the stunted growth of worms in heavily infected birds, relative to the worms in lightly infected birds. Inverse density dependence in worm size was the common pattern across hosts by the end of the experiment, when parasite intensities had fallen below the density-dependence threshold.

Density-dependent constraints on parasite survival, growth and fecundity are thought to be a result of either intraspecific competition for host resources (i.e. space or nutrients) or interactions with host immunological responses (Anderson & May, 1978). The data presented here are not sufficient to distinguish between these alternatives; a comparison of primary and secondary infections is required (Hudson & Dobson, 1997). Stear *et al.* (1995), however, have recently demonstrated that host resistance is important in the density-dependent regulation of the gastrointestinal nematode *Ostertagia circumcincta* infecting sheep. Here, higher parasite intensities caused greater local parasite-specific immunoglobulin A (IgA) responses in the host, reducing worm fecundity through the inhibition of worm growth.

There are 3 possible explanations for the inverse density dependence in *H. gallinarum* fecundity observed below the density-dependence threshold. The first is reduced parasite mating success at low worm intensities. This seems unlikely, however, since we utilized parasite size as an index of fecundity in our analyses. The second explanation is that the low parasite intensities may have occurred only in recently infected hosts where worms had less time to mature. Again, this seems unlikely since no relationship between parasite intensity and the pro-

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portion of worms that were still larval was apparent. The more likely explanation is that host resistance is involved. For example, if variation in host predisposition to infection occurs where host resistance influences both parasite fecundity and parasite survival, one would expect the most lightly infected hosts to also contain the least fecund parasites (Keymer & Slater, 1987). This is the pattern observed in this study. Host resistance being responsible for both the density dependence and inverse density dependence observed in *H. gallinarum* fecundity is not unlikely since the occurrence of multiple resistance mechanisms in the same host organism, having different effects on parasitic nematode populations, has been recorded in other systems. For example, whilst the fecundity of *O. circumcincta* infecting sheep is regulated by IgA responses, *O. circumcincta* intensity in sheep is regulated by immediate hypersensitivity reactions (Stear *et al*. 1995).

Not all studies have demonstrated density dependence in helminth fecundity. The data presented in this study suggest that thresholds in density dependence, in combination with complexity in host resistance, may account for its absence. Some studies may not have included enough hosts with sufficiently high parasite burdens for the detection of density-dependent fecundity to be successful. For example, this is a possible reason why Marcogliese (1997) failed to demonstrate density dependence in the fecundity of the nematode *Pseudoterranova decipiens* infecting grey seals. Here, 1 seal was host to approximately 1600 worms, whilst the remainder had less than 600. The study by Hudson & Dobson (1997) on the caecal nematode *Trichostrongylus tenuis* in red grouse is particularly interesting since there was no evidence of density dependence in worm fecundity despite very high worm burdens. A lack of resistance to infection was also documented in this system, such that worm intensities increased with age and secondary infections occurred at the same rate as primary infections. This implies that, first, density-dependent reductions in nematode fecundity will be more common in hosts with strong immunological responses and, second, that intraspecific competition may have little or no influence on the fecundity of caecal nematodes.

Much controversy exists over the importance of density-dependent fecundity as a regulatory factor of helminth populations (Quinnell, Medley & Keymer, 1990). In the case presented here, the evidence for host predisposition influencing both the fecundity and survival of *H. gallinarum* in the ring-necked pheasant initially suggests that density dependence may not be so important. However, *H. gallinarum* occurs in a highly aggregated distribution in the ring-necked pheasant, possibly as a consequence of variation in host predisposition. Thus, a large proportion of the *H. gallinarum* population occurs at high densities in a few hosts (typically males; Hillgarth, Robertson & Woodburn, 1990). As such, we can expect density-dependent fecundity to be a major regulatory factor of *H. gallinarum* populations.

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