The role of shared parasites in the exclusion of wildlife hosts: *Heterakis gallinarum* in the ring-necked pheasant and the grey partridge

D. M. TOMPKINS*, J. V. GREENMAN†, P. A. ROBERTSON‡ and P. J. HUDSON*

Institute of Biological Sciences, University of Stirling, Stirling, FK9 4LA; †Department of Computing Science and Mathematics, University of Stirling, Stirling, FK9 4LA; and ‡Central Science Laboratory, Sand Hutton, York, YO41 1LZ, UK

Summary

1. A two-host shared-macroparasite model was parameterized from the results of infection and transmission experiments, to investigate whether apparent competition between the ring-necked pheasant (*Phasianus colchicus*) and the grey partridge (*Perdix perdix*), mediated via the shared nematode *Heterakis gallinarum*, could theoretically cause partridge exclusion.

2. Both the model created and the experiments conducted show that the bulk of *H. gallinarum* infection to partridges, when they occur in the same locations as pheasants, will be from the pheasants and not from the partridges themselves. This is due to R_0 for the parasite being 1·23 when infecting pheasants, but only 0·0057 when infecting partridges. Thus, when the pheasant is present in the model the partridge population is impacted by the shared parasite but, when the pheasant is absent, the parasite is lost from the system.

3. Based on best available parameter estimates, the observed impact of *H. gallinarum* on the grey partridge may be sufficient to cause exclusion when the pheasant is present in the model. This supports the hypothesis that the UK grey partridge decline observed over the past 50 years may be partly due to apparent competition with pheasants.

4. Habitat separation between the two host species, where it decreases the rate of *H. gallinarum* transmission from the pheasant to the partridge, may allow them to co-exist in the field in the presence of the parasite. We predict, however, that grey partridge exclusion would still occur if separation was less than 43%.

Key-words: apparent competition, nematode, parasite-mediated competition, *Perdix perdix*, *Phasianus colchicus*.

Journal of Animal Ecology (2000) **69**, 829–840

Introduction

Indirect interactions between species may play a critical role in determining the community structure and dynamics of ecological assemblages (Abrams *et al.* 1995; Menge 1997; Schmitz 1998). One form of such interaction is where two species, which do not compete for resources, share natural enemies (Holt & Lawton 1994; Abrams & Matsuda 1996; Müller & Godfray 1997; Bonsall & Hassell 1998; Hudson & Greenman 1998). Under these circumstances the density of shared enemies supported by each species individually will impact on both species present. Since each of the two species can suffer as a consequence of the presence of the other species, as in competitive situations, this type of interaction has been termed 'apparent competition' (Holt 1977). Apparent competition between two species can lead to the rapid local extinction of one of the two species, with the species that persists being the one that can tolerate the higher densities of shared enemies (Holt & Lawton 1993; Bonsall & Hassell 1997).

Correspondence: Dr Daniel M. Tompkins, Institute of Biological Sciences, University of Stirling, Stirling, FK9 4LA, UK, dmt1@stir.ac.uk, Tel.: 01786 467812, Fax: 01786 464994

Cases where host exclusion can be attributed to the presence of shared parasites are often cited as examples of apparent competition (e.g. Settle & Wilson 1990; Grosholz 1992). However, conclusively demonstrating that host exclusion is due to apparent competition, as opposed to either direct competition or other parasite effects can be difficult to accomplish (as discussed in Hudson & Greenman 1998). A recent series of controlled experiments, conducted on laboratory populations of two moth species (*Plodia interpunctella* and *Ephestia kuehiella*) and the parasitoid *Venturia canescens*, has provided the first explicit demonstration of apparent competition mediated via a shared parasitoid (Bonsall & Hassell 1997, 1998). Providing an explicit demonstration is far harder to accomplish in natural systems, however, due to the logistical constraints involved. In many circumstances the controlled experiments required are unworkable. The majority of field studies to date have thus been descriptive, often failing to disentangle parasite effects from resource competition among hosts (e.g. Schall 1992; Schmitz & Nudds 1994; Hanley, Vollmer & Case 1995).

Definitive proof that apparent competition mediated via shared parasites occurs in the field will require the large scale manipulation of host populations as employed in other gamebird-parasite studies (Hudson, Dobson & Newborn 1998; May 1999). It would be premature to conduct such an experiment with any natural system, however, without first demonstrating that model simulations run with the best available parameter estimates do, indeed, predict that apparent competitive effects are of sufficient magnitude to be detectable. This is in line with the view that mathematical modelling is a valuable precursor to conducting population scale manipulations in the wild, in order to justify the time and expense involved (Tompkins & Begon 1999). The aim of this study is to provide such a demonstration for two gamebirds, the ring-necked pheasant [*Phasianus colchicus* (L.)] and the grey partridge [*Perdix perdix* (L.)], investigating whether the biology of the shared caecal nematode *Heterakis gallinarum* (Schrank) could result in exclusion of the partridge. *H. gallinarum* occurs in several species of galliform birds, transmitted via an actively ingested egg stage (Lund & Chute 1974).

Numbers of wild grey partridge have declined dramatically in the UK within the past 50 years (Tapper 1992). This decline is linked primarily to the intensification of agriculture and increased predation pressure (Potts 1986, 1997; Sotherton 1998), as supported by large scale field experimentation (Rands 1985; Tapper, Potts & Brockless 1996). However, recent studies indicate that apparent competition with the ring-necked pheasant may also be involved (Wright *et al.* 1980; Tompkins, Dickson & Hudson 1999; Tompkins, Draycott & Hudson 2000). The experimental exposure of naive birds to

infection suggests that the pheasant acts as a reservoir host for *H. gallinarum*, the negative impact of which is greater on the partridge (Tompkins *et al.* 1999, 2000). This is supported by fully controlled experiments with singly housed birds, which clearly demonstrate that the parasite is a cause of decreased body condition in the grey partridge and not vice-versa (D.M. Tompkins, in preparation). Therefore, this study will determine whether a two-host shared-macroparasite model, parameterized from the results of infection and transmission experiments, predicts exclusion of the grey partridge due to apparent competition with the ring-necked pheasant.

Methods

THE MODEL EQUATIONS

The model used to describe the two-host shared-parasite system is illustrated in Fig. 1, and is defined by the following equations (for $i,j = 1,2; i \neq j$):

$$dH_i/dt = r_i H_i (1 - H_i/K_i) - (\alpha_i + \delta_i)P_i \qquad \text{eqn 1a}$$

$$dP_i/dt = \phi_i \beta_i W H_i - (\mu_i + b_i + \alpha_i)P_i$$
$$- \alpha_i k_i' P_i (P_i/H_i) \qquad \text{eqn 1b}$$

$$dW/dt = \lambda_1 P_1 + \lambda_2 P_2 - \gamma_0 W - \beta_1 W H_1$$
$$- \beta_2 W H_2 \qquad \text{eqn 1c}$$

The model explores the dynamics of W, the number of free-living stages in the common infective pool, P_i, the adult parasite in the ith host, and H_i, the host population. The natural exponential growth of the host population (r_i) in equation 1a is offset by density dependent host mortality (with K_i being the carrying capacity) and by the parasite induced effects on host survival (α_i) and fecundity (δ_i). The number of free living stages (equation 1c) increases through the deposition of worm eggs (λ_i) and decreases through both natural mortality (γ_0) and ingestion of eggs by the host (β_i). The number of parasitic worms (equation 1b) increases with the ingestion of eggs by hosts (β_i), modified by the proportion that survive to become mature worms (ϕ_i), and decreases due to the combined effects of worm mortality (μ_i), and natural (b_i) and infection-induced (α_i) host mortality. The natural host mortality rate (b_i) incorporates density dependence, whereby $b_i = b_{i0} + r_i H_i/K_i$. The natural host birth rate is a_i, with the net population growth rate at low population levels expressed as $r_i = a_i - b_{i0}$. The last term in equation 1b models parasite mortality arising from parasite-induced host deaths, assuming a negative binomial distribution of parasites among hosts (Anderson & May 1978). Parameter k_i' equals $1 + 1/k_i$, where k_i is the negative binomial parameter, an
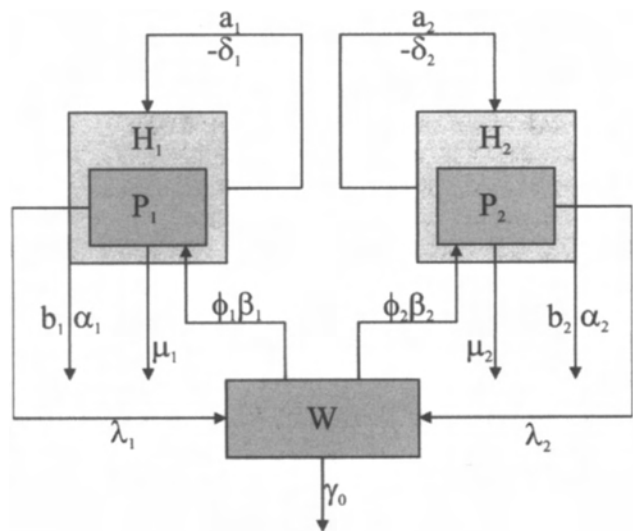
Fig. 1. Flow diagram of the basic two-host/shared-parasite model, where W denotes the number of parasite eggs in a common infective pool while, for the ith host, P_i denotes the adult parasite and H_i the host populations. See Table 1 for parameter definitions and estimates.

inverse measure of aggregation. A full list of parameters is given in Table 1.

In terms of parasite intensity $Z_i = P_i/H_i$ (M. Roberts, personal communication), the equations become (for $i \neq j$):

$$dH_i/\mathrm{dt} = r_iH_i(1 - u_{1i}H_i - u_{2i}Z_i) \qquad \text{eqn 2a}$$

$$dZ_i/\mathrm{dt} = \phi_i\beta_iW - s_iZ_i - e_iZ_i^2 \qquad \text{eqn 2b}$$

$$dW/\mathrm{dt} = \lambda_1H_1Z_1 + \lambda_2H_2Z_2 - \gamma_0W$$
$$- \beta_1WH_1 - \beta_2WH_2 \qquad \text{eqn 2c}$$

where $u_{1i} = K_i^{-1}$ and $u_{2i} = (\alpha_i + \delta_i)/r_i$. These coefficients scale the host density dependence and parasite effect terms, respectively. The transformed equations involve two composite parameters: (i) $s_i = (\mu_i + \alpha_i + a_i)$, measuring the loss of parasite intensity due to both adult parasite and infection-induced host mortality, and dilution through host births, and (ii) $e_i = \alpha_i/k_i - \delta_i$, where δ_i is the parasite-induced reduction in host fecundity. Parameter e_i therefore relates parasite-induced mortality and the reduction in fecundity. The relevance and stability of the point equilibria of model equation 2(a,b,c) have been fully discussed elsewhere (Greenman & Hudson 1999). In

Table 1. Parameter definitions and estimates used in the mathematical model. Empirical values for $\alpha_{\text{partridge}}$ and $\delta_{\text{partridge}}$ have not yet been determined.

Parameter	Symbol	Pheasant value	Partridge value	Units	Source
Natural host fecundity	a	1·55	1·50	year^{-1}	Brittas *et al.* (1992); Tapper *et al.* 1996
Natural host mortality	b	0·65	0·80	year^{-1}	Robertson & Dowell (1990)
Host carrying capacity	K	6	3	home range^{-1}	R.A.H. Draycott, personal com
Mortality of parasite eggs	γ	0·90	0·90	egg^{-1}year^{-1}	Lund (1960)
Ingestion of parasite eggs by hosts	β	$6·70 \times 10^{-5}$	$5·58 \times 10^{-5}$	egg^{-1}host^{-1}year^{-1}	This study
Parasite establishment	ϕ	0·590	0·065	egg^{-1}	This study
Parasite fecundity	λ	26666	2761	eggs year^{-1}	This study
Parasite mortality	μ	4·15	4·17	eggs year^{-1}	This study
Aggregation of parasites in hosts	k	0·30	0·30		Tompkins & Hudson 1999
Parasite increase in host mortality	α	0·00	?	worm^{-1}year^{-1}	Tompkins *et al.* 1999
Parasite reduction in host fecundity	δ	$8·28 \times 10^{-4}$	?	worm^{-1}year^{-1}	M. Woodburn, personal com

this study the relevant results are summarized and applied to the pheasant/partridge system. Parameter estimates were obtained from a combination of infection and transmission experiments (as detailed below), together with sources in the literature and unpublished data. Note that the model is constructed excluding direct interactions between the two host species. Any predicted outcome will thus be due to parasite effects alone.

INFECTION EXPERIMENT

Experimental design

The object of the infection experiment was to determine the establishment success and fecundity of *H. gallinarum* worms in both pheasants and partridges, from which the values for ϕ, μ and λ could be estimated (see Fig. 1). All of the birds used in the infection experiment were reared from day-old chicks on sterilized concrete to ensure that all birds were naïve to parasite infection. At 12 weeks of age, five individuals of each species were culled to confirm the absence of parasites and 30 birds (15 male and 15 female) of each species placed into individual cages with wire mesh floors.

The *H. gallinarum* eggs used in this study were from worms collected from pheasants that had acquired natural infections. Female worms were collected from each bird, maintained for 21 days in 0·5% formalin solution at 21°C to embryonate all viable eggs, and broken down in saline using a small electric blender. Embryonated eggs were then counted in 10 0·1 mL samples, and the volume of saline adjusted to 100 embryonated eggs per ml. Infections were carried out on the 21st day of the embryonation period. Fifteen individuals of each host species were randomly selected and given a single dose of approximately 100 embryonated *H. gallinarum* eggs. Nematode eggs, suspended in 1 mL of saline, were administered orally via a tube into the birds crop. The remaining 15 birds of each species were treated as controls and given 1 mL of saline containing no nematode eggs. An infective dose of 100 eggs was chosen since this was the largest that could be used, whilst avoiding previously documented density-dependent influences on *H. gallinarum* fecundity (Tompkins & Hudson 1999). The birds were maintained for 100 days and supplied with food (gamebird maintenance pellets), water and grit (medium flint grit) *ad libitum*. Preliminary infection trials indicated that 100 days was sufficient to monitor worm life expectancy (unpublished data).

Estimation of parasite establishment success

Previous work has shown that once *H. gallinarum* worms reach maturity (at approximately 30 days in pheasants), they undergo negligible mortality for at

least the following 20 days (Tompkins & Hudson 1999). Thus, to estimate the rate at which ingested *H. gallinarum* eggs survived to become mature parasites within each host species, six birds (three of each sex) from both experimental and control groups were randomly selected, and culled at 40 days post-infection and their worm burdens determined. All worms were removed from both caeca of each bird by sequentially washing the caecal contents through a course sieve (1·4 mm), to remove host tissue, and a fine sieve (0·2 mm), to collect the worms, using standard techniques (Doster & Goater 1997). Worms were counted under a binocular microscope.

Estimation of parasite fecundity

To monitor *H. gallinarum* egg production the number of *H. gallinarum* eggs present in the caecal droppings of individual hosts was counted at 5-day intervals. Half a gram of each sample was suspended in 10 mL of saturated salt solution and eggs counted, using McMasters chambers under ×100 magnification, in five 0·1-mL subsamples. To convert egg counts (expressed as eggs per gram of caecal dropping) into number of eggs expelled per day, they were multiplied by the total mass of caecal droppings produced by that host on that day. The total number of nematode eggs expelled per infected individual was estimated for each host species by multiplying the overall mean number of eggs expelled per day by individuals of that species by 100 days. Each estimate was then converted into the total number of eggs expelled per mature *H. gallinarum* worm infecting that host species, using the previously determined success rates for parasite establishment (see above).

TRANSMISSION TRIAL

Experimental design

The aim of the transmission trial was to determine the rate at which individuals of both host species ingest *H. gallinarum* eggs from the environment, and from this estimate the transmission coefficient β (Fig. 1). Birds were kept for a set period of time in pens (measuring 1·8 × 3·6 m), where the density of worm eggs was known. Prior to the transmission trial, 13 000 embryonated *H. gallinarum* eggs (~2000 eggs m^{-2}) were distributed evenly on the ground in three pens; three other pens were left unmanipulated to control for background levels of nematode eggs. As with the infection experiment, all of the birds used in the transmission trial were reared from day-old chicks on sterilized concrete. At 12 weeks of age, three birds of each species were placed into each of the six pens, where they were maintained for the following 50 days. Fifty days was

considered sufficient time for mature worms to accumulate before either worm mortality or host re-infection occurred. Birds were supplied with food (gamebird maintenance pellets), water and grit (medium flint grit) *ad libitum*. All of the birds were culled at the end of the transmission trial and individual worm burdens determined.

Estimation of parasite transmission rates

To convert worm burdens into transmission rate estimates, calculations were based on the results from the infection experiment. First, to estimate the number of days during which any mature *H. gallinarum* worms found in birds at the end of the transmission trial must have actually initially infected the host, the mean age at maturity for worms in each of the two host species was subtracted from the 50-day exposure period. The mean age at maturity for worms in each host species was estimated as the mean day post-infection at which worm eggs were first observed in host caecal droppings in the infection experiment. This is a valid estimate since the maturation of individuals within a cohort of *H. gallinarum* worms is highly correlated (unpublished data). Secondly, to estimate the number of *H. gallinarum* eggs that would have been ingested by each individual host, the number of mature worms found in each exposed host was divided by the mean success rate of *H. gallinarum* establishment in that host species. The number of mature worms in each host was estimated as twice the number of mature female worms, since mature females can be more accurately distinguished from immature worms (by the presence of viable eggs) than can mature males, and the sex ratio of *H. gallinarum* is 1 : 1 (Tompkins & Hudson 1999). Prior to calculating numbers of eggs, each estimate of mature worms was adjusted to control for infection from background levels of nematode eggs by subtracting the mean number of mature worms observed in the control birds of the appropriate host species from each estimate. A rate of egg ingestion for each host was then calculated by dividing the number of eggs ingested by the number of days during which those eggs could have been taken up. Note that this approach assumes negligible egg mortality.

MODEL PREDICTIONS

Since empirical values for $\alpha_{partridge}$ and $\delta_{partridge}$ (parasite impact on wild partridge survival and fecundity) have not yet been determined (accurate estimation requires controlled population manipulations), we investigated whether a previously documented impact of *H. gallinarum* on grey partridge body condition (Tompkins *et al.* 1999) was of sufficient magnitude for host exclusion to be a possibility. Calculations were made to estimate the levels of

$\alpha_{partridge}$ and $\delta_{partridge}$ in which the observed impact could theoretically result. These calculations were based on the intensively studied gamebird-nematode system, *Trichostrongylus tenuis* in red grouse, where parasites increase host mortality (from a non-parasitized rate of 1·05 year^{-1}) by 3×10^{-4} worm^{-1} year^{-1} and decrease fecundity (from a non-parasitized rate of 1·8 year^{-1}) by 5×10^{-4} worm^{-1} year^{-1} (Hudson & Dobson 1997).

Figure 7·4 of Hudson (1986) illustrates how a sample of 'very thin' grouse (having lost ~70% of their breast muscle mass) had average worm burdens of 7800 *T. tenuis*, a sample of grouse in 'poor condition' (having lost ~50% of their breast muscle mass) had average burdens of 3250 worms and a sample of grouse in 'average to good' condition (having lost ~20% of their breast muscle mass) had average burdens of only 1850 worms. This approximates to a linear reduction in host body condition of $1·12 \times 10^{-2}$ worm^{-1}. Thus, a parasite-induced reduction in grouse body condition of ~1% equates to a ~2·5% increase in the yearly mortality rate and a ~2·5% decrease in the yearly fecundity rate.

Applying this relationship to the grey partridge converts the observed reduction in body condition (Fig. 2) to an increase in partridge mortality of 2·15 $\times 10^{-2}$ worm^{-1} year^{-1} and a decrease in partridge fecundity of $4·04 \times 10^{-2}$ worm^{-1} year^{-1}. The model was run using these parameter values. However, since this is only a rough approximation, sensitivity analyses were conducted to determine the robustness of any finding. Note, this approach does not imply
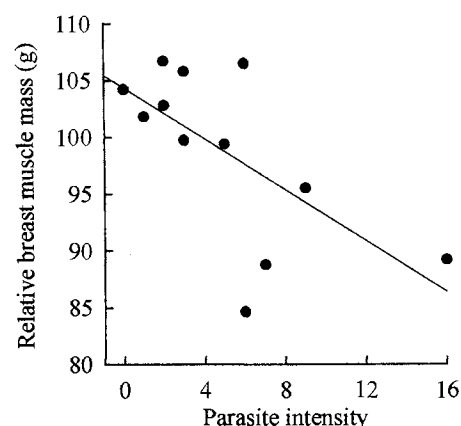

Fig. 2. Relationship between *Heterakis gallinarum* intensity and grey partridge breast muscle mass adjusted for body size (an index of body condition), obtained from an experimental exposure of naive birds to infection. Muscle mass was adjusted to a mean tarsal length of 52·7 mm. Fitting a linear regression to the data illustrates that, on average, there is a drop of 1·076% in body condition for each *H. gallinarum* worm infecting ($n = 12$ birds). Modified from Tompkins *et al.* (1999).

similar pathogenicity of *H. gallinarum* and *T. tenuis*, rather it assumes that the relationship between host body condition and host fitness is similar for the two gamebirds.

SPATIAL SEPARATION

An assumption implicit in the model is that the two host species sharing the same parasite also share precisely the same habitat. This condition, however, will rarely be met for wild systems; it certainly does not hold for the ring-necked pheasant and the grey partridge (Cocchi *et al.* 1990). Since the driving force behind host exclusion due to apparent competition mediated via shared parasites is the transmission of parasites between host species, spatial separation (when it decreases such transmission) would allow species to co-exist that would not otherwise. If the model does, indeed, predict exclusion, the important question is then whether or not the level of separation between the two host species in the wild is sufficient to prevent such exclusion from actually occurring. As a preliminary investigation, parameters of the current model were adjusted to approximate the effect of spatial separation between the pheasant and the partridge. Since the basic reproductive number (R_0; the number of adult female parasites derived from each adult female parasite in a population of uninfected hosts) for *H. gallinarum* infecting pheasants is at least $\times 100$ that for the parasite infecting partridges (see Results), the pheasant is indeed primarily responsible for the spread of infection. Therefore, an approximation of spatial separation was modelled by simply reducing the rate at which the partridges were ingesting the nematode eggs (i.e. a 50% decline in $\beta_{partridge}$ mimics 50% spatial separation between the two host species).

Results

PARASITE ESTABLISHMENT

Forty days after being infected, *H. gallinarum* intensities in pheasants were significantly higher than those in partridges (Fig. 3). Infected pheasants were host to a mean ($\pm$ SE) of 59.00 ± 14.83 worms, whilst infected partridges were host to only 6.50 ± 3.62 worms (Mann–Whitney $U = 5$, $P = 0.04$). Thus, considering that each infected host was given approximately 100 embryonated *H. gallinarum* eggs, 0.590 of the eggs given to pheasants survived to become mature parasites, whilst only 0.065 of the eggs given to partridges survived. None of the control birds of either host species contained any worms.
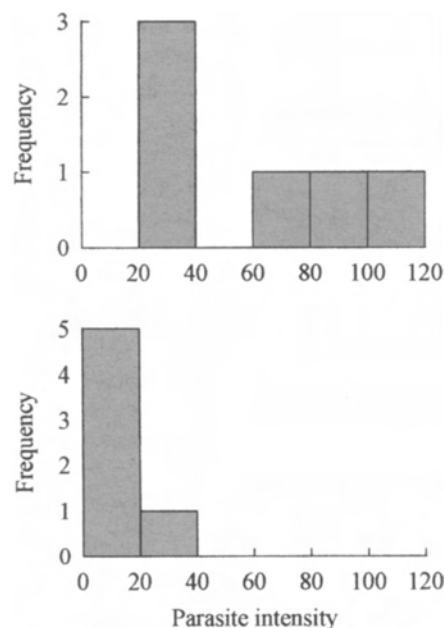

Fig. 3. Frequency distribution of *Heterakis gallinarum* intensity in (a) pheasants ($n = 6$ birds), and (b) grey partridges ($n = 6$ birds), 40 days after being infected with approximately 100 embryonated *H. gallinarum* eggs.

PARASITE FECUNDITY

Over the course of the infection experiment, six pheasants and four partridges were euthanased due to husbandry problems unrelated to parasite infection. In the remainder, the number of worm eggs expelled was significantly higher in the caecal droppings of pheasants than in the caecal droppings of partridges (Fig. 4); infected pheasants expelled a mean ($\pm$ SE) of 3793 ± 3117 eggs day^{-1}, whilst infected partridges expelled only 43 ± 30 eggs day^{-1} ($U = 6$, $P = 0.02$). Since no worm eggs were detected in the caecal droppings of any of the control birds, the average total egg production by each mature *H. gallinarum* worm was estimated as 6429 in pheasants and 662 in partridges.

PARASITE TRANSMISSION

After the transmission trial, *H. gallinarum* intensity was significantly higher in the exposed pheasants ($U = 13.5$, $P = 0.04$) and partridges ($U = 15$, $P = 0.05$) than in the control birds (Fig. 5). Controlling for background infection, the 13 000 worm eggs laid down in each pen resulted in infections of 151.50 mature worms per exposed pheasant and 7.17 mature worms per exposed partridge. Since the mean age of maturity for *H. gallinarum* was estimated as 35 days in pheasants and 42.5 days in partridges (see Fig. 4), only those *H. gallinarum* eggs picked up by pheasants in the first 15 days, or by
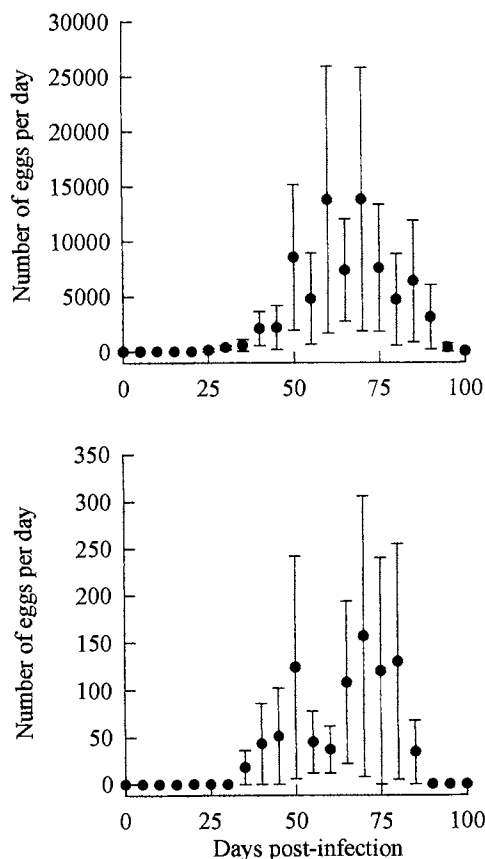
Fig. 4. Number of *Heterakis gallinarum* eggs expelled in the caecal droppings of (a) pheasants ($n = 6$ birds), and (b) grey partridges ($n = 8$ birds), over the 100 days following infection with approximately 100 embryonated *H. gallinarum* eggs. Mean numbers ($\pm$ SE) are shown. Note the different *y*-axis scales.

culled at 100 days post-infection), parasite life expectancy was estimated as a mean of 88 days in pheasants and a mean of 87·5 days in partridges (see Fig. 4).

The values obtained for *H. gallinarum* transmission were converted into instantaneous rates per parasite egg by taking into account the number of eggs to which experimental birds were exposed in the transmission trial (13 000 per pen), adjusting for uptake during the trial. Since the birds were maintained in 6·5 m² pens during the transmission trial, when they would normally occur on home ranges of approximately 5 ha (50 000 m²; R. A. H. Draycott, personal communication), each transmission rate was adjusted to a realistic level by multiplying by 6·5 and dividing by 50 000.

Although captive work has failed to demonstrate an impact of *H. gallinarum* on pheasant body condition (Tompkins *et al.* 1999), parasite removal experiments indicate that there is an impact on their reproductive success in the field, possibly due to an interaction with host nutrition (M. Woodburn, personal communication). A value for *H. gallinarum* induced reduction in pheasant fecundity, of $8 \cdot 28 \times 10^{-4}$ worm^{-1} years^{-1}, was estimated from these experiments. Host carrying capacities for wild populations were estimated from field observations as six pheasants, and three partridges, per 5 ha (R. A. H. Draycott, personal communication).

MODEL PREDICTIONS

When solved for the best available parameter estimates, the two-host shared-parasite model predicts

partridges in the first 7·5 days of the trial, would have developed to mature worms. Daily rates of worm ingestion were thus estimated as 17·12 eggs bird^{-1} for the pheasants, and 14·70 eggs bird^{-1} for the partridges.

MODEL PARAMETERIZATION

Model parameters, and the sources from which they were estimated, are listed in Table 1. The experiments detailed in this study were used to quantify parasite transmission, establishment, fecundity and mortality.

Instantaneous rates of parasite fecundity in both host species were obtained by dividing the mean number of eggs produced by worms in each host (quantified in the infection experiment) by parasite longevity in that host species (measured in years). Assuming that the cessation of egg production in infected individuals represented the age at which infecting worms died (an assumption supported by the fact that no worms were found in hosts when
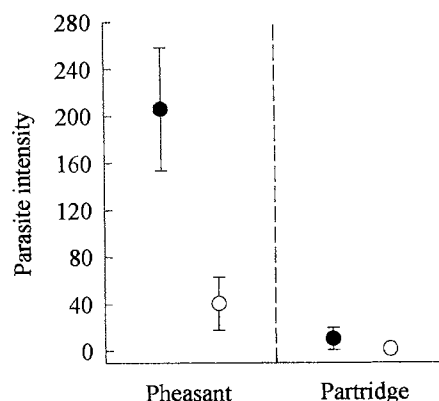
Fig. 5. *Heterakis gallinarum* burdens in both pheasants and partridges after being maintained for 50 days in pens on a grass field. Mean burdens ($\pm$ SE) are shown. Filled points indicate the birds (12 of each species) which were maintained in pens where approximately 13 000 embryonated *H. gallinarum* eggs had been distributed evenly on the ground prior to the transmission trial; open points indicate the birds (six of each species) maintained in unmanipulated pens to control for background levels of nematode eggs.

exclusion of the grey partridge. In general, in the absence of direct competition, host exclusion requires that:

$$S_{0i} > 1 \qquad \text{eqn 3a}$$

where S_{0i} is the 'tolerance' index (Greenman & Hudson 1998, 1999):

$$S_{0i} = (\phi_j W_{i0})/(\phi_i W_{j0}) \qquad \text{eqn 3b}$$

$(i \neq j)$, with:

$$W_{i0} = (d_i/\beta_i)[r_i/(\alpha_i \delta_i)] \qquad \text{eqn 3c}$$

where:

$$d_i = s_i + r_i e_i/(\alpha_i + \delta_i)$$

$$= \mu_i + \alpha_i + b_i + r_i \alpha_i k_i'/(\alpha_i + \delta_i) \qquad \text{eqn 3d}$$

where $d_i > 0$. W_{i0} denotes the equilibrium value of W when host j ($j \neq i$) is absent and density dependence can be ignored. If condition equation 3a holds, host exclusion will occur for:

$$R_{0i} > R_{0i}^* > 1 \qquad \text{eqn 4a}$$

where:

$$R_{0i} = (\phi_i \lambda_i/s_i - 1)(\beta_i K_i)/\gamma_0 \qquad \text{eqn 4b}$$

is the basic reproductive number for the parasite infecting host i and R_{0i}^* is the threshold value of R_{0i} for which the host co-existence equilibrium first

becomes biologically feasible, when all population densities are above zero. Algebraically, R_{0i}^* is found from the condition $Z_2^* = r_2/(\alpha_2 + \delta_2)$, where Z_2^* is the value of Z_2 at this co-existence threshold. Since, by definition, the product of S_{0i} and S_{0j} necessarily equals 1, only one of the two hosts can exclude the other. From the parameter values listed in Table 1, and impacts of *H. gallinarum* on partridge fecundity and survival of 4.04×10^{-2} worm^{-1}year^{-1} and 2.15×10^{-2} worm^{-1}year^{-1} respectively, we find that $S_{0\text{pheasant}} = 7.09$ and $R_0^*_{\text{pheasant}} = 1.12$. Since $R_{0\text{pheasant}} = 1.23$ (see next section) the model predicts that the partridge will be excluded by the pheasant, with the pheasant remaining in co-existence with the parasite.

The closeness in value of $R_{0\text{pheasant}}$ and $R_0^*_{\text{pheasant}}$ suggests that the point in parameter space defined by the empirically determined parameter values lies close to the exclusion–co-existence boundary. This proximity to the threshold was studied in more detail by carrying out sensitivity analysis on individual parameters. Varying the calculated parameters $\delta_{\text{partridge}}$ and $\alpha_{\text{partridge}}$ singly (with all other model equation parameters kept fixed) found a boundary value of $\delta_{\text{partridge}} = 1.02 \times 10^{-2}$ worm^{-1}year^{-1} for the switch in model outcome from exclusion to co-existence, whilst there was no boundary intersection for $\alpha_{\text{partridge}}$. Thus, while a 75% lower $\delta_{\text{partridge}}$ leads to co-existence of the two host species, co-existence cannot be brought about by altering $\alpha_{\text{partridge}}$ alone (Fig. 6).
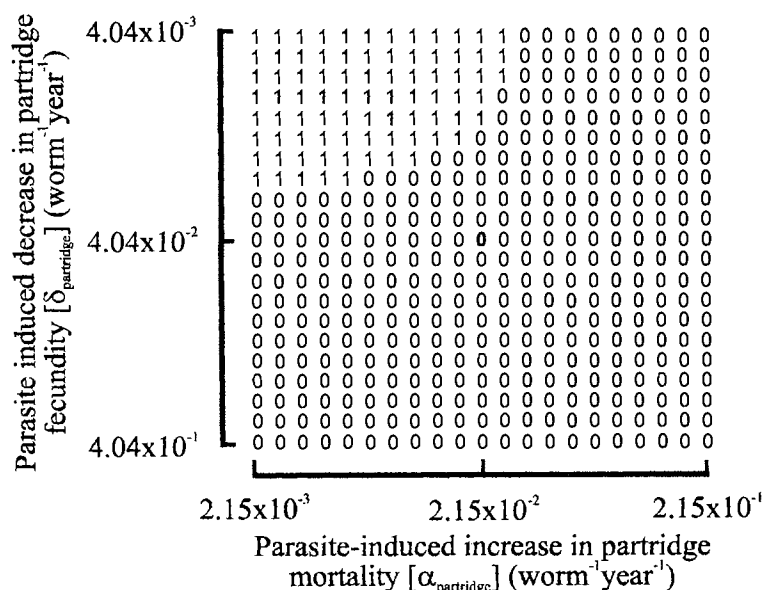

Fig. 6. Exclusion–co-existence boundary curve in the $\alpha_{\text{partridge}}$, $\delta_{\text{partridge}}$ cross-section of parameter space for the two-host/shared parasite model. The parameters on both axes are scaled from one tenth to 10 times their empirical values. '1' denotes co-existence of the two host species, '0' denotes exclusion of the grey partridge. The number in bold text indicates the predicted outcome at the empirically determined values.

Table 2. Threshold values (and percentage change from estimated value) for the change in predicted model outcome, from partridge exclusion to pheasant and partridge co-existence, for those parameters not directly estimated in this study to which the model outcome was considered to be highly sensitive. In all cases, values were determined with all other model equation parameters kept fixed at their estimated levels. Percentage change cannot be calculated for either α_{pheasant} or $\alpha_{\text{partridge}}$ since the estimated value of α_{pheasant} is 0, and there is no threshold value for $\alpha_{\text{partridge}}$

Parameter	Symbol	Pheasant value Threshold	% Change	Partridge value Threshold	% Change
Natural host fecundity	a	1·06	−32%	2·11	+41%
Natural host mortality	b	1·12	+72%	0·25	−69%
Ingestion of parasite eggs by hosts	β	$4{\cdot}23 \times 10^{-4}$	−84%	$3{\cdot}17 \times 10^{-5}$	−43%
Parasite increase in host mortality	α	$5{\cdot}33 \times 10^{-4}$	–	–	–
Parasite reduction in host fecundity	δ	$1{\cdot}65 \times 10^{-3}$	+99%	$1{\cdot}02 \times 10^{-2}$	−75%

Sensitivity analyses were also conducted on α_{pheasant}, δ_{pheasant}, and β, a and b for both host species, since these were the other quantities not directly estimated in this study to which the model outcome was considered to be highly sensitive. However, the model outcome of partridge exclusion was also relatively robust to changes in these values (see Table 2 for a summary of all sensitivity analyses conducted).

SPATIAL SEPARATION

When the model was run for each host species alone with the parasite, the qualitative outcome for the pheasant was unchanged (remaining in co-existence with the parasite), while that for the partridge changed from exclusion of the host to exclusion of the parasite. The model equilibrium, describing an uninfected single host (host *i*) at its carrying capacity, is stable against parasite invasion provided $R_{0i} < 1$. In the two-host simulations, this equilibrium is not stable against invasion by the other host if we assume $r_j > 0$ ($j \neq i$). From the parameter values listed in Table 1 we infer that the pheasant co-exists in equilibrium with the parasite when the partridge is absent since $R_{0\text{pheasant}} = 1{\cdot}23$. The average worm burden is 235 worms host^{-1} and the pheasant population is 0·94 birds ha^{-1}. The basic reproductive number for the parasite infecting the partridge, when the pheasant is absent, is $R_{0\text{partridge}} = 0{\cdot}0057$, i.e. the parasite is excluded. This shift in model outcome, from partridge exclusion when the pheasant is present, to parasite exclusion when the pheasant is absent, demonstrates that the host exclusion predicted by the two-host shared-parasite model is indeed due to the transmission of parasites from the pheasant. These single-host simulations also demonstrate how this parasite-mediated interaction between the pheasant and partridge is not strictly apparent competition, where the presence of either host indirectly affects the density of the other (Holt 1977, 1984), but is an amensal form whereby one host (the partridge) suffers a reduction in density,

while the other (the pheasant) is relatively unaffected. This is because the bulk of *H. gallinarum* transmission to the pheasant is intra-specific, whilst the opposite is true for the partridge.

To determine the level of spatial separation between the two host species at which the model predicts the inter-specific transmission of *H. gallinarum* from the pheasant to the partridge is low enough to allow their co-existence, the model was ran with $\beta_{\text{partridge}}$ set at differing levels below the empirically determined value ($5{\cdot}58 \times 10^{-5}$ egg^{-1} host^{-1} year^{-1}). A boundary value of $\beta_{\text{partridge}} = 3{\cdot}17 \times 10^{-5}$ egg^{-1} host^{-1} year^{-1} was identified, below which the partridge was no longer excluded from the system. This implies that spatial separation of greater than 43% between the two host species will allow the grey partridge to co-exist with the pheasant in the presence of *H. gallinarum*.

Discussion

Based on the best available parameter estimates, the macroparasite model discussed in this paper predicts that apparent competition with the ring-necked pheasant is sufficient to cause exclusion of the grey partridge. This result provides strong evidence for the view that apparent competition plays a vital role in determining the structure of natural communities, and suggests that a population scale experiment with pheasants and partridges could provide proof that apparent competition mediated by shared parasites occurs in the wild.

Apparent competition is but one mechanism by which shared parasites can influence host populations. However, it is fundamentally different from all other parasite effects in that the driving force behind host exclusion, when it occurs, is the presence of alternative host species and not the shared parasite *per se*. It is this characteristic which requires demonstration if the occurrence of apparent competition is to be proven. This study provides such a demonstration for the pheasant/partridge system, confirming that any detrimental effects of the nema-

tode *H. gallinarum* on wild grey partridge populations will, indeed, be due to apparent competition with the ring-necked pheasant. Both the model created and the experiments conducted show that the bulk of *H. gallinarum* infections in partridges, when they occur in the same locations as pheasants, will be from the pheasants and not from the partridges themselves. This is due to the success rate of *H. gallinarum* establishment being nine times greater in pheasants than in partridges, and the fecundity of established worms being approximately 10 times greater in pheasants than in partridges. This results in predicted R_0s of 1·23 for the parasite infecting pheasants and 0·0057 for the parasite infecting partridges. Since the R_0 for *H. gallinarum* infecting grey partridges is much less than unity, the parasite cannot be maintained within partridge populations without the presence of alternative hosts. Thus, when the pheasant is present in the model the partridge population is impacted by the shared parasite, but when the pheasant is absent, the parasite is lost from the system. This clearly illustrates how the force of *H. gallinarum* infection to grey partridges, in areas where pheasants are also present, will be from the pheasants and any resulting impact will be due to apparent competition.

A potential source of error in the calculation of R_0 for the parasite infecting partridges is that the *H. gallinarum* eggs used in the infection and transmission experiments were obtained from worms infecting pheasants. Since adaptations of *H. gallinarum* to particular host species have been previously demonstrated (Lund, Chute & Myers 1970), our calculated value of $R_{0partridge}$ may be an under-estimate. However, work by Lund & Chute (1974) has shown that this is likely not the case. In their trials, where *H. gallinarum* eggs for experimental infections were obtained from a mix of host species, the 'reproductive potential' of the parasite (number of viable eggs produced per embryonated egg infecting) was 243 times less when infecting grey partridges than when infecting pheasants. This difference is of similar magnitude to that between the values of R_0 calculated in the present study (216 times less when infecting grey partridges than when infecting pheasants).

The observed impact of *H. gallinarum* on the grey partridge appears to be sufficient to cause exclusion when the pheasant is present. However, this outcome could be incorrect for at least three reasons. First, it is possible that the values for parasite-induced increase in partridge mortality ($\alpha_{partridge}$) and decrease in partridge fecundity ($\delta_{partridge}$) used in the model, as estimated from the observed impact on body condition, are too high. The true values may not lead to partridge exclusion. However, as the sensitivity analysis shows, the predicted outcome is relatively robust – partridge and pheasant co-existence cannot be bought about in the model by altering $\alpha_{partridge}$ alone, whilst a drop in $\delta_{partridge}$ of 75% is required. Secondly, as outlined earlier, even though the potential for partridge exclusion exists in this non-spatial model, habitat separation between the two hosts in the wild may decrease the transmission of *H. gallinarum* from pheasants to partridges sufficiently for the two species to co-exist in the parasites presence. Our prediction, based on the approximation of spatial separation employed in this study, is that partridge exclusion would still occur if separation was less than 43%. Further work, both modelling and experimental, is required to test this prediction. However, since *H. gallinarum* has been recorded from wild grey partridges (Clapham 1935; Keymer *et al.* 1962), and we have shown here that the parasite cannot be maintained within a population of grey partridges alone, it is apparent that at least some parasite transmission to this species does occur from other sources in the wild. Finally, the model outcome of partridge exclusion may also be incorrect since our estimation of *H. gallinarum* fitness when infecting the pheasant may be too high. This estimate was based on experiments with naive individuals, while evidence suggests there may be some acquired resistance to *H. gallinarum* infection in the ring-necked pheasant (Lund 1967).

Inaccurate estimation of other parameters is another possible source of error in model predictions. The greatest error may result from the manner in which values of ϕ (parasite establishment success) and β (rate of ingestion of parasite eggs by hosts) were estimated. This is not surprising, since transmission rates are generally considered the hardest of epidemiological parameters to quantify (McCallum & Scott 1994). Experiments documented in Tompkins & Hudson (1999) suggest that density dependence may operate to limit the success of establishing *H. gallinarum* worms down to a maximum of approximately 50 worms per dose. Since the highest value of β obtained was only approximately 17 eggs per day, this is unlikely to affect the results of the transmission trial. If slight density dependence was operating at this level, however, our point estimates of β would be slight under-estimates and our model would be predicting partridge exclusion whilst erring on the side of caution. Another possibility is that our infection experiment, where infective doses of 100 eggs were used, may be under-estimating ϕ. However, due to the manner in which β was estimated, and the manner by which both parameters are incorporated into the model, erroneous estimates of ϕ will not affect model output. What may be causing inaccuracies in model output, however, is the linear fashion by which we scaled estimates of β from values obtained when birds were exposed to known numbers of *H. gallinarum* eggs on unrealistically small areas of ground to values applicable to birds on their natural home ranges. This scaling will be highly sensitive to any inaccura-

cies in the estimates of home range size and will only give approximations of β, since nematode infective stages in the wild are not spread evenly over the habitat, but tend to be localized in 'hot-spot' areas of high use (Saunders, Tompkins & Hudson 1999). This uneven spread will increase rates of *H. gallinarum* transmission, making partridge exclusion more likely to occur in the wild than is predicted here.

A further factor which may also increase the possibility of grey partridge exclusion is the pathogenic protozoan *Histomonas meleagridis*. This parasite, which is the causative agent of 'blackhead', can be transmitted between individuals inside the eggs of *H. gallinarum* (Ruff, McDougald & Hansen 1970). Evidence suggests that while partridges which ingest *H. gallinarum* eggs carrying the protozoan are killed by 'blackhead' before the nematode worms mature, pheasants can survive long enough for next-generation nematode eggs carrying the protozoan to be expelled (Lund & Chute 1974; D.M. Tompkins & P.J. Hudson, unpublished data). Thus, when the protozoan is present, the deleterious effects of *H. gallinarum* transmission from pheasants to partridges would most likely be exacerbated. On a more positive note, however, is the fact that the sensitivity analyses indicate that an increase in partridge fecundity of 41% would alter the model outcome from partridge exclusion to pheasant and partridge co-existence. Since this increased value is within the documented range of natural grey partridge fecundity (Tapper *et al.* 1996), it suggests that management techniques that increase partridge breeding success (such as predation control or habitat improvement) would prevent exclusion due to apparent competition. A decrease in pheasant fecundity of just −32% would also shift the model outcome to pheasant and partridge co-existence.

This study has accomplished its goal, in demonstrating that model simulations run with best available parameter estimates predict that apparent competitive effects of sufficient magnitude to be detectable do occur between the ring-necked pheasant and the grey partridge. Furthermore, the deleterious effects of *H. gallinarum* may be sufficient to cause exclusion of the partridge in areas where a high degree of spatial overlap with pheasants occurs. Thus, this parasite may have played a contributory role in the UK grey partridge decline, and may also be hampering current efforts to re-establish and increase wild partridge populations. However, experimental manipulations at the population scale are required before the role of *H. gallinarum* can be unequivocally proven.

References

Abrams, P. & Matsuda, H. (1996) Positive indirect effects between prey species that share predators. *Ecology*, **77**, 610–616.

Abrams, P., Menge, B.A., Mittelbach, G.G., Spiller, D. & Yodzis, P. (1995) The role of indirect effects in food webs. *Food Webs: Integration of Pattern and Dynamics* (eds G. A. Polis & K. O. Winemiller), pp. 371–395. Chapman & Hall, New York.

Anderson, R.M. & May, R.M. (1978) Regulation and stability of host–parasite population interactions: I. Regulatory processes. *Journal of Animal Ecology*, **47**, 219–249.

Bonsall, M.B. & Hassell, M.P. (1997) Apparent competition structures ecological assemblages. *Nature*, **388**, 371–373.

Bonsall, M.B. & Hassell, M.P. (1998) Population dynamics of apparent competition in a host-parasitoid assemblage. *Journal of Animal Ecology*, **67**, 918–929.

Brittas, R., Marcstrom, V., Kenward, R.E. & Karlbom, M. (1992) Survival and breeding success of reared and wild ring-necked pheasants in Sweden. *Journal of Wildlife Management*, **56**, 368–376.

Clapham, P.A. (1935) Some helminth parasites from partridges and other English birds. *Journal of Helminthology*, **13**, 139–148.

Cocchi, R., Matteucci, C., Meriggi, A., Montagna, D., Toso, S. & Zacchetti, D. (1990) Habitat use of grey partridge and pheasant on a reclaimed land in northern Italy. *International Union of Game Biologists*, **12**, 354–356.

Doster, G.L. & Goater, C.P. (1997) Collection and quantification of avian helminths and protozoa. *Host–Parasite Evolution—General Principles and Avian Models* (eds D. H. Clayton & J. Moore), pp. 396–418. Oxford University Press, Oxford.

Greenman, J.V. & Hudson, P.J. (1998) Parasite-mediated and direct competition in a two-host shared macroparasite system. *Stirling Mathematical Ecology Group, Working Paper #11*.

Greenman, J.V. & Hudson, P.J. (1999) Host exclusion and coexistence in apparent and direct competition: an application of bifurcation theory. *Theoretical Population Biology*, **56**, 48–64.

Grosholz, E.D. (1992) Interactions of intraspecific, interspecific, and apparent competition with host-pathogen population dynamics. *Ecology*, **73**, 507–514.

Hanley, K.A., Vollmer, D.M. & Case, T.J. (1995) The distribution and prevalence of helminths, coccidia and blood parasites in two competing species of gecko: implications for apparent competition. *Oecologia*, **102**, 220–229.

Holt, R.D. (1977) Predation, apparent competition, and the structure of prey communities. *Theoretical Population Biology*, **12**, 197–229.

Holt, R.D. (1984) Spatial heterogeneity, indirect interactions and the coexistence of prey species. *American Naturalist*, **130**, 412–430.

Holt, R.D. & Lawton, J.H. (1993) Apparent competition and enemy-free space in insect host–parasitoid communities. *American Naturalist*, **142**, 623–645.

Holt, R.D. & Lawton, J.H. (1994) The ecological consequences of shared natural enemies. *Annual Review of Ecology and Systematics*, **25**, 495–520.

Hudson, P.J. (1986). *Red Grouse*. The Game Conservancy Trust, Fordingbridge.

Hudson, P.J. & Dobson, A.P. (1997) Transmission dynamics and host–parasite interactions of *Trichostrongylus tenuis* in red grouse. (*Lagopus lagopus scoticus*). *Journal of Parasitology*, **83**, 194–202.

Hudson, P.J., Dobson, A.P. & Newborn, D. (1998) Prevention of population cycles by parasite removal. *Science*, **282**, 2256–2258.

Hudson, P. & Greenman, J. (1998) Competition mediated by parasites: biological and theoretical progress. *Trends in Ecology and Evolution*, **13**, 387–390.

Keymer, I.F., Rose, J.H., Beesley, W.N. & Davies, S.F.M. (1962) A survey and review of parasitic diseases of wild and game birds in Great Britain. *Veterinary Record*, **74**, 887–894.

Lund, E.E. (1960) Factors influencing the survival of *Heterakis* and *Histomonas* on soil. *Journal of Parasitology*, **46**, 38.

Lund, E.E. (1967) Acquired resistance to experimental *Heterakis* infections in chickens and turkeys: effect on the transmission of *Histomonas meleagridis*. *Journal of Helminthology*, **41**, 55–62.

Lund, E.E. & Chute, A.M. (1974) The reproductive potential of *Heterakis gallinarum* in various species of galliform birds: implications for survival of *H. gallinarum* and *Histomonas meleagridis* to recent times. *International Journal for Parasitology*, **4**, 455–461.

Lund, E.E., Chute, A.M. & Myers, S.L. (1970) Performance in chickens and turkeys of chicken-adapted *Heterakis gallinarum*. *Journal of Helminthology*, **44**, 97–106.

May, R.M. (1999) Crash tests for real. *Nature*, **398**, 371–372.

McCallum, H. & Scott, M.E. (1994) Quantifying population processes. *Parasitic and Infectious Diseases* (eds M. E. Scott & G. Smith), pp. 29–45. Academic Press, San Diego.

Menge, B.A. (1997) Detection of direct versus indirect effects: were experiments long enough? *American Naturalist*, **149**, 801–823.

Müller, C.B. & Godfray, H.C.J. (1997) Apparent competition between two aphid species. *Journal of Animal Ecology*, **66**, 57–64.

Potts, G.R. (1986) *The Partridge: Pesticides, Predation and Conservation*. Collins, London.

Potts, G.R. (1997) Cereal farming, pesticides and grey partridges. *Farming and Birds in Europe* (eds Pain, D.J. & Pienkowski, M.W.), pp. 150–177. Academic Press. London.

Rands, M.R.W. (1985) Pesticide use on cereals and the survival of grey partridge clutches: a field experiment. *Journal of Applied Ecology*, **22**, 49–54.

Robertson, P.A. & Dowell, S.D. (1990) The effects of hand rearing on wild gamebird populations. *The Future of Wild Galliformes in the Netherlands* (eds J. T. Lumeij

& Y. R. Hoogeveen), pp. 158–171. Gegevens Koninklijke Bibliotheek, Den Haag.

Ruff, M.D., McDougald, L.R. & Hansen, M.F. (1970) Isolation of *Histomonas meleagridis* from embryonated eggs of *Heterakis gallinarum*. *Journal of Protozoology*, **17**, 10–11.

Saunders, L.M., Tompkins, D.M. & Hudson, P.J. (1999) Investigating the dynamics of nematode transmission to the red grouse *(Lagopus lagopus scoticus)*: studies on the recovery of *Trichostrongylus tenuis* larvae from vegetation. *Journal of Helminthology*, **73**, 171–175.

Schall, J.J. (1992) Parasite-mediated competition in *Anolis* lizards. *Oecologia*, **92**, 58–64.

Schmitz, O.J. (1998) Direct and indirect effects of predation and predation risk in old–field interaction webs. *American Naturalist*, **151**, 327–342.

Schmitz, O.J. & Nudds, T.D. (1994) Parasite-mediated competition in deer and moose: how strong is the effect of the meningeal worm on moose? *Ecological Applications*, **4**, 91–103.

Settle, W.H. & Wilson, L.T. (1990) Invasion by the variagated leafhopper and biotic interactions: parasitism, competition and apparent competition. *Ecology*, **71**, 1461–1470.

Sotherton, N.W. (1998) Land use changes and the decline of farmland wildlife: an appraisal of the set-aside approach. *Biological Conservation*, **83**, 259–268.

Tapper, S. (1992). *Game Heritage: an Ecological Review from Shooting and Gamekeeping Records*. Game Conservancy Trust, Fordingbridge.

Tapper, S., Potts, G.R. & Brockless, M.H. (1996) The effect of an experimental reduction in predation pressure on the breeding success and population density of grey partridges *Perdix perdix*. *Journal of Applied Ecology*, **33**, 965–978.

Tompkins, D.M. & Begon, M. (1999) Parasites can regulate wildlife populations. *Parasitology Today*, **15**, 311–313.

Tompkins, D.M., Dickson, G. & Hudson, P.J. (1999) Parasite-mediated competition between pheasant and grey partridge: a preliminary investigation. *Oecologia*, **119**, 378–382.

Tompkins, D.M., Draycott, R.A.H. & Hudson, P.J. (2000) Field evidence for apparent competition mediated via the shared parasites of two gamebird species. *Ecology Letters*, **3**, 10–14.

Tompkins, D.M. & Hudson, P.J. (1999) Regulation of nematode fecundity in the ring-necked pheasant *(Phasianus colchicus)*: not just density dependence. *Parasitology*, **118**, 417–423.

Wright, V.L., Farris, A.L., Graham, D.L. & Fielder, W.R. (1980) Effects of *Heterakis* and *Histomonas* on the survival of juvenile gray partridge. *Proceedings of Perdix II Gray Partridge Workshop* (eds S. R. Peterson & L. Nelson), pp. 156–164. University of Idaho, Moscow.