Differential impact of a shared nematode parasite on two gamebird hosts: implications for apparent competition

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SUMMARY

If the deleterious effects of non-specific parasites are greater on vulnerable host species than on reservoir host species then exclusion of the vulnerable host through apparent competition is more likely. Evidence suggests that such a mechanism occurs in interactions between the ring-necked pheasant (*Phasianus colchicus*), the grey partridge (*Perdix perdix*), and their shared caecal nematode *Heterakis gallinarum*. Modelling of the system predicts that the reduced parasite impact on the pheasant compared to the partridge results in the force of infection transmitted from pheasants to partridges being sufficient to cause partridge exclusion. Since the parasite impacts are currently estimated from correlational work, controlled infections were conducted to experimentally compare the impact of *H. gallinarum* on the two hosts and verify cause and effect. While challenged partridges showed reduced mass gain, decreased food consumption, and impaired caecal activity, in comparison to controls, the only detectable effect of parasite challenge on the pheasant was impaired caecal activity. The impact of *H. gallinarum* on challenged partridges conforms with previous correlational data, supporting the prediction that parasite-mediated apparent competition with the ring-necked pheasant may result in grey partridge exclusion. However, the observed decrease in the caecal activity of challenged pheasants could imply that *H. gallinarum* may also have an impact on the fecundity and survival of pheasants in the wild, particularly if food is limiting. If this is the case, the associated decrease in the force of infection to which the partridge is exposed may be sufficient to change the model prediction from partridge exclusion to pheasant and partridge coexistence.

Key words: *Heterakis gallinarum*, *Perdix perdix*, *Phasianus colchicus*, partridge, pheasant, parasite-mediated competition.

INTRODUCTION

Non-specific parasites can affect the structure of animal communities through indirect effects such as apparent competition, where the presence of one host species adversely affects another simply through the increased presence of shared parasites (Holt, 1977; Holt & Lawton, 1994), or parasite-mediated competition, where infection alters the relative competitive strength of host species (Freeland, 1983; Price *et al*. 1986). The potential of such interactions was noted over 50 years ago by Haldane (1949) when he stated that 'A non-specific parasite … is a powerful competitive weapon'. However, although studies have explicitly demonstrated that such interactions can affect community structure in the laboratory (Park, 1948; Bonsall & Hassell, 1997), evidence for effects on natural communities is limited (Hudson & Greenman, 1998).

There are many instances in which non-specific parasites are believed to be involved in population declines of threatened wildlife species (Tompkins & Wilson, 1998; Daszak, Cunningham & Hyatt, 2000). In the majority of cases, however, evidence for such an involvement is based on either descriptive or correlational work (Hudson & Greenman, 1998). Specifically, the cause and effect of observed negative correlations between parasite infection and components of host fitness has yet to be ascertained in a controlled manner that is comparable between the host species. This is hardly surprising since a reasonable weight of evidence demonstrating that parasites are a cause of decreased fitness in any wild population has only recently been obtained (Gulland, 1995; Tompkins & Begon, 1999).

Evidence suggests that apparent competition between the ring-necked pheasant (*Phasianus colchicus* (L.)) and the grey partridge (*Perdix perdix* (L.)), mediated via the caecal nematode *Heterakis gallinarum* (Schrank), may have played a role in the decline of wild grey partridge populations in the UK over the past 50 years (Tompkins, Dickson & Hudson, 1999; Tompkins, Draycott & Hudson, 2000*a*). This hypothesis is supported by a 2-host shared macroparasite model which predicts partridge exclusion for the current set of parameter estimates (Tompkins *et al*. 2000*b*). However, since this outcome is based only on correlational evidence for parasite effects (which indicates a greater impact on the partridge than on the pheasant), work is required

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to quantify parasite impact on the 2 hosts in a comparable manner and verify cause and effect. Such work was carried out here through the use of controlled infection experiments.

Differential impact is one of the potential causes of the non-reciprocity that is often observed in enemy-mediated apparent competition (Chaneton & Bonsall, 2000). Such asymmetry, however, does not necessarily increase the likelihood that one of the competing species will be excluded. For example, with parasite-mediated apparent competition, exclusion of vulnerable host species is dependent on the transmission of parasites from 'reservoir' host species (Tompkins *et al*. 2000*a*). Hence, if parasite impact were greater on a reservoir host and lesser on a vulnerable host, the probability that such exclusion would actually occur may be reduced through 2 concurrent mechanisms. First, parasite-induced reductions in reservoir population size/density may decrease the 'force of infection' to which the vulnerable host was exposed whilst, second, the lesser impact on the vulnerable host would mean that more infection would be required to cause exclusion. The possibility of exclusion could only be increased if the opposite were true – a lesser impact on a reservoir host may increase the force of infection to which a vulnerable host was exposed whilst a greater impact on a vulnerable host would mean that less infection would be required to cause exclusion. This second scenario describes the role which the differential impact of *H. gallinarum* plays in the pheasant/partridge system – for example, if parasite impact on host fecundity was either 99% higher for the pheasant or 75% lower for the partridge the predicted outcome would switch from partridge exclusion to host coexistence (Tompkins *et al*. 2000*b*). Thus, verification that the impact of *H. gallinarum* is indeed as high on the grey partridge and as low on the ring-necked pheasant as the correlational work suggests is essential to this demonstration of apparent competition.

MATERIALS AND METHODS

Infection experiment

Heterakis gallinarum effects on host body mass, food consumption, and caecal activity were directly quantified for both the ring-necked pheasant and the grey partridge through the controlled experimental infection of individually caged birds. Both species were reared from day-old chicks on sterilized concrete to ensure that all birds were naive to parasites. Absence of infection was confirmed at 12 weeks of age through the culling and detailed examination of 5 individuals of each species.

The *H. gallinarum* eggs used in this study were from worms collected from pheasants that had acquired natural infections on a Scottish gamebird estate where previous sampling had confirmed the absence of *Histomonas gallinarum* (a protozoan parasite that can be transmitted via *H. gallinarum* eggs; Lund & Chute, 1972). Female worms were maintained for 21 days in 0·5% formalin solution at 21 °C to embryonate all viable eggs, and broken down in saline solution using a small electric blender. Embryonated eggs were then counted in $10 \times 0{\cdot}1$ ml samples, and the volume of saline adjusted to 100 embryonated eggs/ml.

Infections were carried out on the 21st day of the embryonation period, when the recipient hosts were 12 weeks of age. Nine individuals of each host species were randomly selected and given a single oral dose of approximately 100 embryonated *H. gallinarum* eggs, via a tube into the birds crop. This is a realistic challenge since *H. gallinarum* burdens in the low hundreds are common (Tompkins & Hudson, 1999; Draycott *et al*. 2000). A single challenge was chosen over a continuous challenge since hosts are likely to be exposed to 'clumps' of *H. gallinarum* eggs in the wild, due to the highly aggregated nature of this parasite (Tompkins & Hudson, 1999; Draycott *et al*. 2000). However, even if this is not the case, work on other gamebird/nematode systems indicates that the infection resulting from a single challenge is not significantly different from that resulting from a continuous challenge (Shaw & Moss, 1989). Nine other individuals of each species were treated as controls and given 1 ml of saline containing no nematode eggs.

All birds were weighed to the nearest 5 g at the start of the experiment and then maintained in their individual cages (on wire mesh floors) for the following 100 days, during which body mass was monitored at 25-day intervals. Preliminary infection trials indicated that the approximate life-expectancy of *H. gallinarum* worms infecting pheasants is 100 days (unpublished data). Food (gamebird maintenance pellets), water and grit were supplied *ad libitum*, with the mass of pellets provided being recorded whenever hoppers were re-filled. The caecal droppings produced by each bird were collected at 5-day intervals and weighed to the nearest 0·1 g. To confirm that experimental birds were successfully infected, 0·5 g of each sample collected was suspended in 10 ml of saturated salt solution and *H. gallinarum* eggs counted, using McMasters chambers under $100\times$ magnification, in $5 \times 0{\cdot}1$ ml subsamples. At the end of the study all birds were culled and their body condition estimated by measuring the breast muscle mass of each bird to the nearest 0·01 g and adjusting for host body size (see Tompkins *et al*. (1999) for further details).

For each host species, the body mass, food consumption and caecal activity of control versus experimentally challenged birds were compared over the course of the experiment using the repeated measures analysis of variance module in Statistica™

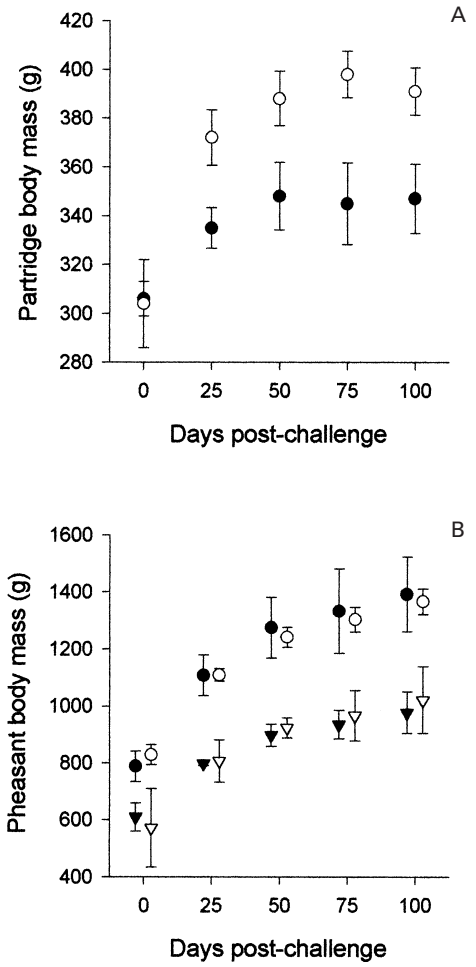

Fig. 1. Comparison of body mass between control birds and (A) partridges and (B) pheasants orally challenged with approximately 100 embryonated *Heterakis gallinarum* eggs. Means and standard errors are shown for 6 control (○) versus 8 experimental (●) partridges, and 6 control (4 male (○) and 2 female (▽)) versus 6 experimental (3 male (●) and 3 female (▼)) pheasants. For the pheasant, body mass was significantly different between the sexes both at the beginning of the study ($F_{1,8} = 20{\cdot}98$, $P = 0{\cdot}002$) and at 25–100 days post-infection ($F_{1,8} = 33{\cdot}65$, $P < 0{\cdot}001$).

v. 5.5, 1999. Both food consumption and caecal dropping production were summed over 25-day intervals for each individual. Host sex was included as a factor in all analyses, being discarded when non-significant.

RESULTS

Over the course of the infection experiment, 6 pheasants and 4 partridges were euthanased due to husbandry problems unrelated to parasite infection. For the remaining birds, observed parasite effects on the host were greater in the grey partridge than in the ring-necked pheasant, even though the level of established infection appeared to be far lower in partridges than in pheasants. During the 100 day trial period, the mean (±S.D.) number of *H. gallinarum* eggs expelled in the caecal droppings of challenged partridges was only 43±84 eggs/day, with eggs detected in the droppings of only 2 of the 8 experimental birds, compared to 3793±7636 eggs/day in the droppings of challenged pheasants (see Tompkins *et al*. (2000*b*) for further details).

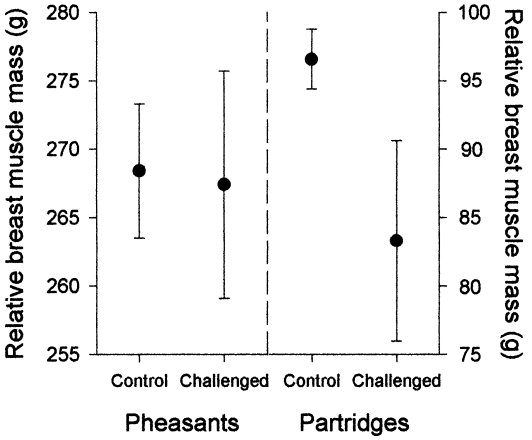

Fig. 2. Comparison of body condition between control birds and both partridges and pheasants orally challenged, 100 days earlier, with approximately 100 embryonated *Heterakis gallinarum* eggs. Means and standard errors are shown for 6 control versus 8 experimental partridges, and 6 control versus 6 experimental pheasants.

Host body mass and condition

While the body mass of control and experimental partridges was similar at the beginning of the study (Fig. 1A; $F_{1,12} = 0{\cdot}01$, $P = 0{\cdot}91$), infection resulted in experimental birds being 11% lighter than control birds at 25–100 days post-challenge (Fig. 1A; $F_{1,12} = 6{\cdot}12$, $P = 0{\cdot}03$). No such pattern was observed for the pheasant, however, where the body mass of control and experimental birds was similar both at the beginning of the study (Fig. 1B; $F_{1,18} = 0{\cdot}00$, $P = 0{\cdot}95$) and post-challenge (Fig. 1B; $F_{1,8} = 0{\cdot}00$, $P = 0{\cdot}99$). The general increase in body mass over the course of the infection trial reflects the last stages of growth of the juvenile birds involved. Thus the parasite reduced the growth rate of partridges but not of pheasants.

The body condition of control versus experimental birds at the end of the infection trial was not significantly different for either the pheasant (Fig. 2A; $F_{1,10} = 0{\cdot}01$, $P = 0{\cdot}91$) or the partridge (Fig. 2A; $F_{1,12} = 2{\cdot}30$, $P = 0{\cdot}15$).

Host food consumption

A significant effect of infection on partridge food consumption was detected as an interaction with days post-challenge (Fig. 3A). There was no significant difference between control and experimental groups over the full 100 days of the study ($F_{1,12} =$

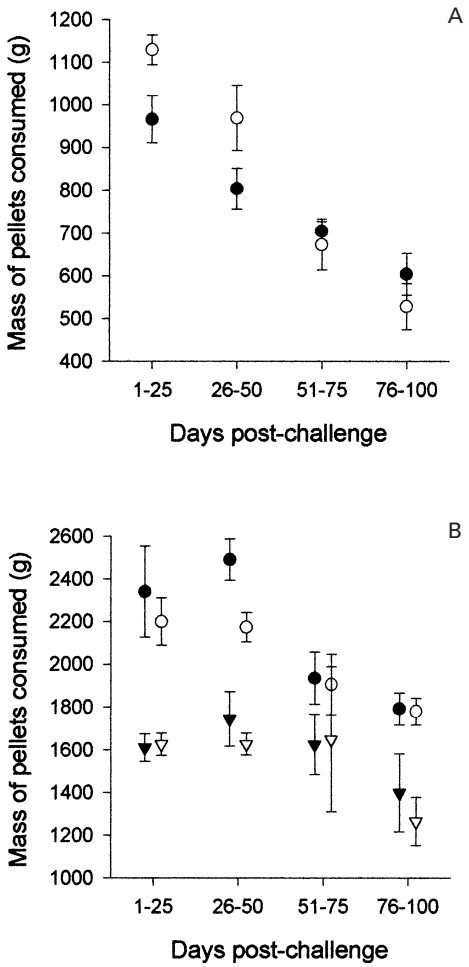

Fig. 3. Comparison of food consumption between control birds and (A) partridges and (B) pheasants orally challenged with approximately 100 embryonated *Heterakis gallinarum* eggs. Means and standard errors are shown for 6 control (○) versus 8 experimental (●) partridges, and 6 control (4 male (○) and 2 female (▽)) versus 6 experimental (3 male (●) and 3 female (▼)) pheasants. For the pheasant, food consumption over the course of the study was significantly different between the sexes ($F_{1,8} = 40{\cdot}40$, $P < 0{\cdot}001$).

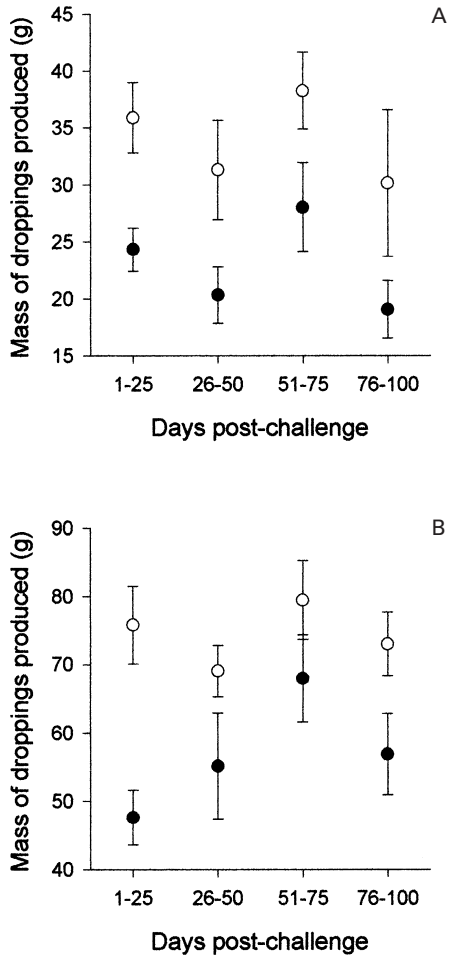

Fig. 4. Comparison of caecal dropping production between control birds and (A) partridges and (B) pheasants orally challenged with approximately 100 embryonated *Heterakis gallinarum* eggs. Means and standard errors are shown for 6 control (○) versus 8 experimental partridges (●), and 6 control (○) versus 6 experimental (●) pheasants.

1·61, $P = 0{\cdot}23$), although control birds consumed 19% more in the first half of the study while experimentally challenged birds consumed 9% more in the second half of the study ($F_{3,36} = 4{\cdot}57$, $P = 0{\cdot}008$).

Again there was no detectable effect of infection on the pheasant (Fig. 3B); food consumption by control and experimental birds was not significantly different ($F_{1,8} = 1{\cdot}20$, $P = 0{\cdot}31$). The general decrease in food consumption over the course of the infection trial may reflect the reduced demands associated with a slow down in growth of the juvenile birds involved.

Host caecal activity

The mass of caecal droppings produced over the course of the study was significantly lower in experimentally challenged birds than in controls for both partridges ($F_{1,12} = 7{\cdot}74$, $P = 0{\cdot}02$) and pheasants ($F_{1,10} = 6{\cdot}64$, $P = 0{\cdot}03$). On average, challenged partridges produced 32% less caecal droppings than control partridges (Fig. 4A) while challenged pheasants produced 23% less caecal droppings than control pheasants (Fig. 4B).

Impact on the grey partridge

Although the body condition of control and experimental partridges at the end of the infection trial was not significantly different (Fig. 2), the body condition of the 2 partridges for which *H. gallinarum* eggs were detected in caecal droppings was significantly lower than that of the other 6 experimental partridges for whom parasite eggs were not detected (Fig. 5; $F_{1,6} = 21{\cdot}01$, $P = 0{\cdot}004$). These birds were also 21% lighter than the other 6 experimental partridges at 25–100 days post-challenge ($F_{1,6} = 48{\cdot}28$, $P < 0{\cdot}001$) and ate 13% less food ($F_{1,6} = 8{\cdot}19$, $P = 0{\cdot}03$), although there was no significant difference in the mass of caecal droppings produced ($F_{1,6} = 1{\cdot}65$, $P = 0{\cdot}25$).

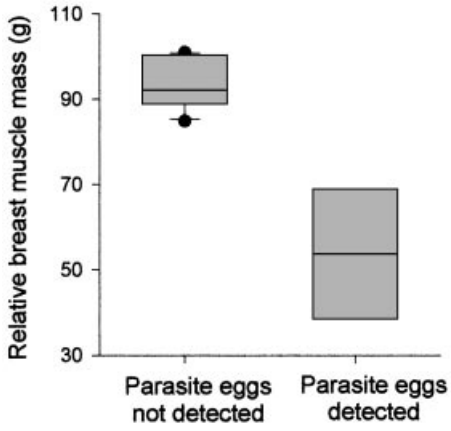

Fig. 5. Box and whiskers plot comparing the body condition of those partridges, orally challenged 100 days earlier with approximately 100 embryonated *Heterakis gallinarum* eggs, for which *H. gallinarum* eggs were detected in caecal droppings ($n = 2$) versus those for which eggs were not detected ($n = 6$).

The impact of *H. gallinarum* was thus greater on 2 of the 8 challenged partridges. However, there was still a deleterious effect of parasite challenge on the other 6, who were 6 % lighter than control partridges at 25–100 days post-challenge ($F_{1,10} = 4{\cdot}89$, $P = 0{\cdot}05$), produced 28 % less caecal droppings ($F_{1,10} = 4{\cdot}82$, $P = 0{\cdot}05$), and for whom there was still a significant effect of treatment on food consumption detectable as an interaction with days post-challenge (control birds consumed 10 % more in the first half of the study, the 6 consumed 6 % more in the second half of the study; $F_{3,30} = 3{\cdot}10$, $P = 0{\cdot}04$).

DISCUSSION

Previous correlational work has suggested that while infections of the caecal nematode *H. gallinarum* have no detectable effect on ring-necked pheasants their impact on grey partridges is of a magnitude likely to cause reductions in host fecundity and survival (Tompkins *et al.* 1999, 2000*a*). In demonstrating that *H. gallinarum* causes a significant reduction in the weight gain and food consumption of partridges, but not of pheasants, the controlled experimental infections conducted in this study show that such a differential impact is indeed the case. There are, however, some detectable effects of *H. gallinarum* on the pheasant – a significant decrease in caecal dropping production was observed in this study for challenged individuals of both host species. Since the avian caecum may play an important role in nutrient absorption (Clench & Mathias, 1995), such an effect on caecal activity could reduce pheasant fecundity and survival in the wild, particularly if food is limiting (Holmes, 1995; Coop & Holmes, 1996). Further work is required to test this hypothesis. Since there is some evidence from parasite removal experiments that nematodes do reduce pheasant reproductive success in the field, a relatively minor impact of *H. gallinarum* on pheasant fecundity was included in the initial parameterization of the pheasant/partridge/*H. gallinarum* model (Tompkins *et al.* 2000*b*). In light of the current experiment, however, the actual impact may be greater.

One possible confounding factor in the results observed here is that, since the *H. gallinarum* eggs used for the experimental infections were derived from naturally infected pheasants, they may have been adapted to this host and thus had a less pathogenic effect on pheasants and a greater pathogenic effect on partridges than they would otherwise. Indeed, adaptations of *H. gallinarum* to particular host species have been demonstrated previously (Lund, Chute & Myers, 1970). However, since the basic reproductive rate (R_0; the number of adult female parasites derived from each adult female parasite in a population of uninfected hosts) for *H. gallinarum* in the partridge is far below unity (estimated as 0·006; Tompkins *et al.* 2000*b*), most, if not all, of the parasites infecting partridges in the wild must be derived from other host species. Since the pheasant is the main reservoir host of *H. gallinarum* (Lund & Chute, 1974; Draycott *et al.* 2000), in which parasite R_0 has been estimated as 1·23 (Tompkins *et al.* 2000*b*), *H. gallinarum* eggs of pheasant origin are the correct ones to use when investigating the impact of this parasite on the grey partridge.

Failure to gain weight, inappetence (reduction in voluntary food intake), and the disruption of normal gastrointestinal function, as observed in this study, are all common clinical consequences of infection by gastrointestinal nematodes (Holmes, 1987). For many hosts, these effects are believed to be principally caused not by the established adult worms but rather by the developing larvae (e.g. Sadun, 1950; Williams *et al.* 1983; Ovington, 1985; Shaw & Moss, 1990). Since, in the current study, food consumption of grey partridges was only reduced up to 50 days post-challenge and increased (possibly in compensation) thereafter, and by 100 days post-challenge there was no significant difference in the body condition of control and challenged partridges, the same may be true for *H. gallinarum* infections (where larvae infecting partridges mature after approximately 40 days; Tompkins *et al.* 2000*b*). Greater deleterious effects of larval as opposed to adult *H. gallinarum* would not be surprising since only the larvae have a tissue stage which can damage the caecal mucosa and possibly elicit host inflammatory responses (Kaushik & Sharma Deorani, 1969). Such damage could potentially affect caecal activity, as was observed.

The lack of a significant difference in the body condition of control and experimental partridges at the end of the infection trial was due to a greater level

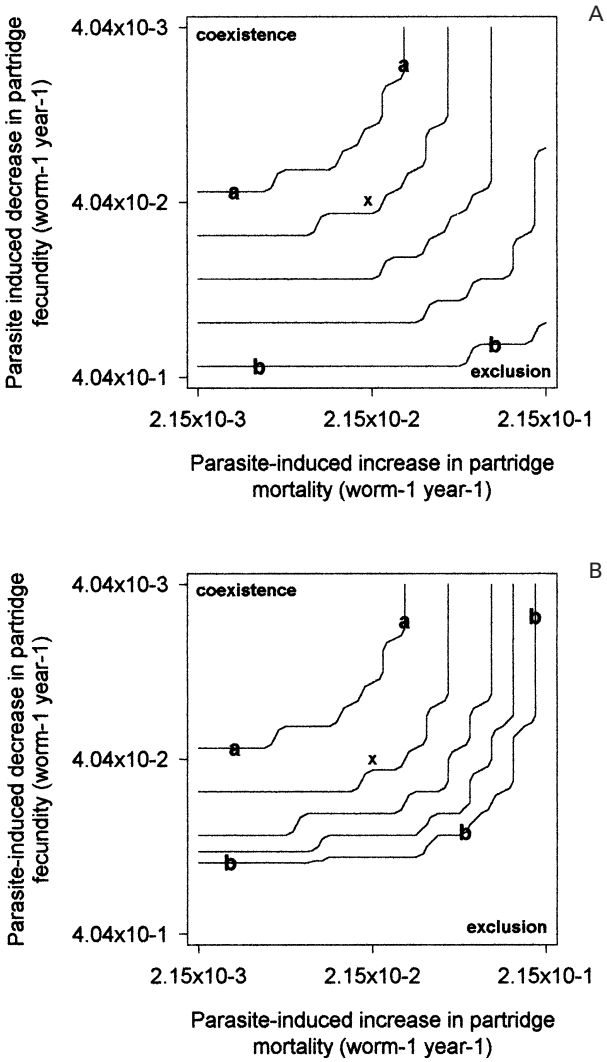

Fig. 6. Exclusion – coexistence boundary curves in the parasite-induced increase in partridge mortality/ parasite-induced decrease in partridge fecundity cross-section of parameter space for the pheasant/partridge/ *H. gallinarum* model detailed by Tompkins *et al.* (2000*b*) illustrating how the boundary shifts as (A) the parasite-induced decrease in pheasant fecundity is increased logarithmically from its estimated value of $8{\cdot}28 \times 10^{-4}$/worm/year (curve a) to $8{\cdot}28 \times 10^{-3}$/worm/year (curve b), and (B) the parasite-induced increase in pheasant mortality is increased linearly from 0·00 (curve a) to $3{\cdot}00 \times 10^{-3}$/worm/year (curve b). The parameters on both axes are scaled from 0·1 to 10 times their empirical values. Crosses indicate the predicted outcome at the empirical values. The outcome shifts from partridge exclusion to host coexistence when either the decrease in pheasant fecundity is increased to $1{\cdot}65 \times 10^{-3}$/worm/year, or the increase in pheasant mortality is increased to $5{\cdot}33 \times 10^{-4}$/worm/year.

of variance in the condition of challenged individuals. Such an increase is a common effect of parasite infection on animal populations (Whitlock, 1961), and in this case was caused by the parasite challenge having greater deleterious effects on some individuals than on others. However, even though not significant, the magnitude of the overall difference was equivalent to that recorded in the previous correlational studies. Since the establishment success of *H. gallinarum* infecting the grey partridge is 6·5% (Tompkins *et al.* 2000*b*), the effects observed in this study (where individuals were challenged with approximately 100 *H. gallinarum* eggs each) are related to a mean established parasite intensity of 6·5 worms. This is equivalent to a decrease in body condition of 2·0 g breast muscle mass/worm, which is consistent with the 1·1–2·5 g/worm range seen in the correlational data (Tompkins *et al.* 1999; Tompkins *et al.* 2000*a*).

In verifying that *H. gallinarum* is a cause of decreased body condition in gamebirds, and clearly showing that it does indeed have differential impacts on the ring-necked pheasant and the grey partridge, this study has accomplished its goal. The experimental results documented here, however, have contrasting implications for the prediction that parasite-mediated apparent competition between the pheasant and the grey partridge results in partridge exclusion. Since the impact of *H. gallinarum* on challenged partridges was of a magnitude similar to that previously suggested by the correlational data, the deleterious effects of this parasite do appear to be sufficient to cause partridge exclusion. However, the possibility that *H. gallinarum* may have an impact on pheasants that is greater than is currently included in the 2 host shared-macroparasite model of the system could mean that the force of infection from pheasants to partridges in the wild may not be sufficient for such exclusion to actually occur. The shift in model outcome from exclusion to host coexistence due to this effect is illustrated in Fig. 6, demonstrating how the inter-specific transmission of parasites from reservoir to vulnerable hosts decreases as the parasite impact on the reservoir host increases.

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