INTERNATIONAL ATOMIC ENERGY AGENCY
UNITED NATIONS EDUCATIONAL, SCIENTIFIC AND CULTURAL ORGANIZATION
INTERNATIONAL CENTRE FOR THEORETICAL PHYSICS
I.C.T.P., P.O. BOX 586, 34100 TRIESTE, ITALY, CABLE: CENTRATOM TRIESTE

UNITED NATIONS INDUSTRIAL DEVELOPMENT ORGANIZATION

INTERNATIONAL CENTRE FOR SCIENCE AND HIGH TECHNOLOGY
c/o INTERNATIONAL CENTRE FOR THEORETICAL PHYSICS 34100 TRIESTE (ITALY) VIA GRIGNANO, 9 (ADRIATICO PALACE) P.O. BOX 586 TELEPHONE 040-224572 TELEFAX 040-224575 TELEX 460449 APH I

SMR.648 - 49

SECOND AUTUMN WORKSHOP ON MATHEMATICAL ECOLOGY

(2 - 20 November 1992)

"Do Parasites Make Prey Vulnerable to Predation? Red Grouse and Parasites"

A.P. Dobson
Dept. of Ecology & Evolutionary Biology
Princeton University
Princeton, NJ 08544-1003
U.S.A.

These are preliminary lecture notes, intended only for distribution to participants.

Journal of Animal Ecology 1992, 61, 681–692

Do parasites make prey vulnerable to predation? Red grouse and parasites

PETER J. HUDSON, ANDREW P. DOBSON* and DAVID NEWBORN

*Game Conservancy, Crubenmore Lodge, Newtonmore, Inverness-shire PH20 1BE, Scotland; and *Ecology & Evolutionary Biology, Princeton University, Princeton, New Jersey, 08544-1003, USA*

Summary

1. An extensive post-mortem survey of grouse revealed that birds killed by predators in spring and summer had significantly greater burdens of the caecal nematode *Trichostrongylus tenuis* than grouse shot during the autumn. Furthermore, grouse that appeared to have died through the effects of parasites carried greater worm burdens than grouse killed by predators.

2. The proportion of grouse with high levels of parasite infection increased with the intensity of predator control as measured indirectly through keeper density. These two empirical observations suggest that predators selectively prey on heavily infected grouse.

3. The interactions between parasites and predators were examined experimentally by reducing the worm burdens of female grouse with an oral anthelmintic. Nests of treated and untreated females were subsequently located either by research workers flushing the incubating female or by dogs trained to locate birds by scent. The dogs found significantly fewer of the treated than control birds, suggesting that female grouse with large parasite burdens emit more scent and are more vulnerable to mammalian predation.

4. A modified mathematical model of the grouse–nematode system is described which incorporates the effects of both random and selective predation of heavily parasitized grouse. An analysis of the model illustrates the importance of interactions between grouse, parasites and predators in determining the relative densities of each. In particular, when predators selectively remove heavily parasitized individuals, then low levels of predation can lead to increases in the size of the host (or prey) population.

Key-words: predator selection, parasite–host dynamics, population cycles, red grouse, parasite-induced effects.

Journal of Animal Ecology (1992), **61**, 681–692

Introduction

A number of studies have shown that predators take a disproportionately large number of prey infected with parasites (Van Dobben 1952; Holmes & Bethel 1972; Vaughan & Coble 1975; Temple 1987). In some of these studies it is not clear whether the parasites were the proximate or ultimate cause of the death. In some cases the parasites significantly alter the host's appearance, or behaviour, to facilitate transmission to the definitive host (Giles 1983; Moore 1984; Dobson 1988), while in other cases, the pathology leads to the death of both host and parasite. Many studies of predation demonstrate that predators tend to forage for more profitable prey items (reviewed by Krebs 1978) and in simple energetic terms it may be to the predator's advantage to select the weaker individuals from the prey population (Mech 1970; Schaller 1972; Temple 1987). As many parasitic infections lead to host morbidity, hosts with high parasite intensities should be more susceptible to predation (Anderson 1979).

Like many ground-nesting birds, red grouse (*Lagopus lagopus scoticus* (Lath.)) are vulnerable to predation when incubating and show various adaptations to avoid capture. For most of the year, grouse emit scent which can be detected by a trained pointing dog at distances of up to 50 m. Female grouse stop producing caecal faeces during the spring incubation period and dogs can only locate incu-

bating females from a distance of less than 0·5 m (St. John 1878). This reduction in scent emission is associated with a change in the function of the bird's caeca that appears to be an adaptation to reduce detection. The parasitic nematode *Trichostrongylus tenuis* (Eberth.) is known to burrow deeply into the caecal mucosa causing disruption of the plicae and bleeding (Watson, Lee & Hudson 1987). This pathology may hinder the control of scent emission and so increase the susceptibility of heavily infected incubating birds to mammalian predators that hunt by scent.

In this study we investigate the population dynamic interactions of predator, prey and parasite in the grouse–*T. tenuis* system using a hierarchical approach that incorporates empirical data, a controlled experiment and mathematical modelling. First we present population data that indicate that predators selectively remove heavily infected grouse from a population and that grouse populations subjected to reduced predation have larger worm burdens. Secondly, we examine experimental data collected at the individual level to test whether infection of grouse with *T. tenuis* made the birds easier for trained dogs to find. We compared the success rate of dogs in finding nests of females with natural infection levels and those with experimentally reduced infections. Finally, we explore the dynamical consequences of selective predation on the grouse–*T. tenuis* system using a model developed by Dobson & Hudson (1992). The model is used to discuss the differences between the empirical patterns observed in this study and studies of grouse and parasite populations in other areas.

Methods

Intensity of infection in grouse shot and killed by predators

The main study area consisted of 17 km^2 of managed grouse moor known as Gunnerside Moor in upper Swaledale, North Yorkshire, England. Regular visits to the study area were made from 1980 to 1984 and all dead grouse found were removed. Each corpse was carefully examined for any external cause of injury and in some instances skinned for signs of bite marks. During this study no grouse had died from 'accidents' since fence lines and overhead cables were absent. Each corpse was classed either as 'killed by predator' or, if there was no external injury, as 'found dead' (after Jenkins, Watson & Miller 1964). Careful examination allowed us to determine whether the grouse had struggled at death indicating predation rather than death followed by scavenging of the corpse. Any grouse found during visits to other moors were treated in the same ways as those from the main study area. All corpses were brought back to the laboratory and, if the remains were in

Table 1. Population parameters for *T. tenuis* and red grouse

Parameter	Symbol	Estimated value (range)
Grouse fecundity	a	1·8 (0–2) $year^{-1}$
Grouse mortality	b	1·05 $year^{-1}$
Parasite fecundity	λ	11 (9·2–11·5) $year^{-1}$
Adult worm mortality	μ_P	1·0 (0·8–1·2) $year^{-1}$
Arrested larvae mortality	μ_A	0·5 $year^{-1}$
Mortality of free-living parasite stages	γ	6·5–13 $year^{-1}$
Duration of arrestment	$1/\Theta$	2–6 months
Parasite pathogenicity	α	3×10^{-4} per worm $year^{-1}$
Parasite reduction in host fecundity	δ	5×10^{-4} per worm $year^{-1}$
Aggregation of parasites in hosts	k	1·0 (0·5–1·8)
Transmission rate	β	0·11 (0·6–1·5 × 10^1) per larvae per host $year^{-1}$
Transmission constant	H_0	60–120 (γ/β)
Predation rate	ρ	100 per fox $year^{-1}$
Parasite-induced increase in susceptibility to predation	π	1×10^{-3} per parasite
Density-dependent reduction in grouse breeding success	w	0·0003–0·03 per grouse $year^{-1}$

The derivation of the parameter estimates for the parasite and grouse are discussed in Hudson, Newborn & Dobson (1992) and Dobson & Hudson (1992). We have assumed that an adult fox will take on average one or two grouse a week, the estimate of π is derived in the main text. The density-dependent parameter w is set at a range of values in Fig. 9 that allows us to examine a wide range of grouse densities km^{-2}.

fit condition, the intensity of *T. tenuis* infection was determined.

Caeca were removed, weighed, and the contents washed with water over a 210 μm gauze. Adult worms were washed from the gauze, subsampled and counted (described by Wilson 1983; Hudson 1986a,b; Hudson, Newborn & Dobson 1992). During the shooting season (12 August–10 December) shot grouse were aged (see Watson & Miller 1976), and the level of *T. tenuis* infection determined in adults (>1 year old) and immature birds (<6 months old). Only data collected from adult birds were used in the comparison of worm burdens. As with most studies of parasitic helminths, the statistical distribution of worm burdens were aggregated (Anderson 1979; Anderson & Gordon 1982). Most transformations of the data were insufficient to allow the use of standard parametric tests, so the data were analysed using a chi-squared test under the null hypothesis that there were no differences between the distributions.

Intensity of predator control

Grouse moors employ gamekeepers primarily to control the number of predators. In northern England, where crows are relatively scarce, foxes are considered by keepers to be the major predator of grouse (Hudson & Watson 1985). Interviews with gamekeepers and estate managers were used to obtain information on the size and position of grouse moors and the numbers of keepers employed on each. The density of keepers was then determined by counting the numbers within 5 km of the centre of each grouse moor and expressing this as the density of gamekeepers per 100 km^2. On estates which employed more than one gamekeeper the process was repeated for each keeper's beat and the average taken for the whole estate. As earlier studies indicate that keeper density varies inversely with the number of foxes shot (Hudson 1986b), keeper density was used as an inverse measure of predation pressure (Hudson & Newborn 1990; Hudson 1992). In the area of northern England where these studies were undertaken, there were no significant confounding associations between keeper density and other aspects of moorland management such as heather burning or percentage of young heather available (Hudson 1986b; Hudson 1992; Hudson & Booth, unpublished).

Treatment of grouse to reduce parasite burdens

In the spring of 1983 and 1984, female grouse were located at night with strong quartz halogen lamps, dazzled and caught (Hudson 1986a). In 1983, alternate females captured were given either 2 ml Levamisole hydrochloride to reduce the infection of *T. tenuis* or 2 ml water as a control. Female grouse were then fitted with a back-tab or radio-transmitter. In 1984, all female grouse caught were treated and fitted with back-tabs and untreated females were used as a comparative group.

Location of grouse nests

The study area was divided into blocks and systematically searched by walking up and down each block with the dogs quartering the ground. Field observers located nests by inadvertently flushing an incubating female, whereas the dogs found the nests by scent; invariably these nests were well hidden from both the dogs and human observers. In 1983, when radio-transmitters were fitted to both treated and untreated females, the general nesting area was located from the radio signal and then searched systematically with the dogs. While searching for radio-tagged females, the observers were unaware whether the females were treated or control birds. Once the nest was located the content of each nest was checked and clutch size recorded.

Dynamics of predation: mathematical model

The mathematical models described in Dobson & Hudson (1992) are modified here to include the effects of predation on the grouse and consequent effects on the parasite population. We consider two models: first the simplest case where the parasites have no effect on the vulnerability of their host to predation, and second the case where the susceptibility of the grouse to predation increases with intensity of parasite burden.

The models of Dobson & Hudson (1992) examined the parasite–host system using three coupled differential equations which describe changes in grouse (H), adult parasites (P), and arrested larval stages (A). Larval arrestment, or hypobiosis, occurs when the parasite larvae enter a period of dormancy immediately after infecting the host; this leads to reductions in the population growth rate of the parasite (Dobson & Hudson 1992). A model was also developed to consider the free-living larval stages of the parasite; here we describe the simpler model without free-living stages. As the free-living stages enter the system as an additional linear expression both models produce essentially similar results. The dynamics of the model can be described according to the following three equations:

$$\frac{dH}{dt} = (a-b)H - (\alpha+\delta)P - \rho HF - \pi\rho FP \qquad \text{eqn 1}$$

$$\frac{dA}{dt} = \frac{\lambda PH}{H+H_o} - (\mu_A + b + \theta + \rho F)A - \alpha\frac{PA}{H} - \pi\rho\frac{PAF}{H} \qquad \text{eqn 2}$$

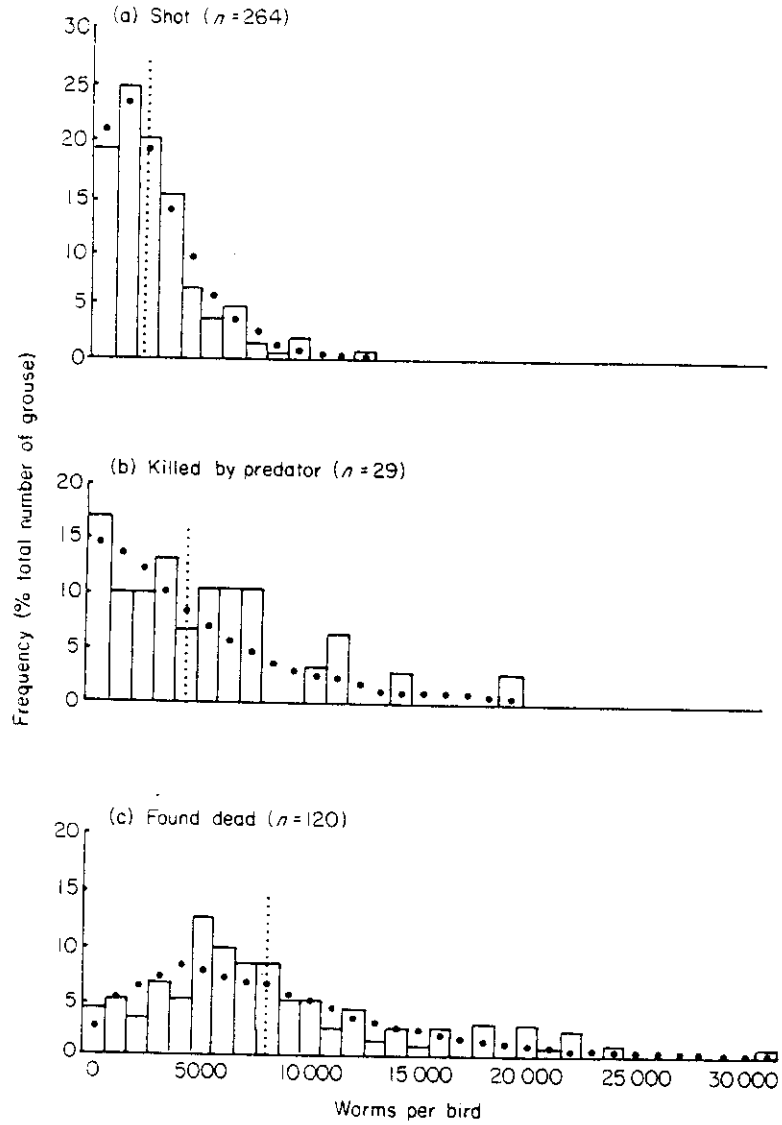

Fig. 1. Frequency distribution of *T. tenuis* in grouse from the main study area 1980–84. Points show the predictions of the negative binomial probability distribution and broken vertical lines the mean worm burden: (a) grouse shot ($n = 264$, mean = 2284, $k = 2{\cdot}2$); (b) grouse killed by predators ($n = 29$, mean = 4759, $k = 1{\cdot}2$); (c) grouse found dead ($n = 120$, mean = 8292, $k = 2{\cdot}4$).

$$\frac{dP}{dt} = \theta A - (\mu_p + \alpha + b + \rho F)P - \alpha k' \frac{P^2}{H} - \pi\rho FH\left[\frac{P}{H} + \frac{P^2}{H^2}k'\right]. \qquad \text{eqn 3}$$

The parameters are described in Table 1. The model assumes that a constant number of predators, F, catch grouse at a rate, ρ, while the *per capita* rate at which parasites increase the susceptibility of their hosts to predation is π. When the parasites have no effect on the host's susceptibility to predation, then π and all the terms including π in equations 1, 2 and 3 are zero. The model assumes that the dynamics of the predator population are independent of the grouse and that predation rates increase with grouse density as a simple linear Type I functional response (Holling 1959). These assumptions seem reasonable for most predators of grouse, and in particular for the fox, a generalist predator which has serious impacts on grouse density (Hudson & Watson 1985; Hudson 1986b, 1992; Hudson & Newborn 1990; Hudson & Booth, unpublished). The more complicated dynamics of prey-driven predator dynamics and variations in the functional and numerical response are considered elsewhere (Dobson & Hudson, unpublished).

Results

Parasite intensity and cause of grouse death

Grouse killed by predators carried significantly higher burdens of parasites than adult grouse shot in autumn, on both the main study area (Fig. 1; $\chi^2 = 13{\cdot}4$, $P < 0{\cdot}005$, df = 3) and in birds collected from all moors (Fig. 2; $\chi^2 = 22{\cdot}5$, $P < 0{\cdot}001$, df = 4). Furthermore, grouse found dead had higher levels of infection than grouse killed by predators on both the main study area (Fig. 1; $\chi^2 = 13{\cdot}3$, $P < 0{\cdot}005$, df = 3), and in the collections from all moors (Fig. 2;

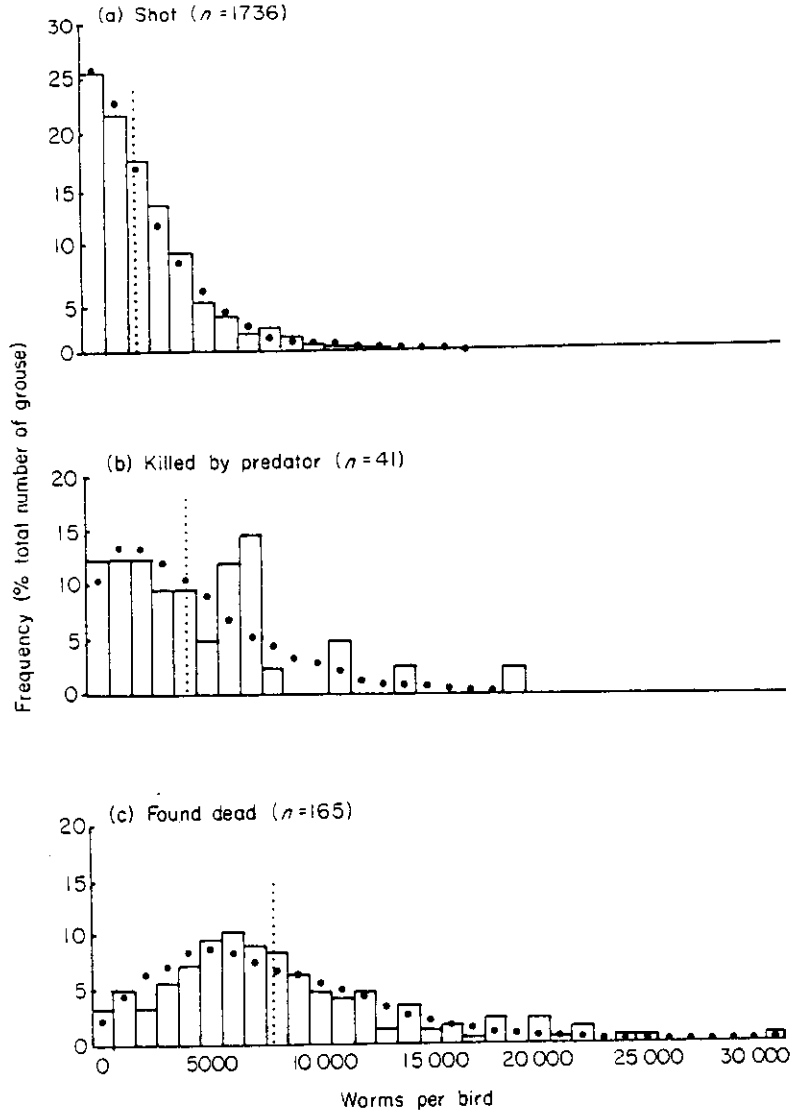

Fig. 2. Frequency distribution of *T. tenuis* in grouse from all study moors in the north of England: (a) grouse shot ($n = 1736$, mean = 2291, $k = 1{\cdot}4$); (b) grouse killed by predators ($n = 41$, mean = 4585, $k = 1{\cdot}8$); (c) grouse found dead ($n = 165$, mean = 8248, $k = 2{\cdot}8$).

$\chi^2 = 20{\cdot}7$, $P < 0{\cdot}001$, df = 5). These results imply that grouse killed by predators tend to be heavily infected and thus vulnerable to predation.

Most of the grouse killed by predators were found during March (24%), April (29%) and May (26%), 3–5 months before the sample of shot birds were obtained. Although a more direct comparison would be attained by comparing parasites from burdens from birds collected at the same time, management policies for grouse populations prohibit shooting before the end of the breeding season. The parasite burdens of adult birds that are shot in the autumn reflect not only the worm populations that have passed the winter in the birds as adults and arrested larvae, but also the worms the bird has acquired during the summer infection period (Hudson & Dobson 1990; Hudson, Newborn & Dobson 1992). As there is a period of parasite infection between the collection of the spring sample of birds found dead and the collection of shot birds in the autumn this creates a bias against a significant result. Despite this bias, shot birds still have lower parasite burdens than birds killed by predators; we feel that the result *underemphasizes* the impact of parasitism on grouse populations. Furthermore, comparisons within a year (1983) found that significantly more of the grouse killed by predators carried high intensities of parasite infection than from the sample of shot birds (Fig. 3, Fisher Exact Test, $P < 0{\cdot}05$), demonstrating that predators selectively killed the heavily infected individuals from the population.

Intensity of predator control and level of infection

The density of gamekeepers could be determined for 44 of the 46 estates from which the intensity of *T. tenuis* were measured; keeper density ranged from 0 to 2·55 keepers $100\,km^{-2}$ on these estates. These data were divided into one of five categories and a frequency distribution of *T. tenuis* per grouse determined for each level of predator control (Fig. 4). There was a significant difference between the

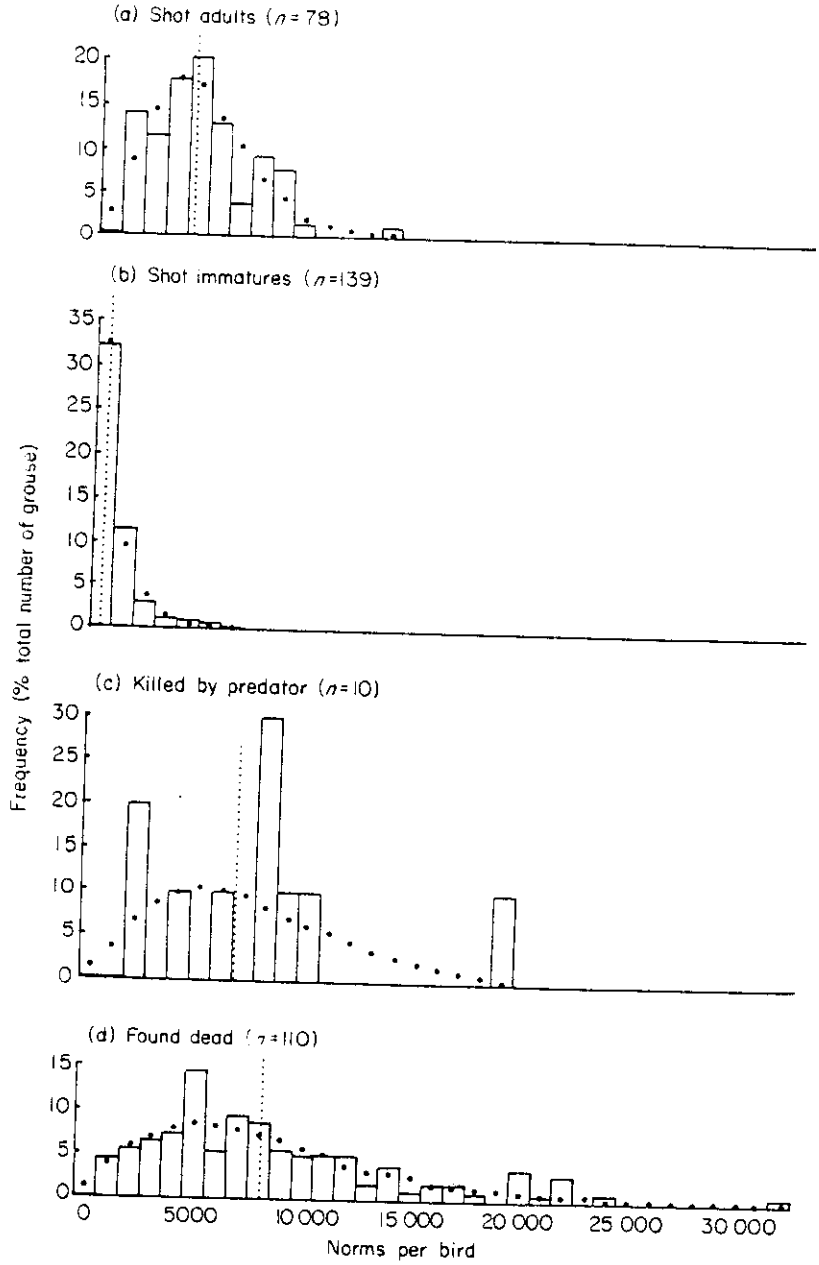

Fig. 3. Frequency distribution of *T. tenuis* in grouse from the main study area during 1983: (a) adult grouse shot ($n = 78$, mean = 4128, $k = 12{\cdot}6$); (b) immature grouse shot ($n = 139$, mean = 604, $k = 0{\cdot}6$); (c) grouse killed by predators ($n = 10$, mean = 7100, $k = 4{\cdot}4$); (d) grouse found dead ($n = 110$, mean = 8636, $k = 3{\cdot}1$).

observed distributions ($\chi^2 = 1487{\cdot}5$; $P < 0{\cdot}001$; df = 24), and an increase in the frequency of birds with high worm burdens (>5000 worms per bird) at high keeper density ($r = 0{\cdot}97$, $P < 0{\cdot}01$, df = 3). Furthermore, on those estates with samples from more than 40 birds there was a positive association between the proportion of birds with high parasite burdens (>5000 worms per bird) and the intensity of predator control measured as density of keepers 100 km^{-2} ($r = 0{\cdot}57$, $P < 0{\cdot}02$, df = 15).

Nests of treated and untreated female grouse

Dogs that were trained to locate nests by scent were significantly better than humans at locating nests incubated by untreated control females with natural levels of infection (Table 2, 1983, $Z = -3{\cdot}374$,

Table 2. Grouse nests found by dogs (scent) and random search (researchers) with respect to treatment of the female with an anthelmintic to reduce parasite burdens

		Number found	
Year	Treatment	Scent dogs	Random search field workers
1983			
Treated	Low worm burden	6	7
Untreated	High worm burden	37	10
1984			
Treated	Low worm burden	9	7
Untreated	High worm burden	29	7

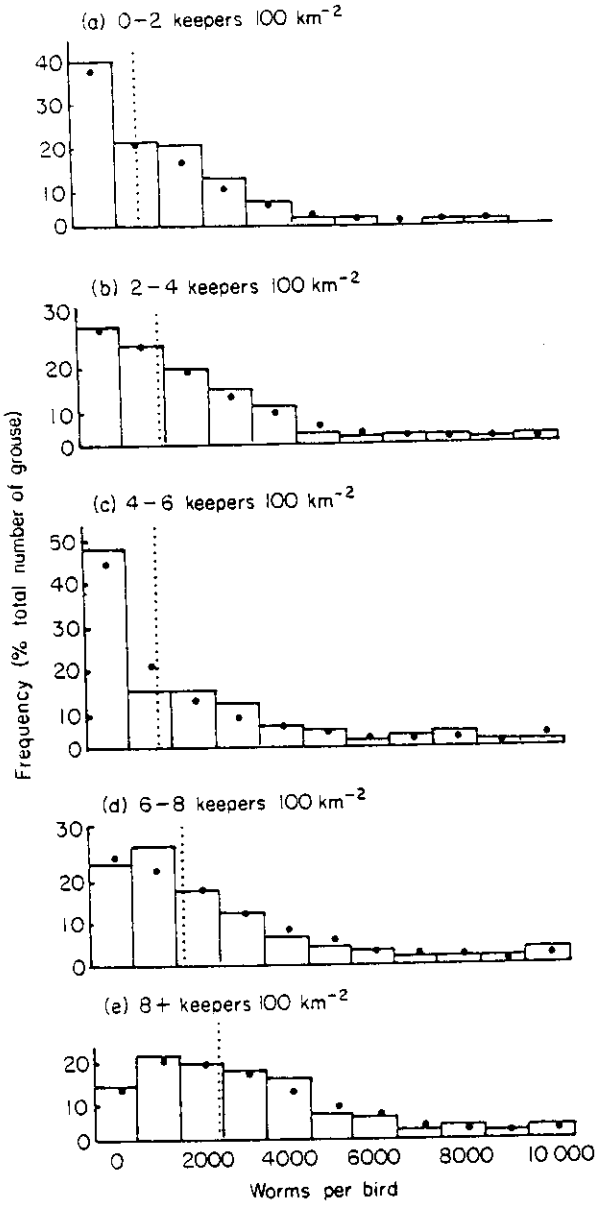

Fig. 4. Frequency distribution of worms per bird from estates for varying intensities of predator control measured as the density of keepers within a 5-km radius: (a) 0–2 keepers 100 km^{-2} ($n = 113$, mean = 1487, $k = 1{\cdot}3$); (b) 2–4 keepers 100 km^{-2} ($n = 349$, mean = 2023, $k = 1{\cdot}5$); (c) 4–6 keepers 100 km^{-2} ($n = 180$, mean = 1844, $k = 0{\cdot}6$); (d) 6–8 keepers 100 km^{-2} ($n = 653$, mean = 2270, $k = 1{\cdot}4$); (e) 8+ keepers 100 km^{-2} ($n = 435$, mean = 2900, $k = 2{\cdot}9$).

$P = 0{\cdot}0004$; 1984, $Z = -3{\cdot}872$, $P = 0{\cdot}0001$). In contrast, there were no significant differences in the ability of humans or dogs to locate nests belonging to treated birds with reduced parasite burdens (Table 2, $\chi^2 = 0{\cdot}04$, $P > 0{\cdot}1$, df = 1). The mean clutch size of females pointed by the dogs was lower than that of the treated females in both years (Table 3; 1983, $t = 2{\cdot}53$, $P < 0{\cdot}01$, df = 58; 1984, $t = 2{\cdot}29$, $P < 0{\cdot}01$, df = 50). As high levels of parasitism have been shown to reduce clutch size (Hudson 1986a,b; Hudson, Dobson & Newborn 1992), these results suggest that grouse with low parasite burdens are more difficult to detect by scent than those with high parasite burdens.

Table 3. Clutch size of nests found by scent (dogs) and random search (research workers)

	Clutch size						
	Found by scent			Found at random			
Year	Mean	SE	*n*	Mean	SE	*n*	Signif.
1983	6·58	1·69	43	7·82	1·76	17	$t = 2{\cdot}53$**
1984	7·63	1·79	38	8·86	1·46	14	$t = 2{\cdot}29$**

** $P < 0{\cdot}01$.

This experiment also allows us to estimate the increase in susceptibility of the hosts to predation due to the parasite. In both years hosts with natural parasite burdens were found three to four times more often than hosts with reduced burdens. Although the reduction in worm burden in 1984 was larger than in 1983 (8996–1200, compared with 2037–900), these data allow us to obtain a coarse estimate for π of 1×10^{-3} ($5 \times 10^{-4} \rightarrow 3 \times 10^{-3}$).

Parasite intensity and size of caeca

The weight of grouse caeca was positively correlated with worm burdens for all adult female grouse examined (Fig. 5; $r = 0{\cdot}60$, $n = 208$, $P < 0{\cdot}001$, df = 216). It would seem likely that this increase in caecal weight was a response to damage caused by the worms to the structure of the caeca (Watson *et al.* 1987).

Dynamics of predation: mathematical model

The dynamical consequence of adding predation to the parasite–host model of Dobson & Hudson (1992) may be initially explored by examining the system at equilibrium, i.e. $dH/dt = dP/dt = dA/dt = 0$. The equilibrium mean parasite burden is given by the following expressions for each model.

Model 1: no increased susceptibility to predation:

$$\frac{P^*}{H^*} = M_p^* = \frac{(a - b - \rho F)}{(\alpha + \delta)} \qquad \text{eqn 4}$$

Model 2: increased susceptibility to predation due to parasites:

$$\frac{P^*}{H^*} = M_p^* = \frac{(a - b - \rho F)}{(\alpha + \delta + \pi\rho F)} \qquad \text{eqn 5}$$

In both instances the presence of predators reduces the mean parasite burden (Fig. 6). If the parasites increase the susceptibility of hosts to predation, then larger reductions in parasite burden are produced since predators selectively remove the heavily infected individuals. In the absence of other mechanisms which may act to counter these effects, increased rates of predation may drive the parasites to extinction by pushing host density below the threshold for parasite establishment (Dobson & May 1986; Dobson & Hudson 1992).

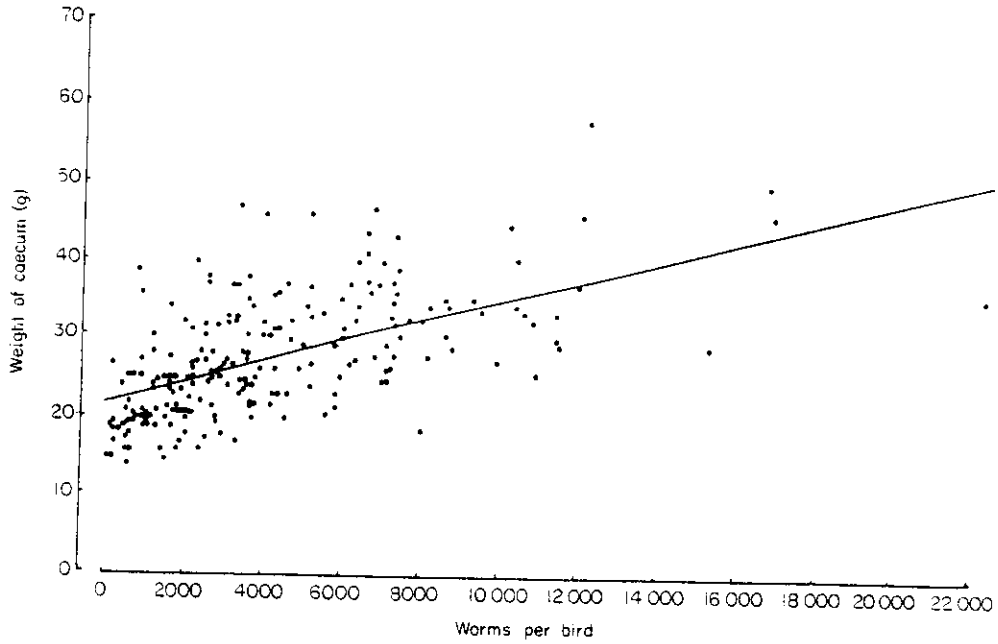

Fig. 5. Relationship between caecal weight and intensity of parasite infection in female grouse showing that individuals with high parasite burdens tend to have larger caeca ($r = 0{\cdot}60$, $n = 208$, $P < 0{\cdot}001$, df = 202).

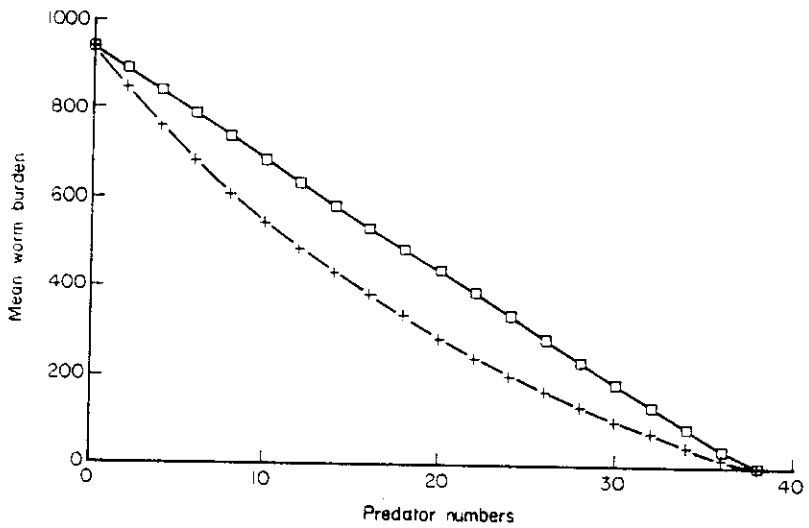

Fig. 6. Relationships between mean parasite burden and predator density for predators that do not selectively take heavily infected individuals (□) and predators that selectively attack infected individuals (+). Parameter values used are $\alpha = 0{\cdot}0003$, $\delta = 0{\cdot}0005$, $\pi = 0{\cdot}001$, $a = 1{\cdot}8$, $b = 1{\cdot}05$. Predator density is expressed as numbers per 100 km^2.

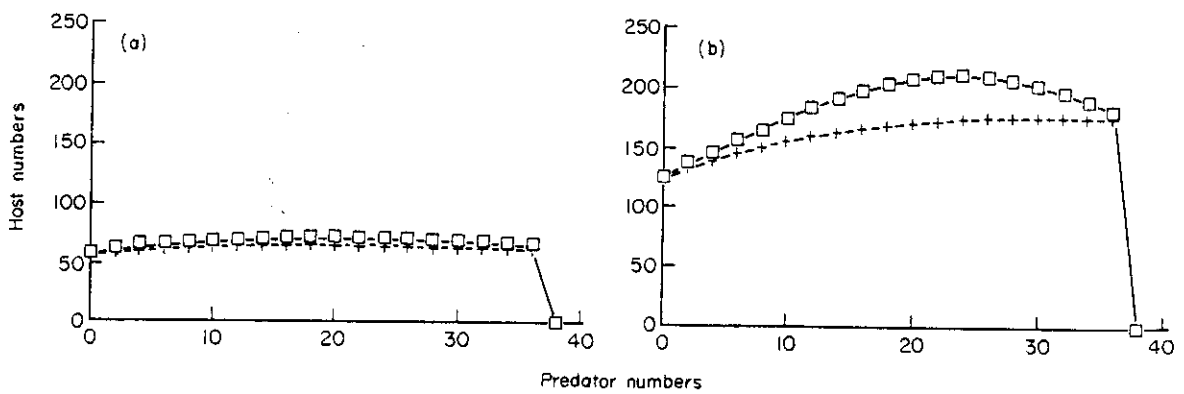

Fig. 7. Relationship between grouse density and predator numbers for predators that do not selectively take heavily infected individuals (□) and predators that selectively take infected individuals (+). Parameter values as in Fig. 6 with $\mu_p = 1{\cdot}5$, $\mu_A = 0{\cdot}5$, $k = 1{\cdot}0$, $\beta = 0{\cdot}1$ and $\rho = 0{\cdot}04$. In (a) the parasites enter a very short period of arrestment ($\Theta = 52$), in (b) arrestment is more prolonged ($\Theta = 3$).

Expressions may also be derived for equilibrium host density in the two models. Host population size at equilibrium is given by:

$$H^* = \frac{\gamma d_1 d_2}{\theta\beta(\lambda - d_1 d_2)} \qquad \text{eqn 6}$$

Where

$$d_1 = (\mu_p + b + \alpha) + \rho F(1 + \pi) + M_p^* k'(\alpha + \pi\rho F),$$

and

$$d_2 = \mu_A + b + \theta + \rho F(1 + \pi M_p^*) + \alpha M_p^*.$$

In the simplest case, with no larval arrestment then the grouse population decreases as additional mortality due to predation is added to the system (Fig. 7). In contrast, if the parasites enter a prolonged period of arrested development, then increased predation can lead to increases in the grouse population. This counter-intuitive result is due to the predators selectively removing a large proportion of the worm population before it has am impact on the host population. This reduces the net impact of the parasite on the grouse population and allows the grouse population to increase to a level determined by the combined effects of low predation and reduced parasitism. Further increases in the numbers of predators ultimately reduces the size of the grouse population and may drive the hosts below the threshold where they can sustain infection by the parasites. The levels of parasite-induced increases in predation observed in the grouse–*T. tenuis* system produce very little difference in the size of the host population when compared to the situation where predation is acting at random.

The transient dynamics of the system are also influenced by predation, most importantly the presence of predators reduces the tendency of the host and parasite populations to cycle (Figs 8 & 9). This occurs principally because predators reduce parasite burdens by removing parasitized hosts from the population, this reduces the delayed density-dependent effects of the parasite on host survival and breeding, and suspends the mechanism that generates the population cycles. This result is in accord with previous explorations of the properties of model of this type (May & Anderson 1978; Dobson & Hudson 1992). The propensity of helminth parasites and their host's populations to oscillate is a function of the ratio of parasite-induced reductions in fecundity to their effects on survival. As this ratio

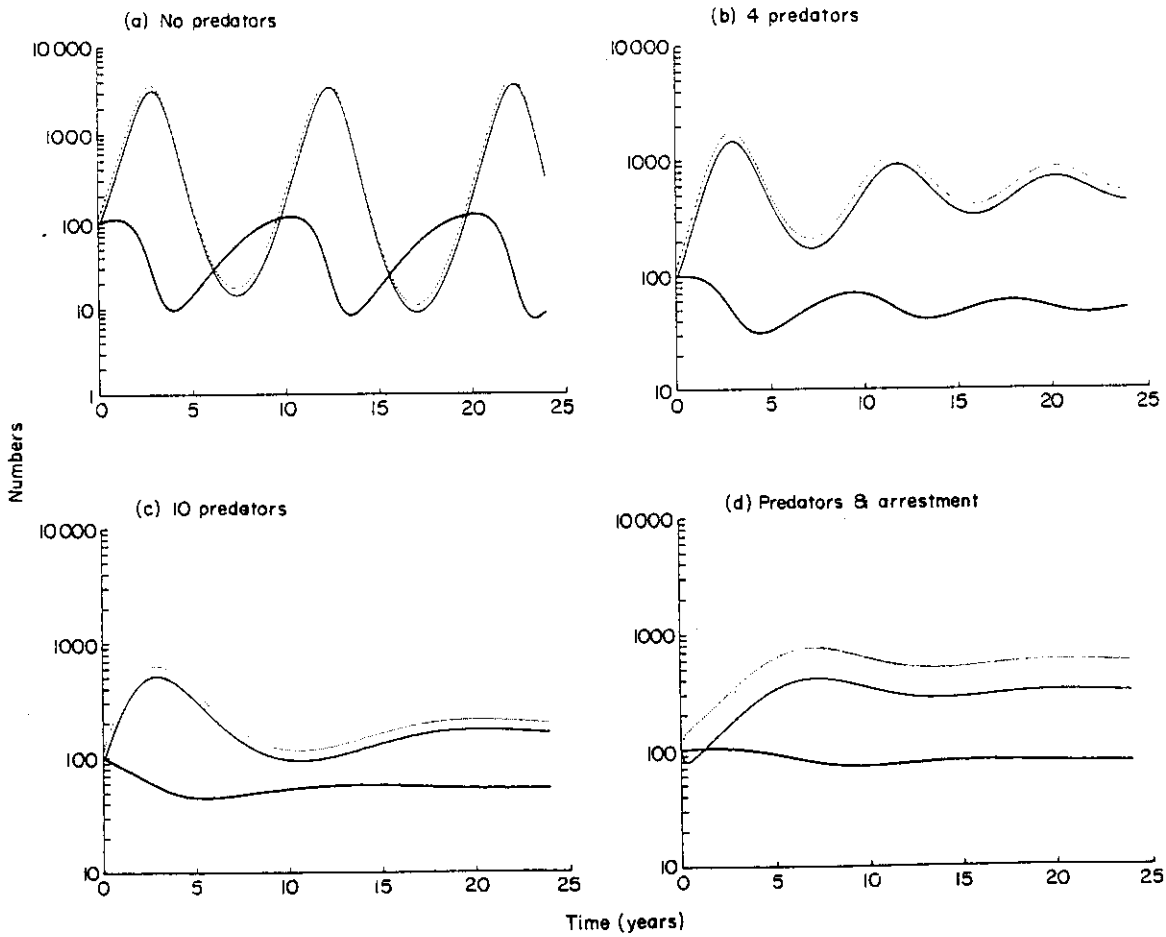

Fig. 8. Transient dynamics of the grouse–parasite systems for different levels of predation: (a) no predators, larval arrestment for 1 month; (b) 4 predators, larval arrestment for 1 month; (c) 10 predators, larval arrestment for 1 month; (d) 4 predators, larval arrestment for 4 months. In each figure the thick line shows grouse numbers, the thinner line gives mean burden of adult worms, and the broken line shows total worm burden (adults + arrested larvae). All parameters are as in Table 1. Predator density is numbers per 100 km^2.

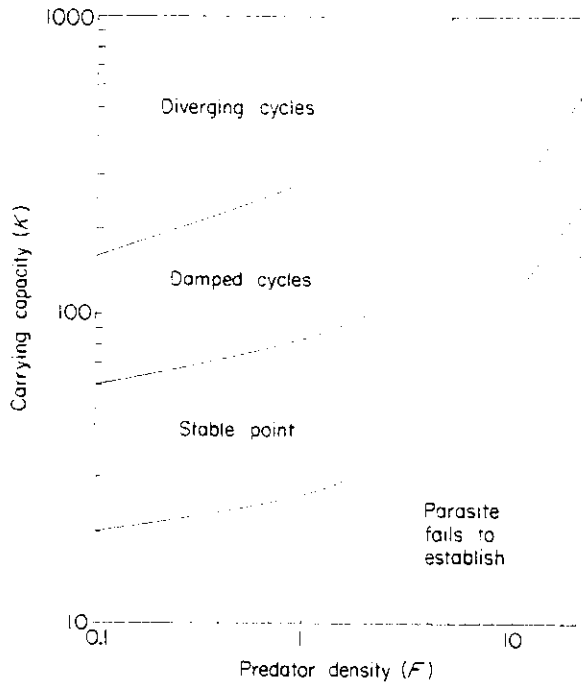

Fig. 9. Stability boundaries of the grouse/parasite/predator model for a range of predator and habitat quality values. The 'quality' of the habitat is modified by altering w, the parameter which determines equilibrium host density in the absence of predators and parasites. Carrying capacity is thus defined as $(a-b)/w$. All the models parameters are set to their mean values in Table 1. The different regions of the graph delineate areas where different patterns of long-term dynamic behaviour are exhibited by the host and parasite populations.

gets larger so the tendency to oscillate increases. The parasite-induced increases in susceptibility to predation reduces the propensity of the system to oscillate by increasing rates of parasite-induced mortality.

Discussion

The experiments and observations described in this paper suggest that predators tend to capture a disproportionately high number of grouse with high parasite burdens. This study did not determine whether these grouse were caught because of the infection or as a consequence of altered social behaviour as suggested by Jenkins *et al.* (1963, 1964) and Watson (1985). However, experimental work has shown that parasites are important in determining survival in this system (Hudson & Dobson 1990; Hudson, Newborn & Dobson 1992).

The experiment using trained dogs to locate the nests of grouse demonstrated that incubating female grouse with reduced parasite burdens were less likely to be found than control birds with natural infections. Although the physiological mechanism controlling the emission of scent is not understood, female grouse with high worm burdens were found to have relatively large caeca which may have emitted more scent during incubation. An alternative possibility is that the parasites compete for the female's reserves during incubation so the female leaves the nest more frequently and this results in more scent being left near the nest of heavily parasitized females. Either way the parasites make incubating females more vulnerable to predation. Since these females were all caught within the same area and treated irrespective of social status, this study would indicate that parasites can indirectly reduce survival of grouse. The clutch size of nests pointed were significantly smaller than those found by human observers, this was probably the result of parasite-induced reduction in the breeding production of the grouse, as demonstrated by Hudson (1986a) and Hudson *et al.* (1992).

The indirect impact of predators on the parasite population has important dynamic consequences for both the parasites and their hosts. As parasitic worms are not distributed randomly in the host population but exhibit an aggregated distribution, the removal of a few heavily infected individuals from the grouse population will have a disproportionately larger effect on the size of the parasite population and reduce the parasite's long-term impact on host numbers. The model demonstrates that this can reduce the regulatory role of the parasites and may increase the size of the host population. This is an interesting example of the counter-intuitive dynamics that simple ecological systems can exhibit.

The model also shows that parasite burdens, predation pressure and grouse density are ineluctably linked (Fig. 9). Changes in predation numbers will influence both parasites and grouse densities irrespective of whether the predators act selectively or not. In this respect the decrease in the proportion of birds with heavy infections on grouse estates with reduced numbers of gamekeepers (Fig. 4) could reflect either the loss of heavily infected individuals through selective predation or simply reduced grouse densities resulting in reduced transmission rates and lower worm burdens. More complex models which explicitly consider the dynamics of the predators give essentially the same results (Dobson & Hudson, unpublished).

The results from the experimental study differ from a previous study by Moss *et al.* (1990). These authors concluded that 'having more caecal threadworms did not make hens more likely to be killed by predators'. However, their study recorded high levels of predation on female grouse and relatively low worm burdens. The model described in this study suggests that high levels of predation would tend to produce reduced parasite burdens. At high levels of predation, the individuals with heavier parasite infections are removed rapidly from the population leading to a decline in observed mean burdens. This is illustrated in Figs 6 & 7 where both the size of the grouse population and the mean parasite burdens were effectively reduced by high

predation rates. A further limitation of the study by Moss *et al.* (1990) is that levels of worm infection were determined indirectly through counts of worm eggs in caecal faeces. In a related study, Hudson & Dobson (unpublished) found high variability in *T. tenuis* egg production in grouse. This effect was further confounded by a tendency for reduced *per capita* egg production at high worm burdens. This would make accurate estimates of worm intensity impossible without large numbers of caecal samples. In general, parasitological workers consider estimates of worm intensity based on these techniques to be strongly biased (Anderson & Schad 1985).

This study has gone some way to quantifying the interaction of parasitism and predation in a field situation; such studies naturally incorporate several interacting factors and present difficulties in interpretation of data. In this respect future research in this field should aim to perform factorial experiments that manipulate levels of parasitism and predation. The control of natural enemies is one of the fundamental tenets of game management (Leopold 1933). Although traditional game management has concentrated on predators, the studies discussed here suggest that parasites may also be important in determining the productivity of game populations (Hudson 1986a,b; Hudson, Newborn & Dobson 1992; Dobson & Hudson 1992). Incorporating these interactions into management schemes for game species will be an important technique for further determining their importance in other wildlife species.

Acknowledgments

We wish to thank The Earl Peel and subscribers to the North of England Grouse Research Project for allowing us to work on their estates and giving us access to their grouse guts. The study would not have been possible without the two dogs, Fennell and Quill. Ian Newton, Dick Potts, Steve Redpath, Nick Giles and two anonymous referees made constructive comments on the manuscript.

References

Anderson, R.M. (1979) The influence of parasitic infection on the dynamics of host population growth. *Population Dynamics* (eds R.M. Anderson, B.D. Turner & L.R. Taylor), pp. 245–281. Blackwell Scientific, Oxford.

Anderson, R.M. (1982) Parasite dispersion patterns; generative mechanisms and dynamic consequences. *Aspects of Parasitology* (ed. E. Meerovitch), pp. 1–40. Institute of Parasitology, McGill University.

Anderson, R.M. & Gordon, D.M. (1982) Processes influencing the distribution of parasite numbers within host populations with special emphasis on parasite-induced host mortalities. *Parasitology*, **85**, 373–398.

Anderson, R.M. & Schad, G.A. (1985) Hookworm burdens and faecal egg counts: an analysis of the biological basis of variation. *Transactions of Royal Society of Tropical Medicine & Hygiene*, **79**, 812–825.

Crompton, D.W.T., Keymer, A.E. & Arnold, S.E. (1984) Investigating over-dispersion: *Monoliformis* (Acanthocephala) and rats. *Parasitology*, **88**, 317–331.

Dobson, A.P. (1988) The population biology of parasite-induced changes in host behaviour. *Quarterly Review of Biology*, **63**, 139–165.

Dobson, A.P. & Hudson, P.J. (1992) Regulation and stability of a free-living host–parasite system, *Trichostrongylus tenuis* in red grouse. II. Population models. *Journal of Animal Ecology*, **61**, 487–500.

Giles, N. (1983) Behavioural effects of the parasite *Schistocephalus solidus* (Cestoda) on an intermediate host, the three-spined stickleback, *Gasterosterus aculeatus* L. *Animal Behaviour*, **31**, 1192–1194.

Holling, C.S. (1959) Some characteristics of simple types of predation and parasitism. *Canadian Entomologist*, **91**, 385–398.

Holmes, J.C. & Bethel, W.M. (1972) Modification of intermediate host behaviour by parasites. *Behavioural Aspects of Parasite Transmission* (eds E.U. Canning & C.A. Wright), pp. 123–149. Academic Press, London.

Hudson, P.J. (1986a) The effect of a parasitic nematode on the breeding production of red grouse. *Journal of Animal Ecology*, **55**, 85–92.

Hudson, P.J. (1986b) *Red Grouse: The biology and Management of a Wild Gamebird*. The Game Conservancy, Fordingbridge.

Hudson, P.J. (1992) *Red Grouse Population Studies*. The Game Conservancy, Fordingbridge.

Hudson, P.J. & Dobson, A.P. (1990) Red grouse population cycles and the population dynamics of the caecal nematode *Trichostrongylus tenuis*. *Red Grouse Population Processes* (eds A.N. Lance & J.H. Lawton), pp. 5–19. British Ecological Society and Royal Society for the Protection of Birds.

Hudson, P.J. & Newborn, D. (1990) Brood defence in a precocial species: variations in the distraction displays of red grouse, *Lagopus lagopus scoticus*. *Animal Behaviour*, **40**, 254–261.

Hudson, P.J. & Watson, A. (1985) The red grouse. *Biologist*, **32**, 13–18.

Hudson, P.J., Newborn, D. & Dobson, A.P. (1992) Regulation and stability of a free-living host–parasite system, *Trichostrongylus tenuis* in red grouse. I. Monitoring and parasite reduction experiments. *Journal of Animal Ecology*, **61**, 477–486.

Jenkins, D., Watson, A. & Miller, G.R. (1963) Population studies of red grouse, *Lagopus lagopus scoticus* Lath. in north east Scotland. *Journal of Animal Ecology*, **32**, 317–376.

Jenkins, D., Watson A. & Miller, G.R. (1964) Predation and red grouse populations. *Journal of Applied Ecology*, **1**, 183–195.

Krebs, J.R. (1978) Optimal foraging: decision rules for predators. *Behavioural Ecology: An Evolutionary Approach*, 1st edn (eds J.R. Krebs & N.B. Davies), pp. 23–63. Blackwell Scientific Publications, Oxford.

Leopold, A. (1933) *Game Management*. Scribners, New York.

May, R.M. & Anderson, R.M. (1978) Regulation and stability of host–parasite interactions. II. Destabilizing processes. *Journal of Animal Ecology*, **47**, 249–268.

Mech, L.D. (1970) *The Wolf*. Natural History Press, New York.

Moore, J. (1984) Parasites that change the behaviour of their host. *Scientific American*, **250**, 208–115.

Moss, R., Trenholm, I.B., Watson, A. & Parr, R. (1990) Parasitism, predation and survival of hen red grouse *Lagopus lagopus scoticus* in spring. *Journal of Animal Ecology*, **59**, 631–642.

St. John, C. (1878) *Wild Sports and Natural History of the*

Highlands. John Murray, London.
Schaller, G.B. (1972) *The Serengeti Lion*. University of Chicago Press, Chicago.
Temple, S.A. (1987) Do predators always capture substandard individuals disproportionately from prey populations? *Ecology*, **68**, 669–674.
Van Dobben, W.H. (1952) The food of the cormorants in the Netherlands. *Ardea*, **40**, 1–63.
Vaughan, G.E. & Coble, P.W. (1975) Sublethal effects of three ectoparasites on fish. *Journal of Fisheries Biology*, **7**, 283–294.
Watson, A. (1985) Social class, socially induced loss, recruitment and breeding of red grouse. *Oecologia*, **67**, 493–498.
Watson, A. & Miller, G.R. (1976) *Grouse Management*. The Game Conservancy, Fordingbridge.
Watson, H., Lee, D.L. & Hudson, P.J. (1987) The effect of *Trichostrongylus tenuis* on the caecal mucosa of young, old and anthelmintic treated wild red grouse, *Lagopus lagopus scoticus*. *Parasitology*, **94**, 405–411.
Wilson, G.R. (1983) The prevalence of caecal threadworms (*Trichostrongylus tenuis*) in red grouse (*Lagopus lagopus scoticus*). *Oecologia*, **58**, 265–268.

Received 23 August 1991; revision received 6 February 1992