

Trophic interactions and population growth rates: describing patterns and identifying mechanisms

Peter J. Hudson^{1*}, Andy P. Dobson², Isabella M. Cattadori¹, David Newborn³, Dan T. Haydon⁴, Darren J. Shaw⁵, Tim G. Benton¹ and Bryan T. Grenfell⁶

¹Department of Biological Sciences, University of Stirling, Stirling FK9 4LA, UK

²Department of Ecology, Evolutionary Biology, Princeton University, Princeton, NJ 08544-1003, USA

³Game Conservancy Trust, Swale Farm, Satron, Gummerdale, Richmond, North Yorkshire DL11 6JW, UK

⁴Department of Zoology, University of Guelph, Guelph, Ontario, Canada N1G 2W1

⁵Centre for Tropical Veterinary Medicine, University of Edinburgh, Easter Bush, Roslin EH25 9RG, UK

⁶Department of Zoology, University of Cambridge, Downing Street, Cambridge CB3 4EJ, UK

While the concept of population growth rate has been of central importance in the development of the theory of population dynamics, few empirical studies consider the intrinsic growth rate in detail, let alone how it may vary within and between populations of the same species. In an attempt to link theory with data we take two approaches. First, we address the question ‘what growth rate patterns does theory predict we should see in time-series?’ The models make a number of predictions, which in general are supported by a comparative study between time-series of harvesting data from 352 red grouse populations. Variations in growth rate between grouse populations were associated with factors that reflected the quality and availability of the main food plant of the grouse. However, while these results support predictions from theory, they provide no clear insight into the mechanisms influencing reductions in population growth rate and regulation. In the second part of the paper, we consider the results of experiments, first at the individual level and then at the population level, to identify the important mechanisms influencing changes in individual productivity and population growth rate. The parasitic nematode *Trichostrongylus tenuis* is found to have an important influence on productivity, and when incorporated into models with their patterns of distribution between individuals has a destabilizing effect and generates negative growth rates. The hypothesis that negative growth rates at the population level were caused by parasites was demonstrated by a replicated population level experiment. With a sound and tested model framework we then explore the interaction with other natural enemies and show that in general they tend to stabilize variations in growth rate. Interestingly, the models show selective predators that remove heavily infected individuals can release the grouse from parasite-induced regulation and allow equilibrium populations to rise. By contrast, a tick-borne virus that killed chicks simply leads to a reduction in the equilibrium. When humans take grouse they do not appear to stabilize populations and this may be because many of the infective stages are available for infection before harvesting commences. In our opinion, an understanding of growth rates and population dynamics is best achieved through a mechanistic approach that includes a sound experimental approach with the development of models. Models can be tested further to explore how the community of predators and others interact with their prey.

Keywords: trophic interactions; red grouse; *Trichostrongylus tenuis*; population cycles

1. INTRODUCTION

A long-term objective of population biology is to explain the spatio-temporal variations in abundance of organisms by understanding the factors that limit both distribution and changes in abundance. In general, theory predicts that a major determinant of distribution and dynamics is the instantaneous population growth rate, presented as r (where $r = \ln(\lambda) = \ln(N_{t+1}/N_t)$). Some models reveal the obvious, such that species will tend not to exist where their

population growth is consistently negative and there is no immigration (see Sibly & Hone 2002). But the models also expose intriguing dynamics; for example, simple single-species nonlinear models reveal that dynamics can vary from stability through oscillatory to chaotic behaviour simply by subtle changes in the population growth rate (May 1976). As such, it is not surprising that when we incorporate interspecific interactions, the stochastic vagaries of environmental conditions and dispersal, we reveal a Pandora’s box of dynamical behaviours.

In the empirical literature, the population growth rate parameter does not enjoy the same importance as it does in the theoretical literature. Rarely do workers make an estimate of the intrinsic growth rate parameter (r) or its empirical equivalent, the maximum growth rate (r_{\max})

* Author for correspondence (p.j.hudson@stir.ac.uk).

One contribution of 15 to a Discussion Meeting Issue ‘Population growth rate: determining factors and role in population regulation’.

which is simply the maximum rate of growth observed within a time-series. Changes in the observed growth rate at a specific time (r_t) may be recorded along with the factors associated with the reproductive output of individuals, but studies tend not to estimate the extent to which the growth rate is reduced by density dependent regulatory factors. This is essentially because estimating the population growth rate may not be simple. Time-series data may not reveal the maximum growth rate simply because the population is close to equilibrium and therefore there is little variation in growth rate. Moreover, we can expect growth rate to fall with density, so by definition we can only obtain an estimate of r when the density is very low, although in some populations the growth rate may be subjected to Allee effects so that it is low or even negative. The growth rate will also be influenced by the age structuring when fecundity or survival varies with age. This population age structure will influence any estimate of growth rate, and a low density population is usually one that has suffered a massive decline in numbers; therefore the age structure is far from stable (Caswell 2001). Other means of estimating r exist, all of which incorporate certain errors. Nevertheless, the point is that if we are to challenge models with data then estimates of growth rates will be needed.

One of the first steps to bridge the gap between theory and empiricism is to examine if some basic predictions from models are supported by data. One approach is to make interspecific comparisons and predict from theory the expected differences in the dynamics between species in relation to variations in population growth rate. Such an approach is taken by Sæther & Engen (2002). An alternative approach is to undertake intraspecific comparisons and make predictions from models of how variations in growth rate will influence dynamics and equilibrium population size between populations of the same species. This is the approach we take in this paper with red grouse (*Lagopus lagopus scoticus*).

The red grouse is particularly suited to this type of study. First, the species is herbivorous with a diet that consists predominantly of the tips of heather (*Calluna vulgaris*), such that its habitat and distribution are limited by the extent of this shrub (Hudson 1992). The productivity of heather is determined by warm and wet conditions, and the quality of the heather is influenced by the underlying rock (Watson *et al.* 1984). Second, we have gathered hunting bag records from 352 individually managed upland estates where the numbers harvested have been carefully recorded for many years, frequently more than 100 years. These are distributed throughout Scotland, northern England and Wales and exhibit large variations in environmental and biotic conditions, such as the presence or absence of predators and tick-borne diseases (Hudson 1992). Third, preliminary evidence indicated that parasites might play an important role, so we have undertaken a series of replicated field experiments manipulating trophic interactions with parasites from the scale of the individual through to the population and community. We believe the combined approach of extensive estimates of population change coupled with experimental manipulations and modelling has allowed us to obtain a unique insight into how natural enemies influence the population growth rate and dynamics of red grouse. We

focus our attention on the natural enemies, partly because we can obtain comparative data between populations and partly because the role of them in the natural regulation of red grouse has been contested (Moss & Watson 2000). We also believe that the majority of studies that have focused on growth rate have tended to apply single-species models, particularly when the population is age structured, so we wished to study trophic interactions and examine how different natural enemies can influence population growth rate.

In this paper, we take two contrasting approaches. The first is an examination of time-series data looking at the patterns theory predicts. We start by addressing the question 'what patterns in growth rate does theory predict we should see in time-series data?' We use models based on the Lotka–Volterra model that have been specifically adapted to understanding the interaction between parasites and their host, in particular the Anderson & May (1978) model and its derivative that has been tailored to the red grouse–*Trichostrongylus tenuis* system (Dobson & Hudson 1992). We use these models partly because we have good evidence to suppose that productivity of red grouse is determined by this macroparasite (Hudson 1986a; Hudson *et al.* 1992b) and partly because the tightly linked relationship between parasites and their hosts is one that lends itself to careful experimentation. However, this approach can only identify some ecological correlates that are associated with variations in maximum growth rates and patterns of red grouse population dynamics. Hence, in the second half of the paper we take an experimental approach and examine variations in the risk of infection between individuals and the consequence of this for population growth rate, population regulation and dynamics. Such an understanding then provides a foundation for us to explore community level questions about how other natural enemies such as predators, viruses and humans can interact to influence population dynamics and observed growth rates.

2. WHAT GROWTH RATE PATTERNS DOES THEORY PREDICT?

The fundamental model of trophic interactions is the Lotka–Volterra model with density dependence in the growth rate of the prey population (Hastings 1996). The model predicts stability in both prey and predator abundance, although the equilibrium is approached through damped oscillations and the predator population may fluctuate to very low levels before the equilibrium is reached. Increasing the growth rate of the prey population has no influence on the size of the equilibrium prey population and all populations stabilize to the equilibrium. This increase in the intrinsic growth rate of the prey population (r) simply feeds through to an increase in the size of the equilibrium predator population in what is sometimes referred to as the paradox of enrichment (Rosenzweig 1971). In other words, fundamental theory of trophic interactions predicts that changes in growth rate of prey populations will have no influence on prey equilibrium population size or dynamics, but may influence the predator equilibrium.

However, this pattern has no influence on the population growth rate on dynamics or equilibrium population

size breaks down once we start to consider nonlinearities in the density dependence, time-lags and specific heterogeneities associated with particular types of natural enemies (e.g. Crawley 1983). If we consider a specific derivative of the Lotka–Volterra model that examines the relationship between macroparasite and host, we see distinct and different patterns emerge. We use here a derivative of the Anderson & May (1978) model of parasite–host relationships developed by Dobson & Hudson (1992) that was specifically designed to describe the dynamics of the red grouse–*T. tenuis* system. The nematode is a directly transmitted macroparasite that is known to induce increased mortality and a reduction in the fecundity of grouse (Hudson *et al.* 1992b). The model examines changes in host, parasite and the population of free-living parasitic stages, and the consequences of changes in host growth rate on host and parasite dynamics. In this model, an increase in the host (or prey) intrinsic growth rate (r) does not lead to the same equilibrium but to some distinct changes in host dynamics, and in the predicted year to year growth rates $-r_t$ (figure 1a).

Several predictions emerge from this model with respect to the growth rate of the host population. When the growth rate in the model is low, then the host population comes to equilibrium with no variation in abundance and consequently no observed variation in growth rate. As growth rate is increased, so instability in the host population increases, variance in observed host growth rate (r_{var}) increases and the maximum growth rate observed (r_{max}) increases to asymptote at the point where $r_{\text{max}} = r$ such that we can assume r_{max} provides a reasonable estimate of the intrinsic growth rate. In real populations this may not be so simple because the host population may have to fall close to zero and below an extinction threshold before r_{max} approaches r . As the intrinsic growth rate increases, so the amplitude increases, leading to severe population crashes such that the minimum growth rate (r_{min}) decreases with r_{max} ; it does so in a nonlinear manner (figure 1a). However, this figure should be seen simply as a section through the broader parasite–host parameter space that corresponds specifically to the grouse–*T. tenuis* system. Variations in the final dynamics will depend on the biological features of the particular system and in particular the tensions between the growth rates of the host and the parasite (figure 2). Nevertheless, the dynamics with high growth rates are essentially oscillatory with cycle periods becoming shorter and amplitudes larger as host growth rate increases.

These predictions are made using the Dobson & Hudson (1992) macroparasite model as applied to the grouse–*T. tenuis* system, but it is gratifying to see that such general predictions hold true from a number of other fundamental models. For example, the single-species, discrete logistic model (Case 1999) provides a similar pattern of observed growth rate (r_t) against (r) (figure 1b). Essentially, general theory provides us with four predictions that should be seen when comparing time-series between species of animals, namely an increase in observed maximum growth rate r_{max} leads to the following:

- (i) an increase in the variance of the observed growth rates;

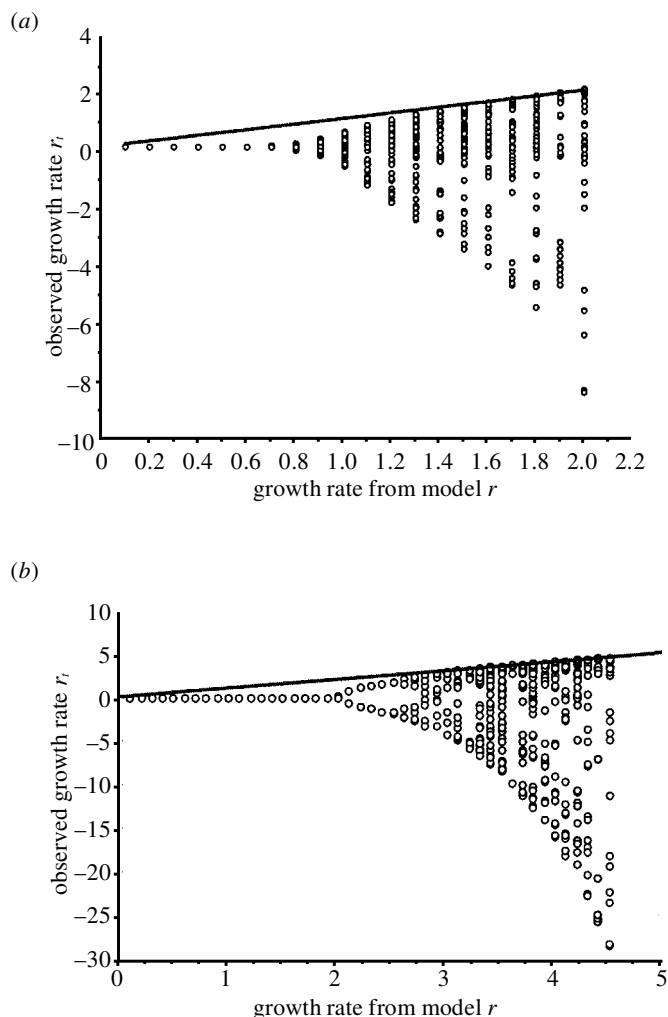


Figure 1. Model outputs predicting the relationship between observed growth rates per annum (r_t) and the intrinsic growth rates (r) included in the model: (a) from Dobson & Hudson (1992) host–macroparasite model showing that an increase in r leads to an increase in the variance of the observed growth rate, with r_{max} rising asymptotically to r and r_{min} decreasing nonlinearly; and (b) a similar result to (a) generated from the lagged logistic model.

- (ii) a nonlinear decrease in the minimum growth rate r_{min} ;
- (iii) shorter cycle periods;
- (iv) cycles with greater amplitude.

Such predictions are not independent, as an increase in the variance of the growth rate is going to be associated with an increase in the minimum growth rate and cycles of lower amplitude.

3. GROWTH RATE PATTERNS IN GROUSE TIME-SERIES

Red grouse inhabit the heather dominant moorland of the United Kingdom, much of which is managed by individually owned private estates to provide a surplus of birds to harvest each autumn (Hudson 1992). The majority of these estates maintain careful hunting records on the precise number harvested each year, providing a spatio-temporal dataset reflecting changes in abundance throughout

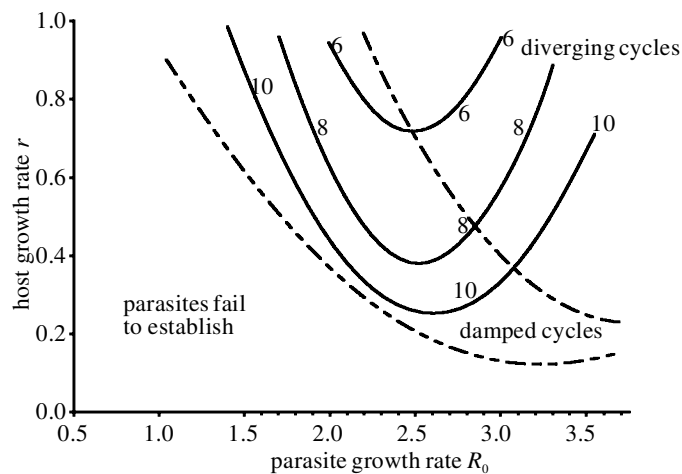


Figure 2. Dynamics of the Dobson & Hudson (1992) host-macroparasite model depend on the tension between the growth rate of the host population and the growth rate of the parasite population. Dashed lines indicate bifurcations from stable dynamics to damped oscillations and from damped to diverging oscillations, increasing instability resulting from an increase in the growth rate of the parasite. Solid lines indicate changes in cycle period of oscillations that decrease as host growth rate increases.

the range of the subspecies. However, the value of any time-series of hunting records depends on the extent to which effort changes with density and how this may vary over time. The system of hunting on the majority of estates is based on driven grouse shooting where the effort per day varies little between days in that the number of hunters and the area harvested per day remains relatively constant, but the number of days, harvesting increases with the overall abundance of grouse. In addition, there is a minimum density below which harvesting is cancelled in order to preserve breeding stock (Hudson 1985, 1992; Hudson & Newborn 1995; Hudson & Dobson 2001; Hudson *et al.* 2002). This minimum tends to lead to an underestimate of abundance when numbers are low and consequently an overestimate of growth rate following recovery (Hudson *et al.* 2002). This minimum is reflected in the general relationship between the numbers harvested per square kilometre and numbers counted on 1 km² sample areas, but there is also a change in the variance of the bags with increasing counts (Hudson *et al.* 1999, 2002).

Interestingly, comparisons of counts within areas show a decrease in spatial variance with mean grouse density, supporting the observation that when abundance is low, grouse tend to be aggregated in small areas, and as density increases so grouse tend towards a more uniform spatial distribution (Hudson *et al.* 1998, 2002). Consequently, harvesting records probably provide a better reflection in abundance of what is happening at the population level than small scale counts. This is supported by a more recent rigorous analysis that examined the power law relationship between mean abundance and variance in both harvest and count data and shows that harvesting records provide a good reflection of the underlying population dynamics, and as such, a reasonable representation of the actual dynamics (I. M. Cattadori, D. Haydon, S. J. Thirgood and P. J. Hudson, unpublished data). Even so, such records will incorporate biases: after all they are col-

lected following breeding, so will tend to reflect changes in breeding production rather than simple changes in breeding density.

The time-series of hunting records were used to estimate growth rates. We obtained 352 harvesting time-series from independently managed estates throughout the United Kingdom. Zeros were not used and annual growth rate (r_t) was estimated as $\ln(N_{t+1}/N_t)$. From each time-series, three growth rate parameters were determined: the maximum growth rate observed (r_{\max}), the minimum growth rate observed (r_{\min}) and the variance in the growth rate (r_{var}).

(a) *What environmental factors are associated with maximum growth rate between populations?*

Maximum growth varied greatly between populations and 92% of the cases had maximum growth rates between 1 and 4. Variations in growth rate were examined in relation to a series of habitat, environmental conditions and biotic factors. Previous studies on ungulates have found that maximum growth rates in herbivores are often associated with food quality (Sinclair & Krebs 2002), and because grouse feed predominantly on *Calluna vulgaris* we examined growth rate in relation to the average climatic factors that would influence heather productivity, length of growing season and heather quality. Heather productivity, measured as the annual production of shoots (H) was estimated according to the relationship derived by Miller (1979) where heather productivity is a function of mean daily temperature between April and August (T in °C) and precipitation during the same period (P in mm):

$$H = 29.3T - 0.168P - 56.0.$$

A similar relationship was used by Albon & Clutton-Brock (1988) in a study of red deer in Scotland, and they proposed that this would reflect the quality of heather feed available to moorland herbivores in general. The growing season was estimated from the relationship between air temperature in degrees centigrade and corrected for altitude in metres as proposed by Smith (in Albon & Clutton-Brock 1988). Growing season will be correlated with heather productivity but was included as a separate variable from productivity. As with previous studies (Picozzi 1966), soil fertility and its presumed influence on heather quality were estimated from an index of base richness of underlying rocks using solid geology maps. Rock type was ranked from low (1) to high (5) base richness and an overall index estimated according to the area of each under each estate (see Hudson 1992). Other environmental conditions included altitude, average number days of snow cover and number of wet days. Predation pressure was estimated indirectly from the density of keepers (Hudson 1986b, 1992). The presence of ticks (*Ixodes ricinus*) and the tick-borne disease louping-ill were determined through a postal survey (Hudson 1992) and were also included as categorical variables. Data were standardized and where necessary \log_{10} transformed.

A general linear model with maximum growth rate as the dependent variable and the independent variables base richness ($F_{1,144} = 6.00$, $p = 0.01$), ticks ($F_{1,142} = 7.98$, $p = 0.005$) and their interaction with heather productivity ($F_{1,138} = 5.85$, $p = 0.02$) explained 54% of the variance in

growth rate. Not surprisingly for a herbivore, the maximum growth rate was associated with quality of the food plants available. The general finding that growth rate between populations was associated with variations in food quality is supported by a field experiment undertaken in northeast Scotland by Watson *et al.* (1984). They fertilized plots of heather moorland and counted the number of grouse and recorded their productivity over a period of years. They found that the grouse populations on the fertilized plots produced more young and that numbers increased faster to a higher density than on control plots, but this treatment did not prevent or reduce the extent of a cyclic decline in abundance. In this respect, food conditions seemed important for determining the maximum production rate and so maximum growth rate, but was not associated with the negative growth rates and the cause of the cycles. Returning to our general linear model, the only natural enemy that entered the model and reduced growth rate was the presence of the sheep tick that carries the viral infection louping-ill that causes significant mortality in grouse (Reid *et al.* 1978; Hudson *et al.* 1995). Again, this may reflect habitat conditions as ticks require wet damp conditions for survival and are frequently associated with poor quality vegetation with a thick mat layer dominated by bracken or other non-palatable vegetation (Hudson 1986c).

(b) Do growth rate patterns reflect predictions from theory?

In § 2, we made four predictions about the general patterns of growth rate in relation to variations between populations. In comparisons between populations of red grouse these predictions were, in general, supported. First an increase in the variance in growth rate was observed with the maximum growth rate (figure 3a; $F_{1,298} = 451.84$, $p < 0.001$, $r^2 = 0.603$), implying that populations became more unstable as population growth rate increases. On the one hand, such a relationship is not too surprising in that we may expect that any increase in the maximum growth rate would lead to an increase in the variance, but on the other hand, if the populations were tightly regulated we may expect to see no increase in variance.

Second, minimum growth rate declined with maximum growth rate in a linear fashion (figure 3b; $F_{1,298} = 1109.25$, $p < 0.001$, $r^2 = 0.432$), whereas a nonlinear relationship was predicted from the models. One explanation is that year to year variation in the transmission parameter may change the shape of this relationship. We incorporated 10% stochastic variation in each of the parameters and found that in every instance the stochasticity reduced the size of the minimum growth rate (r_{\min}), increased the observed maximum growth rate (r_{\max}) and linearized the relationship with the intrinsic growth rate.

To examine the prediction that shorter cycle periods would be found in populations with large maximum growth rates, the tendency for each time-series to cycle and the cycle period were estimated using spectral analysis, full details of which are available in Haydon *et al.* (2002). Cycle period was only estimated for those time-series where there was a distinct and significant peak in the spectrogram. Overall, 57% of populations were cyclic. The tendency to cycle varied between regions such that populations in northeast Scotland were less likely to cycle

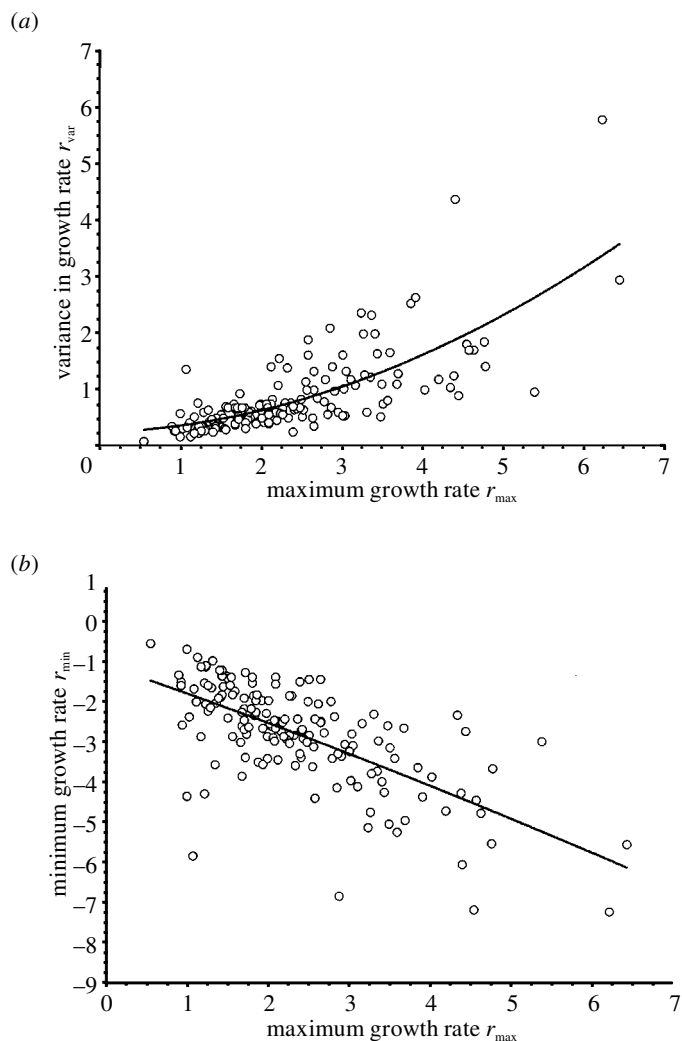


Figure 3. Variations in observed growth rate patterns from red grouse hunting records: (a) the relationship between the observed increase in the variance of annual growth rates with respect to maximum growth rate; and (b) the relationship between the annual observed decline in minimum growth rate and the observed maximum growth rate.

than expected. Among the cyclic populations, cycle periods varied greatly between grouse populations from 3 to 14 years. Overall there was an increase in cycle period with latitude, although the relationship was weak (Haydon *et al.* 2002; $F_{1,163} = 11.962$, $p < 0.001$, $r^2 = 0.092$) with the northern regions of Scotland having significantly longer cycles than populations in northern England (figure 4). As predicted by theory, within the cyclic populations there was a significant decrease in cycle period with maximum growth rate ($F_{1,111} = 12.85$, $r^2 = 0.104$, $p < 0.001$), although the percentage of variance explained was low (figure 5b). Examination of the figure tends to reveal that populations with a relatively high maximum growth rate tend to exhibit short cycle periods, whereas populations with a low maximum growth rate tend to show great variation in the cycle period observed. This may be because populations with low growth rates also have low amplitude and the signal for cycle period becomes difficult to discern (figure 5b).

We estimated the amplitude of each oscillation as the sum of positive growth rates and then expressed the mean

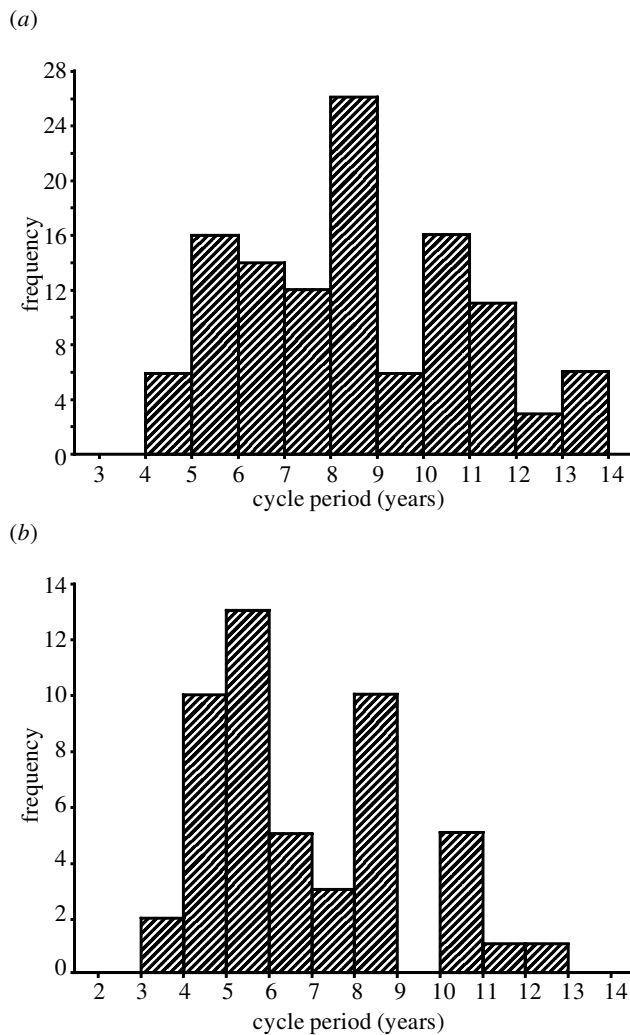


Figure 4. Frequency distributions of cycle periods determined from time-series of grouse harvesting data from (a) Scotland ($n = 119$) and (b) England ($n = 50$). Overall, English grouse populations have shorter cycle periods (mean \pm s.e. = 6.73 ± 0.32) than Scottish populations (mean \pm s.e. = 8.84 ± 0.36).

amplitude for a series as the mean of these positive growth rates. As predicted from the general theory, amplitude increased with maximum growth rate ($F_{1,346} = 227.98$, $p < 0.001$, $r^2 = 0.397$; figure 5a).

When the intrinsic growth rates are high, populations of grouse tend to be unstable. These unstable populations, with their highly variable annual growth rates allow us to examine the mechanisms that influence these growth rates. Not surprisingly, for a herbivorous bird like the red grouse, our comparison of time-series data between populations indicated that part of the variation in the maximum growth rate could be accounted for by the food quality and availability (see § 3a). But such relationships are simply correlations and we have not started to identify the specific processes that lead to negative growth rates. Consequently, we must start to address questions such as: what mechanisms reduce the maximum growth rate?

4. INDIVIDUAL LEVEL PROCESSES IN INFLUENCING PRODUCTION

Population growth rate is in many respects a term that simply integrates the survival and productivity of all indi-

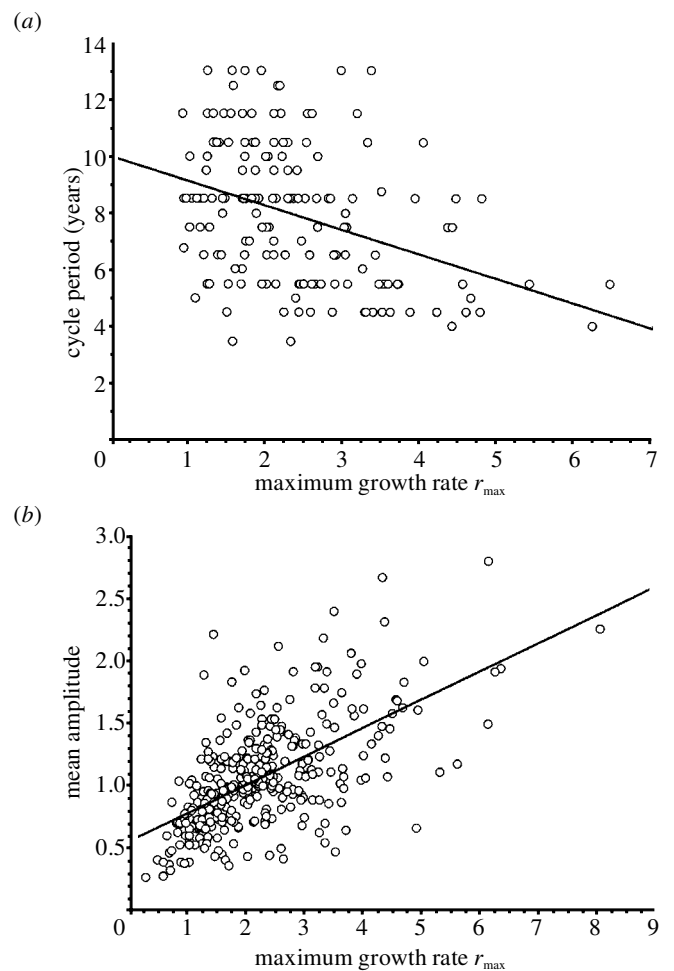


Figure 5. Relationships between grouse cycle period and amplitude with maximum growth rate, England and Scotland; (a) cycle periods of grouse time-series decreased with maximum growth rate although the variance explained was small, and (b) amplitude increased with maximum observed growth rate.

viduals within the population over a period of time. To understand how population growth is reduced, we need to know first how vital rates are influenced by specific factors, and also how the risk of exposure to these factors varies within the population. In this section, we start by describing experiments that examined the impact of parasites on individual productivity and then the consequences of this in the Dobson & Hudson (1992) macroparasite model. Much of this section is based on previously published experiments, so will be presented as a review illustrating how detailed experimental studies at the individual level, integrated with modelling, can provide an insight into the mechanisms influencing growth rates.

(a) *Negative growth rates and productivity*

Early studies on grouse reported that the negative growth rates of grouse were often associated with heavy infections of the caecal nematode *T. tenuis* (Cobbold 1873; Lovat 1911). More recent quantitative, longitudinal studies support this observation and show that within study areas, years with negative growth rate were associated with heavy infections of *T. tenuis* (figure 6a). A more careful inspection of the data demonstrates that both over-

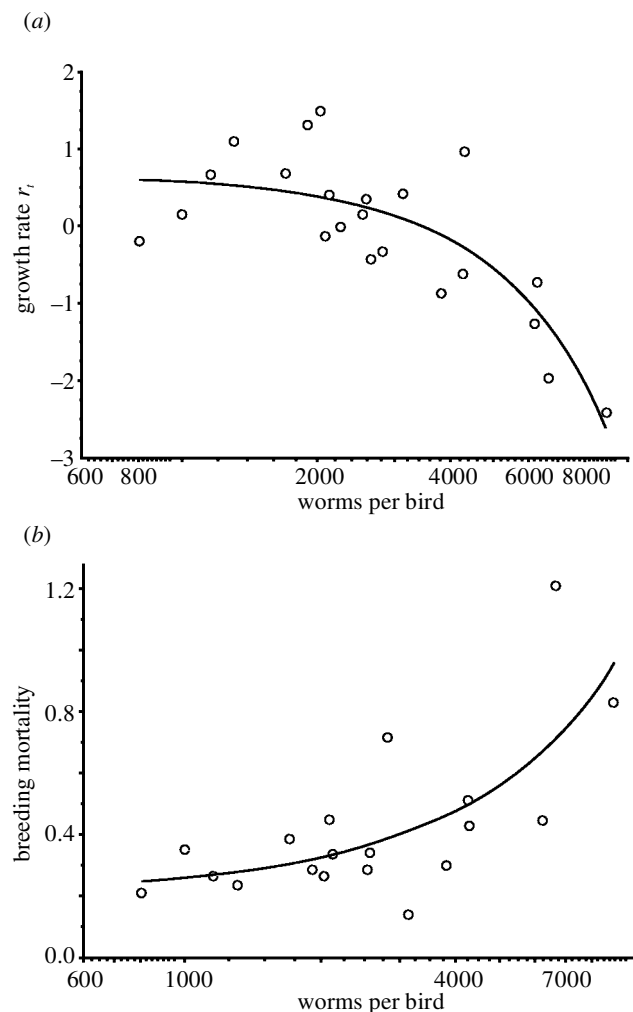


Figure 6. Changes in the population growth rate (a) and breeding mortality (b) of red grouse in relation to mean intensity of infection with the parasitic worm *Trichostrongylus tenuis*. Points represent results from different years during a longitudinal study of population dynamics on a Gunnedside estate, North Yorkshire, England (Hudson *et al.* 1992b).

winter loss (mortality plus net dispersal) and breeding mortality (\log_{10} (maximum clutch size – brood size at 6 weeks of age)) were also associated with the heavy burdens of *T. tenuis* (figure 6b), indicating that parasites may have an important role in the year to year changes in population growth rate (Hudson *et al.* 1992b, 2002). Interestingly, earlier studies (Jenkins *et al.* 1963) dismissed the relative importance of *T. tenuis* in causing negative population growth rates because they focused solely on the possible role of parasites in influencing adult mortality and did not consider the indirect influence parasites may have in increasing grouse breeding mortality.

To determine if parasites reduced host reproduction, we undertook replicated field experiments where we experimentally reduced the natural infections of grouse by treating some birds with an anthelmintic and comparing their reproductive performance with an untreated, placebo control group (Hudson 1986a; Hudson *et al.* 1992b). Both within and between year treatments demonstrated that parasites consistently reduced the reproductive output of the grouse. Parasites also reduced host survival but this

was less severe relative to the impacts on host fecundity (Hudson *et al.* 1992b).

(b) Patterns of infection between individuals

Studies of age-structured populations have illustrated the importance of considering variations in vital rates between age cohorts (Caswell 2001). One advantage of such an approach is that age structured models can then provide predictions of growth rates and future changes in growth rates according to changes in age structure (Caswell 2001). One limitation with such an approach, however, is that this may focus workers solely on age as the predictor of vital rates and not on the biological mechanisms that may influence variations in these vital rates between individuals, and hence their importance at the population level.

An important step in examining the influence of individual vital rates on population growth rate is to consider the frequency distribution of risk between individuals within the population. In terms of natural enemies, that is the risk of becoming infected with a virus, the risk of carrying a heavy parasite burden or the risk of being taken by a predator, and the consequences of these to survival and breeding production. In many species such risks are not evenly distributed through the population: there is frequently a small proportion of the population more susceptible to infection, predation or the impact of some other natural enemy. This is simply illustrated by comparing the frequency distribution of broods in different years where females have heavy and light infections of *T. tenuis* on one study area (figure 7a), or chicks have been exposed to heavy or light infections of the louping-ill virus on another study area (figure 7b). In both instances, mean brood size varied between the sampled years but there are still some individuals that do poorly in good years and some that do well in bad years. To understand how these natural enemies influence population growth rate we must first identify through field experiments that the natural enemies are indeed an important cause of these variations in productivity, and secondly, incorporate this frequency distribution of risk into the models. For both of these natural enemies we have undertaken field experiments that have demonstrated that these differences in breeding production are indeed caused by infection of *T. tenuis* (Hudson 1986a; Hudson *et al.* 1992b) or louping-ill (Laurenson *et al.* 1998).

Most macroparasites exhibit an aggregated pattern of distribution within their host population, with the majority of parasites aggregated in the minority of hosts (Anderson & May 1991; Shaw & Dobson 1995; Wilson *et al.* 2001). In the case of *T. tenuis* in red grouse, the degree of aggregation is relatively low and approaches a random Poisson distribution (Hudson *et al.* 1992b). By contrast, the distribution of the nymph ticks that transmit the louping-ill virus to red grouse chicks is highly aggregated between chicks and broods (Hudson 1992) and, as with many macroparasites, both frequency distributions are best described with the negative binomial distribution (Shaw *et al.* 1998). One consequence of this aggregation is that only a relatively small proportion of the grouse population will be exposed to the virus.

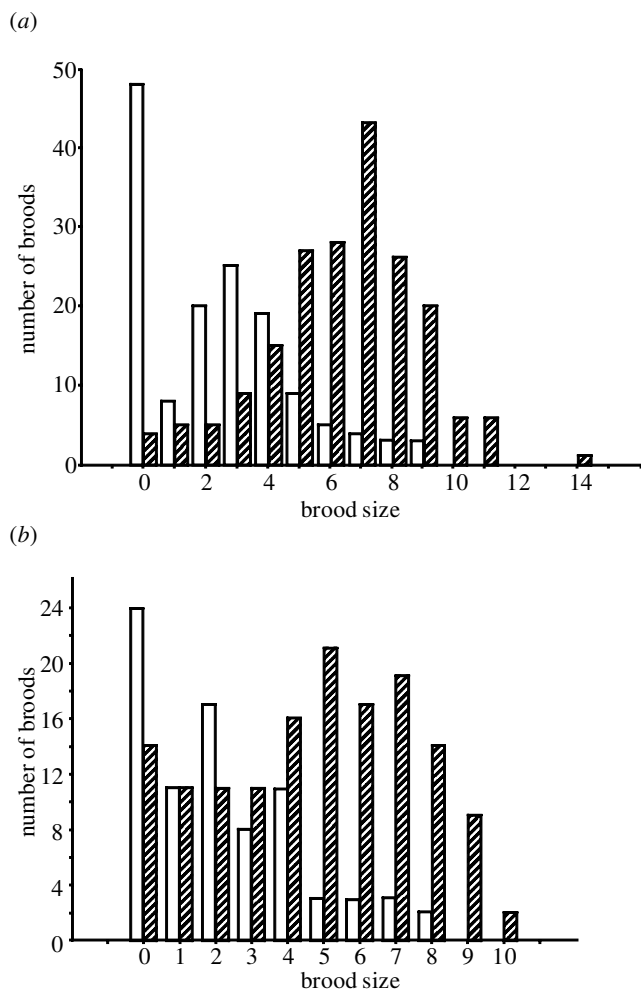


Figure 7. Frequency distribution of red grouse broods in years of high and low infection: (a) years of high (open bars) and low (hatched bars) infection of hen grouse with the nematode *Trichostrongylus tenuis* from Gunnerside, North Yorkshire, England; and (b) years of high (open bars) and low (hatched bars) seroprevalence in young grouse with the tick-borne louping-ill virus from Lochindorb, Morayshire, Scotland.

(c) Modelling the impact of parasites on grouse

The fundamental model of macroparasite–host dynamics that incorporates the essential features of parasite induced reduction in fecundity and the pattern of parasite distribution between hosts is that of Anderson & May (1978). This was extended by Dobson & Hudson (1992) to include the dynamics of the free-living stages and arrested development, and in so doing provided a suitable model of the grouse–*T. tenuis* system. Both studies identified that instability and variations in population growth rate will occur when the parasite-induced reduction in fecundity (δ) is large compared with the parasite-induced reduction in survival (α) with respect to the degree of aggregation as measured by the aggregation parameter k from the negative binomial distribution. More formally, this will occur when the risk of parasite-induced mortality is less than the reduction in parasite-induced fecundity:

$$\alpha/k < \delta.$$

A powerful way to illustrate this is through a graphical representation of the phase-plane analysis of the basic

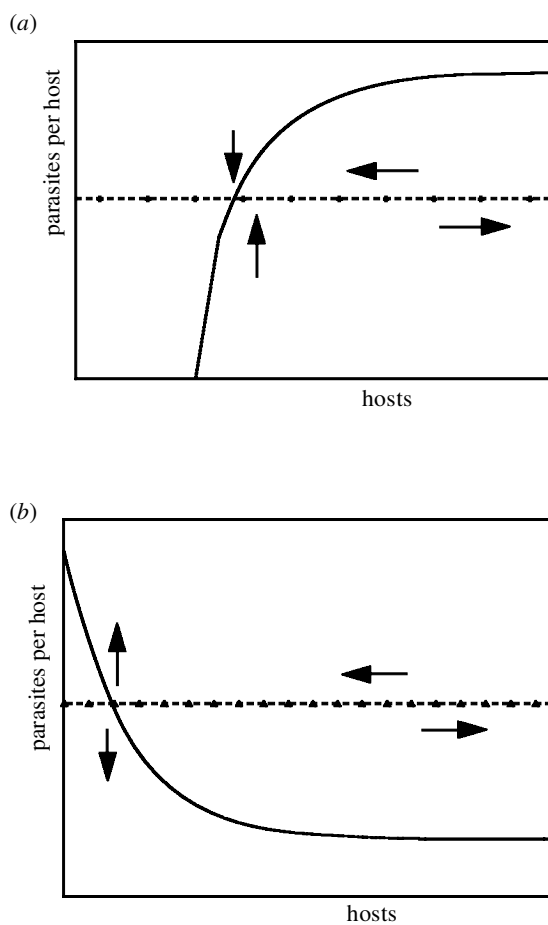


Figure 8. Phase plane analysis of the Anderson & May (1978) macroparasite model with the host (H) isocline $\delta H/\delta t = 0$ represented by the dashed line and the parasites per host (M) isocline $\delta M/\delta t = 0$ represented by the solid line: (a) parasite induced mortality (α) large relative to parasite induced reduction in host fecundity (δ) ($\alpha/k > \delta$) and (b) parasite induced mortality (α) small relative to parasite induced reduction in host fecundity (δ) ($\alpha/k < \delta$). In (a) the isoclines generate stability with populations showing damped oscillations while in (b) the parasite isocline moves and generates unstable oscillations.

Anderson & May (1978) model (figure 8). When the impact of parasites on survival is large relative to that on fecundity ($\alpha/k < \delta$), the phase plane isoclines reflect the pattern seen for the basic Lotka–Volterra predator–prey model with density dependence in the predator population. The vectors also indicate that the dynamics are essentially stable, and perturbations lead to damped cycles that return to the equilibrium represented by the point where the two isoclines intersect (figure 8a). By contrast, when the parasite-induced reduction in survival is low relative to the impact on fecundity ($\alpha/k > \delta$), then the parasite isocline changes and leads to unstable and oscillatory dynamics (figure 8b).

In summary, the parasite-induced reduction on fecundity is particularly important to the stability of the observed growth rates. When this is large relative to the impact on survival, instability and variations in growth rate will be observed. The specific cycle period, patterns of oscillations and amplitude will relate to the specific tension between the growth rate of the host and the parasite (figure 2).

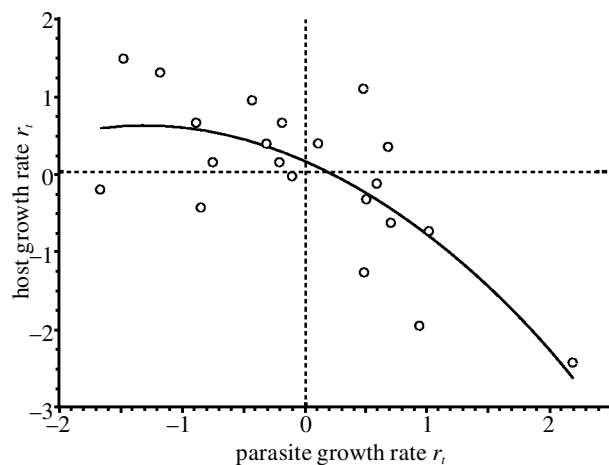


Figure 9. Observed host population growth rate in relation to observed parasite growth rate from a longitudinal study (the same study area as figure 6). Note that host growth rates tend to be positive when parasite growth rates are negative and host growth rates tend to be negative when parasite growth rates are positive.

5. POPULATION LEVEL PATTERNS

In the previous section, we found that when the parasite-induced effects and distribution were incorporated into the model we could predict that the negative growth rates observed at the population level system are essentially caused by the impact of the parasites on host fecundity. This finding is supported by the patterns observed from long-term population monitoring at the population level where negative growth rates and increased breeding mortality occur in years with high worm burdens (figure 6). A clearer illustration is to examine the relationship between observed host growth rates and parasite growth rate; this plot (figure 9) illustrates that host growth rate in this study population tended to be positive when parasite growth rates were negative, and host growth rates negative when parasite growth rates were positive, indicating that the parasites were a regulating factor.

Before we can take our understanding of this system and the interaction with other natural enemies further, we need to test the model predictions and specifically the population level prediction that the parasites are the cause of the negative growth rates. To achieve this, Hudson *et al.* (1998) undertook replicated population level experiments. Initially they used the Dobson & Hudson (1992) model to predict when the growth rate of the study populations would become negative and when treatment should be applied. They also used the model to predict that more than 20% of the population would need to be treated to provide sufficient power to identify a reduction in the size of the negative growth rate. Interestingly, the model predicted that it would be difficult to make growth rates positive and probably impossible to eradicate worms totally. This was because increased treatment would lead to increased survival and productivity, an increase in the density of grouse and a greater force of infection on the grouse population (Hudson *et al.* 1999). Returning to the experiment, a total of six study populations were used; the first two were left as untreated controls for the two population

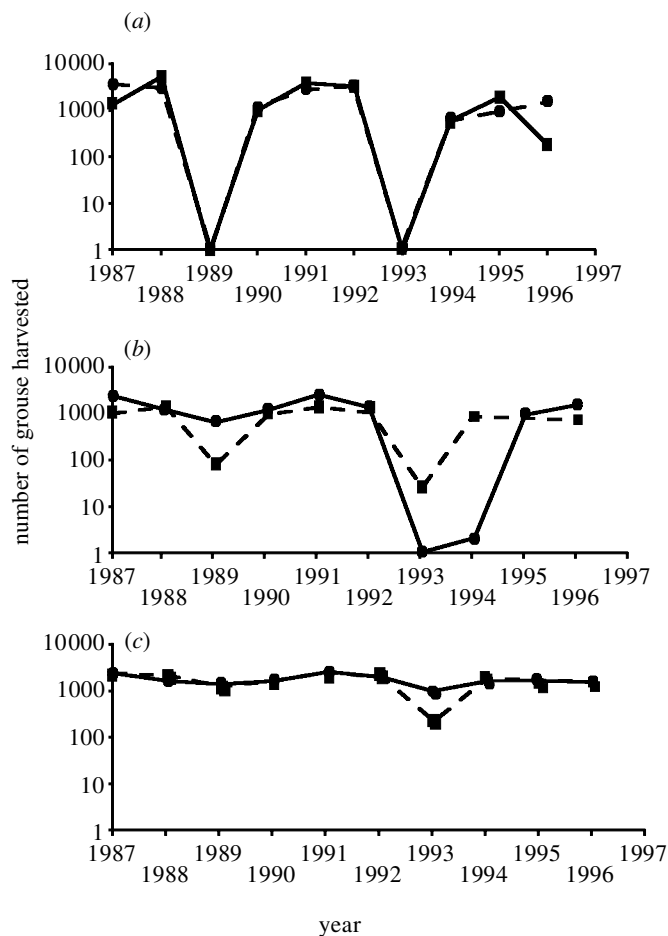


Figure 10. Results from a replicated population level experiment where grouse were treated with an anthelmintic to reduce parasite intensities prior to population growth rates becoming negative in 1989 and 1993 and hunting records recorded: (a) two control populations where no grouse were treated; (b) two experimental populations treated in 1989; (c) two experimental populations treated in both 1989 and 1993. Treatment reduced the negative population growth rates in both years (modified from Hudson *et al.* 1998).

crashes observed. The next two populations were treated prior to the first period of negative growth rate such that the second period of negative growth rate provided a time control. The remaining two populations were treated during both periods of negative growth rate. In each of the six cases of treatment (in four populations), the treated populations exhibited negative growth rates lower than the controls and, overall, experimental treatment significantly reduced the variance in the growth rate of the populations, with two treatments almost completely removing the tendency to cycle (figure 10; Hudson *et al.* 1998), even though the population had been cyclic prior to the experiment. These results support the model predictions that negative growth rates are produced by the influence of parasites on the fecundity of grouse.

6. COMMUNITY LEVEL INTERACTIONS WITH NATURAL ENEMIES

By integrating our findings from experiments, models and through long-term monitoring at the individual and population level, we have obtained an insight into the fac-

tors influencing the growth rate of red grouse populations. The initial comparison of growth rates identified that variations in the maximum growth rates between populations could be accounted for through variations in the factors likely to influence food quality (base richness) and the climatic conditions associated with food productivity, but that variations in annual growth rates between years were a consequence of the impact of parasites on the breeding production of individuals. Given that we now have a fairly good understanding of these factors, we can use the model and comparative data to start exploring the consequences at the community level and consider the range of other natural enemies. We start by examining how selective predators may influence patterns of growth rate and then consider the effects of the louping-ill virus and the influence of hunting.

(a) *Selective predation*

Red grouse are prey to a wide range of mammalian and avian predators, notably red foxes, peregrines, hen harriers and eagles (Hudson *et al.* 1997). On all managed grouse moors there is legal control of most of the mammalian predators, notably the fox and stoat, but the killing of raptors such as peregrine, hen harrier and golden eagle along with the owls is illegal but does occur. In a study of grouse mortality, Hudson *et al.* (1992a) recorded the frequency distribution of parasites in red grouse taken from grouse killed by predators and compared them with a random selection of grouse shot. Grouse killed by predators carried greater intensities of worm infection than a random sample but lower infections than grouse that were found dead and presumably died from the infection. These findings, coupled with other data and experimental studies, indicated that predators were selectively removing the heavily infected individuals from the population, presumably because the individuals had been weakened by the parasites and the predators found it easier to either locate them or catch them (Hudson *et al.* 1992b).

If we incorporate predator selection into the Dobson & Hudson (1992) model then we discover that this selective predation does two things to the grouse population. First, the selection reduces the variance in growth rate and dampens the cyclic tendency of the population. This is essentially because the predation is increasing the parasite induced mortality and in so doing making $\alpha/k > \delta$ so the population no longer oscillates. In this respect the predation is reducing the variance in the growth rate of the host. A reduction in the tendency to cycle was identified in long-term intensive field studies at Langholm moor in southern Scotland where increased predation pressure from hen harriers prevented a predicted cyclic increase in grouse abundance (Thirgood *et al.* 2000). More intriguing is that the model predicts that the equilibrium population size increases (figure 11). At first this appears counterintuitive because we have increased mortality, but this has led to an increased equilibrium. However, the reason for this is quite simply that the selective removal of a few heavily infected individuals from the grouse population effectively removes more parasites than grouse and so reduces the regulatory role of the parasites that causes the instability and also leads to an increase in the equilibrium population size.

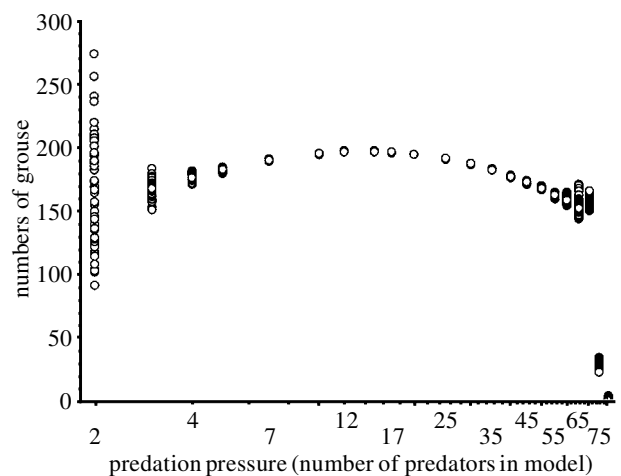


Figure 11. Bifurcation diagram of grouse numbers against predation pressure from the Hudson *et al.* (1992a) macroparasite-host model incorporating selective predation where predators selectively remove the heavily infected grouse. Note that as predation pressure increases so the oscillations dampen and the mean equilibrium level rises, although once predation pressure is high the population subsequently crashes.

(b) *Louping-ill virus*

Louping-ill is a tick-borne virus that causes 80% mortality in exposed grouse (Reid *et al.* 1978), although the evidence is that observed moderate levels of tick infestation have no effect on the grouse chicks (Hudson 1986b). Not all populations of grouse are exposed to ticks and not all of those populations with ticks necessarily have the louping-ill virus circulating in the tick population (Hudson *et al.* 1995). Moreover, the rate of exposure (as estimated indirectly from the proportion seropositive) varied between populations and can range from close to zero to up to 60% (Hudson 1992). We can incorporate this additional mortality into the model as an additive effect, and because we are considering just exposure rate to the virus we do not need to take account of the aggregated distribution of the ticks. The model predicts that increased exposure first leads to increased stability (reduction in the variance of the growth rate) and second to a decrease in equilibrium (figure 12). In contrast to the selective predation, the additional mortality induced by the virus is effectively reducing the population growth rate and through this process reducing infection with the worms, thus leading to stability. The stabilizing effect of louping-ill can also be seen in the time-series hunting record data where the second order partial correlation coefficient of the time-series (an estimate of the strength of the delayed density dependence) of populations with louping-ill was significantly lower than that from populations without louping-ill.

(c) *Harvesting by humans*

While mortality induced by both selective predation and the impact of louping-ill virus were stabilizing, we should note that the hunting records examined in the earlier sections of this paper were highly unstable. In other words,

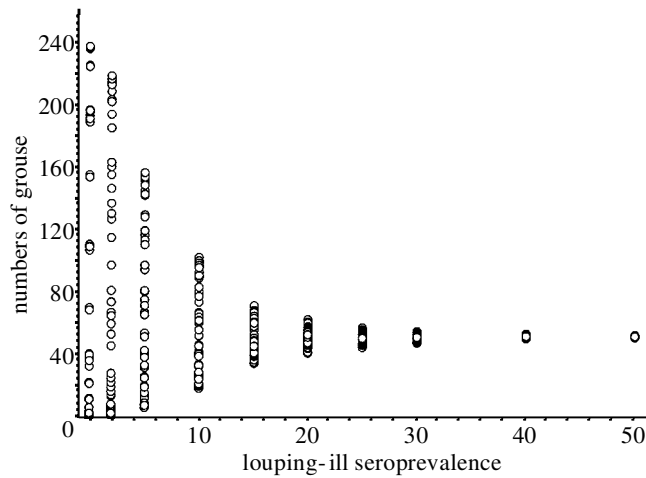


Figure 12. Bifurcation diagram of grouse numbers against seroprevalence of young grouse to louping-ill virus from the Dobson & Hudson (1992) model. Note that increased infection dampens oscillations and reduces mean equilibrium levels.

predation by humans does not appear to be stabilizing the population dynamics of the harvested grouse populations. Harvesting by humans regularly removes up to 50% or more of the population in the autumn, and yet the dynamics are still clearly unstable. This is somewhat surprising as this level of mortality when incorporated into the models should easily stabilize the populations.

One explanation for this observation is that the mechanisms that drive the oscillations occur before the onset of harvesting, such that harvesting reflects the cyclic changes in abundance rather than reducing abundance and infection and so dampening the oscillations. In this respect, it is interesting to note that much of the infection of grouse with *T. tenuis* takes place prior to harvesting (Hudson & Dobson 2001), and even then a large number of infective stages are on the ground and available for infection such that the 'die has been cast' before harvesting commences. If we accept that harvesting has not been highly stabilizing, then this means the important destabilizing effect must occur before harvesting and as such would imply that population changes caused by other mechanisms soon after harvesting, such as changes in spacing behaviour, would in general not influence the population cycles (contra Moss & Watson 2000).

7. CONCLUSION ON STUDYING POPULATION GROWTH RATE

The intrinsic growth rate describes the maximum growth rate of a population and, as such, is the key variable of population models which examine how biological mechanisms or demographic changes can alter observed growth rates. Our comparison of grouse time-series with models is gratifying in that the variation in growth rate can be explained by fundamental predator-prey theory. Furthermore, more than 60% of the variation in the maximum growth rate can be accounted for through food quality. Data analyses, modelling—and especially their synthesis in modern mechanistic time-series approaches—can lead to a focused, testable hypotheses about underly-

ing biological mechanisms. Ultimately though, hypotheses need to be tested through experimentation.

The observed population growth rate is the integral of the birth and death rate processes that occur at the individual level, and the distribution of these risks lead to these processes throughout the population. Risk here is the risk of an individual being killed by a predator, the risk of reduced fecundity through low food availability and the risk of being excluded from a breeding population by the behaviour of others. But the biological mechanisms are the interesting and important aspect of the growth rates that need investigating. This paper has attempted to show this by examining individual level variations in vital rates and how the risk of suffering from natural enemies was distributed through the population. Previous studies led us to suppose parasites may play a role, but as the variance in growth rate was high and parasite induced reductions in fecundity are known to be destabilizing (Anderson & May 1978), we thought it reasonable to suppose that the effects on breeding production would be an initial place to focus. An important component of such studies was to examine the patterns of parasite distribution between individuals and thus consider the overall impact of the parasites at the population level. Indeed, one of the great strengths of the deterministic Anderson & May (1978) model is that it incorporates both the impact of parasites on reduced fecundity and survival, and describes the frequency distribution of this risk within the population. The individual level experiments were incorporated into the model and used to predict population level patterns that could be tested with a replicated field experiment. Such an approach provided a sound framework from which we could start exploring the community level consequences of adding additional natural enemies including selective predation and viral induced mortality.

Clearly, the strength of the experimental approach is that we obtain a foundation of understanding of the processes important at the individual level, and when these findings are incorporated with the modelling this allows us to make predictions at the population level. These predictions can then be tested at the population level: if they are confirmed, we can start to incorporate further complexities from the community level and make predictions that can be tested experimentally. Indeed, this close interaction between experiment, model and then prediction and experiment again provides a good working schedule for examining the important processes that influence growth rate. However, with many natural systems, field experiments on replicate populations may be impractical or unethical. In such cases, the experimental approach may still provide insight from 'model systems'. Such model systems could include free running animals kept in pens in the field (e.g. pheasants and macroparasites) through to laboratory cultures of invertebrates. Careful choice of the model may allow considerable insight into the population dynamics that can then be applied to field studies.

In conclusion, there are three points we wish to make. First, growth rates are an important summary parameter in population models, but considering them in isolation might neglect the many important biological and environmental factors that influence the birth and death process of individuals. These processes should be the focus of our

attention and we need to know the processes influencing birth and death and how the risk of suffering from these is distributed within the population. Age structured models can provide a framework to explore these issues: they should seek to explain the reasons for variations in vital rates between and within age groups, and in so doing, integrate evolutionary life-history studies with demographic ecological studies. Second, time-series analysis, modelling and carefully monitored studies of individuals can provide important insights into the mechanisms influencing population growth rate, but without experiments they can never identify the true mechanisms involved. The ideal approach is simply a combination of all techniques, but they need to be based on a biological understanding that arises from experimental field manipulations. Third, we are keen that the overall purpose of this paper is not misunderstood. This is not a paper only about parasites and grouse, aimed at showing that parasites have some role to play in host population dynamics. Host-parasite interactions are also particularly suitable for exploring general aspects of population dynamics, in particular the ways in which we can tease apart how trophic interactions operate in wild animal populations. Quantifying the risks is not always easy, but this study has illustrated parasite-host trophic interactions are intimate and hence often quantifiable. Moreover, we can use the understanding we obtain at the individual level to make population dynamic predictions that can be tested and then developed to the community level.

We thank all our friends and colleagues who have been very helpful over the years working on the red grouse studies. Particular thanks go to Karen Laurenson, Lucy Gilbert, Simon Thirgood and David Howarth. Grouse moor owners and their keepers have been remarkably helpful with the collection of the bag record data and other extensive information. The Scottish Game Conservancy Trust and NERC supported much of this work. Mick Crawley was particularly helpful in pointing out some simple but important aspects of growth rates while Charley Krebs, Ken Norris and Jim Hone provided helpful comments. We also thank The Royal Society for support to attend the meeting. During the writing of this paper one of our greatest field workers, a German pointer named Fergie, died and this paper is dedicated to her memory.

REFERENCES

Albon, S. D. & Clutton-Brock, T. H. 1988 Climate and the population dynamics of red deer in Scotland. In *Ecological change in the uplands* (ed. M. B. Usher & D. B. A. Thompson), pp. 93–117. Oxford: Blackwell.

Anderson, R. M. & May, R. M. 1978 Regulation and stability of host-parasite interactions. I. Regulatory processes. *J. Anim. Ecol.* **47**, 219–249.

Anderson, R. M. & May, R. M. 1991 *Infectious disease of humans: dynamics and control*. Oxford University Press.

Case, T. 1999 *An illustrated guide to theoretical ecology*. New York: Oxford University Press.

Caswell, H. 2001 *Matrix population models*. Sunderland, MA: Sinauer.

Cobbold, T. S. 1873 The grouse disease. *The Field Lond.*, 15.

Crawley, M. J. 1983 *Herbivory. The dynamics of animal-plant interactions*. Oxford: Blackwell.

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. *J. Anim. Ecol.* **61**, 487–498.

Hastings, A. 1996 *Population biology. Concepts and models*. New York: Springer.

Haydon, D. T., Shaw, D. J., Cattadori, I. M., Hudson, P. J. & Thirgood, S. J. 2002 Analysing noisy time-series: describing regional variation in the cyclic dynamics of red grouse. *Proc. R. Soc. Lond. B* **269**, 1609–1617. (DOI 10.1098/rspb.2002.2053.)

Hudson, P. J. 1985 Harvesting red grouse in the north of England. In *Game harvest management* (ed. S. Beasom & S. F. Roberson), pp. 319–332. Kingsville, TX: Texas A & I.

Hudson, P. J. 1986a The effect of a parasitic nematode on the breeding production of red grouse. *J. Anim. Ecol.* **55**, 85–94.

Hudson, P. J. 1986b *Red grouse. The biology of a managed game bird*. Fordingbridge, UK: Game Conservancy Trust.

Hudson, P. J. 1986c Bracken and ticks on grouse moors in the north of England. In *Bracken: ecology, land use and control technology* (ed. R. T. Smith), pp. 161–170. Carnforth, UK: Parthenon Press.

Hudson, P. J. 1992 *Grouse in space and time*. Fordingbridge, UK: Game Conservancy Trust.

Hudson, P. J. & Dobson, A. P. 2001 Harvesting unstable populations: red grouse *Lagopus lagopus scoticus* (Lath.) in the United Kingdom. *Wildl. Biol.* **7**, 189–196.

Hudson, P. J. & Newborn, D. 1995 *A handbook of grouse and moorland management*. Fordingbridge, UK: The Game Conservancy Trust.

Hudson, P. J., Dobson, A. P. & Newborn, D. 1992a Do parasites make prey vulnerable to predation? Red grouse and parasites. *J. Anim. Ecol.* **61**, 681–692.

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

Hudson, P. J., Norman, R., Laurenson, M. K., Newborn, D., Gaunt, M., Reid, H., Gould, E., Bowers, R. & Dobson, A. P. 1995 Persistence and transmission of tick-borne viruses: *Ixodes ricinus* and louping-ill virus in red grouse populations. *Parasitology* **111**, S49–S58.

Hudson, P. J. N., Newborn, D. & Robertson, P. J. 1997 Seasonal and geographical patterns of mortality in red grouse populations. *Wildl. Biol.* **2**, 79–88.

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

Hudson, P. J., Dobson, A. P. & Newborn, D. 1999 Population cycles and parasitism. *Science* **286**, 2425.

Hudson, P. J., Dobson, A. P. & Newborn, D. 2002 Parasitic worms and the population cycles of red grouse (*Lagopus lagopus scoticus*). In *Population cycles* (ed. A. Berryman), pp. 109–129. New York: Oxford University Press.

Jenkins, D., Watson, A. & Miller, G. R. 1963 Population studies on red grouse, *Lagopus lagopus scoticus* (Lath.) in northeast Scotland. *J. Anim. Ecol.* **32**, 317–376.

Laurenson, M. K., Hudson, P. J., McGuire, K. T., Thirgood, S. J. & Reid, H. W. 1998 Efficacy of acaricidal tags and pour-on as prophylaxis against ticks and louping-ill in red grouse. *Med. Vet. Entomol.* **11**, 389–393.

Lovat, L. 1911 *The grouse in health and disease*. London: Smith, Elder & Co.

May, R. M. 1976 Simple mathematical models with very complicated dynamics. *Nature* **261**, 459–467.

Miller, G. R. 1979 Quantity and quality of the annual production of shrubs and flowers of *Calluna vulgaris* in northeast Scotland. *J. Ecol.* **67**, 109–129.

Moss, R. & Watson, A. 2000 Population cycles in birds of the grouse family (Tetraonidae). *Adv. Ecol. Res.* **32**, 54–111.

Picozzi, N. 1966 Grouse management in relation to the management and geology of heather moors. *J. Appl. Ecol.* **5**, 483–488.

- Reid, H. W., Duncan, J. S., Phillips, J. D. P., Moss, R. & Watson, A. 1978 Studies of louping-ill virus in wild red grouse (*Lagopus lagopus scoticus*). *J. Hygiene* **81**, 321–329.
- Rosenzweig, M. L. 1971 Paradox of enrichment: destabilization of exploitation ecosystems in ecological time. *Science* **171**, 385–387.
- Sæther, B.-E. & Engen, S. 2002 Pattern of variation in avian population growth rates. *Phil. Trans. R. Soc. Lond. B* **357**, 1185–1195. (DOI 10.1098/rstb.2002.1119.)
- Shaw, D. J. & Dobson, A. P. 1995 Patterns of macroparasite abundance and aggregation in wildlife populations: a quantitative review. *Parasitology* **111**, S111–S133.
- Shaw, D. J., Grenfell, B. T. & Dobson, A. P. 1998 Patterns of macroparasite aggregation in wildlife host populations. *Parasitology* **117**, 597–610.
- Sibly, R. M. & Hone, J. 2002 Population growth rate and its determinants: an overview. *Phil. Trans. R. Soc. Lond. B* **357**, 1153–1170. (DOI 10.1098/rstb.2002.1117.)
- Sinclair, A. R. E. & Krebs, C. J. 2002 Complex numerical responses to top-down and bottom up processes in vertebrate populations. *Phil. Trans. R. Soc. Lond. B* **357**, 1221–1231. (DOI 10.1098/rstb.2002.1123.)
- Thirgood, S. J., Haydon, D., Rothery, P., Redpath, S. M., Newton, I. & Hudson, P. J. 2000 Habitat loss and raptor predation: disentangling long- and short-term causes of red grouse declines. *Proc. R. Soc. Lond. B* **267**, 651–656. (DOI 10.1098/rspb.2000.1051.)
- Watson, A., Moss, R. & Parr, R. 1984 Effects of food enrichment on numbers and spacing behaviour of red grouse. *J. Anim. Ecol.* **53**, 663–678.
- Wilson, K. W., Bjørnstad, O. N., Dobson, A. P., Merler, S., Pogliayen, G., Randolph, S. E., Read, A. F. & Skorpington, A. 2001 Heterogeneities in macroparasite infections: patterns and processes. In *The ecology of wildlife diseases* (ed. P. J. Hudson, B. T. Grenfell, J. A. P. Heesterbeek & A. P. Dobson), pp. 6–44. Oxford University Press.