The Royal Society

Host densities as determinants of abundance in parasite communities

Per Arneberg[1]*, Arne Skorping[1], Bryan Grenfell[2] and Andrew F. Read[1,3]

[1]*Department of Biology, Faculty of Science, University of Tromsø, 9037 Tromsø, Norway*
[2]*Department of Zoology, University of Cambridge, Downing Street, Cambridge CB2 3EJ, UK*
[3]*Institute of Cell, Animal and Population Biology, University of Edinburgh, West Mains Road, Edinburgh EH9 3JT, UK*

Several epidemiological models predict a positive relationship between host population density and abundance of directly transmitted macroparasites. Here, we generalize these, and test the prediction by a comparative study. We used data on communities of gastrointestinal strongylid nematodes from 19 mammalian species, representing examination of 6670 individual hosts. We studied both the average abundance of all strongylid nematodes within a host species, and the two components of abundance, prevalence and intensity. The effects of host body weight, diet, fecundity and age at maturity and parasite body size were controlled for directly, and the phylogenetically independent contrast method was used to control for confounding factors more generally. Host population density and average parasite abundance were strongly positively correlated within mammalian taxa, and across all species when the effects of host body weight were controlled for. Controlling for other variables did not change this. Even when looking at single parasite species occurring in several host species, abundance was highest in the host species with the highest population density. Prevalence and intensity showed similar patterns. These patterns provide the first macroecological evidence consistent with the prediction that transmission rates depend on host population density in natural parasite communities.

Keywords: epidemiology; macroecology; metapopulation; patch isolation; transmission rate; parasite abundance

1. INTRODUCTION

Identifying the determinants of species abundance is one of the central problems in ecology (Begon *et al*. 1996). For example, population densities of mammalian herbivores are generally higher in temperate regions than in the tropics, yet the reasons for this variation between communities remains poorly understood (Damuth 1987). Consistent differences in population abundance are also found between communities of parasites. For nematodes in mammals, parasite abundance differs significantly between host species: some mammalian species generally harbour relatively low worm burdens, whereas others have higher nematode densities (Arneberg *et al*. 1997). Here, the prospect for understanding inter-community variation may be better than for many free-living species, because communities can be classified according to characters of host species.

Epidemiological theory points to several characters of host species that may affect densities of macroparasite populations (Anderson & May 1978, 1991; May & Anderson 1978). In particular, for directly transmitted parasites, host population density is assigned a central role, by positively affecting the probability that a parasite transmission stage (e.g. egg/larvae) contacts a host. Looking across helminth communities in host species living at different densities, a positive correlation between host population density and parasite abundance is therefore expected: as host density increases so should the abundances of the parasite populations in the community. Here, we test this prediction using data on parasite abundance in a range of mammalian species gathered from the literature.

2. MATERIALS AND METHODS

(a) *The models*

Dobson (1990) showed that positive relationships between host density and parasite abundance are expected from theory assuming that parasites are short lived compared to the host. In Appendix A, we show that this positive relationship is also expected when this assumption is relaxed, when parasites have no pathogenic effects, and in cases when parasites affect host survival and reproduction. In all cases the model predicts that parasite abundance increases curvilinearly with host density to a plateau (figure 1). Parasite abundance, which expresses the average number of parasites per host, has two components: prevalence, the fraction of hosts in a population infected, and intensity, the mean number of parasites per infected host. If parasite abundance is affected by host population densities, either intensity or prevalence, or both, may be affected.

(b) *Data*

As we are interested in the effects of a host character (population density), we focus on a large and relatively homogenous taxon of mammalian nematodes, the order Strongylida.

*Author for correspondence (pera@ibg.uit.no).

Received 11 March 1998 *Accepted* 16 April 1998

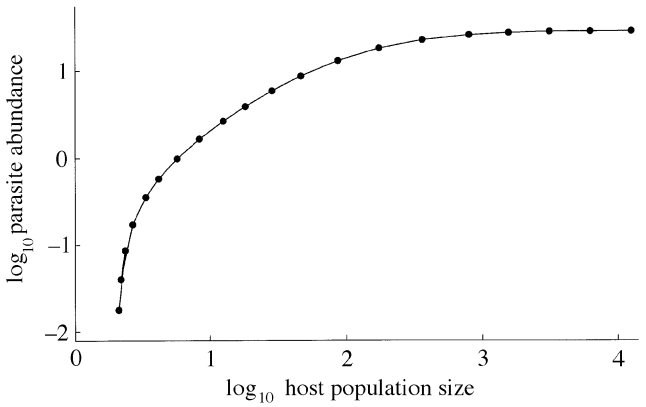

Figure 1. Theoretical relationship between $\log_{10}$ host population size (number of individuals) and $\log_{10}$ parasite population abundance (number of parasites per host individual) when parasites do not affect host survival and reproduction. The parameter ω, which describes the severity of density dependence in host population growth and generates variation in host population density, varied between 10^{-5} and 0.95. The other parameter values were $a=2$, $b=1$, $\mu=2$, $H_0=100$, $\lambda=250$, $k=0.3$. See table 1 for definitions of parameters.

Table 1. *Description of the population parameters used in the models*

parameter	description
a	instantaneous host birth rate (per host per unit of time)
b	instantaneous host death rate, where mortalities are due to causes other than parasites (per host per unit of time)
ω	severity of density dependence in host population growth
α	instantaneous host death rate, where mortalities are due the influence of parasites (per host per unit of time)
β	instantaneous rate of parasite-induced reductions in host reproduction (per host per unit of time)
λ	instantaneous birth rate of parasite transmission stages, where birth results in the production of stages that pass out of the host, and that are responsible for transmission of the parasite within the host population (per parasite per unit of time)
μ	instantaneous death rate of parasites within the host, due to either natural or host-induced (immunological) causes (per parasite per unit of time)
H_0	transmission efficiency constant, varying inversely with the proportion of parasite transmission stages that infect members of the host population
k	parameter describing the negative binomial distribution of parasites within hosts, varying inversely with degree of overdispersion

Compared with variation across all direct life cycle mammalian nematodes, closely related parasite species are more likely to share a range of characters affecting abundance, thus reducing problems of confounding variables. To further reduce extraneous variation, we only consider one habitat within the mammalian body, the gastrointestinal tract, where all strongylid nematodes have direct life cycles (Anderson 1992). As a measure of parasite abundance within a host species, we use the average abundance of all gastrointestinal strongylid nematode species. We also consider the potential effects of a number of traits that are correlated with mammalian population density and that may affect parasite abundance independently of any direct effects of density *per se*. In particular, host body size may be important if, for example, greater food intake results in greater worm intake, or total energy within hosts limits parasite population density. Host diet may also affect worm load if higher food intake of herbivores compared with carnivores leads to higher ingestion rates of parasites. From theory, host birth and death rates can also be important determinants of parasite abundance (Anderson & May 1978; May & Anderson 1978). Finally, the effects of nematode body size are considered. Like free-living species (e.g. Griffiths 1992), densities of smaller-bodied mammalian nematodes are higher than those of larger-bodied species. This size–abundance relationship also captures correlations between abundance and other traits of nematode life history that may affect abundance (e.g. fecundity and generation time (Arneberg *et al*. 1998)).

Studies of mammals where the entire gastrointestinal tract had been examined for strongylid nematodes were included if 30 or more host individuals had been sampled and if only adult parasites were included in the estimates. This yielded data from 19 mammalian species, representing examination of 6670 host individuals and recovery of more than two million individual parasites. Values from single studies of parasite abundance and intensity were logarithmically transformed and prevalence arcsin-transformed before averages were calculated. Within-host-species means were first calculated for each parasite species. Mean parasite abundance within host species was then taken as the average of the parasite species means. Analyses were done on transformed values.

Host population density was measured as the number of individuals per square kilometre, and multiple estimates were averaged within species. Host diet was classified into primary and secondary consumers, and was unclassifiable for two host species (*Procyon lotor* and *Ursus americanus*). Age at maturity is the measure of mammalian life history that best reflects interspecific variation in ecological generation time (Read & Harvey 1989) and was used here as an inverse measure of host death rates. Fecundity was measured as the number of offspring produced per year, following Read & Harvey (1989). Nematode body size was measured as female length, following Skorping *et al*. (1991). Body size of all parasite species was averaged within hosts using geometric means. All continuous measures were logarithmically transformed. Data sources are given in an electronic appendix on the Royal Society's web site (`http://pubs.royalsoc.ac.uk/publish/pro_bs/xxx.htm`), where we also list the host species sampled, the number of studies of each host, the occurrences of each parasite species, and provide summary statistics of the data.

We checked for potential bias from several sources in the data. The following sources of bias were considered: variation in number of host individuals examined for parasites, variation in whether parasites counts were made from the entire gastrointestinal tract or from subsamples of it, and variation in geographic area censused to estimate host population density (see Blackburn & Gaston 1996; Smallwood *et al*. 1996). In addition, data on nematode body size was unavailable for some

species, and we therefore considered whether average size of all parasite species within a host can be meaningfully estimated from a subsample of the parasite species. No significant biases were detected. The analyses are given in the electronic appendix, together with arguments about why we considered it necessary to rule out these sources of bias empirically.

(c) *Analyses*

The data were analysed using both cross-species analyses (i.e. ordinary regression with host species values as independent data points) and phylogenetically independent contrasts. We used a modification of Felsenstein's (1985) independent contrast method (Pagel 1992). Contrasts were calculated using the statistical package CAIC (Purvis & Rambaut 1995), and relationships tested with regression forced through the origin (Harvey & Pagel 1991). Details about how the mammalian phylogeny was assembled are given in the electronic appendix. The branch lengths used adequately standardized contrasts of all variables (for all variables, $p > 0.05$ for the correlation between standardized contrasts and their expected variances). We did not control significance levels for multiple comparisons, because this significantly increases the probability of dismissing real patterns (Rothman 1990).

In addition to calculating contrasts for average abundance of all parasite species within a host, we also compared abundance of the same parasites between closely related host taxa living at different densities. This has the advantage of holding constant potentially important parasite characters, such as body size and behaviour of transmission stages (e.g. Read & Skorping 1995; Arneberg *et al*. 1998). To calculate contrasts with parasite species held constant, we first identified host species sharing parasite species. Some of these hosts shared different nematode species with different host species. Because a host species can only be used once in an analysis, we had to choose between alternative contrasts. To maximize statistical power, we always chose to contrast those taxa that yielded the largest difference in host population density.

3. RESULTS

Host population density and parasite abundance was positively correlated when the effects of other variables were controlled for: looking at comparable host species, nematode abundance tends to be highest in the hosts living at highest population density. This was found both using conventional cross-species analyses (figure 2) and using analyses incorporating phylogenetic relatedness.

(a) *Cross species analyses*

Without controlling for the effects of other variables, parasite abundance was not significantly correlated with host population density ($n=19$ species, $r=0.17$, $p=0.48$). However, host body weight and host population density were strongly negatively correlated ($n=19$, $r=-0.76$, $p=0.0001$). Because density and body weight are both related to parasite abundance in the same direction ($r=0.26$, $p=0.29$ for the correlation between host body weight and parasite abundance), their effects may cancel each other out unless the effect of either variable is controlled for first. Indeed, once the effects of host body size had been removed by partial correlation, parasite abundance was strongly positively correlated with host population density ($n=19$, $r=0.60$, $p=0.008$; figure 2). Thus, nematodes are more abundant in mammals that are relatively common for their body size than they are in rarer host species.

Host diet is another potentially important variable. In a multiple regression model with all four host life-history traits as independent variables, host body weight and diet were significantly correlated with host population density when the three other variables had been controlled for ($n=17$; $r=-0.84$, $p=0.0001$, and $r=0.84$, $p=0.0002$, respectively). Age at maturity and fecundity were not ($n=17$; $r=0.02$, $p=0.95$, and $r=0.17$, $p=0.57$, respectively). Host population density and parasite abundance remained positively correlated when host diet and body weight was controlled for simultaneously ($n=17$, $r=0.65$, $p=0.008$). Nematode body size was not significantly correlated with host population density across mammalian species ($n=17$, $r=-0.25$, $p=0.33$).

A linear model fitted the relationship between host density and parasite abundance better than a curvilinear one after the effects of host body weight had been removed (curvilinear relationship tested for by adding a quadratic term of host density, $n=19$, $R^2=0.36$ and $R^2=0.29$ for the linear and quadratic models, respectively; all host density estimates were scaled to positive values before quadrating, to avoid similar values from negative and positive $\log_{10}$ density estimates).

In addition, host body weight had an independent effect on parasite abundance. After the effects of host population density had been controlled for, host body weight was strongly positively correlated with parasite abundance ($n=19$, $r=0.62$, $p=0.006$). Thus, host species that are large-bodied relative to their population density have a higher parasite abundance than more small-bodied mammals.

(b) *Analyses using host phylogeny*

Using the independent contrasts method, parasite abundance was significantly correlated with mammalian population density without controlling for other variables ($n=14$ set of contrasts, $r=0.76$, $p=0.001$). Thus, among closely related mammalian taxa, subtaxa with the highest population density generally harbour nematode communities with highest population abundances.

Within taxa, no host life-history trait was significantly correlated with host population density ($n=14$; body weight, $r=-0.40$, $p=0.10$; age at maturity, $r=0.10$, $p=0.71$; fecundity, $r=-0.11$, $p=0.67$; for host diet, only two contrasts could be calculated, showing that diet is fairly constant within the mammalian taxa considered). Nematode body size was weakly, but significantly, correlated with host density within host taxa ($n=13$, $r=-0.55$, $p=0.04$). However, the relationship between host population density and parasite abundance was still significant after the effects of nematode body size had been controlled for ($n=13$, $r=0.67$, $p=0.01$).

Host body weight was not significantly correlated with abundance within host taxa without controlling for other variables ($p > 0.8$). However, once the effects of host density were controlled for, host body weight was significantly correlated with parasite abundance ($n=14$, $r=0.60$, $p=0.02$). In comparison, controlling for the effect of host body weight revealed a stronger relationship between host density and parasite abundance ($n=14$, $r=0.85$,

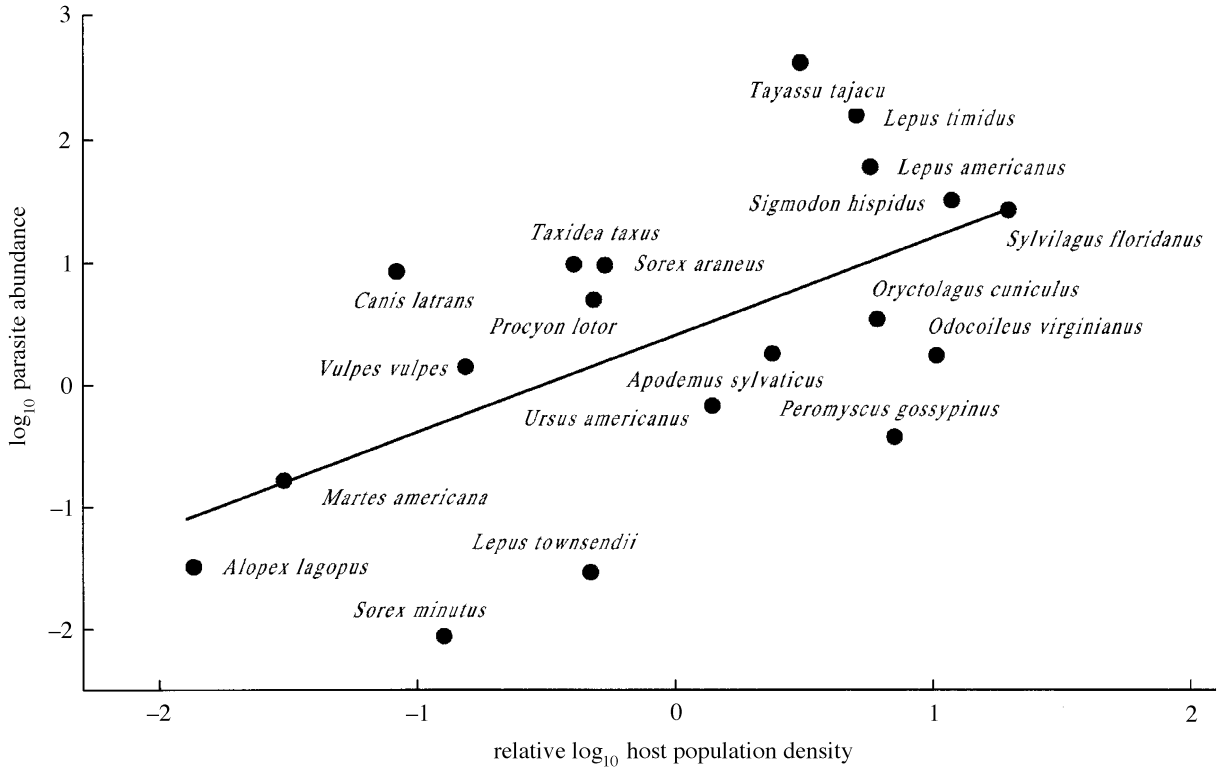

Figure 2. Relationship between relative $\log_{10}$ host population density and $\log_{10}$ abundance of strongylid nematodes across 19 mammalian species. Host density is plotted as residuals from a correlation with $\log_{10}$ host body weight; parasite abundance is the within-host averages of all strongylid nematodes. Names are given for host species.

$p=0.0001$). Thus, although there is a tendency for larger hosts to harbour more parasites when comparing closely related mammalian taxa, this effect is weaker than that of host population density on parasite abundance.

(c) *Analysis using both host and parasite phylogeny*

Most important, when looking at the same parasite species shared by two closely related host species, host population density and nematode abundance were still significantly correlated, despite a limited number of possible comparisons ($n=6$ set of contrasts, $r=0.82$, $p=0.02$). Thus, when a parasite species is found in two closely related host species, its abundance is likely to be higher in the host species living at the higher density.

(d) *Intensity and prevalence*

Intensity and prevalence, the two components of abundance, were also positively correlated with host population density. For example, looking at two comparable host species, a parasite generally infects a larger proportion of the host population in a common host species than in a rarer host. Details of these results are shown in the electronic appendix.

4. DISCUSSION

These analyses show that for strongylid nematodes of mammals, abundance depends on host population density. Host density and parasite abundance were positively correlated when the independent contrast method and host phylogenies were used to control for confounding factors, when host and parasite phylogenies were used jointly to also hold parasite characters constant, or when the relevant mammalian life-history traits had been controlled for directly using cross-species analyses. These patterns all point to a significant biological link between densities of host populations and parasite abundance.

From theory, this link is generated by host densities positively affecting parasite transmission rates: as host densities increase, each parasite egg or larva enjoys an increased probability of contacting a host (Anderson & May 1978; May & Anderson 1978). Here, we found the effect of transmission rates on parasite abundance with contact rates measured only as host population density, i.e. without taking into account other aspects of mammalian behaviour relevant to parasite transmission. This suggests that information on traits, such as host home range size, group size and degree of sociality, may further explain variation in parasite community abundance.

In addition to the propensity of high-density host species to produce high parasite abundance, such hosts may, from theory, also be able to sustain some parasite species which, by virtue of their particular biological characters, can only establish in high-density host populations (May & Anderson 1979). These parasite species may have a lower abundance than others in the community (Dobson 1990). Average abundance of all parasite species in the community, the variable studied here, may therefore contain error variation to the expected theoretical relationship between host population density and parasite abundance. We are currently exploring whether this source of variation, or the

potential influence of other host traits, explains why the observed relationship between host densities and parasite abundance took a linear form (figure 2), and not the curvilinear shape predicted from figure 1.

The assumption that host density is linked to parasite abundance by positively affecting parasite transmission rates is crucial in epidemiological theory, predicting that parasites may regulate host populations and stabilize interactions between herbivore and plant populations. It also plays a critical role in models of intervention strategies against parasites in humans and agricultural systems (Anderson & May 1978, 1991; Grenfell 1988, 1992; Dobson & Hudson 1992; Coyne & Smith 1994). That we were able to unequivocally support this assumption here may be because the comparative approach is better suited to address the question of a link between host population density and parasite transmission rate than are studies of covariation between host density and parasite abundance through time within single host–parasite systems. Despite large numbers of longitudinal studies, correlations between host density and macroparasite abundance are rarely reported in the literature (e.g. Haukisalmi *et al*. 1988; Haukisalmi & Henttonen 1990). This may be because, at the scale of temporal variation, theory neither predicts, nor do observations show, any simple relationship between host density and worm abundance. For example, epidemiological theory, with density-dependent transmission as a central assumption, successfully explains the interrelationship between densities of grouse populations and abundance of the directly transmitted strongylid nematode *Trichostrongylus tenuis*. Yet, only with detailed knowledge of system parameters was it possible to predict the relationship between host density and parasite abundance given the underlying host–parasite dynamics (Dobson & Hudson 1992; Hudson *et al*. 1992). Comparative analyses across communities almost certainly average across such complexities, which may explain why we were more readily able to detect the predicted relationship than is the case in intra-community analyses.

It has been suggested that the primary determinants of population abundance in parasite communities are density-dependent processes within host individuals, such as competition, rather than between-host processes (Holmes 1973). The patterns found here imply that this cannot always be the case, and that transmission rates constrain parasite population growth, at least in some host species. Why else would parasites tend to be rare in a low-density host species? Rather, our findings support the interpretation of parasite community structure as being a consequence of 'supply-side' ecology (Lewin 1986): you have what you are exposed to (Bush 1990), with host density being an important determinant of exposure rates, and, again, most likely so in parasite communities inhabiting low-density host populations. In addition, the tendency for large-bodied host species to harbour more parasites than small-bodied ones may also reflect the role of transmission rates in determining abundance in nematode communities. Higher food intake of larger host species may, for example, lead to higher ingestion rate of parasite transmission stages. If so, we note that host body size still appears to be a less important determinant of transmission rates than host population density, as host size was more weakly associated with parasite abundance.

More broadly, the positive relationship between host densities and parasite density, measured as prevalence, has its parallel to patterns found among free-living animals living in a set of discrete patches. Among such animals, patch occupancy (the fraction of patches occupied, i.e. prevalence) often increases with decreasing patch isolation (i.e. increasing patch density) (e.g. Smith 1980; Harrison *et al*. 1988; Thomas & Jones 1993). These patterns have been found for a wide range of organisms, including insects and mammals, and the biological mechanism appears similar to variation in parasite transmission rates: distantly located (low-density) patches are less frequently occupied because they are harder to colonize (Hanski 1994; Hanski & Gilpin 1997). Such similarities between parasites and free-living animals hold promise for the possibility of broad generalizations about the role of patch (host) density in determining population dynamics of animals exploiting fragmented habitats (Nee 1994; Nee *et al*. 1997).

We thank Ilkka Hanski for thorough and constructive criticism and Sally Montgomery for letting us use unpublished data from her PhD thesis. P.A. and A.S. were supported by a grant from the Norwegian Research Council, B.G. by the Wellcome Trust and NERC, and A.R. by a BBSRC Advanced Research Fellowship.

APPENDIX A

We used the models given by Anderson & May (1978), Anderson (1979), May & Anderson (1978) and Grenfell (1992), describing the dynamics of host (H) and parasite (P) populations for macroparasites with direct life cycles. We assume that (i) host population growth is density dependent in the absence of parasites; (ii) parasite population growth is density dependent in the absence of parasite-induced host deaths; (iii) the frequency distribution of parasites within hosts follows the negative binomial; and (iv) the dynamics of free-living transmission stages occur on a much faster time-scale than changes in host or adult parasite populations. This gives

$$\frac{\mathrm{d}H}{\mathrm{d}t} = (a - b - \varpi H)H - (\alpha + \beta)P \tag{A1}$$

$$\frac{\mathrm{d}P}{\mathrm{d}t} = \frac{\lambda PH}{H_0 + H} - (b + \varpi H + \alpha + \mu)P - (\alpha + \mu)\frac{(K+1)P^2}{kH}. \tag{A2}$$

Parameters are defined in table 1. Here, we are interested in parasite abundance, mean number of parasites per host, $M = P/H$. Rate of change in M becomes

$$\frac{\mathrm{d}M}{\mathrm{d}t} = \frac{\mathrm{d}P/H}{\mathrm{d}t} = \frac{\frac{H\mathrm{d}P}{\mathrm{d}t} - \frac{P\mathrm{d}H}{\mathrm{d}t}}{H^2} = \frac{\lambda MH}{H_0 + H} - (a + \alpha + \mu)M - \left(\frac{\alpha}{k} + \frac{\mu(k+1)}{k} - \beta\right)M^2. \tag{A3}$$

Equilibria for hosts (H^*) and parasites (M^*) are obtained from equations (1) and (3), respectively:

$$H^* = \Omega - \phi M^* \tag{A4}$$

$$M^* = \frac{\frac{\lambda H^*}{H_0 + H^*} - \psi}{\delta}, \tag{A5}$$

where Ω is the carrying capacity of hosts in the absence of parasites, $\phi = (\alpha+\beta)/\varpi$, $\psi = a+\alpha+\mu$ and $\delta = (\alpha/k)+(\mu(k+1)/k) - \beta$.

In the extreme case where parasites are commensals with no pathogenic effects (i.e. $\alpha=\beta=0$), hosts will reach the carrying capacity ($H^* = \Omega$), which gives

$$M^* = \left[\frac{\lambda\Omega}{H_0 + \Omega} - a - \mu\right]\left[\frac{k}{\mu(k+1)}\right]. \tag{A6}$$

When host carrying capacity varies across parasite communities, equation (6) gives a positive relationship between host population density and parasite abundance, with abundance increasing curvilinearly with host density to a plateau $[(\lambda-a-\mu)k]/\mu(k+1)$ (figure 1). The relationship in figure 1 is similar to that generated by Dobson (1990), assuming that adult parasites are short lived compared to the host. Here, we can show that positive relationships can be generated also when parasites and host dynamics occur on similar time-scales, and when parasites affect host survival and reproduction. With pathogenic effects, equilibrium parasite abundance becomes

$$M^* = \frac{\frac{\lambda(\Omega - \phi M^*)}{H_0 + \Omega - \phi M^*} - \psi}{\delta}. \tag{A7}$$

The positive solution for M^* from equation (7) is

$$M^* = \frac{-B - \sqrt{(B^2 - 4AC)}}{2A}, \tag{A8}$$

where A is a constant ($\delta\phi$), and B and C are functions of host carrying capacity (Ω); $B=\delta(H_0+\Omega)+\phi(\lambda+\psi)$ and $C=\Omega(\psi-\lambda)+H_0\psi$. Now H^* can be derived from equation (4) for values of M^*. From equations (4) and (8), this can give positive relationships between host population density and parasite abundance similar to that in figure 1. The easiest interpretation of this model is for a stable host–parasite equilibrium, i.e. where reproductive limitations due to parasitism are not sufficiently strong to throw the system into limit cycles. We are currently exploring the implications for figure 1 of the limit cycle case (i.e. $\delta<0$: May & Anderson 1978). These results are also based on the simplest model, and we are investigating whether relationships between host density and parasite abundance other than the one depicted in figure 1 are possible if biological refinements, such as host immunity and seasonality in host herbivory (Grenfell *et al*. 1987; Roberts & Grenfell 1991), are included.

B.G. was supported by BBSRC and EPSRC.

REFERENCES

Anderson, R. C. 1992 *Nematode parasites of vertebrates: their development and transmission*. Wallingford, UK: CAB International.

Anderson, R. M. 1979 The influence of parasitic infection on the dynamic of host population growth. In *Population dynamics, the 20th symposium of the British Ecological Society* (ed. R. M. Anderson, B. D. Turner & L. R. Taylor), pp. 254–281. London: Blackwell Scientific Publications.

Anderson, R. M. & May, R. M. 1978 Regulation and stability of host–parasite population interactions. I. Regulatory processes. *J. Anim. Ecol*. **47**, 219–247.

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

Arneberg, P., Skorping, A. & Read, A. F. 1997 Is population density a species character? Comparative analyses of the nematode parasites of mammals. *Oikos* **80**, 289–300.

Arneberg, P., Skorping, A. & Read, A. F. 1998 Parasite abundance, body size, life histories and the energetic equivalence rule. *Am. Nat*. (In the press.)

Begon, M., Harper, J. L. & Townsend, C. R. 1996 *Ecology: individuals, populations and communities*. Oxford: Blackwell Scientific Publications.

Blackburn, T. M. & Gaston, K. J. 1996 Abundance–body size relationships: the area you census tells you more. *Oikos* **75**, 303–309.

Bush, A. O. 1990 Helminth communities in avian hosts: determinants of pattern. In *Parasite communities: patterns and processes* (ed. G. Esch, A. O. Bush & J. M. Aho), pp. 197–232. London: Chapman & Hall.

Coyne, M. J. & Smith, G. 1994 Trichostrongylid parasites of domestic ruminants. In *Parasites and infectious diseases* (ed. M. E. Scott & G. Smith), pp. 235–248. San Diego: Academic Press.

Damuth, J. D. 1987 Interspecific allometry of population density in mammals and other animals: the independence of body mass and population energy-use. *Biol. J. Linn. Soc*. **31**, 193–246.

Dobson, A. P. 1990 Models for multi-species parasite–host communities. In *Parasite communities: patterns and processes* (ed. G. Esch, A. O. Bush & J. M. Aho), pp. 261–288. London: Chapman & Hall.

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.

Felsenstein, J. 1985 Phylogenies and the comparative method. *Am. Nat*. **125**, 1–15.

Grenfell, B. T. 1988 Gastrointestinal nematode parasites and the stability and productivity of intensive ruminant grazing systems. *Phil. Trans. R. Soc. Lond*. B **321**, 541–563.

Grenfell, B. T. 1992 Parasitism and the dynamics of ungulate grazing systems. *Am. Nat*. **139**, 907–929.

Grenfell, B. T., Smith, G. & Anderson, R. M. 1987 A mathematical model of the population biology of *Ostertagia ostertagi* in calves and yearlings. *Parasitology* **95**, 389–406.

Griffiths, D. 1992 Size, abundance, and energy use in communities. *J. Anim. Ecol*. **61**, 307–315.

Hanski, I. 1994 Patch-occupancy dynamics in fragmented landscapes. *Trends Ecol. Evol*. **9**, 131–135.

Hanski, I. & Gilpin, M. E. (eds) 1997 *Metapopulation biology: ecology, genetics, and evolution*. San Diego: Academic Press.

Harrison, S., Murphy, D. D. & Ehrlich, P. R. 1988 Distribution of the checkerspot butterfly, *Euphydryas editha bayensis*: evidence for a metapopulation model. *Am. Nat*. **132**, 360–382.

Harvey, P. H. & Pagel, M. D. 1991 *The comparative method in evolutionary biology*. Oxford University Press.

Haukisalmi, V. & Henttonen, H. 1990 The impact of climatic factors and host density on the long-term population dynamics of vole helminths. *Oecologia* **83**, 309–315.

Haukisalmi, V., Henttonen, H. & Tenora, F. 1988 Population dynamics of common and rare helminths in cyclic vole populations. *J. Anim. Ecol*. **57**, 807–825.

Holmes, J. C. 1973 Site selection by parasitic helminths: interspecific interactions, site segregation, and their importance to the development of helminth communities. *Can. J. Zool*. **51**, 333–347.

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. *J. Anim. Ecol*. **61**, 477–486.

Lewin, R. 1986 Supply-side ecology. *Science* **234**, 25–27.
May, R. M. & Anderson, R. M. 1978 Regulation and stability of host–parasite population interactions. II. Destabilising processes. *J. Anim. Ecol*. **47**, 249–267.
May, R. M. & Anderson, R. M. 1979 Population biology of infectious diseases. II. *Nature* **280**, 455–461.
Nee, S. 1994 How populations persist. *Nature* **367**, 123–124.
Nee, S., May, R. M. & Hassell, M. P. 1997 Two-species metapopulation models. In *Metapopulation biology: ecology, genetics, and evolution* (ed. I. Hanski & M. Gilpin), pp. 123–147. San Diego: Academic Press.
Pagel, M. D. 1992 A method for the analyses of comparative data. *J. Theor. Biol*. **156**, 431–442.
Purvis, A. & Rambaut, A. 1995 Comparative analysis by independent contrasts (CAIC): an Apple Macintosh application for analysing comparative data. *Comput. Applic. Biosci*. **11**, 247–251.
Read, A. F. & Harvey, P. H. 1989 Life history differences among the eutherian radiations. *J. Zool*. **219**, 329–353.
Read, A. F. & Skorping, A. 1995 The evolution of tissue migration by parasitic nematode larvae. *Parasitology* **111**, 359–371.
Roberts, M. G. & Grenfell, B. T. 1991 The population dynamics of nematode infections in ruminants: periodic perturbations as a model for management. *IMA J. Math. Appl. Med. Biol*. **8**, 83–93.
Rothman, K. J. 1990 No adjustments are needed for multiple comparisons. *Epidemiology* **1**, 43–46.
Skorping, A., Read, A. F. & Keymer, A. E. 1991 Life history covariation in intestinal nematodes of mammals. *Oikos* **60**, 365–372.
Smallwood, K. S., Jones, G. & Schonewald, C. 1996 Spatial scaling of allometry for terrestrial, mammalian carnivores. *Oecologia* **107**, 588–594.
Smith, A. T. 1980 Temporal changes in insular populations of the pika (*Ochotona princeps*). *Ecology* **61**, 8–13.
Thomas, C. D. & Jones, T. M. 1993 Partial recovery of a skipper butterfly (*Hesperia comma*) from population refuges: lessons for conservation in a fragmented landscape. *J. Anim. Ecol*. **62**, 472–481.

An electronic appendix to this paper appears at (`http://pubs.royalsoc.ac.uk/publish/pro_bs/xxx.htm`).

As this paper exceeds the maximum length normally permitted, the authors have agreed to contribute to production costs.