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Distribution and abundance of parasite nematodes: ecological specialisation, phylogenetic constraint or simply epidemiology?

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We investigate the patterns of abundance-spatial occupancy relationships of adult parasite nematodes in mammal host populations (828 populations of nematodes from 66 different species of terrestrial mammals). A positive relationship between mean parasite abundance and host occupancy, i.e. prevalence, is found which suggests that local abundance is linked to spatial distribution across species. Moreover, the frequency distribution of the parasite prevalence is bimodal, which is consistent with a core-satellite species distribution. In addition, a strong positive relationship between the abundance (log-transformed) and its variance (log-transformed) is observed, the distribution of worm abundance being lognormally distributed when abundance values have been corrected for host body size.
Hanski et al. proposed three distinct hypotheses, which might account for the positive relationship between abundance and prevalence in free and associated organisms: 1) ecological specialisation, 2) sampling artefact, and 3) metapopulation dynamics. In addition, Gaston and co-workers listed five additional hypotheses. Four solutions were not applicable to our parasitological data due to the lack of relevant information in most host-parasite studies. The fifth hypothesis, i.e. the confounded effects exerted by common history on observed patterns of parasite distributions, was considered using a phylogeny-based comparison method. Testing the four possible hypotheses, we obtained the following results: 1) the variation of parasite distribution across host species is not due to phylogenetic confounding effects; 2) the positive relationship between mean abundance and prevalence of nematodes may not result from an ecological specialisation, i.e. host specificity, of these parasites; 3) both a positive abundance-prevalence relationship and a negative coefficient of variation of abundance-prevalence relationship are likely to occur which corroborates the sampling model developed by Hanski et al. We argue that demographic explanations may be of particular importance to explain the patterns of bimodality of prevalence when testing Monte-Carlo simulations using epidemiological modelling frameworks, and when considering empirical findings. We conclude that both the bimodal distribution of parasite prevalence and the mean-variance power function simply result from demographic and stochastic patterns (highlighted by the sampling model), which present compelling evidence that nematode parasite species might adjust their spatial distribution and burden in mammal hosts for simple epidemiological reasons.

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From the eight hypotheses listed in the recent literature, and which may contribute to the explanation of a positive relationship between local abundance and spatial distribution of free organisms (see Gaston 1996 and Gaston et al. 1997 for two recent reviews), we were able to test the adequacy of the four following models:

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(1) the sampling artefact model; the positive correlation between abundance and distribution would be expected from a random process with abundant species having a higher probability to be sampled in the occupied patches (see Brown 1984);
(2) the ecological specialisation model; generally, species exploiting a wide range of resources are more widespread and abundant, or resources used by some species are more extensive than those exploited by other species (Brown 1984, Gaston and Lawton 1990);
(3) the metapopulation models (Hanski 1991a, Gotelli 1991, Gyllenberg and Hanski 1992, Hanski and Gyllenberg 1993); the abundant species tend to be widely distributed and the rare ones tend to have restricted ranges;
(4) the phylogeny-based model; two closely related parasite species exhibit similar traits due to their common ancestry (Gaston 1996, Gaston et al. 1997).

Unfortunately, we were unable to test the four other hypotheses, i.e. range position (Brown 1995), breadth of resource usage (Tokeshi 1990), resource availability (see Hanski and Gilpin 1997) and vital rates, mainly due to the simple fact that relevant data are not available in most host-parasite investigations.

Hanski et al. (1993) evaluated three hypotheses (1, 2 and 3 above) for a large range of taxa (free and associate organisms). Surprisingly, they observed for a parasite data set a negative relationship between local species abundance and parasite prevalence, i.e. spatial occupancy measured as the percentage of infected individuals within the entire host population. In addition, generalist forms were found to have lower prevalence and lower abundance values than specialists, i.e. specific parasites, which is totally in contrast to general results obtained for a variety of free animals and plants. Both theoretical and empirical epidemiological studies have demonstrated the existence of a positive relationship between mean abundance of parasites, i.e. mean number of parasite worms per individual host, and prevalence (Shaw and Dobson 1995). Although the basic mathematical assumptions of metapopulation models are not directly applicable to parasite organisms, we may still consider that metapopulation theory may be applicable to some epidemiological models. Moreover, Grenfell and Harwood (1997) have recently pointed out the need of exchanges between these two research areas since both metapopulation and epidemiological models address the same question of population persistence in a set of habitat (host) patches (Nee et al. 1997). The present work was essentially motivated by the strong discrepancies between observations resulting from free organism and some parasite data bases (see above). We shall see that, for each of the four models we have tested, the consequences are surprising, indeed, since they can be interpreted within the context of simple epidemiological modelling.

Here, we re-investigated the three hypotheses listed by Hanski et al. (1993) using a large data set of adult nematode parasites (Nematoda) inhabiting the gut of different terrestrial mammal species. Because species are not independent and two closely related species are typically similar, e.g. they can have inherited similar traits, we performed a comparative analysis for controlling the effect of phylogeny on further regressions (Harvey and Pagel 1991). Undoubtedly, this property of species to have a common history represents a fourth potential alternative for the positive abundance-spatial occupancy relationship in free organisms (see Gaston 1996, Gaston et al. 1997). This point, of similarity associated with shared ancestry, is grasped intuitively for host-parasite studies since parasite species may have directly cospeciated with their host species (Feliu et al. 1997, Morand 1997, Page and Charleston 1998). We here confirm the existence of a positive relationship between local parasite abundance, i.e. mean number of worms per host, and their spatial distribution, i.e. prevalence of infected hosts within the entire host population. More interestingly, we demonstrate that this observed relationship is essentially due to chance mechanisms revealed by epidemiological modelling. We therefore conclude that, for host-parasite associations, the core-satellite species hypothesis, as defined by Hanski (1982), may follow from the manner in which parasite individuals are distributed among their hosts, and thus predictions made by the theory for host-parasite associations could simply stand within the context of pure epidemiological modelling.

Materials and methods

We compiled data for a total data set of 828 populations of adult nematode worms (belonging to 326 distinct species), recorded from the gut of 66 species of terrestrial mammal species. These data were collected from several sources based on a survey of 90 studies published over the last 30 years (Morand and Poulin 1998, complete references can be sent on request). This data set was relevant for studying the abundance-spatial occupancy relationships since all parasite species are closely related, i.e. nematodes, and ecologically similar, i.e. gut parasites of mammals. Values of prevalence (percentage of individual hosts infected within a population), defined as the measure of host patch occupancy by a given parasite species, were obtained for all 828 cases. Values for average worm burdens (abundance) were only available for 584 cases within the entire data set whereas standard deviation values were given in only 104 cases. We considered that all sample hosts were equally suitable for parasite occupation, a basic assumption needed to fit data with a negative binomial distribution (Brown 1995).

Relationship between prevalence and abundance

Since larger hosts harbour higher worm burdens ($p < 0.001$) (Fig. 1), we controlled for the effect of host body weight (as a measure of body size for each mammal species) on both abundance and prevalence parameters using residual values obtained from standard linear regressions. We checked for normality of variable distribution and homoscedasticity of variance (Zar 1996).

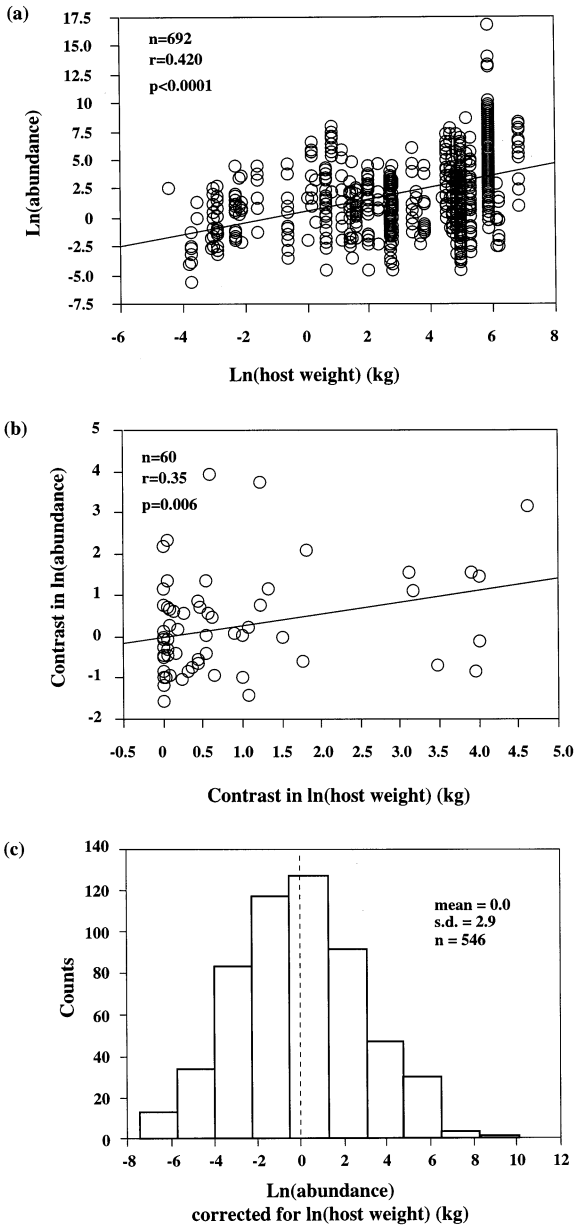

Fig. 1. Relationship between abundance (average worm burden) and host weight, using a) cross species values ($n = 692$), and b) independent contrasts ($n = 60$). c) Abundances are lognormally distributed among host populations giving estimates of mean (0) and standard deviation (2.9) when abundance values (in ln) were corrected for host species weight (in ln).

Relationship between abundance and variance

Taylor's power law describes the spatial heterogeneity of organisms using the empirical relationship:

$$s^2 = aM^b \tag{1}$$

where a represents a constant parameter, b an index of spatial heterogeneity, M the mean abundance and s^2 its variance (Taylor et al. 1978, 1983). Abundance and variance values were fitted to the power function which gives estimates of a and b using the following transformation:

$$\ln(s^2) = b\ \ln(M) + \ln(a) \tag{2}$$

Test of the sampling model

Hanski et al. (1993) proposed the following method for testing the sampling model, which assumes that the prevalence-abundance relationship is a consequence of sampling effects.

Assuming that the distribution of abundance values fits a negative binomial distribution, the fraction of empty patches, here of noninfected hosts, is given by the following relation:

$$p_0 = \left(1 + \frac{M}{k}\right)^{-k} \tag{3}$$

where M is the abundance of parasites, and k the parameter which gives the degree of aggregation. k is expressed by the equation (Hanski et al. 1993):

$$k = 1/(\mathrm{CV}^2 - 1/M) \tag{4}$$

where CV is the coefficient of variation of abundance.

Substituting k in Eq. (3) gives the following relation (after logarithmic transformation):

$$-\ln(p_0) = \frac{1}{\mathrm{CV}^2 - M^{-1}}\,(\ln(M) + 2\ln(\mathrm{CV})) \tag{5}$$

Since the dependence between $1/\mathrm{CV}^2$ and CV is stronger than the dependence between $1/\mathrm{CV}^2$ and ln(CV), the new formulation of Eq. (5) yields the following relation (for large values of M):

$$\ln(-\ln(p_0)) = \ln(\ln(M)) - 2\ln(\mathrm{CV}) \tag{6}$$

Eq. (6) means that the fraction of noninfected hosts within a host population is inversely proportional to the average parasite abundance and proportional to the

CV. We used Eq. (6) to test if CV had a significant positive effect on p_0 when the effect of M has been controlled for.

Test of the ecological specialisation model

All nematodes were exclusively adult forms parasitizing the gut of terrestrial mammals, and thus they did not differ in terms of host habitats (body parts) where they might occur (Morand and Poulin 1998). We then considered host specificity, determined as the number of host species infected by a given parasite species, as a valuable estimate of ecological specialisation. The range of host species parasitized by a given parasite species was derived from our data base. We are aware that this "apparent" calculated host specificity might be simply the product of a sampling artefact, generalist parasites presenting a more familiar distribution among host communities than specialists do (Poulin 1998). For instance, the number of specific parasites might be overestimated if further investigations would report new host species for a given parasite. We accept that the mammals are probably the best surveyed hosts for parasites, which minimizes these potential biases.

First, regressions were performed on both abundance and prevalence values versus the degree of host specificity, i.e. number of known host species for a given parasite. Second, two categories of host specificity were assessed: 1) specific parasite infecting only one host species, and 2) non-specific parasite infecting two or more host species. We then tested the effect of host specificity on the abundance-prevalence relationship.

Epidemiological simulations

In order to test the significance of demographic explanations to observed patterns of bimodality of prevalence, Monte-Carlo simulations were run using epidemiological modelling frameworks (Anderson and May 1985). Epidemiological modelling assumes that the probability distribution of worm numbers per host individual may determine the relationship between the prevalence of infection $P(t)$ and the mean worm burden $M(t)$ at any time t according to the following relation:

$$P(t) = 1 - [1 + M(t)/k]^{-k} \quad (7)$$

where k is the parameter of the negative binomial distribution (Anderson and May 1985). We then used the relationship between k and the two parameters (a and b) of Taylor's power law given in Eq. (1) (Perry and Taylor 1986):

$$1/k = aM^{b-2} - 1/M \quad (8)$$

with a and b the two parameters of Eq. (1).

Eqs (7) and (8) were used to perform simulations on the frequency distribution of parasite populations, with worm burdens following a lognormal distribution according to the empirical distribution. k was calculated using Eq. (8) with the two parameters a and b obtained from Eq. (1). Two thousand populations were simulated and, for each population, prevalence was calculated using Eq. (7).

It can be noted that the two formulae of the sampling model (Eqs 3 and 4) are relatively similar to the two formulae of the epidemiological model (Eqs 7 and 8) in that they both assume a negative binomial distribution of local parasite abundance from which all other patterns are inferred.

Comparative analysis

There are several arguments to support the view that prevalence (and intensity) of worm infections varies in relation to environment (e.g. temperature). However, such epidemiological parameters may also depend on intrinsic characteristics observed within each parasite species, which are in turn non-independent of existing phylogenetic relationships across species (Poulin 1998, Sasal and Morand 1998). Therefore, it seems particularly appropriate, and intuitively not hazardous, to control for the confounded effect exerted by common history on such epidemiological parameters.

We performed an analysis among phylogenetically independent contrasts using the CAIC 2.0 computer package (Purvis and Rambaut 1995). We constructed a phylogeny based on the information in Adamson (1989), Brooks and McLennan (1991) (see Morand 1996) and the recent molecular phylogeny of Blaxter et al. (1998). This phylogeny provided 60 independent contrasts. All correlations between contrasts were forced through the origin, and standardization of contrast values was performed by visual examination of absolute values of standardized contrasts versus their standard deviations (Garland et al. 1992).

Results

Effect of host body size

We found a positive relationship between worm abundance and host body size without and after controlling for phylogenetic effects (Fig. 1a and b, respectively). Therefore, we decided to control all epidemiological parameters for host body size. We found that average worm burden values were lognormally distributed when corrected for host body size (Fig. 1c) with a mean of 0 and a standard deviation of 2.9.

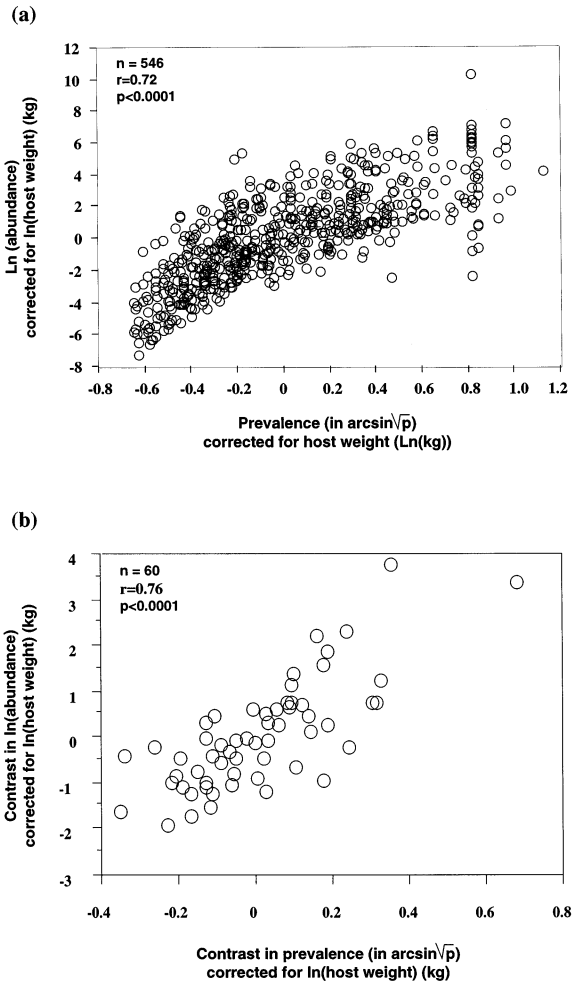

Fig. 2. Variability of abundance (average worm burden) and prevalence of parasite nematodes (828 populations, 326 species) for 66 terrestrial mammal species (obtained from 90 different published sources). Prevalence of parasites plotted against average worm burden shows a strong local abundance-regional distribution relationship as predicted by the core-satellite species hypothesis, using cross species values (584 populations of nematodes), and b) independent contrasts ($n = 60$).

Test of the core-satellite model

After controlling for host body size, we obtained a positive relationship between average worm abundance and prevalence (Fig. 2a and see also Fig. 3b), which may demonstrate that local abundance is linked to spatial host occupancy (here prevalence) as predicted by the core-satellite model. The relationship between prevalence and worm abundance remained statistically significant when controlling for parasite species phylogeny (Fig. 2b).

Additionally, the frequency distribution of the observed worm prevalence showed a bimodal pattern, thus supporting the existence of a core-satellite parasite species distribution (Fig. 3a). As observed in the core-satellite model prediction, regionally rare nematode (satellite) species were observed to be more frequent in the environment than locally abundant (core) species.

Test of the sampling model

The sampling model predicts a positive relationship between parasite abundance and prevalence but, also, a negative relationship between CV and prevalence. Both effects were found for nematode worms of terrestrial mammals (Table 1) without controlling (Fig. 4a), or when controlling (Fig. 4b) for phylogenetic effects.

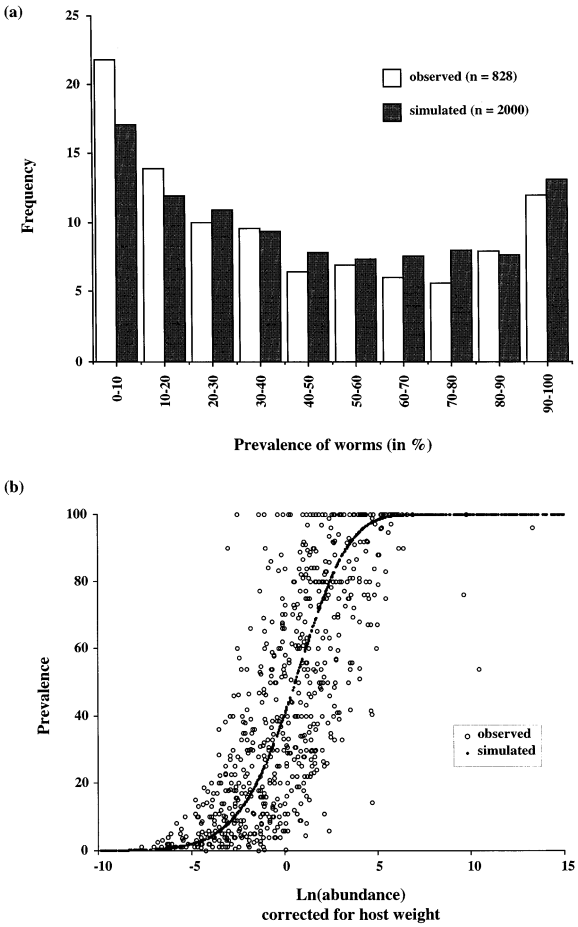

Fig. 3. a) Comparison of observed and simulated values of prevalence of worms showing a bimodal distribution (core-satellite distribution). Simulated prevalence of worms were obtained from a Monte-Carlo simulation with average worm burdens obtained from a lognormal law (mean and standard deviation estimated from Fig. 1), with prevalence calculated using epidemiological models of the form $P(t) = 1 - [1 + M(t)/k]^{-k}$, with k the parameter of the negative binomial law expressed as $1/k = aM^{b-2} - 1/M$ where a and b represent parameters of the power-function (Fig. 6a). b) Relationships between prevalence and abundance of worms. The graph illustrates the comparison between observed values and simulated values.

Table 1. Test of the sampling model for nematodes of terrestrial mammals. The dependent variable is $\ln(-\ln(p_0))$ (where p_0 is the proportion of noninfected hosts, M the abundance of nematodes and CV the coefficient of abundance variation in nematodes; see Materials and methods for further explanation). The sense of effect of independent variables on $\ln(-\ln(p_0))$ is negative for CV and positive for M.

Independent variables	Coefficient	Standard error	p
Intercept	−0.352	0.253	0.167
−2 ln(CV)	0.205	0.032	<0.0001
ln(ln(M))	0.339	0.072	<0.0001

Test of the ecological specialisation hypothesis

In this model, it is implicitly assumed that generalists should exhibit a wider range of distributions and have higher average abundance than specialists. However, nematodes of terrestrial mammals do not support this hypothesis. First, no effect of sampling effort on the level of host specificity was detected ($p > 0.05$). Second, no positive or negative relationship between specificity (host species range) and prevalence was found ($p > 0.05$). Third, we observed no significant relationship between specificity and abundance correcting or not for phylogenetic effects (Fig. 5a and b, respectively).

Finally, our findings show no effect of specificity by category (specific versus non-specific parasites) on the abundance-prevalence relationship (Fig. 6).

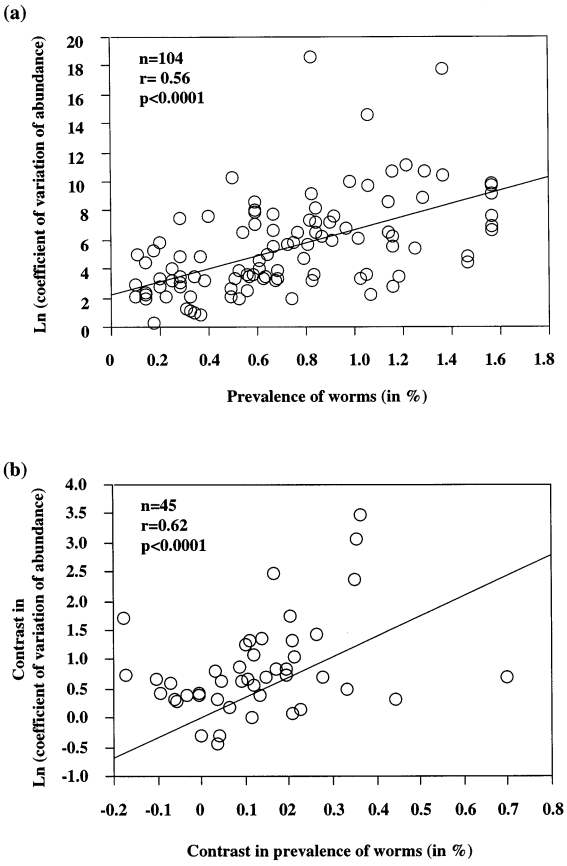

Fig. 4. Relationship between the logarithms of the coefficient of variation of abundance (in ln) and the prevalence of parasites using a) cross species values ($r^2 = 0.32$, $n = 104$, $p < 0.0001$), or b) independent contrasts ($r^2 = 0.39$, $n = 45$, $p < 0.0001$).

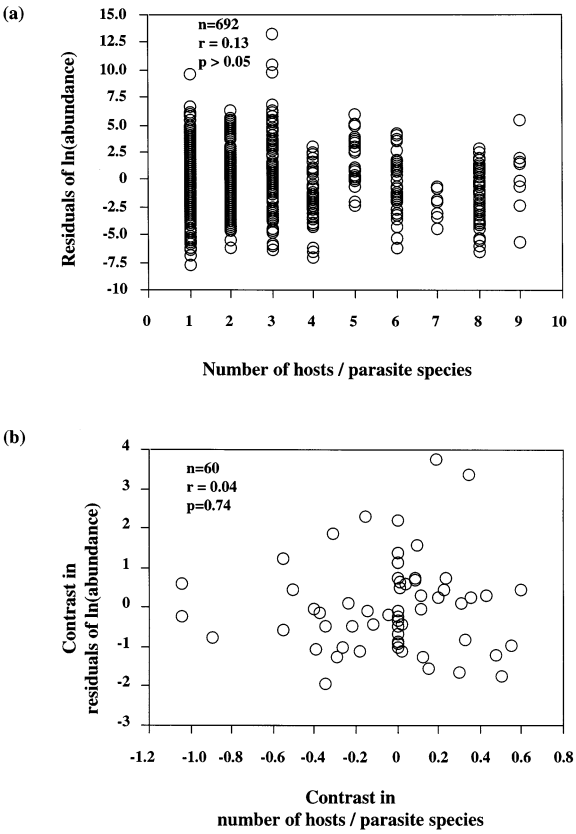

Fig. 5. Relationship between host specificity (expressed as the mean number of infected hosts species per parasite species) and mean abundance using a) cross species values ($n = 692$), and b) independent contrasts ($n = 60$). No significant relationship between host sampling effort, or number of citations, on the level of host specificity was detected ($p > 0.05$).

Taylor's power relationship

The positive relationship between average worm burden and its variance was well fitted to a power function ($p < 0.0001$) yielding estimates of parameters a (3.27 ± 0.17) and b (1.80 ± 0.04) (Fig. 7a). Using independent contrasts, the relationship was also well fitted to a power function ($p < 0.0001$) yielding an estimate of b (1.64 ± 0.06) with a equal to zero since the regression line was forced through the origin (Fig. 7b). A test of comparison between these two regression lines showed no significant difference between slopes ($p > 0.05$).

Simulation studies

Eqs (7) and (8) were used to simulate the frequency distribution of parasite populations, with worm burden following a lognormal distribution according to the empirical distribution of nematode abundance (Fig. 3a). The parameter k of the negative binomial function was calculated using formula (8) with the two parameters (a and b) of the power function being equal to 3.27 and 1.80, respectively (Fig. 4a). Simulated values of prevalence are well fitted to the bimodal distribution of observed values (Fig. 3a). Thus, the number of nematode species, locally rare and spatially aggregated, is higher than the number of parasites locally abundant and spatially well-distributed. Furthermore, simulated values of abundance and prevalence perfectly fit the observed patterns (Fig. 3b).

Discussion

Two different models have been proposed to describe the spatial distribution and local abundance of animals and plants. One is a behavioural model involving natural combination of migratory and aggregating behaviours of organisms (Taylor et al. 1983, Perry 1988, Hanski and Gyllenberg 1993). The other one is a demographic model suggesting stochastic interplay of demographic population characteristics and environmental heterogeneity (Anderson et al. 1982, Nee et al. 1991). These two theoretical models gave rise to the core-satellite species hypothesis (Hanski 1982, 1991a, Hanski and Gyllenberg 1993, Hanski et al. 1995).

The core-satellite species hypothesis explicitly refers to the existence of a spatially structured population (Hanski 1982, Nee et al. 1991) predicting a bimodal distribution of organisms in their environment (most species are present in most patches or in only a small fraction of patches), since the "rescue effect" becomes important for local persistence (Brown and Kodric-Brown 1977, Hanski 1991b, Hanski and Gyllenberg 1993, Hanski and Gilpin 1997). The core species which are both abundant within sites and distributed among many sites have a high probability of producing emigrants. On the contrary, the satellite species which are both in low densities and with a restricted distribution tend to produce few successful dispersers. This mechanism assumes that production of emigrants affects local dynamics by decreasing the probability of local extinction. When incorporating the "propagule rain" into metapopulation models, that is to say the idea that immigration to new sites does not depend basically on regional occurrence but only on the number of unoccupied sites, Gotelli (1991) showed that both the "rescue effect" and "propagule rain" effects might yield bimodal distributions of species.

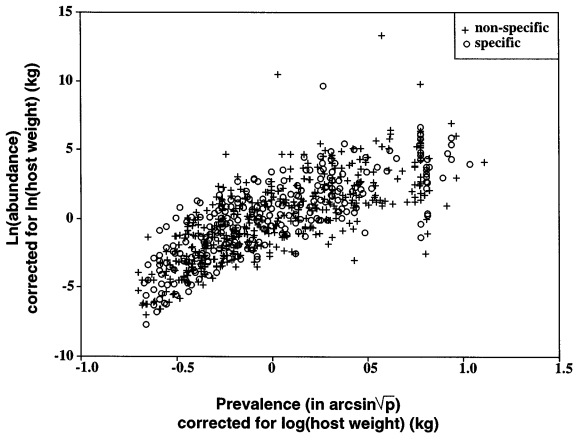

Fig. 6. The relationship between abundance and prevalence of worms for the two categories of parasites, i.e. specific and non-specific, is similar (ANCOVA test, $p > 0.05$) which tends to demonstrate the absence of significant effect of parasite specialisation.

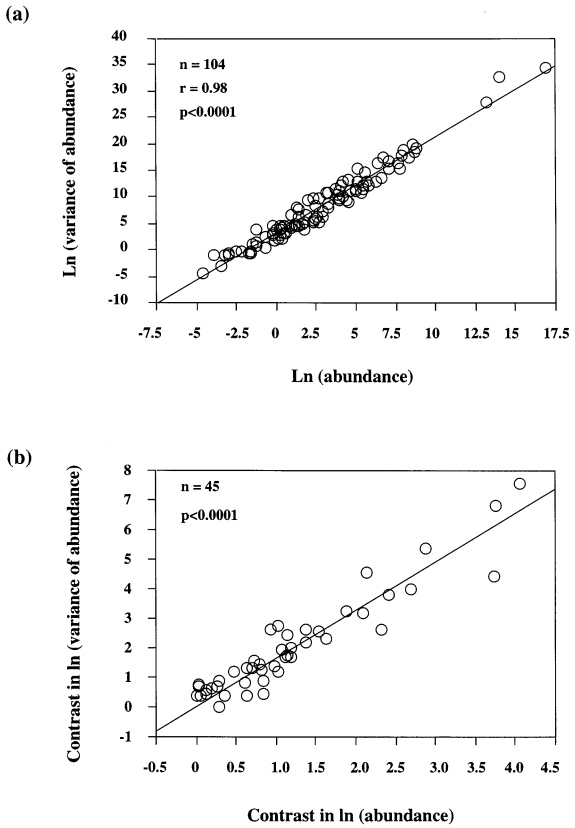

Fig. 7. Relationship between the logarithms of the variance (s^2) and abundance (M) is fitted to a power function using a) cross species values for which $\ln(s^2) = b\ \ln(M) + \ln(a)$ with a (3.27 ± 0.17) and b (1.80 ± 0.04) and b) independent contrasts giving estimate of b only (1.64 ± 0.06) since in this case the regression line is forced through the origin (see Materials and methods).

Taylor's power law has been used to investigate the spatial heterogeneity of organisms in their environment using the power relationship $s^2 = aM^b$ (Taylor and Taylor 1977, Taylor et al. 1978, 1983). The range of values taken by exponent b reflects in some way natural tendencies of organisms to choose between mutual attraction or mutual repulsion (Taylor et al. 1983, Perry 1988). Conceptual models emerging from both the core-satellite hypothesis and Taylor's power law have been strongly criticised (Hanski 1982, 1987, Downing 1986, Gaston and Lawton 1989, Nee et al. 1991), but quite surprisingly, they have not yet been compared, even though both rely on a balance between specific distributive behaviours!

Numerous studies have described the clumped distributions of parasites, usually fitted to the negative binomial distribution (Dobson and Merenlender 1991, Grafen and Woolhouse 1993, Gregory and Woolhouse 1993, Poulin 1993, Shaw and Dobson 1995, Rousset et al. 1996). The empirical power relationship ($s^2 = aM^b$) between mean abundance (M) and variance of mean abundance (s^2) in parasites has been repeatedly used in epidemiology. Although some accounts suggest that values of b could reflect regulation processes in host-parasite systems (Madhavi and Anderson 1985), comparative analyses have rarely attempted to fit data to the power-function relationship (but see Shaw and Dobson 1995). Some parasitological contributions have tried to test the applicability of the core-satellite species hypothesis to host-parasite associations (Bush and Holmes 1986, Dobson 1990, Esch et al. 1990), but they only adapted the terminology of the core (abundant) and satellite (rare) species to parasite data (Dobson 1990, Nee et al. 1991), without any special references to the theory in itself. Further work showed that bimodal distributions predicted by the core-satellite species hypothesis are rarely found in parasitological applications (Nee et al. 1991), questioning the usefulness of this hypothesis in parasitology.

Rejection of the specialisation hypothesis

Our data on parasitic nematodes do not support the specialisation hypothesis which predicts that the degree of specialisation, i.e. host specificity, is correlated either with distribution or abundance. Both average worm abundance and prevalence are independent of host-specificity (see Figs 4 and 5). These results based on a large data set of nematodes across different mammal species contradict two previous investigations based on studies of only a couple of host species and their parasites: the lesser scaup (Bush and Holmes 1986), and two species of voles (Haukisalmi et al. 1988). We see here that for nematode parasites a large proportion of species is either abundant, widespread and generalist or else common, largely distributed and specific. There may be a trade-off between specialisation and generalism for nematodes, and advantages the worms could find for evolving one evolutionary strategy instead of another might depend on something that remains to be discovered and explained more carefully (Poulin 1992, Adamson and Caira 1994)! The practical difficulty hinges now on the fact that parasitological data are still rare, and thus it requires new investigations on large spatial extents in order to test the dispersive behaviour of other groups of parasites.

Rejection of phylogenetic confounding effects

Relatedness of species can seriously bias analysis (Harvey and Pagel 1991). Parasite species are related to one another, and the non-independence of parasite species may influence the statistical tests of most of the relationships and may also alter the patterns of observed epidemiological parameters. Our findings clearly show that parasite relatedness has played no role, at least in our work, in shaping parasite demographic characteristics (i.e. the relationship between prevalence and abundance). Nevertheless, we believe that phylogenetic control is a priori necessary to check for and to get reliable estimates of epidemiological factors.

Sampling model versus epidemiological model

The sampling model was successfully applied to a wide body of animal and plant data (Hanski et al. 1993). However, these studies could not test the validity of the sampling model for parasites because of insufficient data sets. The sampling model we have tested for a large data set of terrestrial mammal nematodes well accounts for the effects of spatial distribution of parasites on abundance values (see Table 1 and Fig. 4). As emphasised by Hanski et al. (1993), this sampling model strongly contributes to the positive relationship between local abundance and spatial distribution. For a given average abundance value, one parasite species may infect more host individuals when either not or poorly aggregated than when strongly clumped. Equivalent predictions were obtained with usual epidemiological models (Anderson and May 1985). In addition, we suggest that the sampling model does not strictly concern a sampling artefact, for which abundant species are more likely to be sampled in patches. It may represent a demographic pattern of parasite populations, in which dynamics of populations interact with spatial and temporal variation in the environment (Anderson et al. 1982, Nee et al. 1991). Hanski and Gyllenberg (1993) and Hanski et al. (1993) have shown that the positive correlation between abundance and distribution would be expected from a simple random process. As pointed out by Brown (1995), it would appear

too easy to conclude that the interspecific relationships between distribution and abundance are trivial consequences of random processes. To a large extent, abundance and distribution reflect the same ecological relationships. The sampling model does not answer the question of what causes the observed degree of clumping. Our results support the notion that both distribution and abundance of parasites are strongly influenced by the stochastic interplay of demographic population characteristics and environmental heterogeneity. Therefore, nematode population characteristics in hosts may be supported by a demographic explanation where average abundance, aggregation/dispersion and spatial parasite distribution within hosts appear to be related by simple relationships.

Our results based on simulation support the demographic hypothesis of species abundance and spatial distribution (Anderson et al. 1982, Nee et al. 1991) because both rare and common species occur at high frequencies, which represents a pattern entirely consistent with a core-satellite distribution of parasites. The number of satellite (rare) nematode species in the environment is higher than that of core (abundant) species, and this pattern of distribution is likely to be linked to the over-representation of so-called tourist species, i.e. non-specific parasites (Nee et al. 1991). Although the commonness of bimodality in parasite data is still questioned (Nee et al. 1991), our simulations demonstrate that this pattern of parasite distribution might be the rule (Fig. 3a). Moreover, the simulations show that the expected abundance-prevalence relationship closely fits the observed relationship (Fig. 3b).

Average worm burden is lognormally distributed supporting previous findings (Shaw and Dobson 1995). The pattern found in parasite communities of single hosts (Dobson 1990) is, hence, also found for parasite nematodes among terrestrial mammals (this study).

Despite the possibility that values of b may differ between species of nematodes, we were unable to test the variability of b between different populations of a given parasite species because only one or a few populations per parasite species was generally available in our data base. Nevertheless, for free-living animals b is not biased by the intra- or inter-species level of investigation (Downing 1986), and analyses of distributions of fish nematodes tend to confirm that intra- and interspecific parasite population comparisons yield similar values of b (Guégan and Morand unpubl.). The range and size of b may vary with the number of samples, and stable and robust estimates ($1 < b < 2$) are usually obtained for large sample sizes ($n > 100$) as in this study (Downing 1986). The value of b we obtained falls within the accepted range of estimates ($1 < b < 2$) for which nematodes are aggregated in their mammal hosts (Taylor 1961). Thus, our data are in good agreement with a demographic model (Anderson et al. 1982). The value of b (without controlling for phylogenetic effects, $b = 1.80$; after controlling, $b = 1.64$) for a single taxonomic group of parasites, i.e. nematodes of terrestrial mammals, is also similar to the value ($b = 1.55$) obtained by Shaw and Dobson (1995) for a large data set of different taxonomic groups of parasites.

Our results have the following implications. First, they suggest that simple relationships can estimate the mean and variability of parasite burdens with up to 96% of the total variance in abundance explained by the average worm abundance.

Second, transmission-mortality models (i.e. epidemiological models) applicable to macroparasites may also account for both a bimodal distribution of prevalence and a positive prevalence-average worm burden relationship. Therefore, it may not be necessary to involve complex mechanisms such as the degree of parasite adaptation (host-specificity) for explaining parasite species patterns in hosts. Rather, there may be a possibility of parasite switching between a satellite state with a more or less clumped distribution in hosts to a core state with a gradually random distribution (Shaw and Dobson 1995). Thus, the core-satellite pattern may eventually break down for host-parasite systems.

Third, the bimodal distribution of nematode species and the power functions may simply be the results of demographic and stochastic patterns due to heterogeneity in probability of infection between hosts, maximum worm burden of hosts (carrying capacities related to host body sizes) and host population sizes. A lognormal distribution of worm burden will arise if worm burden is affected by several factors in a multiplicative fashion (May 1975). The lognormal distribution has been documented for various free-living groups such as birds or plants and reflects statistical properties of the interplay of many independent factors (see also May 1976). Lognormal distributions of worm burden have been observed in specific parasite communities (Dobson 1990). Our findings show that such distributions may be found in both specific and non-specific parasite communities. Additionally, a clumped distribution of parasites among hosts will arise for many reasons such as parasite induced host mortality and heterogeneity in parasite transmission (Anderson and Gordon 1982). All these factors affect the basic reproductive rate of parasites (Dobson 1990) and, consequently, the level and variability of parasitic infection.

Finally, epidemiological characteristics seem to be independent from parasite species phylogeny at least for mammal nematodes. In taking into account the non-independent branching structure of parasite species, this study clearly shows that epidemiology does not seem to be greatly affected by parasite species relatedness.

More interestingly, our data suggest that metapopulation models might be practical tools that could be used to predict the occurrence and variability of parasite species in host populations (see Grenfell and Har-

wood, 1997). The implications for relationships between parasite abundance and distribution are important to be evaluated. How many unparasitized hosts are there since it remains often an open question why a host patch, potentially suitable for a parasite, is empty? First, the host might be entirely suitable but it is free because the parasite population has gone extinct or because it is beyond the dispersal range of any extant parasite population (Guégan and Hugueny 1994). Second, the host is not in fact suitable for colonization in its present state for parasites, e.g. age, body condition, immuno-enhancement. How strong are the consequences of parasite aggregation on host population viability? Furthermore, we can expect that studies on patterns and processes of parasite populations in fragmented landscapes and isolated islands would be of practical interest in the next decades. With these questions, the development of such approaches represents one of the major challenges in general epidemiology.

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