Ecology, 88(7), 2007, pp. 1841–1849

ECOLOGICAL DIFFERENCES AND COEXISTENCE IN A GUILD OF MICROPARASITES: *BARTONELLA* IN WILD RODENTS

Sandra Telfer,[1,2,3] Helen E. Clough,[1,2] Richard J. Birtles,[2] Malcolm Bennett,[2] David Carslake,[1,2] Sarah Helyar,[1] and Michael Begon[1]

[1]*School of Biological Sciences, University of Liverpool, L69 7ZB United Kingdom*
[2]*Department of Veterinary Pathology, Leahurst, University of Liverpool, CH64 7TE United Kingdom*

Abstract. The study of ecological differences among coexisting microparasites has been largely neglected, but it addresses important and unusual issues because there is no clear distinction in such cases between conventional (resource) and apparent competition. Here patterns in the population dynamics are examined for four species of *Bartonella* (bacterial parasites) coexisting in two wild rodent hosts, bank voles (*Clethrionomys glareolus*) and wood mice (*Apodemus sylvaticus*). Using generalized linear modeling and mixed effects models, we examine, for these four species, seasonal patterns and dependencies on host density (both direct and delayed) and, having accounted for these, any differences in prevalence between the two hosts. Whereas previous studies had failed to uncover species differences, here all four were different. Two, *B. doshiae* and *B. taylorii*, were more prevalent in wood mice, and one, *B. birtlesii*, was more prevalent in bank voles. *B. birtlesii, B. grahamii,* and *B. taylorii* peaked in prevalence in the fall, whereas *B. doshiae* peaked in spring. For *B. birtlesii* in bank voles, density dependence was direct, but for *B. taylorii* in wood mice density dependence was delayed. *B. birtlesii* prevalence in wood mice was related to bank vole density. The implications of these differences for species coexistence are discussed.

Key words: Apodemus; *bank vole;* Clethrionomys; *competition; parasite; wood mouse.*

Introduction

While studies of coexisting closely related species are commonplace in ecology generally, there have been very few of parasites (Dobson 1985, Lello et al. 2004; see also Cox 2001 for a general review of "concomitant" infections). Studies of microparasites have been especially rare. This is partly a reflection of a general neglect of parasites by ecologists. But it probably also reflects a common pattern, in which molecular approaches have only recently demonstrated as a complex of different parasite species what was previously described as a single species on the basis of its phenotype (e.g., Baranton et al. 1992, Birtles et al. 1994).

The coexistence of similar microparasite species is of interest beyond a wish simply to establish that processes structuring microparasite communities are similar to those acting on other communities. Most early studies of coexistence addressed issues concerning how, and to what extent, competitors may differ in their exploitation of shared resources; but it has since been recognized that species at the same trophic level may alternatively coexist through apparent competition (Holt 1977), in which prey species differ in their susceptibility to shared predators (and additionally as a result of spatial and/or temporal heterogeneities; see Begon et al. 2006). It appears not to have been noted previously that in the case of parasites, the distinction between resource and apparent competition effectively breaks down. Species are normally affected by resource competition through the trophic level below, and by apparent competition through the trophic level above, but parasites are affected by both through interactions with the same hosts. An individual host, by mounting specific (especially immune) and more general attacks on one parasite species, may make that host wholly or partially unavailable to a second parasite species, decreasing the resources available to the second. Apparent competition reduces resource availability. Thus, investigating coexistence among closely related parasites may pose subtle and previously unstudied questions.

The genus *Bartonella* comprises gram-negative coccobacillary Proteobacteria. They parasitize erythrocytes in a wide range of mammalian species, and several are associated with human or animal disease (Anderson and Neuman 1997, Breitschwerdt and Kordick 2000). Historically, on morphological grounds, a very limited number of species were described, but modern methods have identified increasing numbers, often infecting the same host species (Birtles et al. 1994, Boulouis et al. 2005). Although the transmission mechanisms of bartonellae are not fully understood, arthropod vectors are important (Breitschwerdt and Kordick 2000), and experimental studies have demonstrated that fleas can transmit *Bartonella* spp. between rodents in Britain and Europe (Bown et al. 2004). In a previous, preliminary study of *Bartonella* in two species of woodland rodent in

Manuscript received 13 June 2006; revised 1 December 2006; accepted 6 December 2006. Corresponding Editor: K. Wilson.
[3] E-mail: stelfer@liv.ac.uk

the United Kingdom, *Clethrionomys glareolus*, the bank vole, and *Apodemus sylvaticus*, the wood mouse, several *Bartonella* species were found to infect both (coexisting) species, and mixed infections were also sometimes found (Birtles et al. 2001). Elsewhere, studies have suggested some host specificity among *Bartonella* spp. (Kosoy et al. 1997, 2000), but in general it is clear that different *Bartonella* species can coexist within individual hosts, host species, and geographic sites (Kosoy et al. 1997, 2004, Birtles et al. 2001), though the means by which they do so is unknown.

A key step in addressing this question is to identify differences between the *Bartonella* species: in their prevalence in different hosts, in seasonal patterns, and in their responses to host densities. Here, for the first time, these issues are taken up using longitudinal data from natural populations: those of bank voles and wood mice studied by Birtles et al. (2001). The focus is on patterns in prevalence at the population level. A subsequent analysis will examine questions at the level of the individual host: length of infection, parasite species coexistence within individuals, and other factors.

Specifically, interest concerns a number of aspects of the temporal dynamics of *Bartonella*. First, is there a seasonal pattern in infection probability in either host species? Second, is there evidence of an association between infection probability and host density? To address this question, it is necessary to have taken seasonality into account because it is well known that the densities of these rodent species themselves vary seasonally (Alibhai and Gipps 1985, Flowerdew 1985). It is also important to determine the time delay with which any effect of density operates because, for example, direct and delayed effects of density will reflect very different biological mechanisms (Royama 1992). The typical length of a *Bartonella* infection in both bank voles and wood mice is in the order of two months, (R. Birtles and S. Telfer, *unpublished data*) and therefore infected individuals are likely to have become infected during the previous few months. As it would be biologically naïve to attempt to identify *the* time lag of a density effect, because the underlying mechanisms operate at rates that exhibit natural variation (i.e., an identified time lag indicates only a "central tendency") and densities at nearby time lags are inevitably correlated, we seek simply to distinguish between effects of density that are, biologically, "direct" (density in the previous one to three months being influential), and those that are delayed (density six and/or 12 months ago having the stronger association). Finally, we ask whether either of the host species influences infection prevalence in the other host species, seeking specifically an effect of the density of the alternative species (at a variety of lags).

Methods

Bank voles and wood mice were trapped from April 2001 to May 2003 within a 1-ha plot of mixed woodland in northwest England (53°19′36″ N, 3°3′40″ W). A 10 by 10 grid was marked out with 100 trap stations situated at 10-m intervals, with two Longworth traps at each station. Each site was trapped approximately monthly (primary sessions) for a period of 2–3 days, with traps checked daily (winter) or twice daily (summer). Bedding material and waste was removed from traps containing animals, and they were cleaned with 70% ethanol prior to being reset. Traps were sterilized in an autoclave between primary sessions. All animals were tagged using subcutaneous microchips. On first capture in a primary session, a 20–40 μL blood sample was taken from the tail tip.

DNA extracts were prepared by alkaline digestion (Bown et al. 2003). *Bartonella* DNA was detected using a *Bartonella* genus-specific semi-nested PCR assay (Telfer et al. 2005), in which PCR products from different species are of different sizes (Roux and Raoult 1995). Electrophoretic resolution of PCR products on 3% (mass/volume) agarose gels permitted identification of *Bartonella* species.

To assess whether there are seasonal or host density-related effects on infection probability of the different *Bartonella* species in the two hosts, we combine three approaches, each of which brings different strengths. The first operates at the population level and treats the number of infected animals at a given time as a binomial realization of the number captured in a generalized linear model (McCullagh and Nelder 1989) with a logit link. The binomial model strictly treats observations as independent, but some animals were trapped in more than one session (though 61% were trapped on three occasions or less) and may have been infected on more than one capture. In an attempt to counteract this while maintaining a population-level approach, infection probability at time $t - 1$ was always included as a covariate to explain infection probability at time t. Due to the typical length of infections (see *Introduction*), infection probability at time $t - 1$ was deemed the most appropriate covariate. Thus, we allow for the fact that the same animal might have the same infection in successive sessions. We include the following additional covariates: sinusoidal components to reflect seasonality (e.g., Diggle 1990), and density (both the host and the other species) at a variety of lags (1, 2, 3, 6, and 12 months), with population sizes estimated using the Jolly-Seber method for open populations (Schwarz and Seber 1999; density estimates were also available for the 12 months prior to April 2001). Model selection was performed using the Akaike Information Criterion (AIC), with a decrease in AIC greater than two indicating model superiority (Sakamoto et al. 1986). As a result of the lack of independence, we interpret standard errors from the model cautiously as they may be underestimates.

In a second, supporting analysis we implement a simulation procedure similar to a bootstrap (Efron and Tibshirani 1993). To counteract the problem of repeated

captures, while maintaining a population-level approach, we draw one capture at random from all captures for each animal and fit a GLM to the simulated data. We repeat the process 100 times, and use AICs to compare the "optimal model" (as chosen by the population-based approach on the full data set) with other competing models, assessing for each pair of models the proportion of simulations where the optimal model shows a decrease in AIC greater than two. As repeated captures did not appear in the simulated data sets, the infection probability at time $t - 1$ was excluded from all models. In this analysis, there is an inevitable loss of power brought about by the reduced sample size.

In the final approach, fully respecting the nonindependence of the data, based on covariates found to be significant at the population level we fitted an individual-level (binomial) generalized linear mixed model (GLMM; Venables and Ripley 2002) with an animal-level random effect. As our interest in the present paper is focused on population level patterns and model selection for GLMMs is difficult, we use the individual-level models to strengthen further our findings regarding the association between infection probability and covariates, and for confirming that inference from the population and individual-level models are in broad agreement. All statistical analyses were carried out in the R software package (*available online*)[4] and using the library MASS (Venables and Ripley 2002).

Results

Plots of the estimated densities of bank voles and wood mice throughout the two-year study period (Fig. 1a, b) show clearly that the bank vole population grew appreciably in the second year, whereas the wood mouse population underwent a considerable decline. We identified infections by five Bartonella species: *B. grahamii*, *B. birtlesii*, *B. taylorii*, *B. doshiae* and a *Bartonella* genotype incompletely characterized, but believed to be a new species and subsequently referred to as BGA (Fig. 1d–h). The proportions of tagged bank voles and wood mice positive at least once for each of the species over the study period are shown in Table 1. DNA from more than one species of Bartonella was detected in 5.7% of samples ($n = 172$), indicating that mixed infections were present. Ignoring initially complications introduced by density and seasonal effects, there is evidence that two species were more prevalent in bank voles (*B. grahamii* and *B. birtlesii*), and three were more prevalent in wood mice (*B. taylorii*, *B. doshiae,* and BGA).

For subsequent analyses, we restrict our attention to *B. grahamii*, *B. birtlesii,* and *B. taylorii* in both host species and *B. doshiae* in wood mice only, because *B. doshiae* was rare in bank voles and BGA was rare in both hosts. For each species we determine the extent to which seasonal and/or density effects can be demonstrated. We also revisit apparent differences in prevalence between host species once such effects have been taken into account. For the data sets with greatest power the simulation analyses broadly supported the conclusions from the full data set. Consequently, we only present the results from the full data set population analyses and the individual level analyses, except where the simulations suggested significant difficulties in distinguishing between competing models. AIC values of the most relevant models are quoted in the text. AIC values for all population-level models considered are provided in the Appendix.

B. birtlesii

The optimal model for bank voles includes prevalence at the previous trapping session (positive coefficient), a seasonal effect, and bank vole density at a lag of three months (positive coefficient, AIC = 102.8). Models with densities at lags of two and six months (AICs = 104.4, 103.8) also receive weak support but one with a 12-month lag receives none (AIC = 113.3). Neither is inclusion of wood mouse density supported. Comparable results are found in the individual-level model, and the coefficient for bank vole density three months previously is similar (0.048 ± 0.010 [SE] compared with 0.037 ± 0.011).

For wood mice, the optimal model (AIC = 91.5) includes prevalence at the previous trapping session (positive coefficient) but neither seasonal components nor wood mouse density. It does, however, include a positive effect of bank vole density with a lag of three months. Again, comparable results are found in the individual-level model, and the coefficients for bank vole density with a lag of three months are similar (0.029 ± 0.009 compared with 0.035 ± 0.008).

Fitted values for the two final models are shown in Fig. 2a. The seasonal pattern in bank voles shows an August/September peak and a February/March trough. Throughout most of the study period, in line with initial observations (Table 1), prevalence in bank voles was markedly higher than in wood mice. However, toward the end of the study, following a period when the number of infected bank voles was high, there was a brief period during which prevalence in wood mice was appreciably higher than that in bank voles.

B. grahamii

The optimal model for *B. grahamii* in bank voles includes prevalence at the previous trapping session (positive coefficient) and seasonal variation in infection probability (AIC = 112.1), but there is no support for inclusion of any density effects (typically, AIC is increased to around 114). Inclusion of a seasonal effect but not of density effects is confirmed by the individual-level model.

For *B. grahamii* in wood mice, the optimal model (qualitatively confirmed at the individual level) includes

[4] ⟨http://www.r-project.org⟩

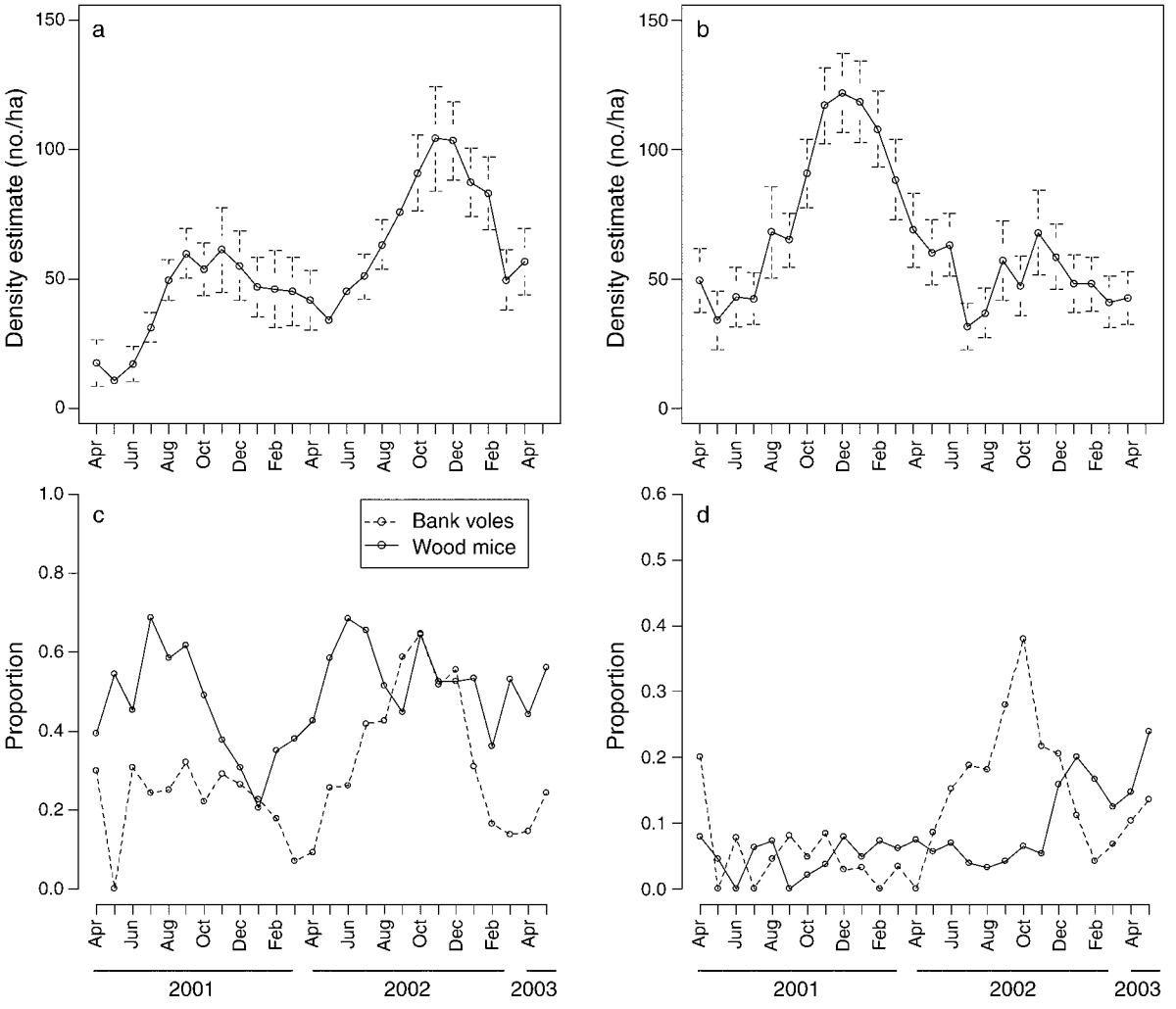

FIG. 1. Estimates of population density, with ±2 SE bars, in (a) bank voles and (b) wood mice, and raw time series data for prevalence of (c) all *Bartonella* species combined, (d) *B. birtlesii*, (e) *B. grahamii*, (f) *B. taylorii*, (g) *B. doshiae* in wood mice only, and (h) the unnamed species BGA in wood mice only.

prevalence at the previous trapping session (positive coefficient), seasonal variation, and a negative association with bank vole density at a lag of six months (AIC = 102.7). However, it is worth noting that a model including a positive association with the density of wood mice in the previous month has an AIC only 2.4 greater (AIC = 105.1), and the simulation analysis based on subsets of the data had difficulty distinguishing between these two models (in only 41% of simulations was the model that included the bank vole density effect better [ΔAIC $>$ 2] than a model with the wood mouse density effect).

Fitted values for the two final models are shown in Fig. 2b. In line with the raw time series (Fig. 1e), there is a clear suggestion that the seasonal peak is extended in bank voles (September/October) compared to wood mice (August/September), and the subsequent trough also occurs later (March/April compared to February/March), whereas the spring increase in prevalence is apparently simultaneous. The consequent prevalence differential between October and March appears to account for the apparently higher prevalence of *B. grahamii* in bank voles than wood mice (Table 1).

TABLE 1. Percentages of tagged bank voles and wood mice that were positive at least once during the study period for each of the five *Bartonella* species.

Bartonella species	Bank voles (n = 346)	Wood mice (n = 405)
B. grahamii	38.7% (n = 134)	25.1% (n = 102)
B. birtlesii	25.4% (n = 88)	16.0% (n = 65)
B. taylorii	17.1% (n = 59)	37.5% (n = 152)
B. doshiae	0.6% (n = 2)	17.5% (n = 71)
BGA	0.6% (n = 2)	7.4% (n = 30)

Note: Two species were more prevalent in bank voles (*B. grahamii*, $\chi^2_1 = 15.26$, $P < 0.0001$; *B. birtlesii*, $\chi^2_1 = 9.56$, $P = 0.002$), and three were more prevalent in wood mice (*B. taylorii*, $\chi^2_1 = 37.73$, $P < 0.00001$; *B. doshiae*, $\chi^2_1 = 59.19$, $P < 0.00001$; BGA, $\chi^2_1 = 19.69$, $P < 0.00001$).

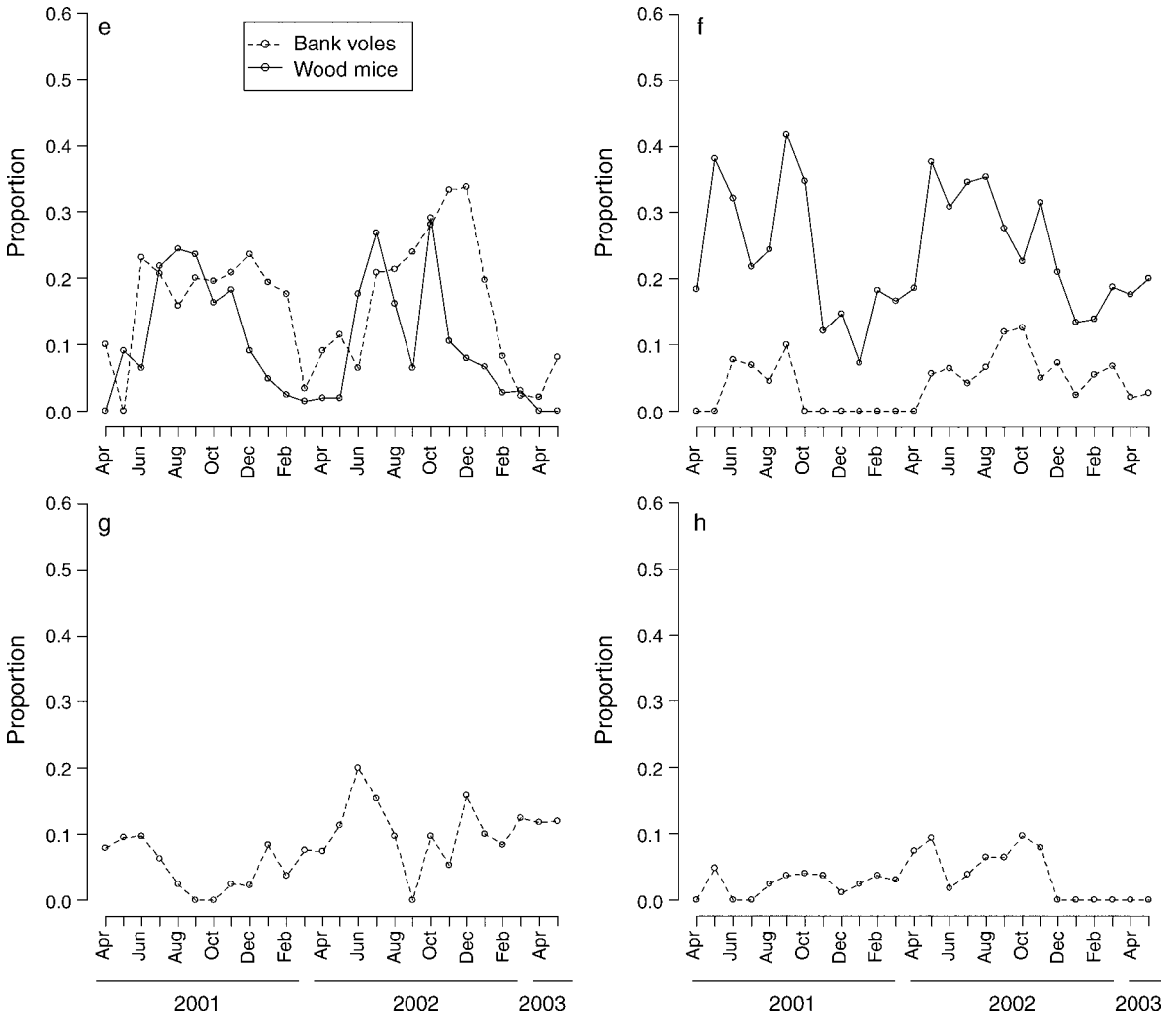

FIG. 1. Extended.

B. taylorii

For *B. taylorii* in bank voles, the optimal population-level model (qualitatively confirmed at the individual level) includes prevalence at the previous trapping session (positive coefficient) and wood mouse density (negative coefficient) with a lag of two months (AIC = 73.1), but there was no additional support for a seasonal component. However, this result should be treated with caution because models including nearby wood mouse density lags (one and three months) were markedly inferior (AIC values of 82.7 and 89.2, respectively). Moreover, in only 26% of simulations was the model including an effect of wood mouse density at a lag of two months better than the null model.

The optimal model for infection probability in wood mice includes prevalence at the previous trapping session (positive coefficient) and a seasonal component, and support is similar for wood mouse densities with a lag of 12 months (AIC = 128.3) and six months (AIC = 128.2). There is no support for densities at shorter lags nor for bank vole densities. These results and the values of coefficients are confirmed at the individual level (12 months, 0.010 ± 0.005 compared with 0.012 ± 0.005; six months, 0.006 ± 0.004 compared with 0.010 ± 0.004).

Fitted values for the two final models are shown in Fig. 2c. The seasonal pattern in wood mice is similar to that for *B. grahamii* in wood mice. Throughout the study period, in line with initial observations (Table 1), prevalence in wood mice was markedly higher than in bank voles.

B. doshiae

For *B. doshiae* in wood mice, the optimal model includes prevalence at the previous trapping session (positive coefficient), a seasonal component, and an effect of bank vole density two months previously (positive coefficient; AIC = 109.6). However, it is worth noting that a model including wood mouse density 12 months previously has an AIC only 2.8 greater. In only

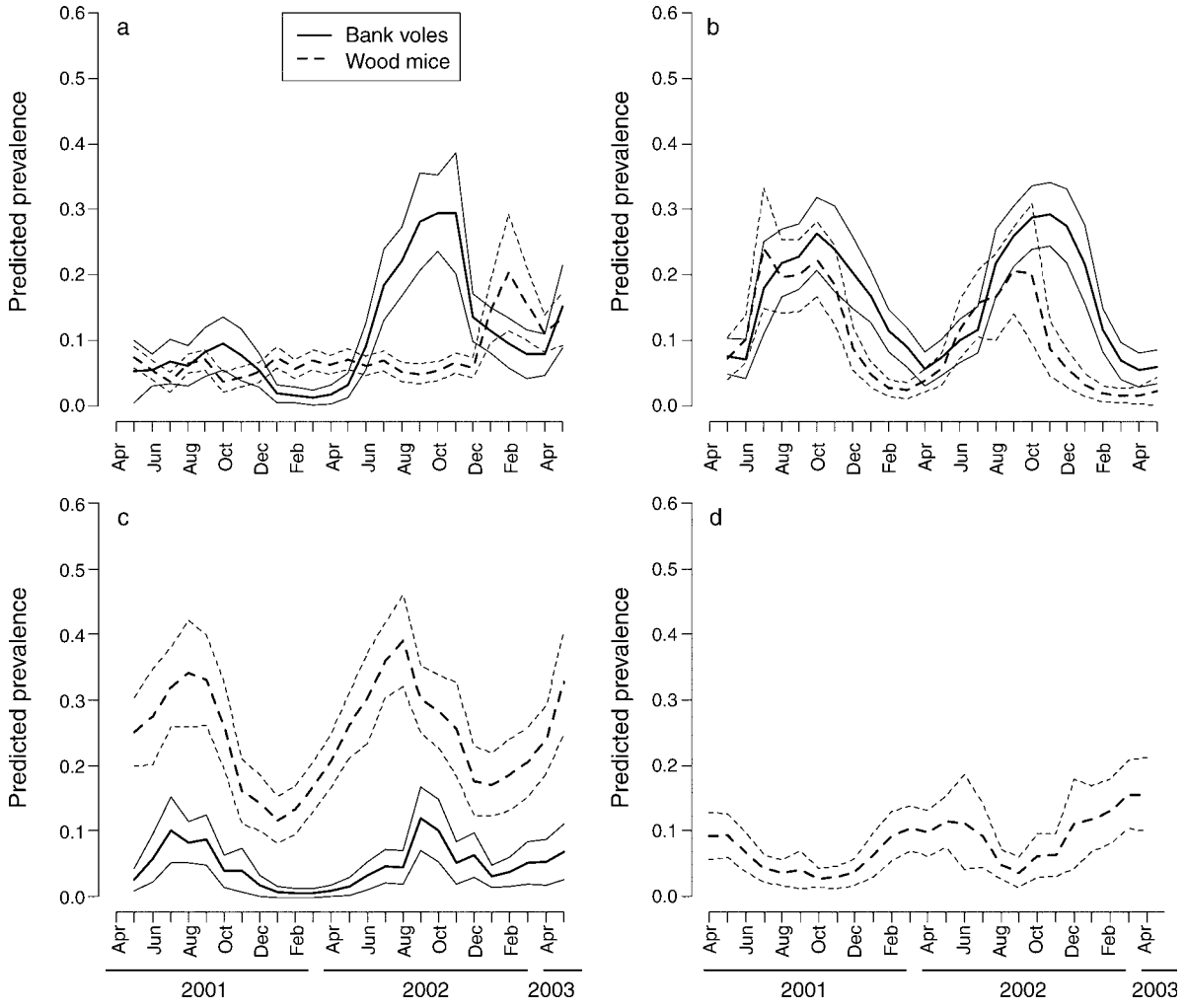

FIG. 2. Predictions of the optimal model, with 95% confidence intervals, for prevalence of (a) *B. bertlesii*, (b) *B. grahamii*, (c) *B. taylorii*, and (d) *B. doshiae* in wood mice only.

5% of simulations was the model including the bank vole density effect better than one including the wood mouse density effect. Moreover, the *B. doshiae* analysis lacks power due to low prevalence; there are strong correlations between bank vole and wood mouse densities ($r = 0.93$); and *B. doshiae* infection is virtually absent in bank voles. Individual level models also qualitatively confirm that distinguishing between an effect of current bank vole density and past wood mouse density is problematic.

Fitted values for the optimal model are shown in Fig. 2d. Note in this case, in contrast to the other *Bartonella* species, that peak prevalence occurs around March with a trough around September.

DISCUSSION

In the study of ecological "guilds"—groups of species exploiting the same class of resources in a similar way (Root 1967)—there has been a common pattern: species that seem ecologically similar initially become increasingly distinct the closer they are examined, suggesting (but not proving) a means by which they might coexist. However, in line with the relative neglect of microparasite communities generally, this pattern seems not to have been observed previously in a guild of microparasites infecting wild animals. In an initial study of our system (Birtles et al. 2001), no differentiation in seasonal pattern, host "preference," or any other feature was apparent. The present study, however, has revealed clear patterns in the distribution and abundance of the various *Bartonella* species and important differences between them in terms of their host specificity, seasonality, and response to host density. We address each of these aspects in turn.

Some of these parasites are consistently more prevalent in one host than the other (though none are found exclusively in only one host). *B. taylorii* and *B. doshiae* (and probably the rare BGA) are more prevalent in wood mice than bank voles, whereas *B. birtlesii* is more prevalent in bank voles. However, the apparently

higher prevalence of *B. grahamii* in bank voles than wood mice is attributable to its fall peak in prevalence being extended for a longer period in bank voles, rather than it ever achieving a higher prevalence; while the pattern for *B. birtlesii* was reversed (prevalence higher in wood mice) for a period toward the end of the study. This emphasizes the necessity of extended studies with repeated sampling, as "snapshots" of host differences in prevalence can clearly be misleading.

In general, a higher *Bartonella* prevalence in one host species than another is likely to reflect either (1) a difference in resistance to infection, or (2) an association between that *Bartonella* species and a species of vector (almost certainly a flea; Bown et al. 2003) coupled with a difference in the prevalence of vector infestation. No data were available on flea infestation rates in the present study, but a previous study at the same site conducted between November and April found four species of flea (*Ctenophthalmus nobilis*, *Amalaraeus penicilliger*, *Rhadinopsylla pentacantha,* and *Hystrichopsylla talpaei*; Bown 1999). However, it is unknown whether these species vary in either their vector competence for different *Bartonella* species or their prevalence on different host species at our site. In an ongoing study at a different site, where field voles (*Microtus agrestis*), bank voles, and wood mice coexist, we are investigating host–vector–*Bartonella* relationships in more detail.

The extended peak in prevalence of *B. grahamii* in bank voles is consistent either with a similar rate of initial infection in the two host species but longer-lived infections in bank voles, or with a longer season of high transmission rates in bank voles. Preliminary investigations of the data found no difference between bank voles and wood mice in the length of *B. grahamii* infections (not shown), but more detailed examination of the data in subsequent analyses will investigate this possibility in detail.

The period of relatively high prevalence of *B. birtlesii* in wood mice coming after a period of high prevalence in bank voles was reflected in the optimal model for prevalence in wood mice including bank vole density at a lag of three months. This suggests "spillover infection" (Power and Mitchell 2004), where infection is maintained in one host species, which is then the main source of infection in a second host species, though spillover infection seems never previously to have been demonstrated directly from time series data. There are two plausible mechanisms: high numbers of infected bank voles may pose a direct threat of infection to wood mice, or high total numbers of bank voles may lead to a high number of shared vectors. Because the selected model for *B. birtlesii* in bank voles included bank vole density (lag 3), either would express itself as an influence of bank vole density on wood mouse prevalence.

Among the *Bartonella* species, two contrasting seasonal patterns were apparent. The more common was shown by *B. grahamii* in both host species, *B. taylorii* in wood mice, and *B. birtlesii* in bank voles: peak prevalence in late summer/fall and the lowest prevalence in late winter/spring. A reversed pattern was shown by *B. doshiae* in wood mice. Variations in seasonal pattern have also been suggested, albeit from twice-yearly (spring and fall) sampling, for *Bartonella* species in field voles in a grassland community of rodents in northern England (Telfer et al. 2007). Again, *B. grahamii* and *B. taylorii* were more prevalent in the fall, and only *B. doshiae* more prevalent in spring. The common pattern fits expectations based on the known biology of rodent *Bartonella* species: prevalence increasing over a period of peak activity of a flea vector (Bown et al. 2003), but declining as flea activity declines because infections are mostly short-lived (Birtles et al. 2001). By contrast, the pattern for *B. doshiae* in wood mice suggests a combination of at least some of the following: increased transmission during the fall and/or winter (due to reliance on a flea species with peak activity during this time or seasonal changes in host behavior that result in significant amounts of transmission by means other than fleas) and long-lived infections, such that these infections continue to accumulate as the cohort of hosts born over the summer ages. Some evidence from another host species indicates that *B. doshiae* infections may indeed be relatively long-lived (R. Birtles and S. Telfer, *unpublished data*). Of 12 field voles captured while infected with *B. doshiae* and taken into the laboratory, 44% were still infected after 10 weeks, compared with $<12\%$ for animals infected with *B. grahamii* ($n = 9$), *B. birtlesii* ($n = 12$), and *B. taylorii* ($n = 12$).

Even after taking seasonal patterns into account, there was, in several cases, a demonstrable effect of host density on prevalence. For *B. birtlesii* in bank voles this effect was direct (densities from the recent past were included in the optimal model), but for *B. taylorii* in wood mice the effect was "delayed" (densities 6–12 months previously were included). There was also some suggestion in wood mice of a direct effect for *B. grahamii* and a delayed effect for *B. doshiae*. Interestingly, the study by Telfer et al. (2007) also found that prevalence of *B. doshiae* (in field voles) showed delayed density dependence. Direct density dependence suggests that transmission rate is itself dependent on host density, the slight "delay" (1–3 months) reflecting the time course of the spread of the infection. Delayed density dependence, on the other hand, suggests a possible role for flea vectors as intermediaries: high host densities leading after a delay to high vector densities and hence high transmission rates.

There was also one further case in which prevalence in one host was clearly associated with the density of the other host (in which the parasite was more prevalent). This positive effect of bank vole density on the prevalence of *B. birtlesii* in wood mice has been discussed already in terms of spillover. Overall, then, we have established that these microparasite species, which were originally indistinguishable morphologically,

and were subsequently distinct molecularly but not ecologically (Birtles et al. 2001), are in fact, on closer examination, distinct ecologically too. As with coexisting guilds of higher organisms, differences between species may reflect the means by which those species coexist, either through resource partitioning or apparent competition, but they may equally have arisen for reasons that have nothing to do with coexistence (Begon et al. 2006). Although this issue appears not to have been addressed previously for microparasites infecting wild animal populations, studies in humans suggest that interactions between malaria species (including interactions mediated by the immune system) are instrumental in allowing coexistence (Bruce et al. 2000, Bruce and Day 2003). Similar processes acting on wild animal microparasite communities may be of both practical importance (because many are of medical, veterinary, or agricultural significance) and difficult to disentangle.

Specifically, the distinction between conventional (resource) and apparent competition in microparasites is not clear-cut. Hosts are a resource for which different parasite species may compete (conventional competition). But equally, the same hosts attack and may eliminate their parasites, and parasites may differ in their ability to withstand these attacks (apparent competition). The *Bartonella* species may compete with one another for resources at the level of the individual (erythrocytes and vascular endothelial cells; Dehio 2005) or the population (susceptible individuals). Coexistence may be favored if they are differentiated in their use of those resources. But equally, a host's immune response to infection by one *Bartonella* species may be effective against other *Bartonella* species (apparent competition). Coexistence may be favored if responses to those attacks are differentiated, especially by virtue of a less than perfect cross-immunity.

The present study was conducted at a single study site and, consequently, some care needs to be taken in interpretation of results. Nonetheless, in the case of the four species examined in detail here, no two showed the same pattern in terms of distribution and dynamics, and hence a basis for coexistence undoubtedly exists. *B. taylorii* and *B. doshiae* were more prevalent in wood mice than bank voles, whereas *B. birtlesii* was more prevalent in bank voles than wood mice. *B. birtlesii*, *B. grahamii,* and *B. taylorii* were all more common in the fall, whereas *B. doshiae* was more common in spring. For *B. birtlesii* in bank voles and possibly *B. grahamii* in wood mice, density dependence was direct, but for both *B. taylorii* and *B. doshiae* in wood mice density dependence was delayed. This puts coexisting Bartonellae in much the same position as coexisting tits (Lack 1971) and warblers (MacArthur 1958) in the 1950s and 1960s. On-going work is aiming to determine how important these (and other) differences actually are for coexistence in this special case of vertebrate microparasites.

Acknowledgments

S. Telfer and H. E. Clough contributed equally to this paper and are "joint first authors." We thank Kevin Bown, Rachel Cavanagh, Trevor Jones, Chris McCracken, and many others for their help in the field and laboratory. Manor Wood was used by permission of Leverhulme Estates. Work was funded by NERC grant GR3/13051 and licensed under home office project licence PPL 40/1813.

Literature Cited

Alibhai, S. K., and J. H. W. Gipps. 1985. The population ecology of bank voles. Symposia of the Zoological Society of London 55:277–305.

Anderson, B. E., and M. A. Neuman. 1997. *Bartonella* spp. as emerging human pathogens. Clinical Microbiology Reviews 10:203–219.

Baranton, G., D. Postic, I. Saint Girons, P. Boerlin, J. C. Piffaretti, M. Assous, and P. A. Grimont. 1992. Delineation of *Borrelia burgdorferi* sensu stricto, *Borrelia garinii* sp. nov., and group VS461 associated with Lyme borreliosis. International Journal of Systematic Bacteriology 42:378–383.

Begon, M., C. R. Townsend, and J. L. Harper. 2006. Ecology. Fourth edition. Blackwell, Oxford, UK.

Birtles, R. J., T. G. Harrison, and D. H. Molyneux. 1994. *Grahamella* in small woodland mammals in the U.K.: isolation, prevalence and host specificity. Annals of Tropical Medicine and Parasitology 88:317–327.

Birtles, R. J., S. M. Hazel, M. Bennett, K. Bown, D. Raoult, and M. Begon. 2001. Longitudinal monitoring of the dynamics of infections due to *Bartonella* species in UK woodland rodents. Journal of Epidemiology and Infection 126:323–329.

Boulouis, H. J., C. C. Chang, J. B. Henn, R. W. Kasten, and B. B. Chomel. 2005. Factors associated with the rapid emergence of zoonotic *Bartonella* infections. Veterinary Research 36:383–410.

Bown, K. J. 1999. Studies on the macro- and microparasites of woodland rodents. Dissertation. University of Liverpool, Liverpool, UK.

Bown, K. J., M. Begon, M. Bennett, Z. Woldehiwet, and N. H. Ogden. 2003. Seasonal dynamics of *Anaplasma phagocytophila* in a rodent-tick (*Ixodes trianguliceps*) system, United Kingdom. Emerging Infectious Diseases 9:63–70.

Bown, K. J., M. Bennett, and M. Begon. 2004. Flea-borne *Bartonella grahamii* and *Bartonella taylorii* in bank voles. Emerging Infectious Diseases 10:684–687.

Breitschwerdt, E. B., and D. L. Kordick. 2000. *Bartonella* infection in animals: carriership, reservoir potential, pathogenicity, and zoonotic potential for human infection. Clinical Microbiology Reviews 13:428–438.

Bruce, M. C., and K. P. Day. 2003. Cross-species regulation of Plasmodium parasitemia in semi-immune children from Papua New Guinea. Trends in Parasitology 19: 271–277.

Bruce, M. C., M. R. Galinski, J. W. Barnwell, C. A. Donnelly, M. Walmsley, M. P. Alpers, D. Walliker, and K. P. Day. 2000. Genetic diversity and dynamics of *Plasmodium falciparum* and *P. vivax* populations in multiply infected children with asymptomatic malaria infections in Papua New Guinea. Parasitology 121:257–272.

Cox, F. E. G. 2001. Concomitant infections, parasites and immune responses. Parasitology 122:S23–S38.

Dehio, C. 2005. *Bartonella*–host-cell interactions and vascular tumour formation. Nature Reviews Microbiology 3:621–631.

Diggle, P. J. 1990. Time series: a biostatistical introduction. Clarendon Press, Oxford, UK.

Dobson, A. 1985. The population dynamics of competition between parasites. Parasitology 91:315–317.

Efron, B., and R. J. Tibshirani. 1993. An introduction to the bootstrap. Chapman and Hall, New York, New York, USA.

Flowerdew, J. R. 1985. The population dynamics of wood mice and yellow-necked mice. Symposia of the Zoological Society of London 55:315–338.

Holt, R. D. 1977. Predation, apparent competition and the structure of prey communities. Theoretical Population Biology 12:197–229.

Kosoy, M., E. Mandel, D. Green, E. Marston, D. Jones, and J. Childs. 2004. Prospective studies of *Bartonella* of rodents. Part II. Diverse infections in a single rodent community. Vector-Borne and Zoonotic Diseases 4:296–305.

Kosoy, M. Y., R. L. Regnery, T. Tzianabos, E. L. Marston, D. C. Jones, D. Green, G. O. Maupin, J. G. Olson, and J. E. Childs. 1997. Distribution, diversity, and host specificity of *Bartonella* in rodents from the southeastern United States. American Journal of Tropical Medicine and Hygiene 57:578–588.

Kosoy, M. Y., E. K. Saito, D. Green, E. L. Marston, D. C. Jones, and J. E. Childs. 2000. Experimental evidence of host specificity of *Bartonella* infection in rodents. Comparative Immunology Microbiology and Infectious Diseases 23:221–238.

Lack, D. 1971. Ecological isolation in birds. Blackwell Scientific, Oxford, UK.

Lello, J., B. Boag, A. Fenton, I. R. Stevenson, and P. J. Hudson. 2004. Competition and mutualism among the gut helminths of a mammalian host. Nature 428:840–844.

MacArthur, R. H. 1958. Population ecology of some warblers of northeastern coniferous forests. Ecology 39:599–619.

McCullagh, P., and J. A. Nelder. 1989. Generalized linear models. Second edition. Chapman and Hall, New York, New York, USA.

Power, A. G., and C. E. Mitchell. 2004. Pathogen spillover in disease epidemics. American Naturalist 164:S79–S89.

Root, R. 1967. The niche exploitation pattern of the blue-grey gnatcatcher. Ecological Monographs 37:317–350.

Roux, V., and D. Raoult. 1995. Inter-species and intraspecies identification of *Bartonella* (Rochalimaea) species. Journal of Clinical Microbiology 33:1573–1579.

Royama, T. 1992. Analytical population dynamics. Chapman and Hall, London, UK.

Sakamoto, Y., M. Ishiguro, and G. Kitagawa. 1986. Akaike information criterion statistics. KTK Scientific Publishers, Tokyo, Japan.

Schwarz, C. J., and G. A. F. Seber. 1999. Estimating animal abundance: review III. Statistical Science 14:427–456.

Telfer, S., M. Begon, M. Bennett, K. J. Bown, S. Burthe, X. Lambin, G. Telford, and R. Birtles. 2007. Contrasting dynamics of *Bartonella* spp. in cyclic field vole populations: the impact of vector and host dynamics. Parasitology 134: 413–425

Telfer, S., K. J. Bown, R. Sekules, M. Begon, T. Hayden, and R. Birtles. 2005. Disruption of a host-parasite system following the introduction of an exotic host species. Parasitology 130:661–668.

Venables, W. N., and B. D. Ripley. 2002. Modern applied statistics with S. Fourth edition. Springer, New York, New York, USA.

APPENDIX

Table of AICs for all models considered (*Ecological Archives* E088-110-A1).