

Species Coexistence and Pathogens with Frequency-Dependent Transmission

Volker H. W. Rudolf* and Janis Antonovics†

Department of Biology, University of Virginia, Charlottesville,
Virginia 22904-4327

Submitted December 14, 2004; Accepted March 17, 2005;
Electronically published April 21, 2005

ABSTRACT: Pathogens that infect multiple hosts are commonly transmitted by vectors, and their transmission rate is often thought to depend on the proportion of hosts or vectors infected (i.e., frequency dependence). A model of a two-host, one-pathogen system with frequency-dependent transmission is used to investigate how sharing a pathogen with an alternative host influences pathogen-mediated extinction. The results show that if there is frequency-dependent transmission, a host can be rescued from pathogen-mediated extinction by the presence of a second host with which it shares a pathogen. The study provides an important conceptual counterexample to the idea that shared pathogens necessarily result in apparent competition by showing that shared pathogens can mediate apparent mutualism. We distinguish two types of dilution effect (pathogen reduction with increasing host diversity), each resulting from different underlying pathogen transmission processes and host density effects. These results have important consequences for understanding the role of pathogens in species interactions and in maintaining host species diversity.

Keywords: multihost systems, community dynamics, cross-species transmission, biodiversity, extinction risk, species coexistence.

Ecologists increasingly appreciate that host-pathogen interactions may greatly influence the dynamics and structure of natural communities. Pathogens can alter competitive interactions between species (e.g., Park 1948; Grosholz 1992; Kiesecker and Blaustein 1999), and they can be crucial for determining the coexistence or exclusion of host species (Anderson and May 1986; McCallum and Dobson 1995). Most pathogens are able to infect multiple species within a community and are commonly transmitted across species (Woolhouse et al. 2001). Recent studies indicate that more than 60% of human diseases may have

a zoonotic origin (Cleaveland et al. 2001; Taylor et al. 2001), and a substantially higher percentage of livestock diseases are probably shared with other wild-ranging hosts (Daszak et al. 2000). Understanding the disease dynamics of such cross-species transmission is an important undertaking in the context of human and wildlife disease (Haydon et al. 2002; Altizer et al. 2003).

There is substantial theory on the dynamics of one-pathogen, two-host systems (Begon et al. 1992; Begon and Bowers 1994; Greenman and Hudson 2000). Most of this theory has been developed on the assumption that transmission is a mass-action phenomenon such that the force of infection (the rate at which uninfected individuals become infected) is proportional to the density of infected individuals in the population (e.g., Holt and Pickering 1985; Begon et al. 1992; Begon and Bowers 1994). In such systems, the probability of uninfected individuals becoming infected is a function of the absolute density of infected individuals within the population (regardless of the number of uninfected individuals). Density-dependent transmission best describes systems in which the pathogen is transmitted through random contact among individuals or by aerial transmission (May and Anderson 1979; Anderson and May 1981).

However, it is increasingly recognized that transmission may often be a function of the frequency (proportion of the population) of infected individuals in a population rather than density. This form of the transmission function most often has been emphasized in the context of sexually transmitted diseases (Getz and Pickering 1983; Thrall et al. 1993; Lockhart et al. 1996). Frequency dependence also provides a reasonable approximation for vector-transmitted diseases when vectors actively search for their hosts and compensate for increased host spacing by increasing their flight distances (Antonovics et al. 1995). Indeed, pathogens that are able to infect multiple hosts are most commonly transmitted by vectors (Woolhouse et al. 2001). Some diseases that are transmitted aerially or by close contact may also have frequency-dependent transmission if the social context creates core groups where the number of interacting individuals is relatively constant (e.g., measles spread within classrooms or disease in mammalian social groups). Under such conditions, transmission functions based on frequency

* Corresponding author; e-mail: vrudolf@virginia.edu.

† E-mail: ja8n@virginia.edu.

dependence rather than density dependence may provide a better fit to observed epidemiological data (Begon et al. 1999; Bjornstad et al. 2002). There is an increasing interest in applying frequency-dependent transmission functions to explain disease occurrence in natural communities (Schmidt and Ostfeld 2001; LoGiudice et al. 2003). Dynamics of disease systems with frequency-dependent transmission differ fundamentally from those with density-dependent transmission (Getz and Pickering 1983; Thrall et al. 1993), yet we are unaware of any investigations that have analyzed the extinction and coexistence dynamics of systems with multiple hosts sharing a pathogen that is transmitted in a frequency-dependent manner.

Here we use a modeling approach to explore how sharing a pathogen that shows frequency-dependent transmission affects host species coexistence. We show that while density-dependent transmission across species often has negative effects on the hosts involved, sharing a pathogen with frequency-dependent transmissions can actually rescue a host from pathogen-mediated extinction under certain conditions. The study therefore provides an important conceptual counterexample to the existing idea that shared pathogens cause apparent competition by showing that shared pathogens can also mediate apparent mutualism. We clarify this idea in the context of the so-called dilution effect (Ostfeld and Keesing 2000).

The Model

The model describes two host populations that share one horizontally transmitted pathogen species. It includes the essential features of a microparasite two-host, one-pathogen system (Holt and Pickering 1985; Begon et al. 1992) but assumes frequency-dependent transmission. In addition, each host population is regulated through intraspecific competition, such that the net reproductive rate of each host declines as a linear function of population size. Getz and Pickering (1983) showed that with frequency-dependent transmission, stable host-pathogen coexistence is only possible when the density of the host population is regulated by factors other than the pathogen. It is important to note that the term “frequency dependent” as used here and in the disease literature (Antonovics and Alexander 1992) refers to the frequency of infected individuals relative to the total population (i.e., infected plus susceptible) as opposed to predator-prey systems, where the term “frequency dependence” refers to the relative frequency of the two prey species.

We used the following equations (and corresponding equations for N_2 and I_2) to describe the change in density

of each host population and of infected individuals in that population:

$$\frac{dN_1}{dt} = N_1 r_1 \left(1 - \frac{N_1}{K_1} \right) - \alpha_1 I_1, \quad (1)$$

$$\frac{dI_1}{dt} = S_1 \beta_{11} \frac{I_1}{N_1 + N_2} + S_1 \beta_{12} \frac{I_2}{N_1 + N_2} - (\alpha_1 + d_1 + v_1) I_1, \quad (2)$$

where N_1 is the total population size of host species 1, I_1 is the number of infected (and also infectious) hosts, and $S_1 = N_1 - I_1$ is the number of susceptible hosts in the population. The growth rate of species 1 is determined by the intrinsic growth rate $r_1 = a_1 - d_1$, where a_1 is the birth rate and d_1 is the death rate, and by intraspecific competition represented as $1 - N_1/K_1$, with K_1 being the carrying capacity. Uninfected individuals can become infected by intraspecific transmission at a rate β_{11} and β_{22} or by interspecific transmission at a rate β_{12} and β_{21} . The pathogen-induced mortality rate is represented by α_1 and recovery rate from disease by v_1 .

Using the models in equations (1) and (2), we set out to answer the specific question of whether the presence of a second host can prevent the elimination of a host-pathogen system that would otherwise go extinct. In all cases we started with an infected population of, say, host species 1, and chose parameters such that the pathogen always led to the extinction of this host (and therefore of itself). We then chose parameters for host species 2 such that when on its own with the pathogen, one of the following three outcomes occurred (Getz and Pickering 1983): the pathogen could not establish ($p/r < 1$); the host and pathogen coexisted ($1 < p/r < \beta/\alpha$, where $p = \beta - [\alpha + d + v]$); or the pathogen drove the system to extinction ($p/r > \beta/\alpha$). We then combined host 1 with all three single-species outcomes of host 2 to generate three different one-pathogen, two-host scenarios and explored how the outcomes differed from the single-species expectations under a range of cross-species transmission coefficients. Interspecific transmission rates (β_{12} , β_{21}) were varied independently, over a range from 0.01 to 0.5, in 0.01 intervals.

For simplicity, we assume that both hosts contribute equally to the total host population and that population carrying capacity of the two populations are equal and independent. Each simulation was run for up to 40,000 generations minimally, assuming a quasi-extinction threshold of 0.001 for both hosts and pathogens; if the number of hosts or infected were below this threshold at

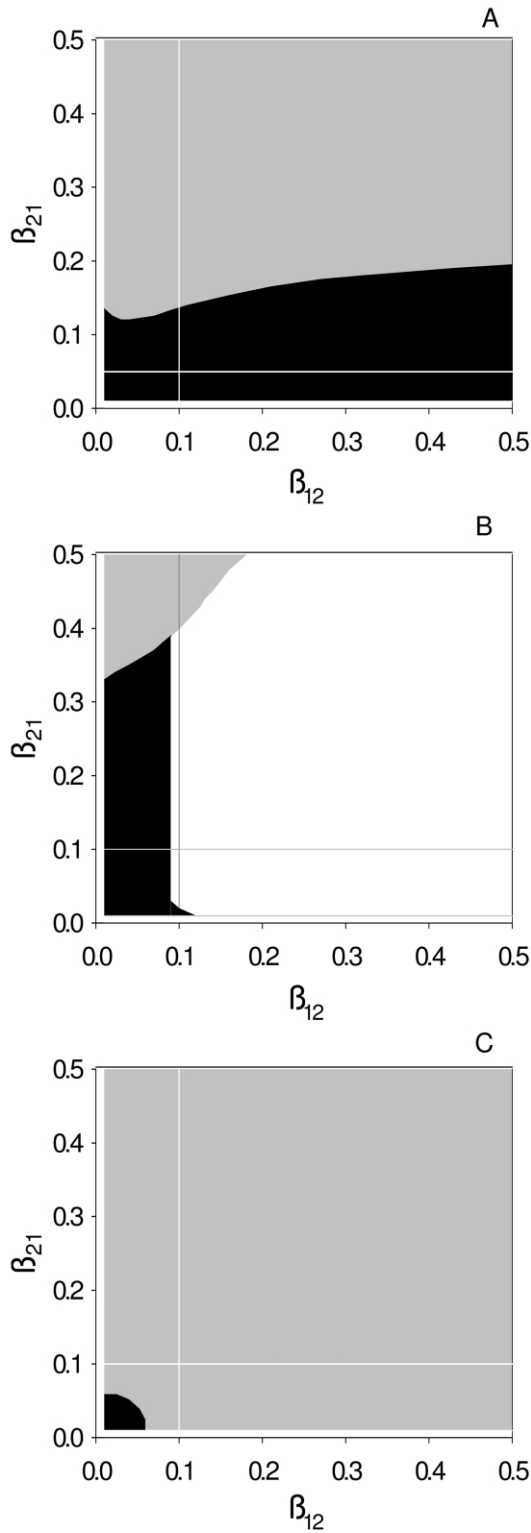


Figure 1: Phase diagrams showing the regions of two-host coexistence (black), elimination of both hosts (gray), and elimination of host 1 only

this time, they were assumed to be extinct. In cases where low-level persistent equilibria were suspected, these were tested by introducing either the host or the pathogen at levels both below and above this threshold. In all models, relatively small parameter values were used for r and α to reduce possible cyclical or chaotic dynamics, and in all cases approach to the equilibrium was asymptotic and not initial value dependent. Simulations programmed independently in Mathematica and Matlab gave identical results, either in continuous or in discrete analog mode.

Results

In all cases, there was a region of parameter space in which host 1 was rescued from extinction by the presence of host 2 (fig. 1). These regions occurred regardless of the single-species host-pathogen relationship in host 2 and occurred most generally when the interspecific transmission coefficients were less than the intraspecific coefficients (represented by the lower left-hand quadrants of the phase diagrams). Given the parameter inequalities investigated, the pathogen always persisted, provided that at least one host was present. Additional simulations indicated that the qualitative aspects of the phase diagrams (i.e., general positions and types of equilibria) did not change for different magnitudes of the coexistence inequalities.

If host 2 could not sustain the pathogen, only two outcomes were possible: coexistence or elimination of both hosts (fig. 1A). Elimination of both hosts occurred when the transmission to host 2 from host 1 (β_{21}) was high; β_{21} reduced the population size of host 2, leading to the elimination of both hosts and the pathogen. However, if β_{21} was low, there were always conditions under which host 1 was protected from extinction by host 2. The reason is that the presence of a resistant host 2 ensures that as the numbers of infected individuals drop in host 1, there will come a point when the overall frequency of infected in-

(white) as a function of the interspecific transmission coefficients. Lines indicate values of intraspecific transmission coefficients for each host. The results are based on a two-host, one-pathogen system with frequency-dependent transmission, in which host 1 (which always becomes eliminated when alone with the pathogen) co-occurs with host 2, where parameters are chosen such that when host 2 is alone with the pathogen: (A), the pathogen is eliminated ($r_2 = 0.01$, $\beta_{22} = 0.05$, $\alpha_2 = 0.05$, $v_2 = 0.05$); (B), there is coexistence of the host and its pathogen ($r_2 = 0.029$, $\beta_{22} = 0.1$, $\alpha_2 = 0.05$, $v_2 = 0.01$), or (C), both the host and the pathogen go extinct ($r_2 = 0.01$, $\beta_{22} = 0.1$, $\alpha_2 = 0.03$, $v_2 = 0.01$). Throughout, $d = 0.01$, $K = 10$, $r_1 = 0.01$, $\beta_{11} = 0.1$, $\alpha_1 = 0.03$, and $v_1 = 0.01$.

dividuals will become sufficiently low to prevent the pathogen from driving host 1 to extinction.

Where the pathogen coexisted with host 2, three outcomes were possible (fig. 1B). In addition to coexistence at low levels of cross-species transmission and host-pathogen elimination at high levels of cross-species transmission, it was still possible for host 1 to go extinct in the presence of host 2 but host 2 to persist. As expected, elimination of host 2 occurred only when β_{21} was high, suggesting that host 1 had a low impact on host 2.

Where the pathogen resulted in extinction when alone with each of the hosts, there were always conditions where there was mutual rescue from extinction if the interspecific transmission rates were sufficiently small (fig. 1C). In contrast to the previous two scenarios, interspecific transmission rates had to be lower than intraspecific transmission rates but not necessarily symmetrically so.

Discussion

Cross-species transmission often is associated with pathogen emergence and therefore has been considered as a negative factor for humans, wildlife, and species of agricultural importance (review in McCallum and Dobson 1995; Woodroffe 1999). Moreover, cross-species transmission is assumed to be a major threat for endangered species because transmission from an abundant, more resistant species to a rarer, vulnerable species can result in extinction of the rarer species (Grosholz 1992; Rushton et al. 2000; LoGiudice 2003). The indirect negative effect of sharing a pathogen with another species has been termed apparent competition (Holt and Pickering 1985). Here, we demonstrate that with frequency-dependent transmission, sharing a pathogen with another species can be beneficial for one or both of the host species involved.

We show that pathogen incidence can be reduced such that, under some conditions, coexistence of two species is possible when they share a pathogen, even though the pathogen would cause the extinction of either species if it were alone with the pathogen. Adding an alternative host decreases the frequency of infected individuals in the total population of hosts (host 1 + host 2), thereby reducing the rate of intraspecific transmission. When this reduction in intraspecific force of infection is not exceeded by additional infection that occurs through cross-species transmission, adding a second host will reduce the total rate of infection. This illustrates that when transmission events are frequency dependent, a pathogen can generate apparent mutualism. This is because, under frequency-dependent transmission, sharing a pathogen among one or more alternative species may actually be limiting the

spread of the pathogen in any one particular host population. Similar results were obtained by a recent study by Dobson (2004) on disease prevalence in multihost systems. Thus, elimination of one or more of these host species, or even a reduction in the population size of one of the species (e.g., through human impact) could result in a cascading effect that leads to the increase of the pathogen in another species and perhaps to its extinction. Therefore, an epidemic on one species may result because of a contextual change in the community without any numerical or genetic change in the host or pathogen. Studying only the target species or the abiotic environment would not reveal the cause of the epidemic.

The term “dilution effect” has been used in a phenomenological sense to describe the situation when there is a decrease in disease frequency with increasing host diversity (Schmidt and Ostfeld 2001; Mitchell et al. 2002; LoGiudice et al. 2003). However, this decrease in disease frequency can occur by two very different mechanisms that often have been confounded. One mechanism, which we term density-dependent dilution, occurs when a decrease in disease incidence does not occur because of an increase in species richness per se but only happens if there is a corresponding decrease in the abundance of the individual species (fig. 2). The other mechanism, which we term frequency-dependent dilution, occurs when there is frequency-dependent transmission, as in our study. Here the decrease in disease incidence is the result of a direct increase in biodiversity and is not caused by changes in the abundance of the individual species. It would occur even if adding species increased the total number of individuals in the community. In contrast, with density-



	<i>Frequency-dependent transmission</i>	<i>Density-dependent transmission</i>
<i>Additive</i> 	less disease	more disease
<i>Substitutive</i> 	less disease	less disease

Figure 2: Effect of transmission mode on disease levels in a target (*shaded*) population following the addition of a second (*unshaded*) species. The outcome in the bottom right-hand panel represents density-dependent dilution, and outcomes in the two left-hand panels represent frequency-dependent dilution. In the former case, we assume that the interspecific disease transmission is less than intraspecific transmission.

dependent transmission this situation would in all cases result in an increase in disease incidence (given that interspecific transmission is not zero). Both dilution mechanisms are facilitated if among-species transmission is less than within-species transmission. This is likely since most pathogens show some degree of physiological specificity and because hosts differ in behavior, size (Davies et al. 1991), and habitat preference (Ezenwa 2003). However, surprisingly, regions of coexistence also sometimes occurred when both interspecific transmission rates (β_{12}, β_{21}) were higher than both intraspecific transmission rates (e.g., fig. 1A, *black part of top right-hand quadrant*). This cannot happen if the model parameters are identical for the two hosts (i.e., fig 1C), but it can occur in specific host combinations when the infection, recovery, and pathogen-induced mortality rates (and hence population equilibria) are asymmetrical. For example, with the parameters in figure 1A, one of the hosts (host 1) is driven to extinction while in the other host (host 2) the equilibrium is a low disease prevalence but with large population size. In that case, host 1 can tolerate higher interspecific infection rates because there are few infected individuals in host 2 relative to the total number of host individuals. Host 2 can tolerate higher interspecific infection rates because there are few infected individuals in host 1 (because of low total population size) relative to total number of hosts and because host 2 is very resistant to infections.

We know of only one study, by Begon et al. (1999), that has attempted to gauge the functional form of interspecific transmission coefficient between wildlife host species. In their study, within- and between-species transmission were best explained by models with frequency-dependent transmission terms (but, significantly, only within species). Our theoretical results show the pressing need for empirical studies on the functional form of interspecific transmission processes in multihost pathogen systems. Only such studies can address the fundamental mechanisms underlying the dilution effect and thereby explain how species diversity interacts with pathogen prevalence and, thereby, ecosystem functioning.

Frequency-dependent models might be best suited for describing systems with vector-based transmission, and most pathogens that infect multiple host species are vector transmitted (Woolhouse et al. 2001). Zoonotic pathogens like those that cause monkey and bird malaria, Lyme disease, or West Nile encephalitis are often transmitted across species by biting arthropods (Van Riper et al. 1986; Davies et al. 1991; Lee et al. 2001). In plant systems, pollinators are common vectors that transfer floral pathogens across species (Antonovics et al. 2002). In vector-transmitted human diseases it also has been observed that vectors such as mosquitoes can be attracted to alternative nonhuman hosts such as cattle or dogs that are not infectious. This

has led to the proposal that the introduction of these alternative noninfectious hosts can reduce the incidence of disease transmission to humans, a process that has been given the name “zooprophylaxis” (Hess and Hayes 1970). In zooprophylaxis, disease reduction is achieved solely by reducing the contact rate between humans by adding an alternative noninfectious host and not through their interaction over generations, as in our model.

The assumptions underlying our model are conservative. They represent a situation in which coexistence of the pathogen and its host are the least likely outcome in a single host-pathogen system (Thrall et al. 1993). To test the generality of our results, we explored a system in which the pathogen does not affect survival but causes sterility of the infected host (Thrall et al. 1993). Again, the frequency-dependent dilution effect allowed the coexistence of both species and the pathogen, supporting the generality of our model. Thus, our results may also be applicable to other less restricted systems.

Vector-based systems are diverse, and the model structure we have used is in many ways a simplification. For example, vector preferences and their effectiveness in transmission have been shown to be critically important in understanding the dynamics of Lyme disease in sylvatic communities (Schmidt and Ostfeld 2001; LoGiudice et al. 2003). Clearly, the biological particulars of any host-pathogen system would change the range of values allowing for coexistence but would not change the qualitative results of our model. It has been pointed out that vector-based transmission is likely to be a more complex function of host density and pathogen frequency because transmission involves a two-stage pathogen loading and unloading process (Anderson 1981; Antonovics et al. 1995). It is also likely that vectors may become limiting at high host densities, such that per capita transmission rates actually fall; there is experimental evidence for this effect in a pollinator-transmitted disease (Antonovics and Alexander 1992; Antonovics et al. 1995). In many host-pathogen systems, the functional form of the intra- and interspecific transmission functions may be different. For example, in a sexually transmitted disease that is shared between two species, the cross-species transmission is likely to be via an alternative, nonsexual transmission mode (Lockhart et al. 1996). It is also possible that species directly compete for a common resource with each other (Park 1948; Grosholz 1992), which could have additional effects on the population dynamics. Clearly, the effects of these and other complications of vector-based transmission would need to be addressed in any real-world system.

In conclusion, our study shows that whether transmission is frequency or density dependent has to be considered an important factor when interpreting correlations between community composition and pathogen preva-

lence. However, while the theoretical issues are resolved relatively easily for specific cases, very few empirical studies have attempted to estimate the form of the transmission functions for any particular host-pathogen system, through either experiment or model fitting. This remains a major challenge for the future.

Acknowledgments

We are grateful to A. E. Dunham for providing critical comments on an earlier version of the manuscript. We wish to thank the National Institutes of Health (grant GM 60766-01) for financial support.

Literature Cited

- Altizer, S., D. Harvell, and E. Friedle. 2003. Rapid evolutionary dynamics and disease threats to biodiversity. *Trends in Ecology & Evolution* 18:589–596.
- Anderson, R. M. 1981. Population dynamics of indirectly transmitted disease agents: the vector component. Pages 14–43 in K. Maramorosch, ed. *Vectors of disease agents: interactions with plants, animals, and man*. Praeger, New York.
- Anderson, R. M., and R. M. May. 1981. The population dynamics of microparasites and their invertebrate hosts. *Philosophical Transactions of the Royal Society of London B* 291:451–524.
- . 1986. The invasion, persistence and spread of infectious diseases within animal and plant communities. *Philosophical Transactions of the Royal Society of London B* 314:533–568.
- Antonovics, J., and H. M. Alexander. 1992. Epidemiology of anther-smut infection of *Silene alba* (= *S. latifolia*) caused by *Ustilago violacea*: patterns of spore deposition in experimental populations. *Proceedings of the Royal Society of London B* 250:157–163.
- Antonovics, J., Y. Iwasa, and M. P. Hassell. 1995. A generalized model of parasitoid, venereal, and vector-based transmission processes. *American Naturalist* 145:661–675.
- Antonovics, J., M. Hood, and J. Partain. 2002. The ecology and genetics of a host shift: *Microbotryum* as a model system. *American Naturalist* 160(suppl.):S40–S53.
- Begon, M., and R. G. Bowers. 1994. Host-host-pathogen models and microbial pest-control: the effect of host self-regulation. *Journal of Theoretical Biology* 169:275–287.
- Begon, M., R. G. Bowers, N. Kadianakis, and D. E. Hodgkinson. 1992. Disease and community structure: the importance of host self-regulation in a host-host-pathogen model. *American Naturalist* 139:1131–1150.
- Begon, M., S. M. Hazel, D. Baxby, K. Bown, R. Cavanagh, J. Chantrey, T. Jones, and M. Bennett. 1999. Transmission dynamics of a zoonotic pathogen within and between wildlife host species. *Proceedings of the Royal Society of London B* 266:1939–1945.
- Bjornstad, O. N., B. F. Finkenstadt, and B. T. Grenfell. 2002. Dynamics of measles epidemics: estimating scaling of transmission rates using a time series SIR model. *Ecological Monographs* 72: 169–184.
- Cleaveland, S., M. K. Laurenson, and L. H. Taylor. 2001. Diseases of humans and their domestic mammals: pathogen characteristics, host range and the risk of emergence. *Philosophical Transactions of the Royal Society of London B* 356:991–999.
- Daszak, P., A. A. Cunningham, and A. D. Hyatt. 2000. Wildlife ecology: emerging infectious diseases of wildlife; threats to biodiversity and human health. *Science* 287:443–449.
- Davies, C. R., J. M. Ayres, C. Dye, and L. M. Deane. 1991. Malaria infection-rate of amazonian primates increases with body-weight and group-size. *Functional Ecology* 5:655–662.
- Dobson, A. 2004. Population dynamics of pathogens with multiple host species. *American Naturalist* 164(suppl.):S64–S78.
- Ezenwa, V. O. 2003. Habitat overlap and gastrointestinal parasitism in sympatric African bovids. *Parasitology* 126:379–388.
- Getz, W. M., and J. Pickering. 1983. Epidemic models: thresholds and population regulation. *American Naturalist* 121:892–898.
- Greenman, J. V., and P. J. Hudson. 2000. Parasite-mediated and direct competition in a two-host shared macroparasite system. *Theoretical Population Biology* 57:13–34.
- Grosholz, E. D. 1992. Interactions of intraspecific, interspecific, and apparent competition with host pathogen population-dynamics. *Ecology* 73:507–514.
- Haydon, D. T., S. Cleaveland, L. H. Taylor, and M. K. Laurenson. 2002. Identifying reservoirs of infection: a conceptual and practical challenge. *Emerging Infectious Diseases* 8:1468–1473.
- Hess, A. D., and R. O. Hayes. 1970. Relative potentials of domestic animals for zoonophylaxis against mosquito vectors of encephalitis. *American Journal of Medicine and Hygiene* 19:327–334.
- Holt, R. D., and J. Pickering. 1985. Infectious disease and species coexistence: a model of Lotka-Volterra form. *American Naturalist* 126:196–211.
- Kiesecker, J. M., and A. R. Blaustein. 1999. Pathogen reverses competition between larval amphibians. *Ecology* 80:2442–2448.
- Lee, H.-I., J.-S. Lee, E.-H. Shin, W.-J. Lee, Y.-Y. Kim, and K.-R. Lee. 2001. Malaria transmission potential by *Anopheles sinensis* in the Republic of Korea. *Korean Journal of Parasitology* 39:1–10.
- Lockhart, A. B., P. H. Thrall, and J. Antonovics. 1996. Sexually transmitted diseases in animals: ecological and evolutionary implications. *Biological Reviews of the Cambridge Philosophical Society* 71:415–471.
- LoGiudice, K. 2003. Trophically transmitted parasites and the conservation of small populations: raccoon roundworm and the imperiled Allegheny woodrat. *Conservation Biology* 17:258–266.
- LoGiudice, K., R. S. Ostfeld, K. A. Schmidt, and F. Keasing. 2003. The ecology of infectious disease: effects of host diversity and community composition on Lyme disease risk. *Proceedings of the National Academy of Sciences of the USA* 100:567–571.
- May, R. M., and R. M. Anderson. 1979. Population biology of infectious diseases. *Nature* 280:455–461.
- McCallum, H., and A. Dobson. 1995. Detecting disease and parasite threats to endangered species and ecosystems. *Trends in Ecology & Evolution* 10:190–194.
- Mitchell, C. E., D. Tilman, and J. V. Groth. 2002. Effects of grassland plant species diversity, abundance, and composition on foliar fungal disease. *Ecology* 83:1713–1726.
- Ostfeld, R. S., and F. Keasing. 2000. Biodiversity and disease risk: the case of Lyme disease. *Conservation Biology* 14:722–728.
- Park, T. 1948. Interspecies competition in populations of *Tribolium confusum* Duval and *Tribolium castaneum* Herbst. *Ecological Monographs* 18:265–307.
- Rushton, S. P., P. W. W. Lurz, J. Gurnell, and R. Fuller. 2000. Modelling the spatial dynamics of parapoxvirus disease in red and grey

- squirrels: a possible cause of the decline in the red squirrel in the UK? *Journal of Applied Ecology* 37:997–1012.
- Schmidt, K. A., and R. S. Ostfeld. 2001. Biodiversity and the dilution effect in disease ecology. *Ecology* 82:609–619.
- Taylor, L. H., S. M. Latham, and M. E. J. Woolhouse. 2001. Risk factors for human disease emergence. *Philosophical Transactions of the Royal Society of London B* 356:983–989.
- Thrall, P. H., J. Antonovics, and D. W. Hall. 1993. Host and pathogen coexistence in sexually transmitted and vector-borne diseases characterized by frequency-dependent disease transmission. *American Naturalist* 142:543–552.
- Van Riper, C. I., S. G. Van Riper, M. L. Goff, and M. Laird. 1986. The epizootiology and ecological significance of malaria in Hawaiian land birds. *Ecological Monographs* 56:327–344.
- Woodroffe, R. 1999. Managing disease threats to wild mammals. *Animal Conservation* 2:185–193.
- Woolhouse, M. J., L. H. Taylor, and D. T. Haydon. 2001. Population biology of multihost pathogens. *Science* 292:1109–1112.

Associate Editor: Michel Loreau
Editor: Donald L. DeAngelis