CHAPTER 2

Extending the principles of community ecology to address the epidemiology of host–pathogen systems

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2.1 Background

Community ecologists grapple with the structure and dynamics of ensembles of species that live in the same habitat, landscape, or region, and so potentially interact (Morin 1999; Lawton 2000). This concern with interspecific interactions has led to sustained interest in several questions. One is to understand how species within a single trophic level, competing for the same resources, manage to coexist (Chesson 2000a; Holt 2001). Other central concerns of community ecology go beyond coexistence within a trophic level to include topics such as understanding the structure and dynamics of food webs, and the relationship between diversity and ecosystem stability (e.g. McCann 2000). In addressing these issues, including the core issue of coexistence, parasites are increasingly recognized as "hidden" but vital constituents of natural communities (Morand and Arias-Gonzalez 1997; Thompson *et al.* 2001). In turn, there is a growing appreciation of the community dimensions of infectious disease epidemiology, as witnessed by the chapters of this volume.

In applied ecology, understanding the community context of infectious disease is critically important (e.g. zoonotic diseases, Ostfeld *et al.* 2001 and Chapter 3, this volume; invasive species, Mitchell and Power 2003; conservation, McCallum and Dobson 1995; Woodroffe 1999; Lafferty and Gerber 2002; Torchin *et al.* 2003). Such understanding can improve our ability to interpret and mitigate the emergence of novel infectious diseases (Daszak *et al.* 2000; Woolhouse 2002). Community structure can influence disease emergence in many ways. Parasites can infect multiple host species, and most hosts are vulnerable to infection by multiple parasite species (Dobson and Foufopoulos 2001, Morgan *et al.* 2004). Even specialist host–pathogen interactions are embedded in complex food webs, generating complex feedbacks. For instance, a generalist predator with a nonlinear functional response to a prey species that itself harbors a specialist parasite can lead to cyclic or chaotic patterns of disease prevalence (Hall *et al.* 2004; Holt, in press; Holt *et al.*, in review). Maintenance of natural host–parasite dynamics may be crucial for maintaining species diversity and facilitating successional dynamics (Gilbert 2002); anthropogenic disruption (e.g. species introductions) can potentially lead to cascading shifts in host–parasite interactions, with devastating effects on community structure.

A key issue at the interface of community ecology and infectious disease epidemiology is how the interdependence of hosts and parasites affects species coexistence. Many processes can influence coexistence, often in idiosyncratic ways, yet coexistence is most broadly understood as arising from the interplay of three factors (Chesson 2000a; Holt 2001): (1) the inherent properties of the species themselves (e.g. feeding specializations), (2) properties of the extrinsic environment (e.g. abundance of food resources), and (3) the dynamic impacts each species in turn has upon the environment (e.g. influence of feeding on future food resources). Trade-offs between species are usually required for coexistence (Kneitel and Chase 2004). For instance, consider two consumer species competing via exploitation for

two discrete, renewable food resources (see, for example, Chase and Leibold 2003). For stable competitive coexistence, where each species can increase when rare, the two species have to differ in terms of their responses to resource availability—an example of niche differentiation. Moreover, the two resources in turn need to respond differently to the two consuming species, such that the two consumer species experience distinct feedback effects arising from their impacts upon the resource supply. Finally, the abundance and renewal rates of the resources cannot be too different; otherwise, despite the existence of niche differences, one species will be able to exclude the other.

When analyzing coexistence, it is often insufficient to consider just pairs of species and their required resources in a local environment over short timescales. Interactions with other trophic levels can permit coexistence (as in keystone predation on competing species, Holt *et al.* 1994), or preclude it (as in apparent competition between prey species, Holt 1977; Holt and Lawton 1994). Noncompetitive interactions such as facilitation can moderate the impact of competition. Spatial flows of resources and species are often central to maintaining species coexistence at broader spatial scales (e.g. Holt 1993; Leibold and Miller 2004), as is temporal variation in resource availability or environmental conditions, when species have different responses to such variation (Chesson 2000a). Increasing the temporal, spatial, and trophic scales of inquiry broadens the range of trade-offs, environments, and feedbacks that can permit (or preclude) coexistence (Chesson 2000a; Holt 2001).

All of these questions in community ecology bear on the issue of understanding the persistence of multispecies assemblages of parasites and their hosts. This is a broad and rapidly evolving topic (see prior syntheses in Grenfell and Dobson 1995; Hudson *et al.* 2002). Because of the large numbers of species in communities, and the complexity and fluidity of the interaction webs that link these species, understanding community dynamics is a substantial challenge, even ignoring parasites and infectious disease! One fruitful approach to unraveling the dynamics of complex food webs is the analysis of "community modules" (Holt 1997a; Persson 1999). A community module is a carefully chosen multispecies extension of pairwise interactions, chosen because the configuration of interactions is found in a wide range of species assemblages. Several familiar modules at the heart of community ecology are shown in Fig. 2.1. Figure 2.2 displays similar community modules involving infectious diseases. The community modules that have received greatest attention are those elucidating the coexistence of species competing for resources. Because hosts provide resources, habitats, and dispersal mechanisms for parasites, models from classical community ecology can be used to elucidate controls on parasite community structure. Toward this end, we will consider here several models based on simple epidemiological modules that address coexistence. We first focus on coexistence of parasites, then turn to the issue of how parasites influence host coexistence.

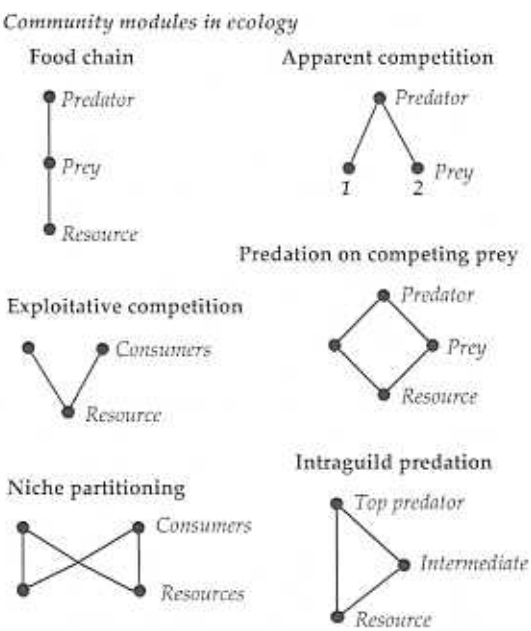

Figure 2.1 Multispecies assemblages involving a relatively small number of species interacting in a defined way provide a useful conceptual waystation between pairwise interactions and highly complex food webs (after Holt 1997a). Note that some of these community modules may go by different names depending on the outcome of interactions; for example, predation on competing prey may be referred to as "keystone predation" if it facilitates prey coexistence, or "predator-mediated competition" if it excludes one prey species (resulting in a "food chain").

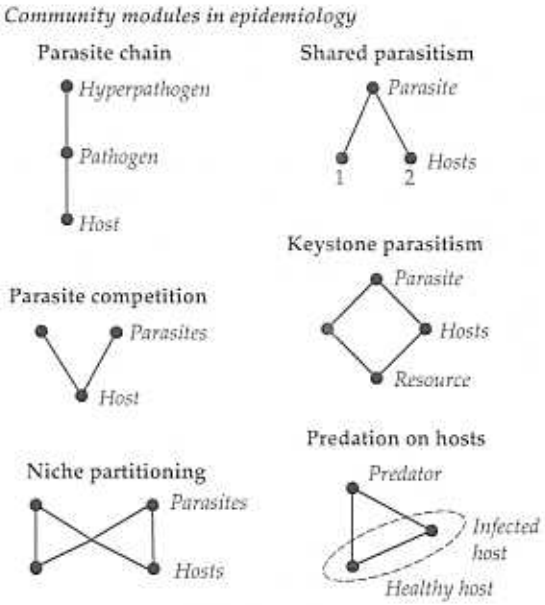

Figure 2.2 Many problems at the interface between community ecology and infectious disease epidemiology can be viewed as modules analogous to those in Fig. 2.1.

2.2 The host community: a templet for the parasite community

A theme of growing interest (and the focus of several chapters in this book) is that parasites can be key determinants of host population dynamics and community organization (e.g. Anderson and May 1978; Hudson *et al.* 1998; Dobson 1999; Hudson *et al.* 2002). For many ecologists (including ourselves), the dramatic impacts of parasites upon their hosts provide the principal "*raison d'être*" for studies of parasite–host ecology. However, for some purposes it may be useful to consider the host community as an arena in which parasite dynamics and interactions play out, without a significant reciprocal affect on host community structure. In the felicitous phrase of Jaenike and Perlman (2002), at times parasites are only a kind of "trophic garnish" on the food web.

The "trophic garnish" perspective on parasites may approximate reality in many cases. Human hosts, for instance, harbor large numbers (at least thousands) of microbial species, mostly bacteria (B. Bohannan and M. McFall-Ngai, personal communication). Some bacteria are always pathogenic, and others can be pathogenic in particular circumstances (e.g. due to wounding or stress), but it is surely true that many of these potential parasites usually have only a negligible impact on their human hosts, in effect being commensals that do not impact host fitness (Levin and Antia 2001). Similarly, nematodes in *Drosophila* may have negligible effects on host fecundity (Perlman and Jaenike 2003). Competitive interactions within individual hosts (e.g. chemically mediated interference among nematodes) can provide density dependence that regulates parasites, even if host fecundity and mortality are not affected (Jaenike 1998; Poulin 1998). Density-dependent resource competition or facultative host defenses may reduce parasite survival or reproduction, without altering host demography (Shostak and Scott 1993; Jaenike 1996). An assumption of fixed host numbers is often made in classical human epidemiology, such as the Kermack–McKendrick (1927) model; such models in effect assume that the combined determinants of host abundance are effectively independent of pathogen impacts. Even harmful pathogens may fail to influence host population size if host population growth is density-dependent. For instance, "damping disease" may reduce seedling survival in a plant cohort (Augspurger 1983), but if the number of survivors exceeds the number of adult plants that can be sustained on a site, the overall course of plant population dynamics may be unchanged. Finally, many parasites that can in principle regulate host numbers do so only in certain contexts (e.g. where hosts experience stressful conditions, Brown *et al.* 2003; Lafferty and Holt 2003).

By assuming that total host numbers are fixed, independent of parasite load, one can identify key mechanisms of coexistence acting primarily on the parasites. Once understood, these mechanisms provide a useful yardstick for interpreting the consequences of relaxing the assumption of fixed host abundances. Given that the host community provides a templet for parasite dynamics, one can then ask how the properties of this templet influence patterns of parasite community organization and richness. This broad question is the focus of much work in classical parasitology (reviewed in Price 1980; Poulin 1998), but we suggest it would be useful to revisit this question more specifically using the conceptual framework

provided by contemporary community ecology. Here we sketch some potentially fruitful applications of community ecology, using simple, illustrative models.

As noted above, a fundamental concern of community ecology is to understand how the strength and pattern of interspecific interactions influence species coexistence. Classical community ecology focused on equilibrial properties of species interactions in closed ecosystems in stable environments. If each of a set of consumer species is limited by the abundance of a single, depletable, limiting factor (as in the "exploitative competition" module of Fig. 2.1), then at equilibrium at most one species is expected to persist (Levin 1970). For instance, in a well-mixed chemostat where algae compete for a single nutrient, theory predicts (and experiments confirm) that a single species of algae dominates, driving inferior competitors to extinction (Tilman 1982; Grover 1997). If multiple species stably coexist, one or more of the conditions required for competitive exclusion must be violated (Chesson 2000a; Holt 2001). We can categorize mechanisms of coexistence in terms of how they differ from this simple limiting case. Box 2.1 provides one typology of coexistence mechanisms.

Box 2.1 A précis of mechanisms of coexistence

Several key mechanisms of coexistence have been identified by community ecologists. These mechanisms depend generally on differences in how species exploit resources, and how the environment responds to such exploitation (Chesson 2000a; Holt 2001). In practice, these mechanisms are neither sharply defined nor mutually exclusive. See the text for further exploration of several scenarios of coexistence among competing parasites.

A. Coexistence in closed, equilibrial communities

Some systems (e.g. terrestrial communities on remote islands) are essentially closed to immigration, and may experience little temporal variation. Much of classical community theory assumes such closed, equilibrial communities.

Classical niche partitioning. In a stable environment with populations at equilibrium, species coexistence requires niche partitioning (e.g. Schoener 1989). Models of niche partitioning assume: (1) the environment is heterogeneous, with multiple potential limiting factors (e.g. a heterogeneous resource base), and (2) species have different requirements (e.g. due to trade-offs in exploitative ability for distinct resources). Such niche differences are necessary but not sufficient for coexistence; species must also be similar in how they respond to general abiotic factors (the "equalizing" conditions of Chesson 2000a), and have differential impacts upon the limiting factors themselves (Chase and Leibold 2003). Often, local coexistence is permitted by subtle variation in microhabitats and in the selection of microhabitats by individuals (e.g. Kotler and Brown 1999).

Localized interactions between individuals. Even in spatially homogeneous communities, dispersal following reproduction can be spatially circumscribed. This increases the impact of intraspecific competition relative to interspecific competition, potentially facilitating coexistence at local scales (Bolker and Pacala 1999).

Food-web effects. Frequency-dependent consumption by natural enemies (e.g. due to predator switching) can promote competitive coexistence. If superior competitors for resources are more vulnerable to predation, coexistence can occur (see the keystone predation module in Fig. 2.1; Holt *et al.* 1994; Leibold 1996; Chase and Leibold 2003). If an inferior resource competitor can directly prey upon the superior competitor, coexistence may be permitted (see the intraguild predation module; Holt and Polis 1997).

Nontrophic mechanisms of population regulation. Many biological mechanisms can influence population regulation, and thus interactions among species. For instance, if superior exploiters also experience strong intraspecific interference, this can permit the continued existence of inferior competitors (Schoener 1976).

B. Closed, nonequilibrial communities

Although much of theoretical community ecology assumes that the environment is constant, many natural systems in fact experience substantial temporal variation in abiotic conditions and resource supply rates. Moreover, populations may have intrinsically unstable dynamics, leading to cycles or chaotic dynamics even in constant environments. Given temporal variability, other mechanisms of coexistence can operate.

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Box 2.1 *continued*

Temporal niche partitioning (storage effects). In variable environments, coexistence may reflect temporal variation in the performance of different species on the same resource. This mechanism requires devices to slow the rate of population decline during bad times, such as seed banks or long-lived adult classes (Chesson 2000a). High recruitment into the seed bank or adult classes during better times can permit the population to persist through poor times. If different species are superior at different times, a large number of species with demographic storage effects can potentially coexist on a shared resource.

Nonlinear dynamics. Often, population growth rates are highly nonlinear functions of the magnitude of limiting factors (e.g. resource availability), leading to unstable dynamics. If competing species have different nonlinear responses to shared limiting factors, nonequilibrial coexistence may occur (Grover 1997; Huisman and Weissing 1999; Chesson 2000a; Abrams and Holt 2002). More subtle mechanisms of coexistence involving nonlinear dynamics reflect shifts between different dynamical regimes in systems with cyclical or chaotic dynamics (e.g. Harrison *et al.* 2001).

C. Open communities

Many natural communities are open, coupled to an external landscape or regional species pool via dispersal (Polis *et al.* 2004; Holyoak *et al.* 2005). This permits the operation of a broad range of coexistence mechanisms involving species movement patterns in response to spatial and temporal variation (Holt 1993; Chesson 2000b).

Migration and habitat selection. Some species (e.g. migratory birds) may circumvent (and exploit) temporal variation by migration or seasonal habitat selection. If different species are regulated at different seasons and/or in different habitats, they can potentially coexist. Moreover, species that disperse at different rates in effect average over spatial variation in different ways; this subtle effect can at times permit coexistence (McPeek and Holt 1992; Debinski *et al.* 2001).

Metapopulation processes. Coexistence may occur if inferior competitors can rapidly colonize and establish populations following disturbance, permitting the exploitation of transient habitats, whereas superior competitors disperse more sluggishly. This mechanism requires recurrent loss of the superior competitor from local sites (e.g. through disturbance).

Source-sink dynamics. An inferior competitor may persist in one community if it is a superior competitor in a nearby community. Dispersal from "source" habitats then permits sustained presence in what would otherwise be "sink" habitats for the inferior competitor (Holt 1993; Leibold and Miller 2004).

2.3 Mechanisms of coexistence in parasite assemblages

We think it is fair to assert that the relative importance of each potential coexistence mechanism (Box 2.1) is not well understood for *any* natural community; parasite communities are certainly no exception. However, there are hints in the literature that many of these mechanisms may influence the structure of parasite assemblages. One basic question is the degree to which parasite species richness reflects processes acting within individual hosts (dubbed the "infracommunity" in parasitology; Holmes 1973, Holmes and Price 1986; Goater *et al.* 1987, Poulin 1998), versus processes acting at the level of entire host populations and communities. Here we focus on the latter.

2.3.1 Simple models for microparasite competition and coexistence

Parasites compete for susceptible hosts and the resources those hosts contain. In principle, analogues of any mechanism in Box 2.1 could help explain parasite coexistence. There is considerable room for developing theory that formalizes these mechanisms in a manner specifically tailored to host–parasite systems. We sketch here several models for microparasites (e.g. viruses, bacteria), illustrating different modes of coexistence.

Parasites abstractly compete at two levels of organization: within individual hosts and between hosts. Microparasites establish populations within individual hosts. Because hosts provide resources that can be consumed, this permits exploitative

competition between multiple species that co-occur within a "patch" (individual host). For the moment, assume that only a single parasite species can persist in an individual host, and that whichever species initially colonizes that host excludes other species; that is, there is no coinfection by multiple species leading to either within-host coexistence or superinfection (where one parasite can supplant another). The absence of coinfection is assumed in many theoretical studies (e.g. Dushoff and Dwyer 2001). (We will relax this assumption below.)

Even if coinfection can at times occur, one can safely ignore it in a number of plausible circumstances. Consider a system in which two parasite species with similar birth and death rates use the same tissues within a host that does not exhibit species-specific immune responses (Iwasa *et al.* 2004). If infective propagules are small and infrequent, then whichever species first colonizes (infects) an individual host is likely to exclude the other species. After the first species has achieved an equilibrium between births and deaths within its host, small and infrequent propagules of a second species cannot invade unless its birth or death rates are relatively favorable. Species with similar birth and death rates will generally be distributed in a checkerboard pattern, with each host infected by just one of these similar parasite species. This outcome of parasite competition for host tissues within an individual parallels that of competition for host individuals within a population, as modeled in Equation (2.1) below. In other systems, assuming no coinfection can be viewed as a simple limiting case of a more complex epidemiological model. For instance, if coinfection rapidly leads to host mortality, few host individuals will in practice be coinfected. Alternatively, if encounter rates are low, hosts infected by any single parasite species are likely to recover or die before encountering a host infected with another parasite. Finally, for our purposes, assuming no coinfection is conceptually useful, because it permits us to identify various mechanisms operating at the host level to influence parasite coexistence.

Assuming two parasite species and no coinfection, we can divide a host population into one uninfected class and two, nonoverlapping classes of individuals infected with either parasite 1 or parasite 2. Recall that for the moment we are assuming the host is regulated at its carrying capacity, K, by factors other than parasitism. A simple "SI" model describing "susceptible" and "infected" hosts and the exploitative competition between two species of microparasite spread by density-dependent transmission includes the following terms for the dynamics of each parasite:

$$\begin{aligned} \frac{dI_i}{dt} &= (\beta_i S - d_i) I_i, \quad i = 1,2 \\ K &= S + I_1 + I_2 \end{aligned} \tag{2.1}$$

Here, S is the density of susceptible hosts, I_i the density of hosts infected with parasite species i, β_i the transmission rate for parasite species i, and d_i the rate of parasite loss from the host population (including death and recovery of infected hosts). The second equation states that total host numbers are fixed at carrying capacity by factors other than parasitism.

When parasite i occurs alone, the first equation in model (2.1) reveals that the equilibrial density of susceptible hosts is $S^* = d_i/\beta_i$. Parasite j can invade if $dI_j/dt > 0$; that is, if $\beta_j S^* - d_j > 0$ or $d_i/\beta_i > d_j/\beta_j$. If this is true, when parasite j is alone at equilibrium, parasite i cannot invade. Thus, model (2.1) predicts competitive exclusion, and the winning parasite is the one that can persist at the lowest density of susceptible hosts. This model parallels in its essential features standard resource–consumer models (Grover 1997), which formalize the idea that, at equilibrium, a single species will dominate any single limiting resource. Here, the resource is the susceptible host subpopulation. See Allen *et al.* (2004) for more complex epidemiological models of this type.

If we substitute $S = K - I_1 - I_2$ into the first equation in (2.1), and do this for each of the two parasite species, we generate a competition model of Lotka–Volterra form, with nonintersecting zero-growth isoclines, corresponding to competitive exclusion. This very simple model predicting competitive exclusion provides a springboard for more complex models that illuminate how different aspects of host population and community properties influence parasite coexistence. Next, we sketch some plausible scenarios corresponding to the coexistence mechanisms of Box 2.1. Due to limitations in

space and in the current state of theory, we consider some potential mechanisms in more detail than others; these mechanisms are not necessarily more important in natural communities.

2.3.1.1 Classical niche partitioning

In a multispecies host community, if each parasite species is specialized to a different host species, and the hosts do not themselves compete, parasite coexistence is trivial, as it is determined entirely by the independent responses each parasite has to its own host (e.g. each respective host should exceed the threshold density for its parasite). In effect, this scenario assumes a rigid niche partitioning among parasites. But in many natural systems, parasites are shared by multiple host species (Cleaveland *et al.* 2001; Dobson 2004; Woolhouse 2002; Woolhouse *et al.* 2001), and hosts harbor multiple parasites. In a species-rich host assemblage, heterogeneity among parasites in how they use different host species can in principle permit the sustained coexistence of multiple species of parasites.

There is now a rich theoretical literature on the dynamics of multi-host, one-pathogen systems (e.g. Holt and Pickering 1985; Bowers and Begon 1991; Begon *et al.* 1992; Begon and Bowers 1995; Bowers and Turner 1997; Greenman and Hudson 2000; Dobson 2004). Understanding cross-species transmission can be of great importance for addressing applied issues, and ignoring such transmission can lead to erroneous conclusions. For instance, Hess (1994) provocatively argued that increasing connectivity in a metapopulation might not always be a helpful conservation strategy, because connectivity also facilitates movement of pathogens. Several authors have noted that this result may be moot if transmission is generally facilitated by alternative, "reservoir" hosts (Gog *et al.* 2002; McCallum and Dobson 2002).

A simple listing of known hosts does not quantify the dynamical importance of multiple host species for parasite dynamics and coexistence. Compilations of parasite "host range" conflate several alternative dynamical scenarios. First, cross-species transmission may be only of historical or biogeographic importance. For HIV, contact with the original source host (presumably an African primate) was historically crucial but is now irrelevant in determining the subsequent dynamics of the disease in humans. Many emerging diseases of economically important plants involve single introductions, not recurrent infection within a single community (Anderson *et al.* 2004). Second, in the case of recurrent cross-species transmission, the incidence of the disease in a focal host can be influenced by the presence, abundance, and epidemiological properties of alternative hosts. In this case, it is useful to distinguish several alternative scenarios (elaborating on a suggestion by Antonovics *et al.* 2002; see also May *et al.* 2001):

1. The focal host species may be a permanent demographic sink for the parasite, in that each primary infection in the focal host generates less than one secondary infection within its own population ($R_0 < 1$). Under this scenario, focal host infections are generally due to "spillover" of the parasite from a source host. Reciprocal transmission back to the source host (Antonovics *et al.* 2002) may alter prevalence in both source and sink hosts. This scenario is particularly likely when no host species alone is sufficiently dense to sustain the infection.
2. The focal host species may be able to sustain the parasite entirely on its own ($R_0 > 1$), but recurrent infection from alternative hosts may nonetheless significantly perturb dynamics within the focal host population.
3. As an intermediate case, the focal host may be an intermittent sink, such that R_0 varies through time. Alternative hosts may then be particularly important for ensuring parasite persistence through times of low R_0 in the focal host. In some ways, this scenario is reminiscent of the "migration and habitat selection" mechanism in Box 2.1.

Density-dependent disease transmission implies a threshold host population size, below which $R_0 < 1$. A host may be a sink for a parasite not because of the poor physiological suitability of the host, but because of ecological factors influencing host abundance or background mortality rates, such as microhabitat or resource availability, predation, or competition with other species. In model (2.1) above, the rate at which an uninfected individual becomes infected is proportional to the density of infectives in the host population. In some sexually transmitted or vector-transmitted diseases, however,

the rate of infection depends upon the frequency of infection in the host population (i.e. the fraction of individuals infected). With pure frequency-dependent disease transmission, there is not a threshold host population density. But more realistic models of frequency-dependent transmission suggest that density-dependence often emerges at sufficiently low host numbers (Antonovics *et al.* 1995). So, it is likely that a threshold host density describes a wide range of infectious disease systems.

With recurrent cross-species infection, determining the criterion for microparasite invasion ($R_0 > 1$) requires a more complex approach than considering R_0 in each host alone (Dobson and Fofopoulos 2001; Holt *et al.* 2003; Dobson 2004). Here we exemplify one approach, and extend it to consider competition and effective niche partitioning among parasite species.

We can generalize model (2.1) to include two parasite and two host species, as follows:

$$\begin{aligned}
\frac{dI_1}{dt} &= \beta_{11}S_1I_1 + \beta_{12}S_1I_2 - \Gamma_1 I_1 \\
\frac{dI_2}{dt} &= \beta_{22}S_2I_2 + \beta_{21}S_2I_1 - \Gamma_2 I_2 \\
\frac{dI_1'}{dt} &= \beta_{11}'S_1I_1' + \beta_{12}'S_1I_2' - \Gamma_1' I_1' \\
\frac{dI_2'}{dt} &= \beta_{22}'S_2I_2' + \beta_{21}'S_2I_1' - \Gamma_2' I_2'
\end{aligned} \qquad (2.2)$$

The first two equations describe dynamics of parasite 1 in host species 1 and 2; the second two equations (with primes) describe parasite 2. The quantity β_{ij} denotes transmission of infection from infected individuals of species j to susceptible individuals of species i; Γ_i scales loss rates (mortality plus clearance or recovery) of infected individuals of host species i.

To complete the model, we must describe dynamics of the rest of the host populations. As before, we assume each host is regulated by strong density dependence (e.g. territoriality) independent of parasitism, so $K_i = S_i + I_i + I_i'$, $i = 1,2$. [Note that with this assumption about host regulation, we preclude apparent competition (Holt 1977; Holt and Pickering 1985; Hudson and Greenman 1998; Bowers 1999). We turn to such indirect interactions between hosts below.] Alternative models include exponential or logistic growth of hosts, regulated at least in part by parasitism. For example, Begon *et al.* (1992) assume $dN_i/dt = r_iN_i(1 - N_i/K_i) - \alpha_i I_i$, $i = 1,2$, for each of two host species; the first term is logistic growth experienced by all individuals in species i, and the second denotes additional mortality experienced by infected individuals. In Box 2.2, we describe how a model with fixed host density (2.2) leads to isoclines that describe qualitatively the conditions for invasion of each parasite species; these isoclines can then be used to characterize some necessary conditions for parasite coexistence.

The zero-growth isoclines depicted in Box 2.2 characterize, for a system of two fixed-density host species, the densities of susceptible hosts that permit shared parasites to increase when rare. With this graphical tool in hand, we can now address some aspects of competitive coexistence without wallowing in complex algebra. Each parasite has its own zero-growth isocline. By jointly plotting each species' isocline, one can qualitatively characterize necessary conditions for coexistence of a pair of parasite species competing for susceptible individuals of two host species. If the isocline for parasite i lies entirely inside the isocline for parasite j, then i can invade the system and depress susceptible host density enough to keep j out. Thus, a necessary condition for parasite coexistence is that the two isoclines cross. (To characterize sufficient conditions, one must also consider host properties, such as the degree of regulation of hosts by parasitism.)

Parasites can coexist through different types of niche partitioning. Coexistence may be related either to the capacity each parasite has for using individual hosts, or to the pattern of transmission within and among host species. If parasite transmission is similar within and among host species, the zero-growth isoclines are straight lines. In this case, for these isoclines to cross, each parasite species must experience a lower loss rate in a different host species (Fig. 2.3 (a) and (b)). For example, parasites may coexist if each can better resist clearance from a different host. Such niche differentiation may be due to differential tolerance of induced or constitutive host defenses. Alternatively, if parasites have equivalent loss rates in both host species, they can coexist only if each has its highest transmission rate in a

Box 2.2 A graphical model for parasite invasion: zero-growth isoclines

Before addressing coexistence in the community represented by model (2.2), we need to characterize persistence conditions for each parasite species alone. If we assume host abundance is fixed (e.g. each host is at its respective carrying capacity), we can ask whether a given parasite (say species 1) can invade the host community. To determine the answer analytically, it is useful to rewrite the equations for parasite 1 (shown in main text) in the form

$$\frac{d}{dt}\begin{bmatrix} I_1 \\ I_2 \end{bmatrix} = \begin{bmatrix} (\beta_{11}S_1 - \Gamma_1) & \beta_{12}S_1 \\ \beta_{21}S_2 & (\beta_{22}S_2 - \Gamma_2) \end{bmatrix} \begin{bmatrix} I_1 \\ I_2 \end{bmatrix}$$

from which it is clear that the growth rate of the parasite is represented by the matrix. In fact, the asymptotic growth rate of the parasite is equivalent to the dominant eigenvalue of this matrix. The dominant eigenvalue equals zero (i.e. $R_0=1$) when the determinant of this matrix (det[]) is zero, resulting in the following condition for zero parasite growth:

$$\det\begin{bmatrix} (\beta_{11}S_1 - \Gamma_1) & \beta_{12}S_1 \\ \beta_{21}S_2 & (\beta_{22}S_2 - \Gamma_2) \end{bmatrix} = (\beta_{11}S_1 - \Gamma_1)(\beta_{22}S_2 - \Gamma_2) - \beta_{12}S_1\beta_{21}S_2 = 0$$

When this condition is plotted in relation to the densities of susceptible hosts (S_1 and S_2, as in Fig. 2.3), it describes the "zero-growth isocline" for the parasite. If the combination of susceptible host densities lies outside the isocline, then the parasite can invade; conversely, if susceptible host densities are between the isocline and the origin, the parasite will decline toward extinction. The shape of the zero-growth isocline reflects the pattern of parasite transmission resulting from the type of interaction among host species (see Holt *et al.* 2003, and main text). In this particular model, host-specific loss rates, Γ_i, influence the intercepts, but not the curvature of the parasite's zero-growth isocline.

There is a subtle distinction between the zero-growth isoclines in Fig. 2.3 and the more familiar isoclines of resource–consumer theory (Grover 1997). In a typical resource–consumer model, the current growth rate of the consumer is simply a function of current resource abundance, with no explicit effect of time. In a host–parasite system, susceptible host abundance is indeed a "resource" for the parasite. But the growth rate in question here is the *asymptotic* growth rate for the parasite, after enough time has passed following invasion for the parasite to settle into its stable pattern of distribution across the host species.

The zero-growth isoclines derived above and depicted in Fig. 2.3 generalize the concept of a minimum host density to two host populations, and encapsulate graphically the minimal host community configurations permitting parasite invasion. One can also plot additional isoclines of constant R_0 as a function of susceptible host densities (Fig. 2.4). The zero-growth isocline is that set of susceptible host densities for which $R_0=1$; that is, although total host densities are fixed in model (2.2), parasites may regulate susceptible host densities. At equilibrium with a given parasite, the density of susceptible hosts will be depressed to levels somewhere on the zero-growth isocline for that parasite. This equilibrium then determines the initial array of susceptible host densities that a second parasite species will face when it attempts to invade the host community. By plotting isoclines for each parasite simultaneously, we can begin to characterize conditions for exclusion, versus coexistence. With similar parasite transmission rates within and among host species, the isoclines are straight lines (Fig. 2.3(b)). If most transmission is within host species, the isoclines instead bow out from the origin (Fig. 2.3(d)). Begon *et al.* (1999) analyzed transmission dynamics of the cowpox virus in mixed populations of bank voles and wood mice, and showed that despite their close co-occurrence, transmission between host species was negligible, so convex isoclines are quite plausible in this system, and doubtless many others as well.

In Fig. 2.3 and in model (2.2) in the text, both zero-growth isoclines have negative slope; this is not necessarily the case for systems with frequency-dependent transmission, vector-mediated transmission, or free-living infectious stages (Holt *et al.* 2003; J. Antonovics, personal communication). Figure 2.4 compares the isoclines describing contours of constant R_0 as a function of host density for several frequency-dependent systems, each with two host species. With multiple host species, frequency-dependent transmission can buffer disease outbreaks, leading to the "dilution" effect described by Ostfeld and Keesing (2000), while density-dependent transmission usually leads to enhanced potential for parasite establishment and outbreak. When the frequency-dependent case is expanded to explicitly consider transmission vectors for the parasite, then the "height" of each R_0 contour varies approximately with the square root of vector abundance (Dobson 2004). So increasing vector density increases the potential for an epidemic. This effect helps explain why vector control has been so effective in controlling diseases such as malaria and yellow fever.

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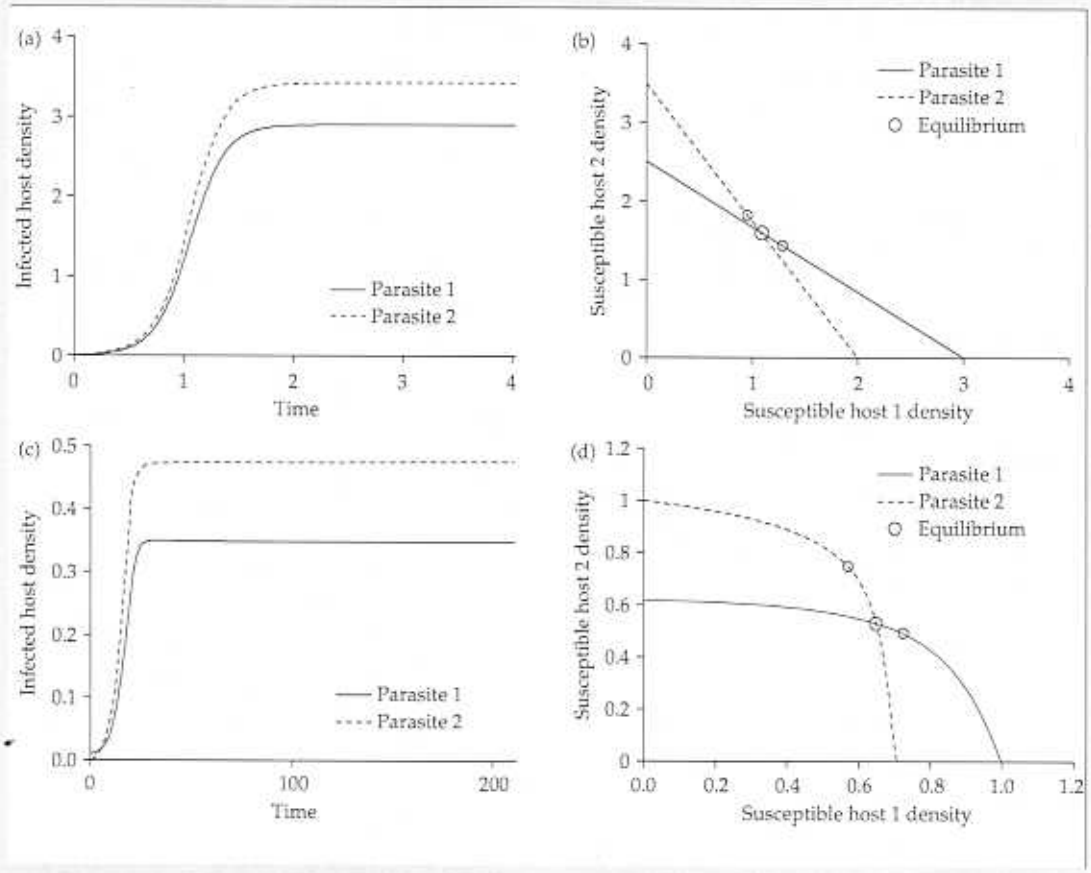


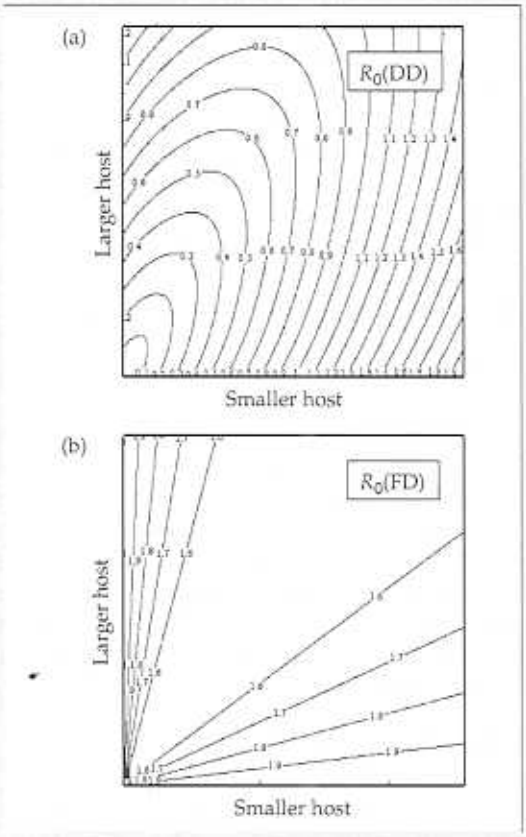
Figure 2.3 Examples of isocline shapes and coexistence for two parasite species competing for susceptible individuals of two (fixed-density) host species. In (a) and (b), transmission is similar within and between host species, but each parasite persists longer in a different host. In (c) and (d), parasite loss rates are similar between species, but there is more intra- than interspecific transmission. The time plots (a) and (c) are numerical simulations demonstrating that the parasites can coexist. In the isocline plots (b) and (d), the smaller dots indicate equilibrial densities of susceptible hosts when each parasite occurs alone; in these examples, the equilibria shift so as to facilitate invasion by the other parasite. The specific parameters are as follows: (a) Total number of hosts infected by each parasite, starting with host 1 infected by each parasite at a density of 0.001. $\beta_{11}=\beta_{12}=\beta_{21}=\beta_{22}=\beta_{11}'=\beta_{12}'=\beta_{21}'=\beta_{22}'=1$, $\Gamma_1=3$, $\Gamma_2=2.5$, $\Gamma_1'=2$, $\Gamma_2'=3.5$, $K_1=4$, $K_2=5$. (b). Isoclines for parameters in (a). When both parasites are present, the system approaches the point at which the isoclines cross. (c and d). Same as (a) and (b), but $\beta_{11}=0.5$, $\beta_{12}=0.1$, $\beta_{21}=0.4$, $\beta_{22}=0.8$, $\beta_{11}'=0.7$, $\beta_{12}'=0.3$, $\beta_{21}'=0.1$, $\beta_{22}'=0.5$, $\Gamma_1=\Gamma_2=\Gamma_1'=\Gamma_2'=0.5$, $K_1=K_2=1$.

An important subtlety arises with pathogens that use ticks as vectors. The abundance of ticks may be tightly coupled to the abundance of their hosts—hosts that also harbor the pathogens that ticks transmit. In this case, increases in host abundance may lead to both increased vector abundance and enhanced amplification of disease transmission. If factors other than host availability can regulate vector numbers when hosts are common, nonlinear isoclines can readily occur.

In systems where vector transmission is by mosquitoes or tsetse flies, whose abundance is independent of host abundance, an increase in host numbers may dilute the per capita vector attacks and the per capita production of fresh infections from any given infected host (R_0). Models that

continues

Box 2.2 *continued*



include a free-living pool of pathogens can generate isoclines with positive slope (Holt *et al.* 2003). If different pathogens have very different isoclines in these two-host systems, the interactions between them may not resemble familiar competition isoclines at all.

The isocline approach to analyzing coexistence of parasites in host communities provides useful insights, but it may be difficult to apply to particular empirical systems. In natural systems it can even be difficult to show that threshold densities exist (Begon *et al.* 2003). Detailed case studies seem to reveal that parasite persistence is not governed so much by average host density, as by the detailed spatial structuring of infection processes (e.g. Keeling and Gilligan 2000). An important task for future work is to articulate the impact of alternative forms for transmission dynamics, and the spatial structuring of transmission, on the coexistence conditions for parasites potentially competing for the same suite of host species.

Figure 2.4 Zero-growth isoclines resulting from density-dependent (a) or frequency-dependent (b) transmission. These isoclines with nonlinear or positive slopes depict contours of constant R_0 for a single parasite species infecting two hosts (see Dobson 2004). Were one to simultaneously plot isoclines for two parasite species infecting these two hosts, it is clear that the resulting figures would not match those of classical competition theory (details not shown).

different host, corresponding to nonlinear isoclines (Fig. 2.3 (c) and (d)). Transmission rates can differ in this way for a variety of reasons. Each parasite species may increase contact behavior in a different host species, or each may be specialized to exploit contacts in a different host.

Crossing isoclines are necessary but not sufficient for parasite coexistence. For coexistence, host carrying capacities cannot differ too greatly. A parasite that is better at exploiting a host with a low carrying capacity is vulnerable to exclusion by a parasite that is better at exploiting a host with a higher carrying capacity. This effect arises in model (2.5) below, which is a limiting case of model (2.2).

It should be noted that in model (2.2), we assume that hosts that are susceptible to infection by one parasite species in general are also susceptible to the other parasite. If hosts have specialized immune responses, then host individuals that recover and become immune to one parasite species may still be available for infection by a second parasite species. This leads to a kind of niche partitioning within a single host species, which can facilitate the coexistence of competing parasites (Pej Rohani, personal communication).

2.3.1.2 Spatially localized competition

Roberts and Dobson (1995; see also Dobson 1985) explored a model for competing macroparasites that are characteristically aggregated within the host population. This model incorporates both exploitative competition for hosts, and direct interference. Parasite aggregation can facilitate coexistence,

particularly given negative cross-species correlations in the parasite distributions. The model of Roberts and Dobson (1995) assumed that hosts are solely regulated by the parasites (which increased host mortality). If, as in model (2.1), we instead assume that host density is constant and regulated by factors other than parasitism, the macroparasite equations presented in Roberts and Dobson (1995, p. 194) can be re-written as the familiar Lotka–Volterra competition equations, with parameters such as the competition coefficient expressed as a function of epidemiologically relevant quantities such as parasite fecundity and aggregation strength (details not shown). This simplification of the model reveals that the coexistence of competing macroparasites requires that they not be too dissimilar in their inherent ability to use the host. For example, macroparasites with similar rates of growth when rare may coexist via this "equalizing mechanism" (*sensu* Chesson 2000a). This simplification of the model also reveals that competing macroparasites must differ in their patterns of aggregation. If aggregation patterns ensure that intraspecific interference exceeds interspecific interference, then macroparasites may coexist via this "stabilizing mechanism" (*sensu* Chesson 2000a).

We are not aware of parallel theoretical studies directly pertinent to microparasites. However, we note that the degree of aggregation created in any host macroparasite system reflects the interplay between heterogeneities in host susceptibility (immunological, spatial, and genetic) and parasite virulence. In general, parasites that are more virulent will exhibit lower levels of aggregation and thus will be less likely to coexist with other species (Shaw and Dabson 1998) (barring spatial heterogeneities in parasite transmission efficiency, which would tend to promote parasite coexistence).

2.3.1.3 *Frequency-dependent mortality*

As in community ecology in general, food-web interactions may influence which parasite dominates and whether there will be exclusion or coexistence, depending upon the detailed structure of the trophic interactions (see Chapter 9, this volume, for further treatment of parasites in food webs). In model (2.1), the loss rate of infected hosts implicitly incorporates losses due to predation. In that case, predation can potentially influence parasite community structure if different parasite strains lead to different (fixed) predation rates for infected hosts; for example, by affecting host behavior.

In models that allow the death rate of infected hosts to increase with the density of infected hosts, coexistence may be promoted (Pugliese 2002). Assume that parasite species 1 has higher transmission rates and also causes its host to attract generalist predators, who respond facultatively to the abundance of the infected prey. We can modify model (2.1) to account for this effect by allowing infected host deaths to increase directly with their own abundance:

$$\frac{dI_1}{dt} = (\beta_1 S - d_1(I_1))I_1 \tag{2.3}$$

Now assume that parasite 2 has lower transmission rates and does not affect host predation rates, so that host dynamics follow model (2.1). For simplicity, assume the death rate of hosts with parasite 1 is a linear function of their density, $d_1(I_1) = d_1 + d'I_1$, whereas hosts with parasite 2 have a fixed death rate; moreover, assume that each parasite can invade when alone with the host. The condition for parasite coexistence is:

$$\frac{\beta_1}{d_1} > \frac{\beta_2}{d_2} > \frac{\beta_1 + d'}{Kd' + d_1}$$

The left-hand inequality describes when parasite 1 can invade, given that parasite 2 is present and at equilibrium. The right-hand inequality describes when parasite 2 can invade, given that parasite 1 is present and at equilibrium. The full condition reveals that coexistence is more likely with larger host carrying capacity and larger effects of parasite 1 on host predation rates.

Packer *et al.* (2003) and Ostfeld and Holt (2004), building upon earlier work by Dobson (1988) and Lafferty (1992), have recently emphasized the importance of predation as a factor governing host–parasite dynamics, even if predators act simply as density-independent mortality agents upon various classes of prey. There are many reasons to focus on systems combining predation and parasitism. When predators attack both healthy and infected prey individuals, the full relationship corresponds to intraguild predation (Holt and Polis 1997; see Fig. 2.1), because predators both

compete with parasites for healthy hosts and inflict mortality upon parasitized hosts. Generalist top predators in many communities seem particularly at risk to anthropogenic impacts. If disruption of natural predator–prey interactions reduces prey mortality rates, one indirect consequence could be the unleashing of host–pathogen interactions present in lower trophic levels. This could involve both increases in disease incidence in species that are already sustaining the pathogen, and spread to novel hosts (Packer *et al.* 2003; Ostfeld and Holt 2004; Hethcote *et al.* 2004; Hall *et al.* 2005; Holt, in press). If selective predation promotes parasite coexistence, as in model (2.3), predator removal may also bring certain parasites to dominance.

An interesting example comes from studies by Hudson and colleagues on the interplay between gamekeepers, predators, grouse, and a parasitic nematode (Hudson *et al.* 1992, 1998). Predators selectively attack heavily infected grouse. The job of a gamekeeper is to reduce predator numbers, in order to increase the game available for hunters. Across sites, Hudson found that the percentage of grouse heavily infected with worms actually *increases* with the density of gamekeepers! This suggests that although there may indeed be more grouse where gamekeepers are doing their job, those grouse on average are wormier. This effect should be expected whether or not predators differentially prey upon wormier grouse; with fewer predators, parasite-infested prey can simply live longer and generate more secondary infections.

We should caution that the impacts of predator removal upon disease dynamics may differ dramatically from the descriptions above. For instance, predators may themselves be hosts, or be transmission agents for the parasite (as is true in many complex life cycles, Parker *et al.* 2003; see also Lafferty 1992 and Chapters 9 and 10, this volume). Predator removal may then disrupt transmission dynamics and reduce disease incidence among the remaining hosts. Moreover, prey behavioral responses to predation can alter transmission dynamics directly (Dobson 1988; Lafferty 1992). If prey tend to be less mobile in the presence of predators, a reduction in predation may lead to an increase in prey contacts and disease transmission. If reduced predation leads to higher prey densities and more competition, prey may become more vulnerable to infection (Keesing *et al.*, in preparation). Caveats aside, we suggest that the elimination of top predators can often lead to an upsurge in the abundance of infected prey and the potential for disease transmission across host species.

2.3.1.4 Storage effects

Temporal fluctuations in the environment can promote parasite species coexistence via the storage effect if, for instance, competing parasite species have different abiotic optima for reproduction, and are able to produce long-lived resting stages. Baculoviruses with a long-lived resting stage may coexist via this storage effect. We are unaware of any formal theory addressing storage-effect mechanisms for parasite species coexistence.

2.3.1.5 Temporal fluctuations and nonlinearities

If transmission rates do not vary with host abundance or quality, then (using time-averaging techniques, Holt 1997a) it can be shown for model (2.1) that coexistence cannot be facilitated by temporal fluctuations in the host. However, there is growing evidence in many systems that transmission rates can be nonlinear functions of host density (Hochberg 1993; McCallum *et al.* 2001). If one parasite transmits better at low host densities, and the other transmits better at high host densities, it is conceivable that fluctuations in host density could promote parasite coexistence.

2.3.1.6 Alternating habitats

Many macroparasites have complex life cycles, making coexistence feasible if different species are regulated at different stages of life history (e.g. see Chapters 9–11, this volume). A complication here is that one must also characterize how the different hosts are themselves regulated, and if this regulation depends upon parasitism. This scenario is ripe for theoretical development.

2.3.1.7 Competition–colonization trade-offs

In model (1), we assumed that there is no coinfection. If individual hosts can harbor multiple

parasite species then coexistence may occur even if there is just a single host species. As a simple example, consider the following modification of model (2.1):

$$\begin{aligned}\frac{dI_1}{dt} &= (\beta_1 S - \beta_2 p I_2 - d_1) I_1,\\ \frac{dI_2}{dt} &= (\beta_2 (S + p I_1) - d_2) I_2,\\ K &= S + I_1 + I_2\end{aligned} \tag{2.4}$$

Here, the parameter p describes the rate of superinfection of hosts by parasite 2, or the ability of parasite 2 to infect individuals already infected with parasite 1 (relative to susceptibles). Model (2.4) in effect ignores coinfection by assuming that parasite 2 quickly supplants parasite 1 in any hosts that become jointly occupied.

With this modification in transmission dynamics, coexistence can now occur, if parasite 1 can persist at a lower host density than can parasite 2. Model (2.4) is similar in form to familiar models of competition–colonization trade-offs in metapopulations. This scenario is also in a sense a form of intraguild predation; both parasites compete for healthy hosts, and in addition the superior within-host parasite "preys" upon the other parasite. Hochberg and Holt (1990) explore a version of this model in which the host is regulated by parasites. That model can also be viewed as a variant of intraguild predation; in general, if a predator consumes the required resource of its prey, that prey species must be superior in exploitative competition for it to persist (Holt and Polis 1997).

2.3.1.8 *Spatial subsidies*

A simple mechanism for enriching local communities of parasites inhabiting a given host species in one location is "spillover" from other locations or other species. Model (2.1) can be modified to illustrate such spillover effects, as follows:

$$\begin{aligned}\frac{dI_1}{dt} &= (\beta_1 S - d_1) I_1\\ \frac{dI_2}{dt} &= (\beta_2 S - d_2) I_2 + S\beta' I'\\ K &= S + I_1 + I_2\end{aligned} \tag{2.5}$$

Here $\beta' I'$ is the net force of infection of susceptible hosts occurring from external sources ("spillover"). Note that we have assumed that parasite species 1 is maintained solely by local dynamics, and there is just one host species available.

If parasite species 2 is locally inferior (i.e. $d_2/\beta_2 > d_1/\beta_1$), then it will surely be excluded in a closed community. In an open community with spillover, it will persist and equilibrate at

$$I_2^* = \frac{I'\beta'(d_1/\beta_1)}{d_2 - d_1(\beta_2/\beta_1)}$$

This equilibrium follows from examination of the first and second equations in model (2.5), which give us $S^* = d_1/\beta_1$ and $I_2^* = S^* I'\beta'/(d_2 - S^*\beta_2)$, respectively. Because we have assumed fixed host abundance, we can express the abundance of the resident parasite as $I_1^* = K - I_2^* - S^*$. Because I_1^* declines with I_2^*, and I_2^* is directly proportional to $I'\beta'$, the equilibrial abundance of parasite 1 declines linearly with the force of spillover infection from the alternative host for parasite 2.

At low spillover rates, one will observe coexistence. The coexistence mechanism in this case reflects habitat partitioning at a broader spatial scale; parasite 1 is locally superior, but parasite 2 can persist elsewhere and so be maintained locally in the focal community. However, if spillover is too great, the locally superior parasite can be excluded. This is particularly likely for host species with low K. Host–parasite models with density-dependent transmission predict minimum host population sizes, below which specialist parasites are likely to disappear. Spillover infections can amplify this trend, by permitting inferior generalists (maintained in large measure outside the focal host) to supplant specialist parasites in hosts that are low in abundance.

2.4 Back to parasite-driven host dynamics

We have explored some ways in which the standard repertoire of coexistence mechanisms that are of such enduring interest in community ecology may pertain to parasite species coexistence, and sketched simple models illustrating several mechanisms. We deliberately made the simplifying

assumption that the abundance of each host species was set by ecological factors, independent of parasitism itself. But one reason for the increasing attention to parasites is that they can drive host population and community dynamics. Relaxing the assumption that hosts are regulated independently of parasitism increases the degrees of freedom that must be considered in analyzing community dynamics. Host species may themselves go extinct, and so one must ascertain the conditions for the joint maintenance of diversity in hosts as well as in their parasites. Moreover, unstable dynamics become more likely, permitting nonequilibrial influences on species exclusion and coexistence to arise.

Each of the modules shown in Fig. 2.2 warrants considerable treatment, well beyond the space limits of this chapter, and examples are beginning to appear in the literature (Hochberg and Holt 1990; Grenfell 1992; Holt and Hochberg 1998; Taylor *et al.* 1998, Dwyer *et al.* 2004). Here we highlight just a few simple points about regulation by parasites.

Consider again the basic model (2.1) of exploitative competition between parasites for a single host species, but now allow the parasite-free host population to grow in an unlimited fashion, following $dS/dt = rS - \beta_1 I_1 S - \beta_2 I_2 S + e_1 I_1 + e_2 I_2$. The last two terms describe contributions to susceptibles due to recovery or reproduction by infected individuals. The winning parasite species is still the one persisting at the lowest value of $S^* = d_i/\beta_i$. But the equilibrium may not exist; adding the equations for infected hosts, one finds that a condition for host population regulation is that $d_i < e_i$; that is, the death rate of infected hosts must exceed their combined rates of recovery and reproduction (Holt and Pickering 1985). Moreover, there is now a tendency for dynamic instability; for example, the lower the reproductive rate of infected hosts, the longer one observes damped oscillations when the system is perturbed.

2.4.1 Apparent competition

As noted above, one plausible mechanism for the coexistence of parasites is niche partitioning among host species. But if parasites influence host dynamics, a rich array of outcomes become possible. Embedded within the niche partitioning module (Fig. 2.2) is the module of shared parasitism. If a parasite can limit the numbers of each of several host species, apparent competition between hosts may occur, leading to reduced host abundance or even elimination of some host species from the community (Holt and Lawton 1994; Hudson and Greenman 1998). Shared parasitism could play important roles in community structure and biodiversity, and in the emergence of disease. For instance, Power and Mitchell (2004) experimentally demonstrated that wild oats facilitated spillover of the yellow dwarf virus onto several other host species, reducing their abundance through apparent competition (see also Chapter 5, this volume).

Holt and Pickering (1985) provided a formal treatment of apparent competition due to shared parasitism for a standard susceptible-infected (SI) model in which hosts are not regulated independently of parasitism, and in which disease transmission was density-dependent within and between host species. By examining conditions for each species to increase when rare, they identified conditions that led to exclusion of one host species, and conditions permitting coexistence. These conditions combine intrinsic host properties, impacts of disease on host fitness, and patterns of transmission. Host species with low intrinsic growth rates are vulnerable to exclusion; for robust coexistence, such that each host species can increase when rare, within-species disease transmission must exceed between-species transmission.

The potential for indirect exclusion via shared parasitism arises in almost any model in which parasites can regulate host numbers. For instance, Bowers and Begon (1991) examined a model for two hosts interacting via a pathogen with a free-living infectious stage. The single-host case can lead to a stable equilibrium or limit cycles. Despite the seeming potential for nonequilibrial coexistence, coexistence did not occur. At equilibrium, the host contributing most to the free-living pool of pathogens won out. This result parallels that of predator-mediated competition models in which

the winning competitor is often the one that can sustain more predators (Holt *et al.* 1994). Begon and Bowers (1995) showed again in a model of multiple host species that one species would exclude all others if both transmission and density-dependent host growth were in proportion to total host density. In general, host coexistence in the face of a single, shared parasite seems to require the classical niche partitioning suggested by Holt and Pickering (1985); that is, stronger intraspecific than interspecific disease transmission.

Bowers and Begon (1991) also observed that nonequilibrial dynamics may occur, allowing the winner of competition between hosts to depend on the starting conditions. Since then, further evidence has mounted that even simple one-pathogen, two-host models can display a rich diversity of dynamical outcomes. Greenman and Hudson (1997) demonstrated that the Holt–Pickering model could generate alternative equilibria; under the same parameter values, one host species could exclude the other or, if the second host were introduced in sufficient numbers, both hosts could persist in a stable limit cycle. Similar phenomena can arise even with direct intraspecific density dependence in host dynamics (as in Begon *et al.* 1992; Begon and Bowers 1995). However, if such intraspecific density dependence is sufficiently strong, the system settles into a stable equilibrium. Intraspecific density dependence depresses host productivity, reduces the equilibrial level of infection, and weakens the indirect interaction between host species.

There are often substantial differences among host species in transmission rates and other key parameters reflecting body size, diet, and other important ecological dimensions. Figure 2.5 illustrates the long-term transient dynamics of a system of four host species when rates of interspecific transmission vary across four orders of magnitude (after Dobson 2004). In this system we have scaled the dynamics of the host species by their body sizes; these allometric rescalings allow numerical examination of more complex systems for realistic ranges of parameter values (DeLeo and Dobson 1996). When interspecific transmission is low, each host species exhibits persistent epidemic cycles of a frequency determined by the demographic speed with which the pool of susceptible hosts recovers; host species with small body size exhibit faster epidemic cycles (Fig. 2.5(a)). As interspecific transmission increases, outbreaks are dominated by the dynamics of smaller species until the system stabilizes and each species reaches a constant abundance. Eventually, interspecific transmission is sufficiently high to allow the smaller (least susceptible) to drive the larger (more susceptible) species to local extinction (Fig. 2.5(b)).

The theoretical studies discussed above focus on microparasites. Comparable phenomena arise in macroparasite models (Dobson 1990; Dobson and Pacala 1992; Greenman and Hudson 2000). Although such models are relatively complex and difficult to analyze, qualitatively similar messages emerge. In a wide range of circumstances, apparent competition mediated by macroparasites can lead to host exclusion. Adding direct competition exacerbates this trend, as suggested by data on the decline of the partridge in the United Kingdom (Tompkins *et al.* 1999, 2000, 2001).

2.4.2 Keystone parasitism

When there is competition among host species, parasites may facilitate coexistence—a "keystone parasitism" effect. For a parasite to play this role, the host species that is superior in competition must also be more vulnerable to parasitism. This situation can emerge if competitive dominance translates into high population size, and specialist parasites are more likely to evolve on abundant hosts. A specialist parasite can regulate the abundance of a superior competitor below the level set by resources, thus freeing resources for inferior competitors that can escape parasitism (e.g., Packer and Clay 2000). One suggestive line of evidence for the potential ubiquity of keystone parasitism comes from invasion biology. Introduced species often carry fewer parasites than inflict them in their native ranges, which may permit them to explode to high abundances in their new ranges, reducing the abundances of competing native species (Mitchell and Power 2003; Torchin *et al.* 2003). If invasive species suffer more parasitism and occur at lower densities within their native ranges than

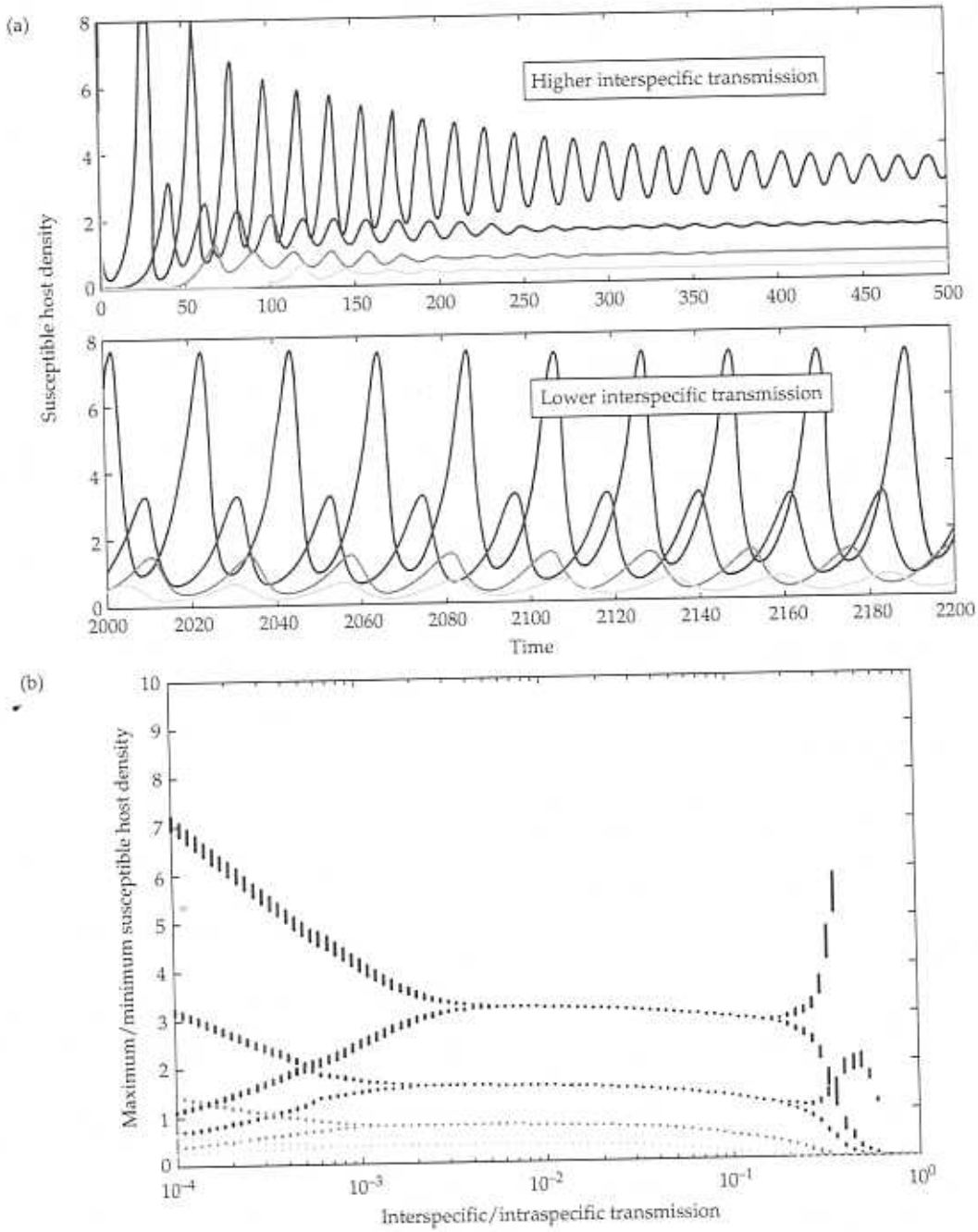


Figure 2.5 Examples of transient dynamics, coexistence, and exclusion in hosts sharing parasites. The examples shown are based on parameter values arising from the allometric scaling relationships noted by DeLeo and Dobson (1996). (a) An increase in interspecific transmission from low to intermediate rates may stabilize dynamics. (b) Further increases in transmission can lead to exclusion of hosts with low intrinsic growth rates (e.g. hosts with large body sizes).

within their introduced ranges, then keystone parasitism may be keeping these competitive dominants in check within their native ranges.

2.5 Future directions

Ultimately, everything described above suggests that ecologists need to reconsider the role that parasites play in communities (Marcogliese and Gone1997; Dobson *et al.* 2005). In Chapter 9 of this volume, Lafferty *et al.* quantify the abundance of parasites in a salt marsh community. This work reinforces earlier studies by Memmott *et al.* (2000) and others suggesting that consideration of parasites increases the diversity of species in a community by between 50% and 100%, often quadrupling the number of links in a food web. The methods described here illustrate how we can illuminate aspects of these complex systems by breaking them into individual modules of multiple interacting species. Quantifying the relative importance of the different types of modules in different communities remains an important task for the future of parasite-host community ecology.

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