

INFECTIOUS DISEASE AND SPECIES COEXISTENCE: A MODEL OF LOTKA-VOLTERRA FORM

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One principal goal of community ecology is to identify and gauge the relative importance of those factors that govern the coexistence of species and, ultimately, the species richness of communities. An extensive body of theory and empirical work is built on the assumption (which is often quite reasonable) that either competition for resources (e.g., Tilman 1982; Schoener 1983) or predation (e.g., Connell 1975; Zaret 1980; Holt 1984) or a blend of the two is responsible for constraining or allowing species coexistence. Recently, ecologists have begun to examine the influence of factors other than competition and predation on community composition (e.g., Price 1984). One such factor is infectious disease.

There is growing evidence that parasites often substantially reduce the density of their host populations (Anderson 1979a; Price 1980; Anderson and May 1982a). Mathematical models that couple the theory of population dynamics with classical epidemiology have highlighted the potential ability of parasites to regulate their host populations, even in the absence of other regulatory agents (Anderson and May 1978, 1979, 1981; Anderson 1979a, 1982; May and Anderson 1978, 1979; Bremermann 1983). Anderson and May (1980), for example, have argued that host-parasite interactions could generate the long-term population cycles observed in many temperate forest insects.

At an abstract level, any factor that regulates a single population can also be a mechanism of interspecific interaction influencing community composition (Levin 1970). In particular, by supporting a population of natural enemies, a given host or prey species may indirectly depress the abundance of alternative hosts or prey, even to the point of local extinction (Williamson 1957; Holt 1977, 1984). Several authors have suggested that parasites—broadly defined to include viruses, bacteria, protozoans, and helminths—influence community composition through their effect on the coexistence of closely related host species. Barbehenn (1969) hypothesized that shared parasites could limit mammalian species diversity. Cornell (1974) suggested that parasitism may explain distributional gaps between allopatric species. Freeland (1983) has argued that the influence of parasites on coexistence should be more pronounced for closely related host species than for

distantly related species, because the overlap in susceptibility to the available parasite community increases with increasing phylogenetic relatedness. These qualitative arguments are supported by several field examples. For vertebrate hosts, a classic example is the exclusion of moose and caribou from areas in which a meningeal worm (*Parelaphostrongylus tenuis*) is supported by populations of white-tailed deer; infection with the parasite is fatal in moose and caribou, but not in deer (R. C. Anderson 1972; Saunders 1973; Embree 1979). For invertebrate hosts, Pickering and Gutierrez (unpubl. data) found that differential mortality induced by a fungus (*Erynia neoaphidis*) infecting two sympatric species of *Acyrtosiphon* aphids can lead to dramatic switches in their relative population densities.

We find the verbal arguments of Barbehenn, Cornell, and Freeland compelling, and we suspect that parasites often have a substantial, if as yet poorly recognized, effect on community composition. In this paper we develop a simple formal theory for predicting species coexistence and exclusion when the only inter-specific interaction occurring is a shared infectious disease. We first briefly review a model of host-parasite population dynamics developed by Anderson and May for a single host species. We then extend this model to two species of hosts, derive a condition for sustained coexistence, and show that this condition has a natural interpretation, analogous to the familiar coexistence criteria in the Lotka-Volterra model of direct interspecific competition. Although other workers have emphasized the potential role of host-specific parasites in promoting the coexistence of species competing for limited resources (see particularly the review in Burdon and Shattock 1980; Janzen 1970), we emphasize instead the possibility that inhibitory interactions arising from cross-species infection may lead to the exclusion of species from communities, even when resources are not limiting.

ONE-HOST MODEL

In a recent series of papers, Anderson and May (1981 and references therein) have shown that infectious disease, in the absence of any other regulatory factor, can stably regulate an invertebrate population that otherwise would show unbounded, exponential growth. The model we use as the basis for our two-host model (model B in Anderson and May 1981) has the following form:

$$\begin{aligned} dS_1/dt &= (a_1 - b_1)S_1 - \beta_{11}S_1I_1 + a_1(1 - f_1)I_1 + \gamma_1I_1, \\ dI_1/dt &= \beta_{11}S_1I_1 - (\alpha_1 + b_1 + \gamma_1)I_1. \end{aligned} \quad (1)$$

Here, S_1 and I_1 are, respectively, the densities of individuals in host species 1 that are susceptible to, and infected with, the disease. The per capita birth rate of uninfected hosts is a_1 , and $a_1(1 - f_1)$ is the per capita birth rate of infected individuals. This model assumes that there is no vertical transmission (so that offspring of infected mothers are healthy and susceptible at birth), and that there is no acquired immunity. The parasite-induced per capita death rate of infected individuals, α_1 , is assumed to be additive to an inherent death rate of susceptibles, b_1 . (For a contrary point of view, see the work of Holmes [1982], who argued that

mortality from parasites is often compensatory to other mortality factors.) The parameter γ_1 measures the per capita rate at which infected individuals recover and reenter the susceptible portion of the population.

The transmission dynamics are described by $\beta_{11}S_1I_1$, where β_{11} scales the rate of infection of susceptibles as a quadratic function of the density of both susceptibles and infectives. This "mass action" term should be most applicable as a representation of infection when the latent period of infection is short, infection does not occur via a pool of long-lived, free-living parasites external to the host population, and transmission is not affected by density-dependent stresses altering the vulnerability to infection of host individuals. (For a discussion of alternative models of transmission dynamics, see Getz and Pickering 1983.)

As Anderson and May (1981) explained, the absence of permanent immunity in invertebrate host-disease systems allows such systems to be characterized by just two variables, namely, the densities of susceptible and infected individuals. Vertebrate systems require more-complicated models with an additional variable if individuals that recover from infection acquire immunity to future infection. For simplicity we assume that there is no acquired immunity, and that individuals that recover from infection become immediately susceptible to reinfection. Hence, the model presented here should be most literally appropriate for invertebrate hosts, although our qualitative conclusions should be more broadly applicable to vertebrate and plant hosts as well.

To simplify the two-species model developed below, it is helpful to combine several of the above parameters. Let $r_1 = a_1 - b_1$ be the intrinsic rate of growth of host species 1, $d_1 = \alpha_1 + b_1 + \gamma_1$ be the rate of depletion of the infected fraction of the population, and $e_1 = a_1(1 - f_1) + \gamma_1$ be the rate of entry of new susceptibles stemming from the infected portion of the population (r_1 , d_1 , and e_1 are all per capita rates). The quantity d_1 characterizes the rate of decline of the pool of infective individuals through deaths (induced by parasites and other causes) and recoveries, and e_1 describes the entry into the susceptible pool of individuals either that are born to infected hosts or that recover from infection. The quantity $d_1 - e_1$ measures the extent to which the death rate of infected individuals exceeds the rate at which they give birth to healthy offspring.

With these notational changes, model (1) becomes

$$\begin{aligned} dS_1/dt &= r_1S_1 - \beta_{11}S_1I_1 + e_1I_1, \\ dI_1/dt &= \beta_{11}S_1I_1 - d_1I_1. \end{aligned} \quad (2)$$

Using $\hat{}$'s to denote equilibrium, we have

$$\hat{S}_1 = \frac{d_1}{\beta_{11}} \quad \text{and} \quad \hat{I}_1 = \frac{r_1d_1}{\beta_{11}(d_1 - e_1)}. \quad (3)$$

The equilibril ratio of infected to susceptible individuals is

$$\hat{I}_1/\hat{S}_1 = r_1/(d_1 - e_1). \quad (4)$$

The equilibrium exists if and only if $d_1 > e_1$. Anderson and May (1981) showed that $d_1 > e_1$ also ensures the local stability of the equilibrium.

If $e_1 = 0$, model (2) reduces to a form identical to the classical, neutrally stable

Lotka-Volterra model for a one-predator, one-prey interaction. This familiar model exhibits perpetual oscillations around a point equilibrium, with no tendency for the system to return to this equilibrium, or to any particular cycle, once displaced from its original state. In model (2), these neutrally stable cycles are expressed as recurrent waves of epidemics coursing through the population. The full condition for local stability in model (2) is thus

$$d_1 > e_1 > 0. \tag{5}$$

Examining the eigenvalues of the model linearized near equilibrium shows that the equilibrium is approached with damped oscillations if

$$d_1(\sqrt{r_1/4d_1} + 1)^{-1} > e_1$$

and it is approached monotonically if this inequality is reversed.

In other words, population stability requires that the death rate of infectives exceed their own rate of birth, and that infected individuals either give birth to uninfected individuals or recover from the infection so as to reenter the susceptible class. As long as $r_1 > 0$, the birth and death rates of susceptibles do not directly influence the presence or absence of stability in the population, but they do affect its density, the prevalence of the disease at equilibrium, and the pattern of convergence to this equilibrium following a disturbance.

TWO-HOST MODEL

We now extend the above model to an infectious disease attacking two sympatric host species. The model is

$$\begin{aligned} dS_1/dt &= r_1S_1 - \beta_{11}S_1I_1 - \beta_{12}S_1I_2 + e_1I_1, && \text{species 1} \\ dI_1/dt &= \beta_{11}S_1I_1 + \beta_{12}S_1I_2 - d_1I_1; \\ dS_2/dt &= r_2S_2 - \beta_{22}S_2I_2 - \beta_{21}S_2I_1 + e_2I_2, && \text{species 2} \\ dI_2/dt &= \beta_{22}S_2I_2 + \beta_{21}S_2I_1 - d_2I_2. \end{aligned} \tag{6}$$

The term $\beta_{ij}S_iI_j$ characterizes the transmission dynamics. The quantity β_{ij} scales the rate at which susceptible individuals of species i acquire the disease from infected individuals of species j . We assume that there is no direct competition, either within or between species, so that each host population increases exponentially in the absence of the disease. Furthermore, we assume that the disease can regulate each host species in the absence of the other host, that is, $d_i > e_i > 0$.

The rate of change of the total population of host i is

$$dN_i/dt = r_iS_i + (e_i - d_i)I_i, \tag{7}$$

where $N_i = S_i + I_i$. Let I_i^* and S_i^* , respectively, be the equilibril density of infectives and susceptibles in species i when both species are present, and \hat{I}_i and \hat{S}_i the same quantities when species i is alone (see eqs. 3). It immediately follows that at equilibrium the ratio of infected to susceptible individuals is

$$I_i^*/S_i^* = r_i/(d_i - e_i) = \hat{I}_i/\hat{S}_i. \tag{8}$$

Therefore, the equilibril ratio of infected to uninfected individuals in each species is independent of the alternative host.

When host 1 is at equilibrium, $dN_1/dt = 0$, and hence $I_1 = S_1 r_1 / (d_1 - e_1)$. Assuming host 2 is present, after substitution into equations (6) we find that a necessary condition for equilibrium in host 1 is

$$r_1 e_1 / (d_1 - e_1) = \beta_{11} I_1 + \beta_{12} I_2$$

which can be compactly rewritten as

$$\hat{I}_1 = I_1 + \frac{\beta_{12}}{\beta_{11}} I_2. \quad (9)$$

Similarly, a necessary condition for host 2 to be in equilibrium is

$$\hat{I}_2 = I_2 + \frac{\beta_{21}}{\beta_{22}} I_1. \quad (10)$$

The equilibril densities of infectives are the simultaneous solutions of (9) and (10):

$$I_1^* = \frac{\hat{I}_1 - (\beta_{12}/\beta_{11})\hat{I}_2}{1 - \beta_{12}\beta_{21}/\beta_{11}\beta_{22}} = \frac{\beta_{22}d_1r_1/(d_1 - e_1) - \beta_{12}d_2r_2/(d_2 - e_2)}{\beta_{11}\beta_{22} - \beta_{12}\beta_{21}} \quad (11)$$

and

$$I_2^* = \frac{\hat{I}_2 - (\beta_{21}/\beta_{22})\hat{I}_1}{1 - \beta_{12}\beta_{21}/\beta_{11}\beta_{22}} = \frac{\beta_{11}d_2r_2/(d_2 - e_2) - \beta_{21}d_1r_1/(d_1 - e_1)}{\beta_{11}\beta_{22} - \beta_{12}\beta_{21}}.$$

These expressions, together with equation (8), specify the full, joint equilibrium.

When do the two species of hosts coexist at this point equilibrium? The necessary conditions for an equilibrium given by equations (9) and (10) can be represented as straight lines in a phase space with axes I_1 and I_2 . In figure 1, we show the three qualitative ways these lines may be drawn; these graphs capture the essence of the three ways in which a shared infectious disease may influence species coexistence. In figure 1A, the lines do not intersect, and the line for host-species 1 lies outside the line for species 2. The algebraic conditions for this to occur are

$$\hat{I}_1 > (\beta_{22}/\beta_{21})\hat{I}_2 \quad \text{and} \quad (\beta_{11}/\beta_{12})\hat{I}_1 > \hat{I}_2. \quad (12)$$

When these inequalities hold, an equilibrium with both species present at constant densities does not exist. Similarly, if the line of species 2 were to lie completely outside the line for species 1, there would be no joint, nontrivial equilibrium. If the two lines intersect, they may do so in two fundamentally different ways. In figure 1B,

$$\frac{\beta_{11}}{\beta_{21}} > \frac{r_1 d_1 / (d_1 - e_1)}{r_2 d_2 / (d_2 - e_2)} > \frac{\beta_{12}}{\beta_{22}} \quad (13)$$

or, equivalently,

$$\frac{\beta_{22}}{\beta_{21}} > \frac{\hat{I}_1}{\hat{I}_2} > \frac{\beta_{12}}{\beta_{11}}. \quad (14)$$

This implies that $\beta_{11}\beta_{22} > \beta_{12}\beta_{21}$.

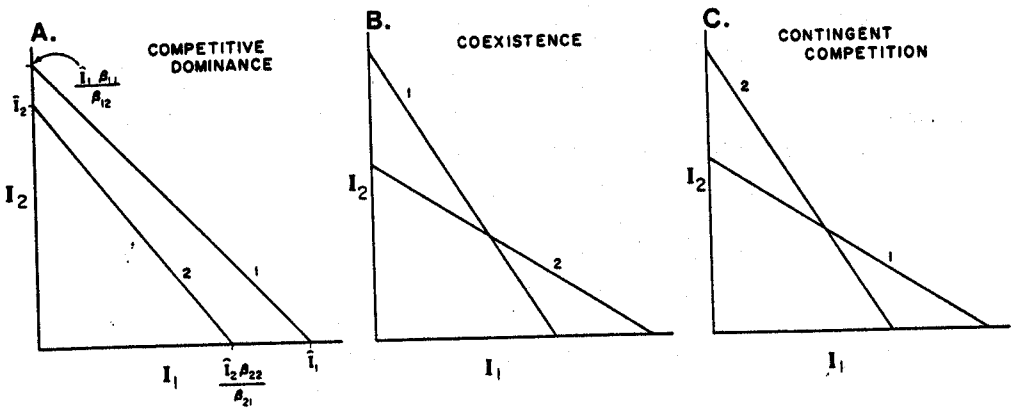


FIG. 1.—Necessary conditions for equilibria in the two-host model. Axes are the density of infected individuals in each host species. Numbered lines demarcate for each host species that combination of densities of infectives, I_1 and I_2 , that permit the species to be in equilibrium (i.e., both zero growth rate and equilibrium incidence of the infectious disease). The quantity \hat{I}_i is the equilibrium density of infected individuals in host species i when isolated from the other host species. The following assertions are justified in the Appendix: A, host-species 1 excludes species 2 (condition 12 in text); B, each species can increase when rare, so coexistence is assured (condition 14 in text); C, either host species can exclude the other, the winner being determined by the initial conditions, i.e., condition (15) in text.

Figure 1C depicts the final possibility,

$$\frac{\beta_{22}}{\beta_{21}} < \frac{\hat{I}_1}{\hat{I}_2} < \frac{\beta_{12}}{\beta_{11}} \tag{15}$$

which can be true only if $\beta_{11}\beta_{22} < \beta_{12}\beta_{21}$. A point equilibrium with both species coexisting exists only if either (14) or (15) holds.

This graphical representation of necessary conditions for coexistence at constant densities in our model is reminiscent of the familiar graphical treatment of coexistence and exclusion in the Lotka-Volterra competition model (see, e.g., Hutchinson 1978). In the Lotka-Volterra model, the growth rate of, say, species 1 is

$$dN_1/dt = r_1N_1(1 - N_1/K_1 - \alpha_{12}N_2/K_1)$$

where r_1 is the intrinsic growth rate of species 1, K_1 is its carrying capacity, and α_{12} is the competition coefficient—a measure of the inhibitory effect of species 2 on species 1, scaled by the effect of species 1 upon itself. Two competing species stably coexist if and only if

$$1/\alpha_{21} > K_1/K_2 > \alpha_{12}.$$

A priority effect is observed if both these inequalities are reversed, and if only one is reversed, one species unilaterally excludes the other.

Comparing this expression with equations (14) and (15), we see that K_i is formally comparable to \hat{I}_i , and α_{ij} to β_j/β_{ii} . The only interactions occurring in our

system are within-species and cross-species infections. From the point of view of species i , the only individuals in species j that matter are infected individuals, of which there are \hat{I}_j at equilibrium when species j is alone. This explains why the quantity \hat{I}_j , rather than total population size, $\hat{N}_j = \hat{S}_j + \hat{I}_j$, might be expected to enter into the criterion for coexistence. The ratio β_{ij}/β_{ii} describes the efficiency with which species i is infected by species j , relative to the efficiency with which species i infects itself. This ratio thus has an interpretation similar to the usual definition of the competition coefficient in the Lotka-Volterra model.

So far, all we have done is to point out some formal analogies between the Lotka-Volterra model of direct competition and our model of two hosts sharing an infectious disease. In graphical treatments of competition theory, the zero-growth isocline of competitor 1 is defined to be that set of joint densities at which $dN_1/dt = 0$, plotted in a phase space with axes (N_1, N_2) , which in the case of the Lotka-Volterra model is the straight line

$$K_1 = N_1 + \alpha_{12}N_2. \quad (16)$$

Although equations (9) (or 10) and (16) have the same linear form, they differ in that (16) is a sufficient condition for competitor 1 to be in equilibrium, whereas (9) is only a necessary condition for equilibrium in host-species 1. One might therefore wonder whether conditions (12), (14), and (15) have any bearing on coexistence at all. For the Lotka-Volterra competition model, the graphical representation of the zero-growth isoclines analogous to figure 1A implies that competitor 1 unilaterally excludes competitor 2, which is to say that competitor 2 is unable to increase when rare and competitor 1 can always invade. Similarly, the representation comparable to figure 1B implies that the two species coexist at a stable point equilibrium, so that each species can increase when it is rare and the other is present at carrying capacity. The figure resembling figure 1C implies that there is a priority effect in which either species can exclude the other, the initial conditions determining the winner. For our model, we examine in the Appendix the stability of each of the two equilibria $(\hat{I}_1, \hat{S}_1, 0, 0)$ and $(0, 0, \hat{I}_2, \hat{S}_2)$. We show that results similar to the Lotka-Volterra model hold: the graphical characterizations presented in figure 1 in fact encapsulate the analytic conditions for sustained coexistence or exclusion. In short, we show that host-species 1 can exclude host-species 2, when condition (12) holds (fig. 1A); that each species can increase when rare, ensuring sustained coexistence, when condition (14) holds (fig. 1B); and that there can be contingent exclusion, in which either host, if already established, can exclude the other through their shared disease, when condition (15) holds (fig. 1C).

This analysis of conditions for invasion does not fully solve the problem of describing the dynamic behavior of the model. It is difficult to exclude, in general, the possibility of limit cycles or other more-complicated kinds of sustained oscillatory behaviors. In extensive numerical simulations, however, such behaviors have not been observed. Moreover, in analyzing species coexistence it may often be adequate to determine the conditions that allow each species to increase when rare, for this ensures that the composition of the community is robust to large perturbations in population densities. Condition (14) compactly describes the

criterion for such robust coexistence in model (6). We now examine some illustrative special cases.

Case 1. Uniform Transmission ($\beta_{11} = \beta_{12} = \beta_{21} = \beta_{22}$)

Uniform transmission of the disease both within and between species implies that sustained coexistence cannot occur unless (from inequality 13)

$$r_1 d_1 / (d_1 - e_1) = r_2 d_2 / (d_2 - e_2)$$

which has essentially zero probability of being true. The host species that maintains the higher density of infectives when alone (its \hat{I}_i) unilaterally excludes the alternative host. This dominance may reflect either a higher intrinsic growth rate, a lower death rate when infected, or a higher rate of recovery of infected individuals. In a like manner, if one species is uniformly more vulnerable to infection than the other (viz., $\beta_{11} = \beta_{12} = \beta$, $\beta_{22} = \beta_{21} = \beta'$, $\beta \neq \beta'$), the species with higher \hat{I}_i always wins. Because \hat{I}_i is inversely dependent on β_{ii} (see 3), one host by virtue of its lower infectivity alone may also be able to exclude an alternative host.

Case 2. High Death or Low Birth or Recovery Rates of Infectives

If the parasite severely reduces fecundity and there is little recovery by infected individuals, such that $d_i \gg e_i$ for both species, condition (13) reduces to

$$\frac{\beta_{11}}{\beta_{21}} > \frac{r_1}{r_2} > \frac{\beta_{12}}{\beta_{22}}.$$

(This is also the condition for coexistence in the special case $d_1/e_1 = d_2/e_2$.) In this limiting case, the parameters d_i and e_i that determine stability in the single-host model drop out of the criterion for coexistence. The relative death rates of infected individuals in the two species are thus irrelevant in determining their coexistence. Even if individuals of species 1 are better able to withstand the disease (i.e., $d_1 < d_2$), species 1 may be eliminated from the community if species 2 has a relatively high intrinsic growth rate ($r_2 > r_1$) or if the transmission efficiency for species 2 is greater for interspecific than for intraspecific transmission (i.e., $\beta_{12} > \beta_{22}$).

Case 3. Symmetrical Transmission

If the two hosts are phylogenetically related and have similar population structures, we might expect within-species transmission to be similar for both (i.e., $\beta_{11} = \beta_{22} = \beta$). If the two species only partially overlap in space, however, then interspecific transmission might be less likely than intraspecific transmission. For simplicity, let interspecific transmission be a fraction E of intraspecific transmission (i.e., $\beta_{12} = \beta_{21} = E\beta$). The quantity E can be thought of as an index of spatial overlap. Figure 2 displays the condition for coexistence for this special case. At high overlap ($E \cong 1$), coexistence requires improbably similar values of \hat{I}_i . By contrast, at low overlap ($E \cong 0$) neither species is likely to exclude the other. This suggests that the coexistence of alternative host species may be promoted by habitat partitioning, which should reduce the rate of between-species infection as compared to within-species infection.

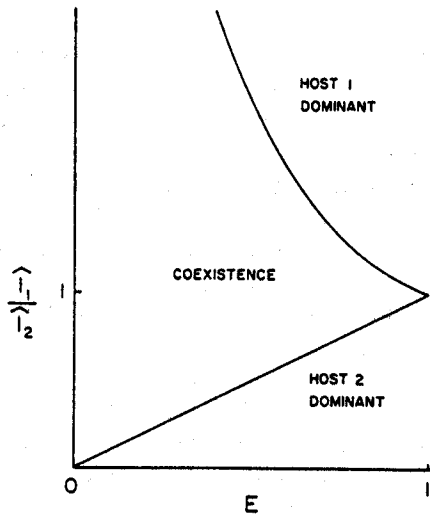


FIG. 2.—Coexistence conditions for symmetrical transmission dynamics. The quantity E scales between-species transmission as a function of within-species transmission (see text). Coexistence requires the ratio \hat{l}_1/\hat{l}_2 to lie within the indicated bounds.

Case 4. Priority Effect

In some circumstances it is reasonable to expect a priority effect to occur. Consider two species with equal intrinsic growth rates and similar demographic responses to an infective disease, so that $\hat{l}_1 = \hat{l}_2$. From (15), either species can potentially exclude the other if $\beta_{11}\beta_{22} < \beta_{12}\beta_{21}$. In the symmetrical case, we let $\beta_{11} = \beta_{22} = \beta$ and $\beta_{12} = \beta_{21} = \hat{\beta}$. The transmission coefficient β_{ij} reflects many facets of the biology of the hosts and the disease organism. If disease transmission is a function of the physical proximity of hosts, individuals might be selected to reduce disease transmission by avoiding each other. If previously isolated host species were to come into contact after a history of selection for intraspecific but not interspecific spacing behavior (e.g., territoriality), cross-species infection might occur more frequently than within-species infection. If so, $\beta < \hat{\beta}$, and the initially rarer species becomes locally extinct. Since each species can preclude invasion by the other, this could lead to parapatric distributions with each species stably excluded from the other's geographic range because of a single infectious disease held in common.

Case 5. Nonregulatory Disease

If the disease cannot regulate either host species ($d_1 < e_1$, and $d_2 < e_2$), then it is obvious that it cannot lead to the exclusion of either host. If the disease regulates species 1 ($d_1 > e_1$) but not species 2 ($d_2 < e_2$) and if $\beta_{12} > 0$, then eventually species 1 will be excluded from the community, while species 2 grows exponentially.

DISCUSSION

We have shown that when an infectious disease is the only factor regulating population size, one host species can exclude another by means of a shared infectious disease. This is true even though both host species, in isolation from each other, are regulated at positive densities by the same disease. The model analyzed here suggests that apparent competitive dominance can result if individuals of one species, as compared to individuals of the other species, have a higher growth rate when uninfected, are less susceptible to becoming infected, or have a higher tolerance to the disease. The higher tolerance to disease of individuals of one species may result from their faster recovery, lower death rates, or higher reproductive rates.

This model leads to several key conclusions that are likely to hold qualitatively for other host-parasite models as well. First, in a given host species the proportion of infected to uninfected individuals—a measure of the prevalence of infection—is, at equilibrium, independent of the presence or absence of alternative hosts. This result generalizes readily to a much broader class of models, including any number of host species and models for transmission other than $\beta_{ij}S_iI_j$. Consider any characterization of between- and within-species transmission rates, such as the $\beta_{ij}S_iI_j$ terms in model (6), consistent with the assumption that the infectious disease can regulate each host population separately (for examples of possible models of transmission rates, see Anderson 1979*b*, 1982; Bailey 1975; Anderson and May 1981; Yorke et al. 1979; but see Getz and Pickering 1983 regarding the relation between the model of transmission and the assumption that infectious disease can regulate host population density). For any variant of model (6) that incorporates such transmission terms (possibly with infections stemming from multiple host species), we can still express the change in the total population density of species i by equation (7). Hence, because equation (8) follows immediately from (7), we conclude that the equilibrium prevalence of infection in species i is independent of the presence of other species sharing the infectious disease. This may be important in interpreting empirical observations, for it suggests that the absence of a positive correlation between infection levels and the presence or abundance of alternative hosts does not preclude the existence of an interaction resulting from a shared infectious disease. Moreover, if the prevalence of infection in a given host species is found to vary in accord with the presence or absence of alternative host species, and the host is at equilibrium, then either per capita birth, or death, or recovery rates must be functions of population size. Thus, if the prevalence of infection differs between areas with and without alternative hosts, we can conclude either that some of the populations are not at equilibrium, or that the infectious disease is not the sole factor regulating population size.

Secondly, the sustained coexistence of alternative hosts regulated solely by a shared disease requires within-species disease transmission to be stronger than between-species transmission (i.e., $\beta_{11}\beta_{22} > \beta_{12}\beta_{21}$). If all the β_{ij} coefficients are equal, then the host that when alone sustains the higher density of infected individuals can exclude the alternative host. As noted above, if infection depends

TABLE 1
A CLASSIFICATION OF TRANSMISSION PATTERNS

	Within-Species Transmission		Between-Species Transmission	
	β_{11}	β_{22}	β_{12}	β_{21}
A. Independent hosts	+	+	0	0
Alternative hosts				
B. Reciprocal interaction	+	+	+	+
C. One-way interaction	+	+	0	+
D. Incidental host	+	0	0	+
E. Alternate hosts	0	0	+	+

on host individuals being in close physical proximity, the relative magnitudes of within- and between-species infection should depend on the degree of spatial overlap between the two hosts. With complete overlap, the probability of within-species infection should approximately equal that for between-species infection. By contrast, with spatial segregation, it seems likely that within-host contacts will be more frequent than between-species contacts, and hence that coexistence will be achieved more readily. Our analysis also raises the possibility that whichever species first occupies an area can exclude the other host species (viz., if $\beta_{11}\beta_{22} < \beta_{12}\beta_{21}$ and the ratio \hat{I}_1/\hat{I}_2 is appropriately bounded, as in condition (15)). These observations suggest that one of the outcomes of a shared infectious disease might be habitat partitioning or nonoverlapping geographical ranges. A number of examples from a wide range of communities implicate parasites in the failure or success of invasions started deliberately or accidentally by humans (Dobson and May, in press).

The importance of the relative magnitudes of within- and between-species transmission suggests a natural classification of the interactions arising between alternative hosts because of infectious disease. Table 1 schematically describes five possible transmission patterns. In A, the two hosts are independent. In B, the disease potentially regulates either host and can lead to the exclusion of one of them. This has been the principal focus of this paper. Categories C and D describe two distinct patterns of amensalism. In C, the disease is regulatory in both hosts if $d_1 > e_1$. Because the interspecific interaction is one-way, however, the condition for coexistence is the single inequality $\beta_{22}\hat{I}_2 > \beta_{21}\hat{I}_1$. In D, individuals of species 2 can be infected by species 1, but the infection is not transmitted through individuals of species 2 at all. Even though the disease cannot regulate species 2 when this incidental host is alone, species 2 may nevertheless be excluded from the community given that $\beta_{21}\hat{I}_1 > r_2/(d_2 - e_2)$; this requires that $d_2 > e_2$. Note that were there within-species transmission in species 2 (i.e., $\beta_{22} > 0$), then species 2 could in fact be regulated by the disease.

Finally, we consider systems involving alternate hosts (category E). A host-parasite system with alternate hosts is one in which the parasite requires the sequential infection of two host species to complete its life cycle. This implies that there is between-species infection, but not within-species infection. For $\beta_{ii} \rightarrow 0$ ($i = 1, 2$), with $\beta_{ij} \neq 0$ ($i \neq j$), the transmission dynamics of our model would seem

to describe a host-parasite system with alternate hosts. For instance, vertebrates and mosquitoes are alternate hosts for the malarial parasite *Plasmodium*, which persists only if both alternate hosts are present. In this case, each species can transmit the infection to the other species, but not to itself. Clearly, when just a single host species is present, the disease necessarily becomes extinct. When the disease has disappeared, the alternate host can then invade. Because the disease persists only if both species are already present, the analysis of conditions for invasion presented in the Appendix is not adequate for characterizing the behavior of the system. Extensive numerical studies of model (6) with alternate hosts suggest that if both hosts are present with the infectious disease, then the joint equilibrium is unstable, and one host eventually is driven to extinction by the shared disease, while the other host grows exponentially. The model thus appears to be inadequate for portraying the stable coexistence of alternate hosts. Yet such host-parasite systems are commonly observed (e.g., malaria, trypanosomiasis, and schistosomiasis). For these systems to stably persist, it would appear that regulatory factors other than a shared infectious disease must influence the dynamics of one or both host species. Alternatively, it is conceivable that more complex epidemic models than the "mass action" model we have used can lead to the stable coexistence of alternate hosts, each regulated by the same disease. Aron and May (1982) reviewed models of the population dynamics of malaria; the transmission term used in these models is indeed quite different from that used in equations (6).

The model presented can be generalized in numerous ways, as Anderson and May (1981) have done for the one-host model given by equations (1). These include incorporating a free-living infectious stage; allowing direct within- and between-species density-dependent mortality or fecundity of the hosts; adding an immune class for vertebrate hosts that recover from infection; altering the functional form for the transmission rate; and considering the effect of interactions among different parasite species within hosts (Toft 1985). Moreover, gene-for-gene interactions and other evolutionary phenomena that are manifestly important in one-host, one-parasite systems (Clarke 1976; Levin and Pimentel 1981; Hamilton 1980, 1982; Anderson and May 1982*b*; Bremermann and Pickering 1983; May and Anderson 1983; Levin 1983) are also likely to be important components for understanding the full significance of infectious disease for species coexistence in natural communities. Such considerations, however, are beyond the scope of this paper. We believe that the relatively simple model developed here concisely illustrates one way in which infectious disease can act as a powerful mechanism leading to the exclusion of species from communities, and to the determination of relative abundances for coexisting host species.

SUMMARY

There is a growing body of evidence suggesting that infectious disease may influence the species composition of natural communities. We examine the effect of a shared infectious disease on species coexistence in a differential equation model that generalizes to two host species, a one-host one-disease model explored by Anderson and May (1979). In the Anderson-May model, the transmission rate

is described by a "mass action" term; there is no acquired immunity; and the infectious disease is the only factor regulating population growth. These assumptions, which are generally more applicable to invertebrate than to vertebrate hosts, are carried over to our two-host model. We show that, just as in the familiar Lotka-Volterra model of direct competition, there are three possible outcomes to the interaction: (1) one host species may unilaterally exclude the other; (2) the two host species may coexist; or (3) either host may exclude the other, with the outcome depending on initial conditions. These outcomes are graphically expressed with isoclines similar to those generated by the Lotka-Volterra model, and the necessary and sufficient condition for species coexistence is given by an expression formally parallel to the coexistence criterion of the Lotka-Volterra model. The numbers of infected individuals sustained by a host species when alone and at equilibrium are shown to be comparable to carrying capacity in the Lotka-Volterra model. Similarly, we show that the ratio of between-species to within-species infection rates is analogous to the competition coefficient.

The model identifies three ingredients that must be assessed to predict the consequences of shared infectious disease for species coexistence: the intrinsic capacity for increase of each host; the per capita birth, death, and recovery rates of infected individuals; and the pattern of within- and cross-species infections. The model also leads to two additional conclusions that appear to apply in a broader range of models. First, given certain assumptions, including the assumption that infectious disease is the only factor regulating population growth, at equilibrium the ratio of infected to uninfected individuals in any particular host species is independent of the presence or absence of alternative host species. Second, the basic model does not lead to stable coexistence for an infectious disease that is only transmitted between host species and not within host species (i.e., host-parasite systems involving alternate hosts, such as infectious diseases carried between definitive hosts by intermediate hosts). The paper concludes with some suggestions about how this model can be extended to a broader class of infectious disease systems.

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APPENDIX

The Jacobian of system (6) is

$$Q = \begin{pmatrix} r_1 - \beta_{11}I_1 - \beta_{12}I_2 & -\beta_{11}S_1 + e_1 & 0 & -\beta_{12}S_1 \\ \beta_{11}I_1 + \beta_{12}I_2 & \beta_{11}S_1 - d_1 & 0 & \beta_{12}S_1 \\ 0 & -\beta_{21}S_2 & r_2 - \beta_{22}I_2 - \beta_{21}I_1 & -\beta_{22}S_2 + e_2 \\ 0 & \beta_{21}S_2 & \beta_{22}I_2 + \beta_{21}I_1 & \beta_{22}S_2 - d_2 \end{pmatrix}. \quad (A1)$$

A full, local stability analysis requires determination of the signs of the eigenvalues of this matrix evaluated at each of the three possible equilibria $(I_1, S_1, 0, 0)$, $(0, 0, I_2, S_2)$, and $(I_1^*, S_1^*, I_2^*, S_2^*)$, where in each case $S_i = I_i(d_i - e_i)/r_i$ (see text for I_i and I_i^*).

To analyze sustained coexistence, we ask whether each species can increase when rare. At the equilibrium $(0, 0, I_2, S_2)$, the matrix (A1) takes the simpler form

$$J = \begin{pmatrix} r_1 - \beta_{12}I_2 & e_1 & 0 & 0 \\ \beta_{12}I_2 & -d_1 & 0 & 0 \\ 0 & -\beta_{21}S_2 & r_2 - \beta_{22}I_2 & -\beta_{22}S_2 + e_2 \\ 0 & \beta_{21}S_2 & \beta_{22}I_2 & 0 \end{pmatrix}. \tag{A2}$$

If one or more of the eigenvalues has a positive real part, then the equilibrium $(0, 0, I_2, S_2)$ is unstable, which is to say that species 1 can invade. Conversely, if all eigenvalues have negative real parts, then the equilibrium is stable and species 1 is excluded from the community. Let $A = J - \lambda I$, where λ is an eigenvalue and I the identity matrix. To find the eigenvalues of J , we solve the determinantal equation $|A| = 0$. Matrix A has the form

$$A = \begin{pmatrix} B & C \\ D & E \end{pmatrix},$$

where the four entries are 2×2 submatrices. It is convenient to note that $|A| = |B| |E - DB^{-1}C|$. In our case, C is the zero matrix; hence, $|A| = |B| |E|$. The eigenvalues of E are those corresponding to species 2 living by itself. These two eigenvalues have negative real parts if $d_2 > e_2$. The remaining two eigenvalues (from B) are the roots of the quadratic equation

$$\lambda^2 + a_1\lambda + a_2 = 0$$

where

$$a_1 = -(r_1 - d_1 - \beta_{12}I_2)$$

and

$$a_2 = -d_1r_1 + \beta_{12}I_2(d_1 - e_1).$$

Both roots are negative if $a_1 > 0$ and $a_2 > 0$; otherwise, one root is positive. The quantity $a_2 > 0$ if and only if

$$d_1r_1/(d_1 - e_1) < \beta_{12}I_2 \tag{A3}$$

or if

$$d_1r_1/\beta_{11}(d_1 - e_1) < \beta_{12}I_2/\beta_{11},$$

which is to say that

$$I_1/I_2 < \beta_{12}/\beta_{11}. \tag{A4}$$

Assume this to be true. We have also assumed that $d_1 > e_1$, i.e., the disease can regulate species 1. This implies that $r_1 < d_1r_1/(d_1 - e_1)$. Together with inequality (A3), this leads to $r_1 - \beta_{12}I_2 < 0$ or to $a_1 = d_1 - (r_1 - \beta_{12}I_2) > 0$. Therefore, if inequality (A4) holds, both a_1 and a_2 are positive, and the final two eigenvalues have negative real parts. Conversely, if (A4) does not hold, $a_2 < 0$, and at least one of the eigenvalues is positive.

In other words, if (A4) holds, species 1 is excluded when rare. If (A4) is false, species 1 can invade. We now repeat this analysis for species 2. We find that species 2 is excluded (i.e., the edge equilibrium $[(I_1, S_1, 0, 0)]$ is stable) if and only if

$$\beta_{22}/\beta_{21} < I_1/I_2. \tag{A5}$$

If inequality (A4) is false but inequality (A5) is true, then species 1 can invade when rare, whereas species 2 cannot. This is the case depicted in figure 1A. Similarly, if inequality (A5) holds but inequality (A4) does not, then species 2 can invade, but species 1 cannot.

These correspond to cases of unilateral dominance of one species by another. If both inequalities (A4) and (A5) are false, coexistence is guaranteed, and each species increases when rare (see fig. 1B).

Finally, if both inequalities (A4) and (A5) are true, then each species, given that it is initially at its solitary equilibrium density, can exclude the other. This contingent competition is represented in fig. 1C.

Manipulation of inequalities (A4) and (A5) leads to conditions (12), (14), and (15) in the text.

This analysis supports our interpretation of the interspecific interaction described by model (6) in terms of the coexistence criterion of the Lotka-Volterra competition model. This of course does not address the issue of possible cyclic behavior in the model's dynamics. In principle, we could determine the local stability of the joint equilibrium (I_1^* , S_1^* , I_2^* , S_2^*) by applying the Routh-Hurwitz criteria to the characteristic equation (a quartic) of the matrix Q . In practice, this procedure leads to cumbersome algebraic expressions. Our working hypothesis is that the simple condition (14) for mutual invasibility is also the necessary and sufficient condition for the local stability of the joint equilibrium. Numerical studies to date strongly suggest (but of course cannot conclusively prove) that this is true.

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