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THE COEXISTENCE OF COMPETING PARASITES. I. THE ROLE OF CROSS-SPECIES INFECTION

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It is widely recognized that both macroparasites and microparasites (in the sense of Anderson and May 1979) can have a considerable impact on host population dynamics (see, e.g., Anderson and May 1979, 1981). The importance of parasites in community dynamics has been widely disputed (Barbehenn 1969; Rohde 1979; D. Brooks 1980; Holmes and Price 1980; Holmes 1982; Freeland 1983; review in Price et al. 1986), but in principle, their effects could greatly influence the structure of species assemblages (Levin 1970).

To this end, theoretical studies (Dobson 1985; Holt and Pickering 1985; Anderson and May 1986; Hochberg et al. 1989) have examined the dynamics of multispecies systems. Two of these studies (Holt and Pickering 1985; Anderson and May 1986) have considered various cases of two host species that share an infectious disease. They found that the parasite can play a substantial role in determining species coexistence or exclusion, whether or not the host species interact in the absence of the parasite. Dobson (1985) has investigated the competitive dynamics of a pair of parasite species sharing a common host. He found that the parasites' pathogenicities, transmission abilities, and frequency distributions among individual hosts are all instrumental in determining the competitive outcome.

Most of the past modeling work on multispecies systems of microparasites (Levin and Pimentel 1981; May and Anderson 1983; Anderson and May 1986) has concentrated on the special case of invariably lethal parasites; infected hosts are often assumed to suffer no loss in reproduction because of the disease. However, many studies of invertebrates indicate that some infected hosts may survive the initial onslaught of the infection (Salt 1970) and/or reproduce at slightly to greatly decreased rates while infected (McLaughlin 1965; Breed and Olsen 1977). Levin (1983*a*, 1983*b*) briefly considered models incorporating this effect and suggested that decreased rates of reproduction of infected hosts and their recovery to the susceptible state might give rise to sustained population cycles.

The models studied by Levin and others (May and Anderson 1983; Anderson and May 1986) are also relevant to the current debate on the relative contributions

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of predation and competition to community structure and dynamics (Connell 1975; Tilman 1982; Schoener 1983; Holt 1984). Multiple parasitism, in which two or more parasite species or strains attack the same host individual, presents a rather intriguing combination of predatory (i.e., consumptive) and competitive effects. When interference competition between parasite species occurs within infected individuals, this may formally provide an example of intraguild predation at the population level (Polis et al. 1989). The interference interaction among parasites that generates such intraguild predation can be parasitic (see, e.g., Siegel et al. 1986; for a review of hyperparasitism, see Sullivan 1987), competitive (see, e.g., Beegle and Oatman 1975; Hochberg, in press), or a combination of the two (Cossentine and Lewis 1988). For instance, Siegel et al. (1986) found that infection by the microsporidian *Nosema pyrusta* of both the braconid parasitoid *Macrocentrus grandi* and its lepidopteran host *Ostrinia nubilalis* resulted in fewer parasitoids surviving to adulthood. Cossentine and Lewis (1988) found that the same microsporidian had no significant effect on the ability of the tachinid parasitoid *Lydella thompsoni* to eclose, but a related microsporidian in the genus *Nosema* did. Apparently, the latter pathogen directly affected the parasitoid larvae, whereas the former did not. Previous theoretical studies (Schoener 1976, 1978; Vance 1985) have recognized that interference interactions (e.g., aggression, intraguild predation) can influence competitive outcomes and even permit the persistence (and hence a higher diversity) of competing species. Here we extend this basic insight to host-parasite systems.

In this article we explore, in more depth, the role of cross-species infection in a multiparasite system, similar to the one examined by Levin (1983*a*, 1983*b*). The models considered here are most appropriate for characterizing microparasitic (including viral, fungal, bacterial, and protozoan) infections of vertebrates or invertebrates; however, the models could be suitably modified to encompass other parasitic taxa (e.g., macroparasites and parasitoids). In particular, we aim to compare and contrast three distinct dynamic roles that a parasite can play: (1) a sort of "predator" of the host, (2) a predator of hosts that harbor a competing parasite (i.e., an "intraguild" predator), and/or (3) a competitor with another parasite for a limiting resource (i.e., healthy hosts). First, we briefly review the dynamics of the one-host, one-parasite model of Anderson and May (1981). We then consider the consequences of invasion by a second parasite species and examine (1) the possible outcomes of invasion, (2) the effects of successful invasion on the abundances of the host and its parasites, and (3) the stability of the two-parasite equilibrium. Finally, we discuss the implications of our results for the structure of simple parasite communities. Throughout the study, emphasis is laid on intuitive explanations of the findings. A separate study focuses on hyperparasitism, in which one parasite can infect hosts only if another parasite species is already present (R. D. Holt and M. E. Hochberg, unpublished manuscript).

ONE-HOST, ONE-PARASITE MODEL

In this section, we briefly review the basic one-host, one-parasite model of Anderson and May (1981). The model does not take explicit account of long-lived

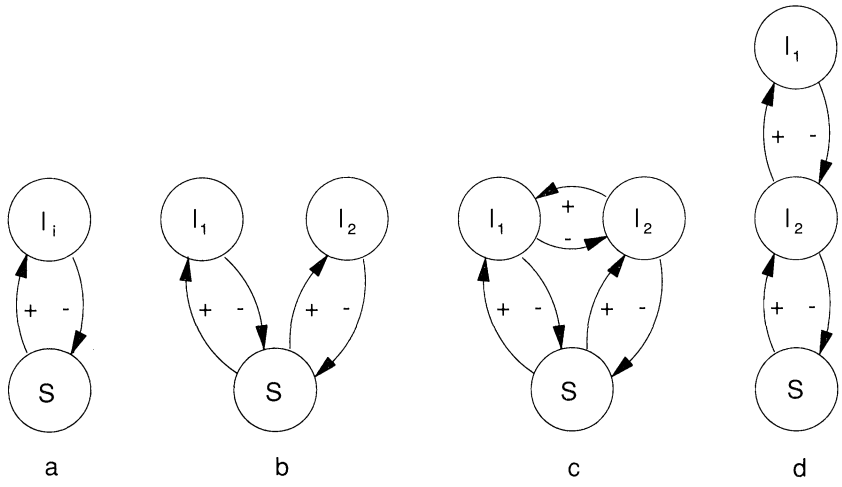

FIG. 1.—Schematic diagrams of some host-parasite interactions: *a*, one host and one parasite; *b*, one-host, two-parasite system with pure exploitative parasitism between the parasites; *c*, one host and two parasites with cross-transmission (facultative hyperparasitism); *d*, obligate hyperparasitism. *S*, Susceptible hosts; I_i, hosts infected with parasite *i*; I_1, hosts infected with parasite 1; I_2, hosts infected with parasite 2.

parasitic stages, latent periods of infection, or immunity. The assumptions of the model are appropriate for many, but by no means all, infections of vertebrates or invertebrates.

Susceptible hosts of density S are assumed to reproduce and die at per capita rates of a and b, respectively. These hosts can be infected, at a per capita rate of $\beta_i I_i$, by parasites transmitted from infected hosts (of density I_i). The term for overall transmission rate, $\beta_i I_i S$, assumes that the population mixes homogeneously and that there are no long-lived external stages of the parasite. Once infected, a host may recover to the susceptible state at a per capita rate of v_i, give birth to susceptible offspring at a per capita rate of a_{i1}, vertically transmit the parasite to its offspring at a per capita rate of a_{i2}, or die from natural or disease-induced causes at per capita rates of b and m_i, respectively. The total per capita rate of reproduction of infected individuals is thus $a_{i1} + a_{i2}$. The basic interaction is schematically illustrated in figure 1*a*.

The model system is

$$dS/dt = (a - b)S + (v_i + a_{i1})I_i - \beta_i I_i S, \tag{1a}$$

$$dI_i/dt = \beta_i I_i S - (b + m_i + v_i - a_{i2})I_i, \tag{1b}$$

which can be further simplified by letting $r = a - b$, $e_i = v_i + a_{i1}$, and $d_i = b + m_i - a_{i2} + v_i$. Here, parameter r is the familiar intrinsic rate of increase of the susceptible host, e_i is a measure of the contribution (by birth or recovery) of parasitized individuals to the healthy subpopulation, and d_i represents the net rate of loss of parasitized individuals. System (1a) and (1b) becomes

$$dS/dt = rS + e_i I_i - \beta_i I_i S, \tag{1c}$$

$$dI_i/dt = \beta_i I_i S - d_i I_i . \tag{1d}$$

Thus, although seven parameters are required to describe the mechanistic details of the system, four compound parameters encapsulate its dynamics. When $a_{i2} = 0$, the expected length of time an individual stays infected is $1/d_i$.

Equations (1c) and (1d) have a unique equilibrium, S_i' and I_i', given by

$$S_i' = d_i/\beta_i , \tag{2a}$$

$$I_i' = rd_i/\beta_i(d_i - e_i) . \tag{2b}$$

The effect of most of the parameter values on the equilibrium levels of the host (given by $S_i' + I_i'$) is straightforward. Increases in r and e_i and decreases in β_i result in higher populations of the host at equilibrium. The effect of parameter d_i, however, is more complicated. Though not elaborated upon here, maximum depression of the total host population occurs for parasites exhibiting intermediate levels of pathogenicity; that is, $d_i \approx e_i + (re_i)^{1/2}$.

The equilibrium exists and is stable if and only if $r > 0$ and

$$d_i > e_i > 0 . \tag{3}$$

In other words, stability requires that the intrinsic rate of increase of the parasitized portion of the host population be negative (i.e., $e_i - d_i < 0$; thus, $b + m_i > a_{i1} + a_{i2}$; the death rate of infected individuals exceeds their own birthrate) and that parasitized individuals contribute to the growth of the susceptible population (i.e., $e_i > 0$) by their recovery or births. Population trajectories converge fastest and without oscillations to the equilibrium point for $d_i \approx e_i$; however, when this limit is exceeded ($d_i < e_i$), the disease no longer regulates its host. If $e_i = 0$ (e.g., because of a low recovery rate), the model leads to neutrally stable dynamics. Indeed, the model in this limit is structurally identical to the classic Lotka-Volterra predator-prey model. The two possible dynamic behaviors leading to a positive host-parasite equilibrium are illustrated in figure 2.

TWO PARASITES COMPETING FOR A SHARED HOST

We now expand the one-host, one-parasite model to two parasite species or strains competing for the same host. The conditions under which one parasite is able to invade and/or resist extinction when competing with the second parasite are characterized, and the consequences of successful parasitic coexistence for the equilibrium host and parasitic abundances and for the long-term dynamics of the system are then considered.

Model Development

The model presented here takes a form broadly similar to the models discussed by Levin (1983*a*, 1983*b*). The basic interactions are the same as for the one-host, one-parasite model, except that in addition to infecting susceptible hosts, one or both of the parasites can be transmitted to hosts infected with the opposing parasite species (fig. 1*c*).

The differential equations for healthy hosts (S), hosts infected with parasite

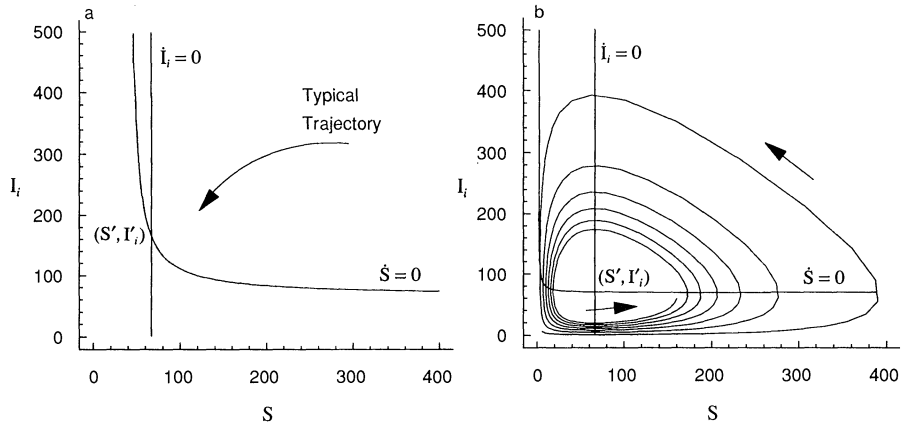

FIG. 2.—Phase-space and typical trajectories for the one-host, one-parasite interaction. *a*, Monotonic damping of the approach to the equilibrium point ($e_i = 0.6$); *b*, cyclic damping ($e_i = 0.02$). Other parameter values: $r = 1$, $\beta_i = 0.015$, $d_i = 1$. $\dot{S} = 0$ and $\dot{I}_i = 0$ are isoclines for susceptible hosts and hosts infected with parasite i, respectively. The host-parasite equilibrium is given by S', I'_i.

species 1 (I_1), and hosts infected with parasite 2 (I_2) take the form

$$dS/dt = rS + e_1 I_1 + e_2 I_2 - \beta_1 I_1 S - \beta_2 I_2 S, \tag{4a}$$

$$dI_1/dt = \beta_1 I_1 S - d_1 I_1 + \delta(\beta_1, \beta_2) I_1 I_2, \tag{4b}$$

$$dI_2/dt = \beta_2 I_2 S - d_2 I_2 - \delta(\beta_1, \beta_2) I_1 I_2. \tag{4c}$$

As before, the parameters r, e_i, and d_i compound several distinct biological processes. The cross-transmission term δ may be, in general, an increasing function of the rate of transmission to susceptible hosts, as represented by $\delta(\beta_1, \beta_2)$. However, the two processes need not be coupled, for example, in obligate hyperparasitism (for which $\delta > 0$ but $\beta_1 = 0$; see fig. 1*d* and, for further discussion, R. D. Holt and M. E. Hochberg, unpublished manuscript). Parameter δ can be interpreted abstractly as a form of predation between the parasites, since (as represented in eqq. [4]) cross-transmission entails the elimination of the inferior parasite from multiply infected hosts. Positive values of δ reflect parasite 1's superiority in cross-transmission, whereas parasite 2 is superior when $\delta < 0$. The above model assumes that one parasite rapidly displaces the other in hosts parasitized by both.

The Outcomes of the Competitive Interaction

A basic quantity characterizing the demographic attributes of a parasite is its basic reproductive rate, denoted by R_i for parasite i. This is defined as the average number of secondary infections produced by a single infected host during the time that the host remains infected (i.e., before dying or recovering; Anderson 1980). Were S constant, an individual infected with parasite i would infect an additional $\beta_i S$ individuals and would stay infected an average length of time $1/d_i$. Hence,

TABLE 1

SOME LIMITING CASES FOR INVASION AND COEXISTENCE OF THE PARASITES ASSUMING THAT $\beta_2 d_1 > \beta_1 d_2$

Case	Invasion by Parasite 1	Invasion by Parasite 2	Coexist?
1. $\delta = 0$	never	always	no
2. $dS/dt = 0$	$1 - \frac{e_2}{d_2} < \frac{r\delta}{\beta_2 d_1 - \beta_1 d_2}$	$1 - \frac{e_1}{d_1} > \frac{r\delta}{\beta_2 d_1 - \beta_1 d_2}$	yes
3. $\frac{e_2}{d_2} = \frac{e_1}{d_1} = \frac{e}{d}$	$1 - \frac{e}{d} < \frac{r\delta}{\beta_2 d_1 - \beta_1 d_2}$	$1 - \frac{e}{d} > \frac{r\delta}{\beta_2 d_1 - \beta_1 d_2}$	no
4. $e_1 = 0$	$1 - \frac{e_2}{d_2} < \frac{r\delta}{\beta_2 d_1 - \beta_1 d_2}$	$1 > \frac{r\delta}{\beta_2 d_1 - \beta_1 d_2}$	yes
5. $e_2 = 0$	$1 < \frac{r\delta}{\beta_2 d_1 - \beta_1 d_2}$	$1 - \frac{e_1}{d_1} > \frac{r\delta}{\beta_2 d_1 - \beta_1 d_2}$	no
6. $e_2 = d_2$	$\delta > 0$	$1 - \frac{e_1}{d_1} > \frac{r\delta}{\beta_2 d_1 - \beta_1 d_2}$	yes
7. $e_1 = d_1$	$1 - \frac{e_2}{d_2} < \frac{r\delta}{\beta_2 d_1 - \beta_1 d_2}$	$\delta < 0$	no
8. $r\delta = \beta_2 d_1 - \beta_1 d_2$	always	never	no
9. $\delta = \beta_1$	$1 - \frac{e_2}{d_2} < \frac{r\beta_1}{\beta_2 d_1 - \beta_1 d_2}$	$1 - \frac{e_1}{d_1} > \frac{r\beta_1}{\beta_2 d_1 - \beta_1 d_2}$	yes
10. $\beta_1 = 0, \delta > 0$	$1 - \frac{e_2}{d_2} < \frac{r\delta}{\beta_2 d_1}$	does not apply	yes
11. $\beta_2/d_2 = \beta_1/d_1$	$\delta > 0$	$\delta < 0$	no

$R_i = \beta_i S/d_i$. Parasite i, when growing alone with the host, depresses host densities to $S = d_i/\beta_i$ (eq. [2a]), at which density the parasite's basic reproductive rate is unity. For simplicity, in our analysis of the two-parasite system, we let parasite 2 be the species with the higher R_i (i.e., $\beta_2/d_2 > \beta_1/d_1$).

If both parasites are present at positive densities—that is, $I_1 > 0$ and $I_2 > 0$—and each is able to regulate the host in the absence of its competitor (i.e., condition 3 holds for parasite 1 and parasite 2 alone with the host; thus, $d_1 > e_1$ and $d_2 > e_2$), we can evaluate the conditions for their sustained coexistence through an analysis of the conditions for invasion. Some special cases are presented in table 1.

Parasite 2 can invade the equilibrium between parasite 1 and the host only if equation (4c) is positive when evaluated at the equilibrium $S = S_1'$, $I_1 = I_1'$, and $I_2 \approx 0$ or when

$$r\delta/(\beta_2 d_1 - \beta_1 d_2) < 1 - e_1/d_1 . \tag{5a}$$

Invasion is always successful if $\delta < 0$; in this case, parasite 2 is the more successful infective agent for both susceptible hosts and hosts already infected with the other parasite. Given that $\delta > 0$, invasion by parasite 2 is promoted by (1) the high pathogenicity of parasite 1 (i.e., low e_1, high d_1), (2) parasite 2's being a sufficiently superior exploiter of susceptible hosts, compared with parasite 1, over

their respective life spans, and (3) a small intrinsic rate of increase of the susceptible host population.

In like manner, if parasite 2 is near its equilibrium with the host, invasion by parasite 1 requires

$$r\delta/(\beta_2 d_1 - \beta_1 d_2) > 1 - e_2/d_2 . \tag{5b}$$

Invasion is impossible if $\delta < 0$. Parasite 1 is more likely to invade if (1) it has a sufficient advantage at interference, (2) its transmission success (as measured by $\beta_2 d_1 - \beta_1 d_2$) is not too different from that of parasite 2, (3) parasite 2 is marginally regulatory, and (4) the intrinsic rate of increase of susceptible hosts is large. In contrast to the case of invasion by parasite 2, increased pathogenicity of the resident parasite may prevent invasion by parasite 1.

Were we to assume that $\beta_2 d_1 < \beta_1 d_2$, note that a system marginally regulated by its parasite (i.e., $e_i \approx d_i$) is virtually impervious to invasion by a competing parasite unless the invader is better both at transmitting infection to healthy hosts and at ousting the competitor from infected hosts (see table 1, cases 6 and 7).

It is useful to consider some special cases of this model (summarized in table 1).

Purely exploitative competition between parasites.—If $\delta = 0$, the parasite species interact only indirectly through their joint exploitation of a shared host population (fig. 1*b;* table 1, case 1). From the above inequalities and the assumption that $\beta_2/d_2 > \beta_1/d_1$, it follows that species 2, if present at equilibrium, excludes species 1 and can invade when it itself is rare. Sustained coexistence is thus impossible. Given that parasite i is present, the equilibrium density of healthy hosts is d_i/β_i. Hence, the parasite that depresses healthy host numbers to the lower level when alone (i.e., has the higher R_i) wins in competition with the alternative parasite. This result parallels the usual interpretation of competitive dominance in exploitative competition for a limiting resource (Tilman 1982), in which the winner is the species that can persist at the lowest resource density.

Steady-state dynamics: effects on healthy hosts.—Assume that the healthy host population equilibrates more quickly than do the infected host populations, such that in equation (4a), $dS/dt \approx 0$ (see table 1, case 2). We do this in order to compare the competitive interaction between the parasites with more traditional analyses of two-species competitive and predator-prey systems. This separation in time scales implies that

$$S(I_1, I_2) \approx S \approx (e_1 I_1 + e_2 I_2)/(\beta_1 I_1 + \beta_2 I_2 - r) . \tag{6}$$

Steady-state assumptions are commonly used in the study of enzyme kinetics (Segel 1988) and multispecies interactions (MacArthur 1970) to illuminate the behavior of complicated dynamic systems.

Expressing the density of susceptible hosts as a function of infected hosts in this fashion makes sense only if $r < \beta_1 I_1 + \beta_2 I_2$. This means that the rate of growth of the healthy population from self-recruitment is less than the rate at which it is becoming infected (the demographic accounting stays in balance because of recoveries and births to infected individuals). The quantity $S(I_1, I_2)$ describes how the availability of susceptible hosts is dynamically influenced by the abundance of the two kinds of infected hosts. Infection by parasite i depresses host availabil-

ity (the terms $\beta_i I_i$ in the denominator), whereas recovery and births enhance host availability (the $e_i I_i$ terms in the numerator). Even though the two parasites clearly compete exploitatively for hosts, this representation of their interaction reveals more complexity than is contained in most models of exploitative competition (e.g., Schoener 1976; Tilman 1982).

If $S(I_1,0) > S(I_1,I_2)$, the net effect of parasite 2 is to decrease the availability of susceptible hosts. This occurs when

$$(e_1I_1 + e_2I_2)/(\beta_1I_1 + \beta_2I_2 - r) < e_1I_1/(\beta_1I_1 - r) \equiv Q(I_1) \tag{7a}$$

or, rearranging terms, when

$$e_2/\beta_2 < Q(I_1)\,. \tag{7b}$$

Conversely, if $S(I_1,0) < S(I_1,I_2)$, parasite 2 enhances host availability, on balance, which requires that

$$e_2/\beta_2 > Q(I_1)\,. \tag{7c}$$

Q declines monotonically with I_1 (for $I_1 > r/\beta_1$) toward the limiting value of e_1/β_1. Similar inequalities describe the impact of parasite 1 on host availability. Manipulating these expressions reveals that parasite 2 always depresses host availability if $e_2/\beta_2 < e_1/\beta_1$, but that if $I_2 > re_1(e_1\beta_2 - e_2\beta_1)$, parasite 1 actually increases the availability of susceptible hosts. Conversely, if $e_2/\beta_2 > e_1/\beta_1$, parasite 1 uniformly diminishes host availability, whereas parasite 2 increases it at sufficiently high densities of I_1. Parallel conclusions follow from an examination of the marginal effect of each parasite on host availability (i.e., the sign of $\partial S(I_1,I_2)/\partial I_i$). The quantity e_i/β_i measures the degree to which the pool of susceptible hosts is replenished from individuals infected with parasite i, scaled against depletion by infection. At sufficiently high I_i, recoveries of and births to infected hosts are the principal source of recruitment into the pool of susceptible hosts. The positive effect of infection on host availability is one-sided; one of the two parasites always depresses healthy-host availability. This component of the interaction thus shifts from competitive $(-,-)$ at a low density of the competing parasite to predatory $(+,-)$ at a high density of one of the parasites. Unlike in most competitive interactions, an increase in the density of one of the "competitors" in infectious-disease systems can (in the short term) *benefit* the other.

Steady-state dynamics: effects on parasitized hosts.—In our model, the complex effects of the parasites on the exploitable resource (i.e., healthy hosts) are overlain by a sort of predator-prey interaction resulting from cross-infection (assuming that parasite 1 is the predator). Substituting approximation (6) into equations (4b) and (4c) leads to

$$\frac{dI_1}{dt} = I_1\left[\frac{\beta_1(e_1I_1 + e_2I_2)}{\beta_1I_1 + \beta_2I_2 - r} - d_1 + \delta I_2\right], \tag{8a}$$

$$\frac{dI_2}{dt} = I_2\left[\frac{\beta_2(e_1I_1 + e_2I_2)}{\beta_1I_1 + \beta_2I_2 - r} - d_2 - \delta I_1\right]. \tag{8b}$$

At low values of I_1 and I_2, the overall interaction between the parasites is clearly competitive $(-,-)$; at high densities, it becomes predatory $(+,-)$.

The isoclines of this model are hyperbolas. The examples depicted in figure 3 combine elements of standard textbook representations of competitive and predator-prey interactions (figs. 3*a*,*b*). In each case, the isocline of species 2 has a negative slope. At the limit $\delta = 0$ (purely exploitative competition), the isoclines are nonintersecting straight lines with negative slopes (fig. 3*a*). At the limit $\beta_1 = 0$ (obligatory hyperparasitism), the isocline of parasite 1 is perpendicular to the I_2-axis, emanating from $I_2 = d_1/\delta$ (fig. 3*b*). In the intermediate cases, with both $\delta > 0$ and $\beta_1 > 0$ (figs. 3*c*–*f*), the isocline of species 1 often shows two distinct behaviors, having a negative slope at low densities of parasite 2 (i.e., like a standard competition isocline) but bending into an isocline of positive slope at high densities of parasite 2 (as in a predator's isocline). For instance, in figure 3*c*, parasite 1 unilaterally excludes species 2, regardless of initial densities. However, the population trajectories differ from those expected given either pure predation or pure competition. At low densities of parasite 2, parasite 1 in effect attacks only susceptible hosts. Because parasite 2 is a superior exploiter of healthy hosts, small increases in its density depress the growth rate of parasite 1. As parasite 2 increases in density, there are fewer healthy hosts available, and the ability to cross-infect enjoyed by parasite 1 becomes relatively more important. Lowering the carrying capacity of parasite 1, or increasing that of parasite 2, may lead to sustained coexistence at a stable node (fig. 3*d*).

Sometimes the isocline of parasite species 1 breaks into two disjunct pieces, one with a negative slope, the other with a positive slope. Figure 3*e* shows an example of this, further illustrating a case in which initial densities determine which of the two parasites excludes the other. And finally, combinations of parameters can lead to a stable point equilibrium and to species coexistence in this reduced system (eqq.[8]) yet imply an unstable point equilibrium in the original three-species system (eqq.[4]; fig. 3*f*). Nonetheless, the collapsed system suggests some of the range of behaviors to be expected in the full model (e.g., oscillations).

The Conditions for Sustained Coexistence

Figures 4 and 5 show various possible outcomes of the three-species system. The conditions under which both parasites simultaneously invade the system can be compactly restated from inequalities (5a) and (5b) as (fig. 4, *region D;* fig. 5*b*):

$$1 - e_2/d_2 < r\delta/(\beta_2 d_1 - \beta_1 d_2) < 1 - e_1/d_1 . \tag{9}$$

This also ensures the existence of a unique three-species equilibrium point:

$$S^* = (d_1 e_2 - d_2 e_1)/\Omega , \tag{10a}$$

$$I_1^* = \tau_1/\Omega , \tag{10b}$$

$$I_2^* = \tau_2/\Omega , \tag{10c}$$

where for notational convenience

$$\Omega = \beta_2(d_1 - e_1) - \beta_1(d_2 - e_2) - r\delta , \tag{10d}$$

$$\tau_1 = rd_2 - (\beta_2 d_1 - \beta_1 d_2)(d_2 - e_2)/\delta , \tag{10e}$$

$$\tau_2 = (\beta_2 d_1 - \beta_1 d_2)(d_1 - e_1)/\delta - rd_1 . \tag{10f}$$

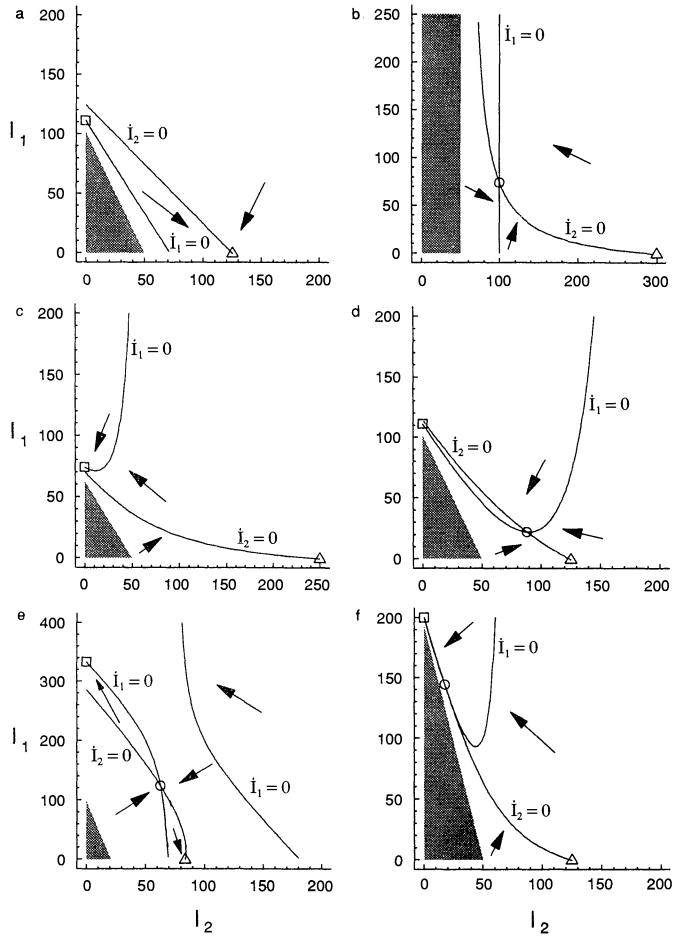

FIG. 3.—Phase diagrams of dynamic outcomes of one-host, two-parasite interaction in the I_2, I_1 plane when $dS/dt = 0$. *a,* Standard competitive interaction resulting in parasite 2 competitively excluding parasite 1 ($\delta = 0$); *b,* standard predator-prey interaction resulting in coexistence ($e_2 = 0.8$, $\beta_1 = 0$, and $\delta = 0.01$); *c,* unilateral exclusion of parasite 2 by parasite 1 ($e_1 = 0.7$, $e_2 = 0.4$, and $\beta_2 = 0.05$); *d,* coexistence at constant population densities ($\delta = 0.005$); *e,* exclusion of parasite 2 based on initial population densities ($e_1 = 0.7$ and $e_2 = 0.4$); *f,* stable point in the reduced system (this figure and eqq. [8a] and [8b]) but unstable point in the three-species system (eqq. [4]; $e_1 = 0.001$, $\beta_1 = 0.005$, and $\delta = 0.014$). Isoclines for infected hosts ($\dot{I}_1 = 0$ and $\dot{I}_2 = 0$ for parasites 1 and 2, respectively) and typical trajectories (given by arrows) are based on equations (8a) and (8b). *Shaded areas,* Regions of infeasibility under the assumption that $dS/dt = 0$. The equilibrium of each parasite in the absence of the other: *open square,* host and parasite 1 $(0, I_1')$; *open triangle,* host with parasite 2 $(I_2', 0)$; *open circle,* the equilibrium between both parasites (I_2^*, I_1^*). Parameter values, unless otherwise specified, $r = 1$, $d_1 = 1$, $d_2 = 1$, $e_1 = 0.1$, $e_2 = 0.6$, $\beta_1 = 0.01$, $\beta_2 = 0.02$, and $\delta = 0.004$.

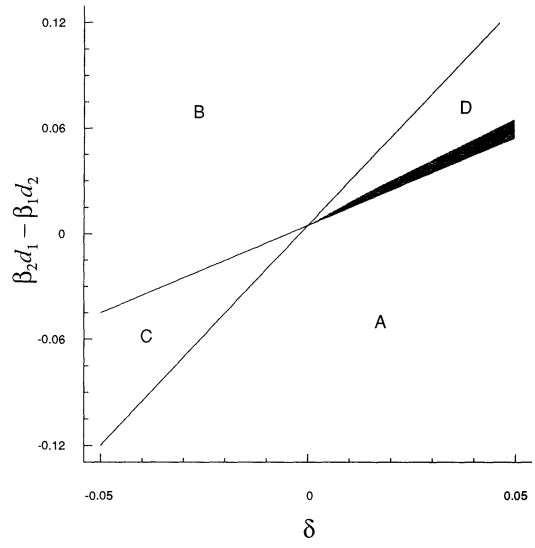

FIG. 4.—Effects of net interference of parasite 1 (δ) and net exploitative advantage of parasite 2 ($\beta_2 d_1 - \beta_1 d_2$, with β_1, d_1, and d_2 held constant) on the outcome of competition between two parasites when each regulates the host in the absence of the competitor. *A*, Parasite 1 wins; *B*, parasite 2 wins; *C*, competition contingent on initial densities; *D*, coexistence at constant densities. *Shaded region*, Parameter space for which an unstable point equilibrium occurs, resulting in persistent oscillations. Parameter values: $e_1 = 0.001$, $\beta_1 = 0.005$, $e_2 = 0.6$. Parameters r, d_1, and d_2 are each set to unity.

Note that the positivity of the constants τ_1 and τ_2 is equivalent to satisfying the conditions for invasion, inequalities (5a) and (5b), respectively.

A necessary condition for inequalities (9) to hold is that

$$e_2/d_2 > e_1/d_1 . \tag{11}$$

Thus, for coexistence, the parasite with the higher R_i must be less pathogenic than its competitor. Moreover, given our assumption that $d_1/\beta_1 > d_2/\beta_2$,

$$\delta > 0 \tag{12}$$

is also necessary for coexistence. The condition $d_1/\beta_1 > d_2/\beta_2$ means that one species is superior at exploiting the healthy host population, whereas the other is superior at cross-infecting individuals already infected. This represents a kind of niche partitioning between the two parasites.

Since the quantities $1 - e_1/d_1$ and $1 - e_2/d_2$ are constrained to be between 0 and 1 (because each parasite is assumed capable of regulating the host), the ratio in the center of inequality (9) is likewise constrained by

$$r\delta < \beta_2 d_1 - \beta_1 d_2 . \tag{13}$$

In other words, the advantage enjoyed in infecting healthy hosts by parasite 2 must sufficiently offset the interference advantage that parasite 1 possesses.

Finally, coexistence requires intermediate values for the intrinsic rate of in-

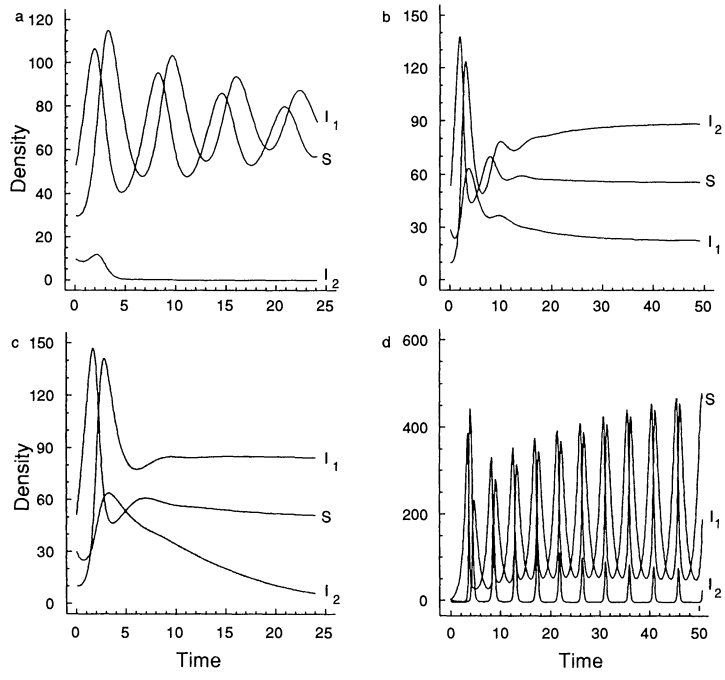

FIG. 5.—Population dynamics of the three-species system. *a*, Parasite 1 excludes parasite 2; *b*, coexistence at constant population densities; *c*, parasite 1 excludes parasite 2 on the basis of initial densities ($S = 50$, $I_1 = 30$, and $I_2 = 10$); *d*, persistent oscillations. Parameter values as for the respective cases in fig. 3*c–f*. *S*, Susceptible hosts; I_1, hosts infected with parasite 1; I_2, hosts infected with parasite 2.

crease of the susceptible hosts, or

$$(1 - e_2/d_2)(\beta_2 d_1 - \beta_1 d_2)/\delta < r < (1 - e_1/d_1)(\beta_2 d_1 - \beta_1 d_2)/\delta. \qquad (14)$$

The parameter r can be thought of as a measure of host productivity. Thus, for this system, parasite species' richness is maximal at intermediate levels of host productivity.

Note that if only one of the inequalities (9) holds, then one parasite unilaterally excludes its competitor (fig. 4, *region A* or *B;* fig. 5*a*). The winner depends on which of the inequalities holds. For instance, if inequality (5b) is true and inequality (5a) is false, parasite 1 always eliminates parasite 2 from the system. If both conditions (5a) and (5b) are violated, then the system is reciprocally impervious to invasion, and the winner depends on the initial population densities (fig. 4, *region C;* fig. 5*c*).

If parasites differ in their transmission rates but not in their demographic impact on the hosts (i.e., $\beta_2 > \beta_1$, $\delta > 0$, $d_1 = d_2$, $e_1 = e_2$), then from inequalities (9) there can be no coexistence. One simple prediction of the model is that if two parasite strains coexist in a single host population, the parasite that is better at cross-infection should be more virulent (as measured by e/d) and have a lower R_i.

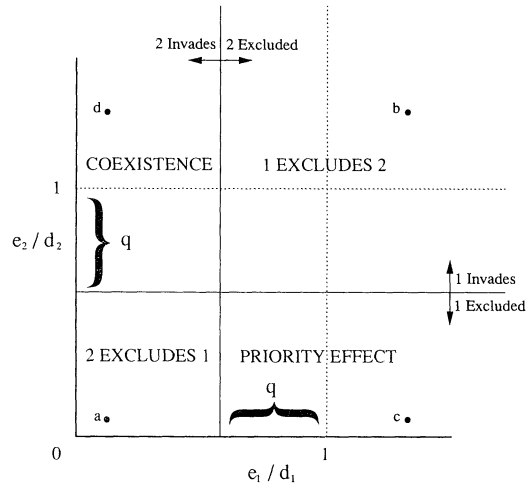

FIG. 6.—Possible states of the one-host, two-parasite system. The quantity $q = r\delta/(\beta_2 d_1 - \beta_1 d_2)$ and is constrained to the bounds of zero and unity. If $q > 1$, then parasite 1 always excludes parasite 2. *Dotted lines,* limits of parameter combinations in which the parasites can stably regulate the host population ($d_1 > e_1 > 0$). *a,* The host can be regulated by either parasite but shows weakly damped convergent oscillations to the point equilibrium. *b,* The host cannot be regulated by the parasites. *c,* Parasite 2 can regulate the host, but parasite 1 cannot. These roles are reversed at point *d.* (See the text for further discussion.)

The quantity $q = r\delta/(\beta_2 d_1 - \beta_1 d_2)$ in inequalities (9) compounds host productivity and parasite cross-infectivity, scaled against a measure of the relative impact each parasite has on the susceptible host population. Figure 6 shows the various outcomes of this model stated in terms of q. As shown there and discussed below, the rules describing the possible long-term state of the system also apply when the host is not stably regulated by the parasite.

As in the familiar Lotka-Volterra model of competition, one parasite may in general unilaterally exclude the other, the two may coexist, or there can be a priority effect such that either parasite can exclude the other if it is initially present in equilibrium with the host. Parasite coexistence hinges on three factors: (1) host productivity must lie in an intermediate range (at low r, the parasite better at direct exploitation of the host dominates; at high r, the parasite better at within-host competition wins); (2) the parasite that is more effective at cross-infection must be less effective at depressing the abundance of healthy hosts (i.e., have a lower R_i); (3) this parasite must also be more pathogenic (i.e., have a lower e_i/d_i). By contrast, a priority effect is seen at intermediate host productivities if the parasite with a lower R_i is less pathogenic.

The Consequences of Invasion for Species Abundance

It is useful to consider how the equilibrium densities of the host and its parasites depend on the model. Several examples are shown in figure 7. We see that the populations of parasite 1 and the healthy host have similar reactions to many parameter changes: both benefit from increases in r, β_1, d_2, δ, e_1, and e_2 and from

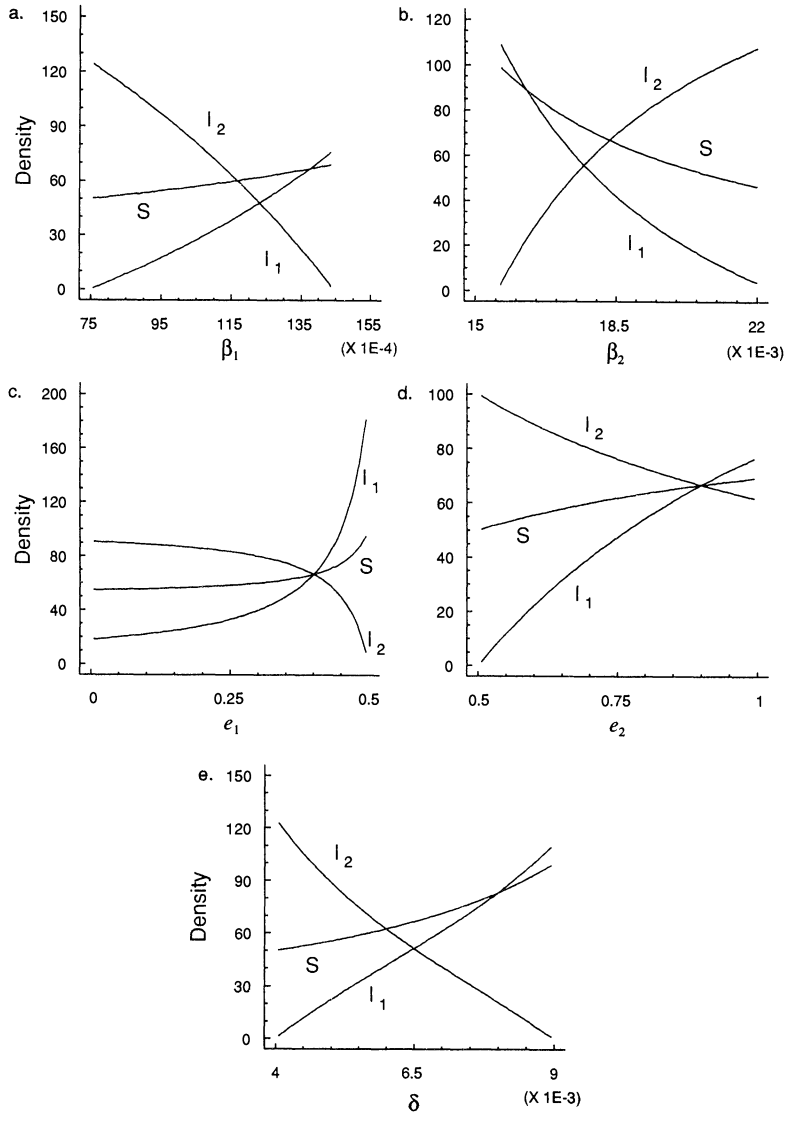

FIG. 7.—Effects of parameter values on the equilibrium abundance of host and parasites. *a*, Transmission efficiency of parasite 1; *b*, transmission efficiency of parasite 2; *c*, recovery and reproduction of hosts harboring parasite 1; *d*, recovery and reproduction of hosts harboring parasite 2; *e*, net interference benefit for parasite 1. Parameter values common to all figures as for fig. 3*f*. *S*, Susceptible hosts; I_1, hosts infected with parasite 1; I_2, hosts infected with parasite 2.

decreases in β_2 and d_1. The population of parasite 2, the dominant parasite in terms of exploitative competition, reacts to these changes in just the opposite way. Some interesting patterns emerge from these figures. Variation in β_1, for instance, has a negligible effect on the density of healthy hosts but sharply influences the relative density of the two parasites (fig. 7*a*).

We can restate condition (9) in terms of the equilibrium densities of the single- and multiple-parasite systems to give

$$I_1'/S_1' < (I_1^* + I_2^*)/S^* < I_2'/S_2'. \tag{15}$$

This implies that, in the absence of a competitor, the parasite with the greater transmission rate will always be more prevalent than the better interferer (i.e., cross-transmitter). When the parasites coexist at a point equilibrium, the total prevalence of infection is intermediate to the equilibrium single-species prevalences.

Alternatively, condition (9) can be expressed in terms of susceptible host densities at equilibrium. Coexistence implies that

$$S_2' < S^* < S_1'. \tag{16}$$

The invasion of parasite 2 into a host population regulated by parasite 1 always depresses the equilibrium of susceptible hosts, whereas, by contrast, the susceptible-host density increases if parasite 1 invades a system regulated by parasite 2.

Invasion also entails a decrease in the density of the initially resident parasite, regardless of which of the two parasites is the invader. This is intuitively obvious when parasite 1 invades, since the better interferer usurps hosts from the population of parasite 2. For this same reason, one might expect the density of parasite 1 to increase upon invasion by parasite 2. However, because of parasite 2's advantage at transmission ($\beta_2 d_1 > \beta_1 d_2$), parasite 1 has fewer total hosts to exploit. The net effect of parasite 2 is thus to depress the population of parasite 1. Hence, when examined at equilibrium, this system is clearly competitive in the usual sense of the word, in that each parasite population depresses the abundance of the alternative parasite, even though there are density combinations away from equilibrium in which one parasite benefits from the other (see above).

The Stability of the Three-Species Equilibrium

Through numerical studies of the local stability criterion (see the Appendix), we found that the equilibrium point defined by equations (10) may be unstable, giving rise to what appear to be limit cycles (see figs. 4, 5*d*, 8). More complicated oscillatory behavior (e.g., chaos) was not observed.

Limit cycles tend to occur if parasite 1 is at the limit of being able to regulate the host in the absence of parasite 2, or $e_1 \approx 0$. When parasite 2 is added to this interaction, persistent cyclic behavior results if parasite 2 has a slow but positive growth rate, such that $r\delta \approx \beta_2 d_1 - \beta_1 d_2$; or, in other words, the behavior becomes cyclic as the destabilizing competitor (parasite 1) comes to dominate the system.

The approximate criteria for an unstable three-species equilibrium leading to limit cycles can be expressed as

$$e_1 \approx 0 \tag{17a}$$

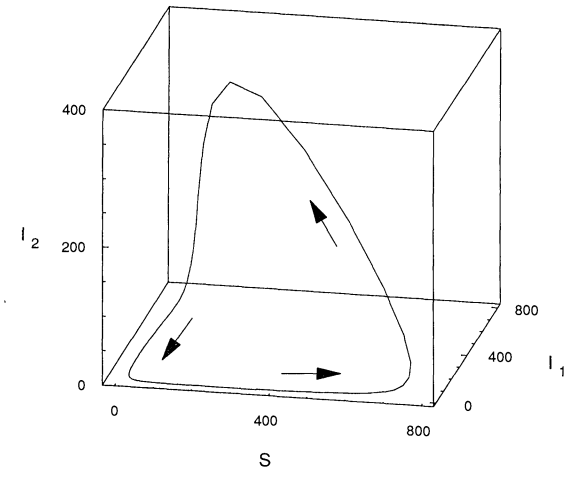

FIG. 8.—Final trajectory (approximate) of limit cycle in three-dimensional space. Parameters as for fig. 3*f*. *S*, Susceptible hosts; I_1, hosts infected with parasite 1; I_2, hosts infected with parasite 2.

and

$$\delta/d_1d_2 \approx \beta_2/rd_2 - \beta_1/rd_1 . \tag{17b}$$

Thus, the advantage of parasite 1 at cross-transmission must be comparable in magnitude to parasite 2's superiority at transmission. As the difference in the transmission efficiencies of the two parasites grows, so too does the requirement that the transmission efficiency of parasite 1 to hosts infected with parasite 2 (i.e., δ/d_1d_2) exceed the transmission efficiency of parasite 1 to healthy hosts (i.e., $\beta_1 rd_1$).

Alternatively, limit cycles ensue if the net effect of each parasite on the population of susceptible hosts is negative and if the links between the host and each of the parasites individually are stronger than the links among the three species taken together. These conditions are elaborated upon in the Appendix.

The oscillations produced by the model can be understood intuitively as follows. The pathogenic parasite, parasite 1, depletes both of its resources (i.e., healthy hosts and hosts that harbor parasite 2) and then declines to densities that are too low to regulate the host. The host is then able to escape temporarily the regulatory influence of its parasites and grows at a nearly exponential rate. This eruption of the susceptible host population is not immediately checked because hosts infected by parasite 2 can be quickly cross-infected by parasite 1. Because parasite 2 is better at directly infecting susceptible hosts, this reduces the regulatory potential of the combined parasite populations. The population of susceptible hosts is then ultimately depleted by the combined actions of parasite 2 and parasite 1. Our numerical studies suggest that these cycles are stable limit cycles, although the possibility remains that more complex dynamic behavior could occur.

Nonstable Systems

In the above analyses, we assumed that each pathogen could, when alone, regulate the host population at a locally stable equilibrium, which is to say that $d_i > e_i > 0$. This assumption could be violated in either of two ways: the equilibrium might exist but not be locally stable (i.e., $e_1 = e_2 = 0$), or the parasite might not be able to check the growth of its host (i.e., $d_i < e_i$). We consider each of these in turn.

Neutrally stable one-host, one-parasite systems.—When $e_1 = e_2 = 0$, the system reduces to the classic Lotka-Volterra predator-prey model. This model exhibits neutrally stable oscillations, a behavior that disappears with slight changes in the model's structure. This structural instability makes the model unsuitable as a subject for exhaustive analysis, but this limiting case does highlight several factors likely to be important in understanding the coexistence or exclusion of parasites. When the quantities e_i are quite small (e.g., as at point *a* in fig. 6), the algebra presented above suggests that if $q < 1$, parasite 2 excludes parasite 1, whereas if $q > 1$, parasite 1 excludes parasite 2. Using the time-averaging technique of Puccia and Levins (1985), one can readily show that if $q < 1$ and parasite 2 and the host are fluctuating cyclically around the point equilibrium, averaged over the cycle, parasite 1 declines when rare; by contrast, parasite 2 shows a positive per capita rate of increase averaged over the cycles exhibited by parasite 1 and the host. Thus, parasite 2 unilaterally excludes parasite 1. This dominance is reversed if $q > 1$. Thus, the conditions for invasion derived assuming that the resident species are at equilibrium also describe the long-term behavior of the system away from equilibrium.

This special case of the model suggests that parasite coexistence is unlikely when the pathogens greatly depress host fecundity and prevent host recovery. It is somewhat counterintuitive that the recruitment of healthy hosts (by recovery or birth) from infected hosts may be a necessary ingredient in parasite coexistence.

These statements about coexistence apply to closed systems viewed over the long term. If the system is away from equilibrium for small e_i, it shows a long period of gradually damped oscillatory behavior, with fluctuations in both total host numbers and disease prevalence. There can be transient phases when one parasite increases following invasion, even though it will ultimately be excluded from the local community near equilibrium. Such nonequilibrium behaviors open the possibility of persistence of an inferior local competitor as a result of dispersal among populations in a metapopulation of local host-parasite interactions, each occasionally perturbed from equilibrium by localized disturbances.

Nonregulatory parasites.—When neither parasite can regulate the host, total host population size ($N = S + I_1 + I_2$) increases in an unbounded fashion. If for parasite *i*, $e_i > d_i$, it cannot stabilize the host population. Anderson and May (1981) showed that in this case the host population asymptotically grows exponentially, with an ever-increasing fraction of the hosts being infected; the number of healthy hosts, however, is bounded. Consider point *b* in figure 6, which corresponds to the case for which neither parasite can regulate the host. If parasite 1 is initially rare and if the host and parasite 2 are growing unchecked, eventually

there will be a density of hosts infected with parasite 2 above which parasite 1 has a positive per capita growth rate, irrespective of the density of healthy hosts. By contrast, if parasite 2 is present, eventually the density of healthy hosts will reach an asymptote, whereas the density of hosts infected with parasite 2 continues to increase; this implies a continual increase in the rate at which parasite 2 is supplanted by parasite 1, without a corresponding increase in infections of healthy hosts by parasite 2. This suggests that parasite 1 supplants parasite 2 in this unstable host-parasite interaction.

Now consider point *c* in figure 6. Once again, species 1 does not regulate the host and, when initially present with the host, can increase to a density such that species 2 is excluded. If species 2 is initially present at equilibrium with the host, however, there are too few of it present to allow invasion by species 1. Thus, the system can exist in either of two alternative states: parasite 1 and the (total) host population growing exponentially, with parasite 2 excluded, or parasite 2 and the host at a stable equilibrium, with parasite 1 excluded.

Finally, consider point *d* in figure 6. If parasite 2 is alone with the host, the system is unstable; parasite 1 eventually invades when parasite 2 reaches sufficiently high densities. Conversely, if parasite 1 is alone with the host at their joint, locally stable equilibrium, parasite 2 can invade. Hence, the two parasites should be able to coexist. Elsewhere (R. D. Holt and M. E. Hochberg, unpublished manuscript), we discuss in more detail how, in this case, adding a second parasite can either destabilize or stabilize a one-host, one-parasite interaction.

The essential point to emerge from these observations is that the criteria demarcating the four possible states of the system (fig. 6) appear to be independent of the parasites' abilities to regulate the host population. Of course, if parasites are nonregulatory in a natural host population, their interaction must overlie other forms of density dependence, such as food limitation, that are responsible for host regulation; this is likely to modify the conditions for parasite coexistence and exclusion.

DISCUSSION

We have shown that the outcome of competition between two parasite species or strains depends on their respective transmission abilities, pathogenicities, and degree of interference within the host individual. Although the models considered in this study are most appropriate for microparasitic infections (in the sense of Anderson and May 1979), the general results are likely to apply more broadly to a wide range of parasitic taxa (e.g., helminths, parasitoids).

The invasion of a competing parasite into an established host-parasite system may result in one parasite's unilaterally excluding the other from the system, stable or oscillatory persistence of both parasites, or competitive exclusion of either parasite with the winner contingent on initial population densities. Our analyses extend and generalize the results of other studies on multiparasite systems (Levin and Pimentel 1981; Levin 1983*a*, 1983*b*; May and Anderson 1983; Dobson 1985; Anderson and May 1986). For instance, persistent oscillations in the three-species system are feasible if one parasite acts as a sort of intraguild

predator of the other parasite, as well as a predator (or pathogen) of healthy hosts. Our results indicate that high pathogenicity (in terms of low reproductive and recovery ability on the part of diseased hosts) helps an easily transmitted parasite to exclude competitively the less readily transmitted parasite, whereas the better competitor within an individual host is more apt to exclude the easily transmitted parasite from the system if its own pathogenicity is low.

The importance of low pathogenicities as a regulatory factor in simple host-parasite models has been stressed in previous theoretical studies (e.g., Anderson and May 1981). For many invertebrate diseases, total recovery by infected individuals or the ability to reproduce is not possible. Under such circumstances, the single-parasite model with $e \approx 0$ predicts that neutrally stable cycles should ensue. Of course, other modifications of the model not considered here—such as a more realistic structure of the parasite population (Hochberg 1989), nonlinear rates of transmission (M. E. Hochberg, unpublished manuscript), or the presence of refractive states of the host (Brown 1984)—could damp the oscillations in the system.

More generally, for competitive coexistence to occur, one parasite must be better at transmission, whereas the competing parasite must dominate at interference competition within multiply infected hosts. This result is similar to studies that have focused on species coexistence in parasitoid guilds (e.g., Force 1970; Hassell 1978; Zwölfer 1979; Price 1980; May and Hassell 1981), in which some parasitoids are better extrahost competitors (e.g., they have higher encounter rates with healthy hosts), and others are better intrahost competitors (e.g., they are better at ousting competitors from within the host). Unlike parasitoids, however, true parasites need not kill their hosts to complete their life cycle; hence, host reproduction and/or recovery from infection is possible. Thus, low to moderate parasite pathogenicity blurs the precision of this intrahost/extrahost trade-off. The addition of nonrandom distributions of parasites among hosts (Dobson 1985), or complex structures of the parasite population (Hochberg 1989; Hochberg et al. 1989), could permit competitive coexistence in cases in which one parasite species is both the better intrahost and extrahost competitor. Therefore, we would expect that a simple trade-off between intrahost and extrahost competitive abilities is unlikely to be a strict requirement for coexistence in natural parasite communities.

The interference interaction considered here is an interesting example of facultative hyperparasitism, analogous in some ways to intraguild predation (Polis et al. 1989). Such an interaction could manifest itself in a number of ways. For instance, immunosuppression by one parasite may permit the establishment within the host of a second opportunistic parasite (Roitt 1976; Jenkins and Behnke 1977; Cox 1978). The outcome of competition may depend on which parasite was present first (Kennedy 1980) and/or which one completes its development first (D. Levin et al. 1981). In addition, there is a plethora of examples of invertebrate host-microparasite-parasitoid interactions in which the presence of one of the competitors within the host can influence the ability of the second competitor to infect the host (Weseloh et al. 1983) and survive within and successfully exit from the host (W. Brooks 1973; Irabagon and Brooks 1974; Beegle and Oatman 1975; Powell

et al. 1986; Cossentine and Lewis 1988). For instance, Beegle and Oatman (1975) found that the competitive outcome between the ichneumonid endoparasitoid *Hyposoter exiguae* and a nuclear polyhedrosis virus (NPV) within larvae of the lepidopteran host *Trichoplusia ni* depended on the relative timing of attacks. If the NPV infected the lepidopteran host before parasitization, then the parasitoid larvae always succumbed to infection along with the host. (In these cases, the virus directly affected parasitoid development.) But if the virus entered the host after the parasitoid, then some or all of the parasitoids could survive. As revealed by our analysis, even if the direct interaction between the parasites appears to be predatory, the total population interaction becomes truly competitive through indirect interactions with healthy hosts.

Another interesting result of our analysis is that priority effects may dominate some of the invasion parameter space (fig. 4, *region C*). Such effects may be crucial in determining system structure. Recently, several studies have shown that in the early stages of habitat colonization, the particular set of random events that occurs (i.e., invasions of particular species) may determine the successional progression and climax structure of that community (Robinson and Dickerson 1987). The models considered here can give rise to this same effect, suggesting that the order of parasitic invasions may be crucial in determining the final community. For such a priority effect to be feasible, the better-transmitting parasite also has to be more pathogenic than the better interferer. It is also possible that the invasion of an additional host species into the simple system considered here could change a mutually invadable interaction between the parasites into the exclusion of one of the parasites, either unilaterally or contingent on the population densities at the time of invasion. This basic idea has recently been explored for systems in which two competing or noncompeting hosts share an infectious disease (Holt and Pickering 1985; Anderson and May 1986).

In studying these models of a simple two-parasite system, analytical tractability was maintained through the omission of a wide array of other potentially important factors, such as nonrandom transmission, long-lived parasitic stages, host immunity, and vector dynamics. Several of these have already been considered in detail for one-host, one-parasite interactions (Anderson and May 1981) but have not as yet been applied to multispecies systems. For instance, preliminary analysis of a model in which the inferior transmitter produces long-lived external stages suggests that cross-transmission is still necessary for both of the parasites to coexist. In other cases, when one of the parasites is an obligate hyperparasite, the introduction of the hyperparasite into a primary parasite-host system may result in the damping of intrinsic oscillations or in the destabilizing of an otherwise stable system (R. D. Holt and M. E. Hochberg, unpublished manuscript). We believe that these and other extensions would yield a wealth of insight into the roles that parasites may play in population and community dynamics.

SUMMARY

Both theoretical and empirical evidence suggest that infectious disease can be a major determinant of the dynamics of host populations and, more broadly, of the

species composition of plant and animal communities. Previous studies of host-parasite systems have, for the most part, laid emphasis on epidemiological interpretations of the host-parasite interactions. In this article, we extend previous models of multiparasite systems and relate these models to general concepts of predator-prey and competitive interactions. In particular, we explore in some detail three different roles that parasites may play: (1) a predator or true parasite of healthy hosts, (2) an intraguild predator of a competing parasite, and (3) a competitor with a second parasite species or strain. Our analyses uncover a varied and diverse array of outcomes, in terms of both species composition and ensuing population dynamics. It is found that the two parasites are most likely to persist when there are trade-offs in exploitative (e.g., transmission) and interference (e.g., cross-transmission) competition. Regulation of the host population occurs when both parasites act as true parasites (i.e., infected hosts may recover and/or give birth to healthy hosts), whereas when one parasite acts as a predator (or pathogen) persistent oscillations may ensue. Additional realistic modifications of the models considered should greatly increase our knowledge about host-parasite systems.

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APPENDIX

Local Stability Analysis

Here we present a partial local-stability analysis for the general one-host, two parasite system. The Jacobian matrix at the three-species equilibrium is

$$\mathbf{J} = \begin{pmatrix} r - \beta_1 I_1^* - \beta_2 I_2^* & e_1 - \beta_1 S^* & e_2 - \beta_2 S^* \\ \beta_1 I_1^* & 0 & \delta I_1^* \\ \beta_2 I_2^* & -\delta I_2^* & 0 \end{pmatrix}. \tag{A1}$$

If inequalities (9) hold, then $\mathbf{J}$ has one of two different sign structures:

$$\begin{pmatrix} - & - & - \\ + & 0 & + \\ + & - & 0 \end{pmatrix} \tag{A2a}$$

or

$$\begin{pmatrix} - & + & - \\ + & 0 & + \\ + & - & 0 \end{pmatrix}. \tag{A2b}$$

In community matrix (A2a), the susceptible host and both parasites can have negative effects on susceptible-host growth at equilibrium, whereas the presence of the susceptible host always has a positive effect on the parasites. The parasites have no net effect on their own growth rates. One parasite must always benefit from the presence of its competitor at the expense of that competitor.

Matrix (A2b) demonstrates that the presence of infected hosts can have a positive impact on the population growth rate of the susceptible host population at equilibrium. The parasite that causes this positive effect on the susceptible host population must also have the upper hand at cross-infection. Otherwise, coexistence is not possible.

For local stability, all three eigenvalues of the system must have negative real parts. The Routh-Hurwitz conditions of stability are

$$\alpha_1 > 0, \tag{A3a}$$

$$\alpha_3 > 0, \tag{A3b}$$

$$\alpha_1\alpha_2 > \alpha_3, \tag{A3c}$$

with

$$\alpha_1 = -(r - \beta_1 I_1^* - \beta_2 I_2^*), \tag{A4a}$$

$$\alpha_2 = -(e_2 - \beta_2 S^*)(\beta_2 I_2^*) + (e_1 - \beta_1 S^*)(\beta_1 I_1^*) - \delta^2 I_1^* I_2^*, \tag{A4b}$$

$$\alpha_3 = -(e_1 - \beta_1 S^*)(\delta\beta_2 I_1^* I_2^*) - (e_2 - \beta_2 S^*)(\delta\beta_1 I_1^* I_2^*) - (r - \beta_1 I_1^* - \beta_2 I_2^*)(\delta^2 I_1^* I_2^*) \tag{A4c}$$

(May 1974, p. 196). If any one of these is violated, then the system is unstable. Condition (A3a) must hold, since at equilibrium the right-hand side of equation (A4a) equals $-(e_1 I_1^* + e_2 I_2^*)/S^*$; hence, equation (A4a) is positive and condition (A3a) is true. Condition (A3b) is violated in cases of contingent competition (i.e., both inequalities [9] are reversed). Limit cycles are possible only through the violation of inequality (A3c). In these cases, the sign structure of the Jacobian matrix (A1) always conforms to case (A2a). However, when the equilibrium is stable, the sign structure (A1) may conform to either one of the two cases (A2a) or (A2b).

Inequality (A3c) can be restated in terms of the population effects of parasite 1 on itself and parasite 2 on itself (at the three-species equilibrium), via the possible path loops through the three-species system. Violation of condition (A3c) and the ensuing limit cycles can occur only if the strengths of the links involving all three species are smaller than those involving just the host and one parasite. This can be stated symbolically as

$$(I_1 \to S \to I_2 \to I_1) + (I_2 \to S \to I_1 \to I_2) < (I_2 \to S \to S \to I_2) + (I_1 \to S \to S \to I_1), \tag{A5}$$

where $i \to j$ represents the effects of species i on species j at equilibrium.

We have not been able to simplify the Routh-Hurwitz conditions into compact, easily interpretable inequalities. Instead, we have conducted extensive numerical studies of the parameter space, examining the conditions for local stability. For example, take the case illustrated in figures 5*d* and 8 of sustained population cycles. Solving for the equilibrium-point densities gives

$$S^* = 147, \tag{A6a}$$

$$I_1^* = 139, \tag{A6b}$$

$$I_2^* = 19.1; \tag{A6c}$$

and the Jacobian matrix (A1) is

$$\mathbf{J}_{ex} = \begin{pmatrix} -0.08 & -0.73 & -2.34 \\ 0.67 & 0 & 1.94 \\ 0.38 & -0.27 & 0 \end{pmatrix}. \tag{A7}$$

Finally, Routh-Hurwitz condition (A3c) is violated since

$$\alpha_1 = 0.07, \tag{A8a}$$

$$\alpha_2 = 1.89, \tag{A8b}$$

$$\alpha_3 = 0.14. \tag{A8c}$$

In the special case of hyperparasitic systems ($\beta_1 = 0$), from (A3a)–(A3c) we have $\alpha_1 > 0$ (because $r - \beta_2 I_2^* < 0$), and $\alpha_3 = \delta I_1^* I_2^* (\beta_2 I_2^* - \delta r - e_1 \beta_2) > 0$ if and only if $\beta_2(d_1 - e_1) > \delta r$. The condition $\alpha_1\alpha_2 > \alpha_3$ reduces to $\beta_2 I_2^*(r - \beta_2 I_2^*)(-\beta_2 S^* + e_2) > -e_1\delta\beta_2 I_1^* I_2^*$; because $r - \beta_2 I_2^* < 0$ and $-\beta_2 S^* + e_2 < 0$, this is always true. Hence, given that a point equilibrium exists, the sole criterion that must be met for local stability is $\beta_2(d_1 - e_1) > \delta r$. A necessary condition for local stability is that $d_1 > e_1$ (more details in R. D. Holt and M. E. Hochberg, unpublished manuscript).

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