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THE DYNAMICS OF HOST-PARASITOID-PATHOGEN INTERACTIONS

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One of the principal objectives in ecology is to identify and understand the role of factors responsible for the regulation of plant and animal populations. A number of regulatory mechanisms are currently recognized, including interspecific and intraspecific competition for limited resources, herbivory, predation, parasitism, and disease. For the most part, the contribution of these processes to population dynamics is understood from the analysis of relatively simple, two-species population models (e.g., Lotka 1925; Volterra 1926; Nicholson 1933; Watt 1959; Hassell and May 1973; Beddington et al. 1975; Murdoch and Oaten 1975).

Recently, increasing attention has been paid to models of multispecies systems in which different types of interaction are combined. These include one predator and several prey types (e.g., Parrish and Saila 1970; Cramer and May 1972; Roughgarden and Feldman 1975; Comins and Hassell 1976; Hanski 1981), competing predator species attacking one prey species (Nicholson and Bailey 1935; Hassell 1978; May and Hassell 1981), specialist and generalist natural enemies attacking a common prey (Hassell and May 1986), competing parasitic species or strains parasitizing a single host species (e.g., Dobson 1985; Hochberg and Holt 1990), and host-parasitoid-hyperparasitoid (host-parasite-hyperparasite) interactions (Beddington and Hammond 1977; Hassell 1978; May and Hassell 1981; Holt and Hochberg, MS). Although the dynamics in these cases are often no more than expected from a detailed knowledge of the pairwise interactions, quite unexpected dynamics can also arise. For instance, when generalist and specialist natural enemies attack a common prey species, alternative stable states can occur that are not readily predicted from the separate two-species interactions (Hassell and May 1986). Understanding the role of population dynamics in community structure is thus likely to be a challenging task.

Despite this interest in the dynamics of multispecies interactions and the considerable literature on host-pathogen interactions (e.g., Anderson and May 1979, 1980, 1981), there have been few attempts to model interactions between pathogens and other natural enemies sharing a common host. An exception to this is a

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simple predator-pathogen-host system (Anderson and May 1986), showing that invasion of a pathogen into an established predator-prey interaction can depress the prey population to levels too low to sustain the predator population. A similar model in which a host is attacked by two separate parasitic species or strains yields similar results (Hochberg and Holt 1990).

If, as this body of work suggests, pathogens are important determinants of structure in simple communities, their increasing popularity in biological-control programs runs the risk of influencing other beneficial species such as parasitoids, predators, or other pathogens (Turnbull and Chant 1961; Watt 1965; Huffaker et al. 1976; Kekehashi et al. 1984). Since empirical studies indicate that natural enemies, usually insect parasitoids, are often capable of both depressing host population levels and maintaining these levels over long periods of time (for a review, see Waage and Hassell 1982), it is clearly important only to introduce a different type of natural enemy if the risks of disrupting the biological control are small. This in turn requires some fundamental knowledge of the dynamics of interactions between pathogens, parasitoids, and a shared host species.

This paper is divided into two main sections. First, we present a model for a host and a parasitoid, whose attacks can vary from random to highly clumped. A new model is also proposed for a host and its pathogen, where the pathogen is transmitted via external stages. The second section unites these single-natural-enemy models and takes explicit account of the relative competitive abilities of the pathogen and parasitoid within the host. We consider the outcomes of invasion of one natural enemy species into a system that is regulated by the other. The invasion can be rebuffed, lead to unilateral exclusion of the resident natural enemy, or give rise to constant, periodically fluctuating, or chaotic populations of both natural enemies and the host. We then show how the characteristics of the natural enemy affect host abundance and the dynamics of the interaction when both enemies coexist. Finally, we discuss these results in the light of community structure and biological control.

INTERACTIONS WITH A SINGLE NATURAL ENEMY

One Host–One Parasitoid

The dynamics of coupled host-parasitoid interactions have been well studied in the literature (e.g., Hassell and May 1973, 1974; Murdoch and Oaten 1975; Hassell 1978; May 1978; May and Hassell 1981; Chesson and Murdoch 1986). We assume that the host has discrete and nonoverlapping generations and is attacked by a parasitoid during some interval of its life cycle. Although other regulatory factors such as intraspecific competition could be included (Beddington et al. 1978; Hassell 1978), they are not considered here in order to make clearer the role of parasitoids in the dynamics of the interaction. The recursive equations from generations t to $t + 1$ take the form

$$N_{t+1} = FN_t f(P_t), \tag{1a}$$

$$P_{t+1} = cN_t[1 - f(P_t)]. \tag{1b}$$

Here N and P refer to the host and parasitoid population densities, F is the host's finite rate of increase, c is the average number of female parasitoid progeny per host attacked (henceforth assumed to be one), and $f(P_t)$ is the fraction of hosts that escape parasitism, which is here based on a negative-binomial distribution of attacks (May 1978; see also Chesson and Murdoch 1986; Perry 1988):

$$f(P_t) = (1 + aP_t/k)^{-k}. \tag{2}$$

The parameter a is a measure of the per capita searching efficiency of the parasitoid; k characterizes the degree of the clumping of parasitoid attacks, which are highly clumped as k approaches zero and become random (Poisson) as k tends to infinity. Although we have assumed the distribution of parasitoid attacks to be a constant, a more general model would represent k as a function of both host and parasitoid densities (Hassell 1980).

The Equilibrium and Population Dynamics

The parasitoid regulates the host population around steady equilibrium densities:

$$N_P' = P'F/(F - 1), \tag{3a}$$

$$P' = k(F^{1/k} - 1)/a, \tag{3b}$$

if and only if $F > 1$ and $k < 1$ (May 1978). Here N_P' and P' refer to the equilibrium densities of the host and parasitoid, respectively. The host equilibrium density tends to be relatively low if the parasitoids tend to attack randomly (relatively large k) and/or if the host's finite rate of increase is low. When $k > 1$, the equilibrium point is unstable and both host and parasitoid populations exhibit diverging oscillations, which should result in the eventual extinction of both host and parasitoid populations. Note that very small values of k, which promote regulation of the host population, also result in larger host populations at equilibrium.

One Host–One Pathogen

Whereas parasitoids are able to search for their hosts, pathogens must rely on the physical environment (Young and Yearian 1986) or on vectors (for a review, see Entwistle 1982) for horizontal transmission or on infected hosts for vertical transmission to the offspring (Martignoni and Milstead 1962; Tatchell 1981). Past models of pathogens attacking a host with discrete and nonoverlapping generations have assumed that the pathogen is transmitted directly from host to host and/or is very short-lived outside the host (May 1985). These assumptions translate into the requirement that the pathogen must somehow reinvade the host population at the beginning of each infectious season in order to persist. In this section, we present a new model (similar to that of May 1985) with the refinement that the pathogen produces long-lived external stages, as is widely observed in nature (see Fuxa and Tanada 1987 and references therein). The model system is presented schematically in figure 1 and discussed more fully in Appendix A. (A differential-equation variant of the model, for hosts exhibiting continuous generations, is discussed in Hochberg 1989.)

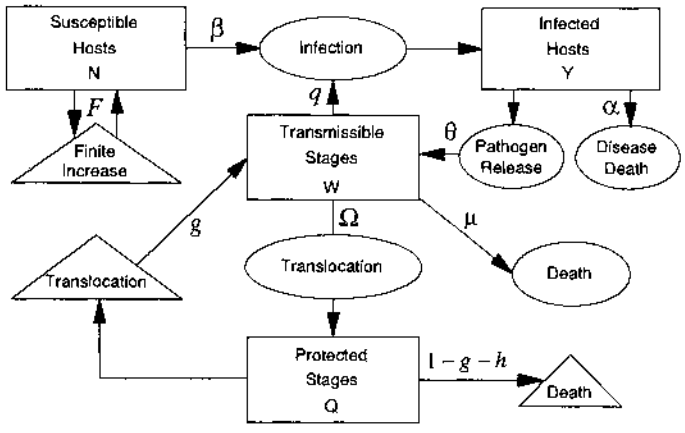

FIG. 1.—Diagrammatic flow chart of the host-pathogen model for susceptible hosts (N), infected hosts (Y), transmissible stages of the pathogen (W), and protected stages of the pathogen (Q). *Ovals*, Processes that occur during the infectious season; *triangles*, processes that occur between infectious seasons. See the text for details.

The infectious season is assumed to begin at some determined time in the host's life cycle (such as at egg hatch). During the infectious season, hosts of density N are infected by pathogen stages of density W at a rate βNW. The latter term, sometimes called the "random-mixing law" (Anderson and May 1979), assumes that the instantaneous risk of infection to a given susceptible host is a linear function of pathogen density at that instant. We also assume that the infection is invariably lethal, with infected hosts (of density Y) surviving for an average period of $1/\alpha$ (α reflects the virulence of the pathogen). Infective stages are produced within infected hosts and then liberated (at a net per capita rate θ) into the host's "accessible" environment, such as on the surface of leaves in the case of herbivorous insects. One of three different fates may befall these liberated propagules. First, when infection occurs, a number q are consumed along with the food source. Second, some stages may perish because of vagaries of the environment (e.g., ultraviolet radiation) at a per capita rate of μ. Finally, some may be transferred at a per capita rate of Ω from the leaf surface into a separate reservoir (of density Q), such as the soil. Once stages are transferred into this pool, we assume (for simplicity) that they neither suffer mortality nor can be translocated back to the leaf surface for transmission in the same season.

We assume that at the end of the infectious season all infected hosts have perished and that no propagules remain on the surface of the leaves. Hence, the length of the infectious season is determined by the persistence of the pathogen stages that are accessible to the host. This assumption is most reasonable when (1) accessible pathogens are very short-lived (because of the combination of mortality on, and removal from, leaves) in comparison with the expected life span of the vulnerable stages of the host and (2) the infectious season is essentially complete before the invulnerable stages of the host appear (e.g., before pupation).

The differential equations for the infectious period are then

$$dN/dt = \beta NW, \tag{4a}$$

$$dY/dt = \beta NW - \alpha Y, \tag{4b}$$

$$dW/dt = \theta Y - \Omega W - \mu W - q\beta NW, \tag{4c}$$

$$dQ/dt = \Omega W. \tag{4d}$$

The initial conditions are $N_t = N_0$, $Y_t = 0$, $W_t = W_0$, and $Q_t = Q_0$.

Equations (4) fully characterize the dynamics of the host-pathogen interaction during the infectious period. To include this set of equations in the broader framework of a dynamic model, we assume that, at the beginning of the next infectious season, a fraction g of the pathogen reservoir will have been translocated back to the leaves and that a fraction h will have remained. Hence, the sum $g + h$ must always be less than or equal to unity, since some propagules may have died or been leached from the soil.

The recursive equations for the population of hosts from generation t to $t + 1$ may now be expressed as

$$N_{t+1} = FN_t(1 - I), \tag{5a}$$

$$W_{t+1} = g[Q_t - (\Omega/\beta)\ln(1 - I)], \tag{5b}$$

$$Q_{t+1} = hW_{t+1}/g. \tag{5c}$$

The fraction of susceptible hosts that survived the epidemic, $1 - I$, is defined implicitly by

$$1 - I = \exp\{-[N_t I + W_t/(\theta/\alpha - q)]/N_T\} \tag{5d}$$

and, as expected, is an increasing function of both host and pathogen densities. These results are derived in Appendix A. The constant N_T is sometimes called the "epidemic threshold" (see, e.g., Anderson and May 1981):

$$N_T = (\mu + \Omega)/\beta(\theta/\alpha - q). \tag{5e}$$

We see that the pathogen population (the sum of eqs. 5b and 5c) is an increasing function of the density of propagules in the soil (Q_t), the course of the previous epidemic ($1/[1 - I]$), and the survival rate of the stages between epidemics ($g + h$). From the denominator of equation (5e), it is apparent that a positive threshold density, and thus the persistence of the pathogen, occurs only if $\theta/\alpha > q$. This translates into the trivial requirement that, on the average, more propagules must be produced per infected host than are required to initiate an infection. In addition, an increasing fraction of hosts comes down with the disease when the transmission efficiency and the production of free-living stages are high (the denominator of eq. 5e) and the rates of pathogen death and transfer to the soil reservoir are low (the numerator of eq. 5e).

The epidemiological meaning of N_T for equations (5) differs from the usual interpretations (Anderson and May 1981). For models in which the pathogen is not able to span the inter-epidemic gap (e.g., May 1985), the initiation of an epidemic requires simply that the host density exceed N_T. In the model defined by equations (5), the allowance for long-lived external stages tends to blur the threshold. Rather, the threshold now applies to epidemics that are initiated by very low densities of the pathogen stages. This is illustrated in figure 2 for various values of W_t.

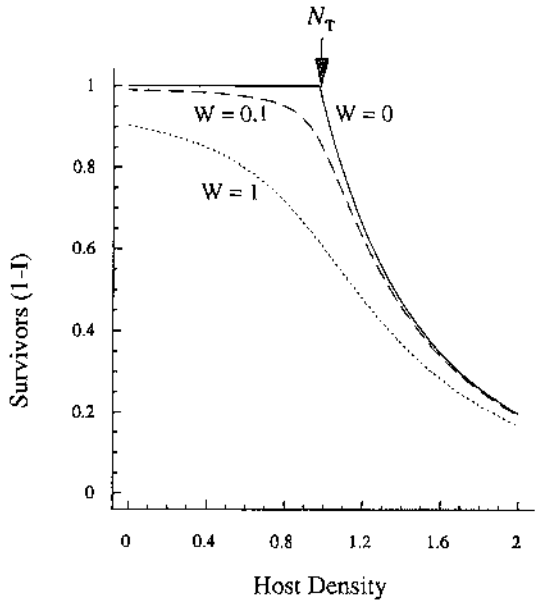

FIG. 2.—Effect of host and parasite densities on the fraction of hosts that survive the disease. If the pathogen is unable to move from the reservoir to the transmissible subpopulation (i.e., $g = 0$ and $W_t = 0$ for any t), then the model behaves in much the same way as the basic Kermack-McKendrick model (May 1985). N_T, The epidemic threshold given in equation (5e). Parameter values are $F = 3$, $g = 0.01$, $h = 0.9$, $\alpha = 1$, $\Omega = 0.1$, $\mu = 0.4$, $\beta = 0.05$, $q = 0$, $\theta = 10$.

The Equilibrium and Effects of the Pathogen on Host Abundance

The system defined by equations (5a,b) has a single positive equilibrium for hosts (N'_W), transmissible pathogen (W'), reservoir pathogen (Q'), and the fraction of hosts surviving the epidemic ($1 - I'$), given by

$$N'_W = N_T(1 - \tau)F\ln F/(F - 1), \tag{6a}$$

$$W' = g\Omega\ln F/\beta(1 - h), \tag{6b}$$

$$Q' = h\Omega\ln F/\beta(1 - h), \tag{6c}$$

$$1 - I' = 1/F. \tag{6d}$$

Here τ characterizes the persistence of pathogen stages on the leaf surface and the soil, both within and between infectious seasons, and is thus a measure of the total survival rate of the pathogen. It is given by

$$\tau = \frac{g}{1 - h}\frac{\Omega}{\Omega + \mu} \tag{6e}$$

and is constrained to the interval zero to one for all h less than one. The first factor reflects the between-infectious-seasons survival rate of the pathogen; the second factor is the within-season survival.

We see from equation (6a) that host population levels decrease with increases in

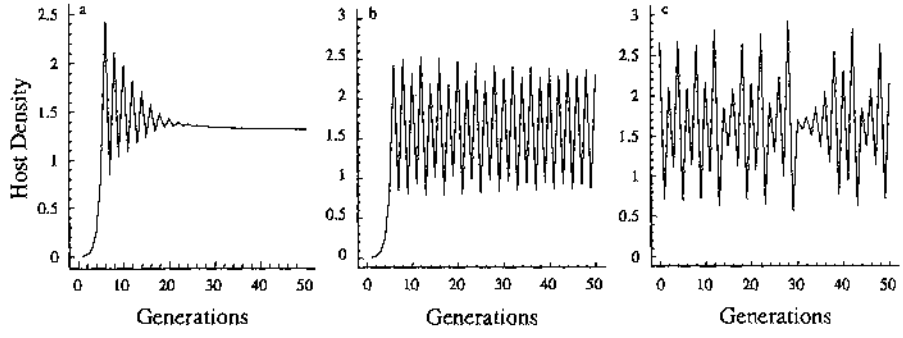

FIG. 3.—The effects of pathogen recycling on the dynamics of the populations. *a*, Damped oscillations ($g = 0.1$); *b*; two-point limit cycles ($g = 0.01$); and *c*, apparent chaos ($g = 0.0001$). Other parameters as for figure 2.

total pathogen survival and a reduced epidemic threshold (high transmission efficiencies and large numbers of stages produced per dying host). The equilibrium density of the pathogen (sum of eqs. 6b and 6c) increases with increases in g, h, Ω, and F and with decreases in β. The last relationship reflects the fact that higher transmission rates result in reductions in host and thus pathogen population density. Note that the equilibrium prevalence of infection, $1 - I'$, is a simple function of the host's finite rate of increase, F.

Population Dynamics

The stability of the equilibrium (eqs. 6), and hence the propensity for constant or oscillatory population dynamics, is determined only by pathogen survival and by the host's finite rate of increase (fig. 3). The equilibrium point is insensitive to small perturbations only if

$$[1 + \tau(1 - h)/(1 + h)]/(1 - \tau) > (F + 1)\ln F/2(F - 1). \quad (7)$$

In other words, constant dynamics require that the pathogen stages are sufficiently invulnerable to mortality both within and between infectious seasons. As in the host-parasitoid model, the "exploitation" parameters (i.e., β and $\theta/\alpha - q$) do not influence the stability of the equilibrium between the host and the natural enemy.

The ramifications of inequality (7) are illustrated by the examples in figure 3. As total survival of the pathogen, τ, decreases (or F increases) and the inequality is violated, the pathogen first requires two host generations to bring host densities to minimum levels. Further decreases in τ, and/or increases in F, result in a series of period-doubling bifurcations and finally a regime of apparent chaos (May 1976). This tendency toward oscillatory behavior is due to the more pronounced threshold level of hosts required for an epidemic to occur (see also May 1985).

Moreover, as τ and/or F approaches unity, the populations once again exhibit oscillations, but now the cycles are of long period. Here, the pathogen produces a "seed bank" with each epidemic, resulting in the subsequent depression of the host population. When the seed bank is finally diminished (because of a paucity of

hosts), the host population grows until the next epidemic, whereupon the seed bank is replenished. The period of the oscillations increases as the stages remain in the soil reservoir (i.e., as $g \to 0$, $h \to 1$), whereas shorter-period oscillations ensue as the stages are more frequently transferred back to the host's food source ($g \to 1$, $h \to 0$). Persistent oscillations, however, were not observed to occur in either case.

HOST-PARASITOID-PATHOGEN INTERACTIONS

The preceding section dealt with models of hosts that are subject to only a single source of mortality. In reality, a given species may be subject to an array of mortality factors, some of which may contribute (often in unapparent ways) to its regulation. In this section, we explore the dynamics of a host attacked by a parasitoid and a pathogen.

Model Development

Whenever more than one mortality factor acts as some function of host density in the host's life cycle, it is crucial to know both the sequence in which they occur (Wang and Gutierrez 1980; May et al. 1981) and the outcome of any interference competition within co-infected hosts. In the case of host-macroparasite interactions, Dobson (1985) has pointed out that interference usually acts asymmetrically. This same conclusion has been reached for interspecific competition in insects (Lawton and Hassell 1981). However, for microparasites and gregarious parasitoids (both of which produce many progeny within a particular host from a single infection), direct competition may not be an all-or-nothing phenomenon. Rather, some fraction of the potential number of progeny of each competitor may survive in co-inhabited hosts (e.g., Beegle and Oatman 1975; Vail 1981; Powell et al. 1986). There is some evidence that the final outcome of competition is determined by the relative timing of attacks (e.g., J. Anderson et al. 1977), suggesting that (in the absence of within-host interactions between the competitors) the respective developmental rates within the host figure heavily in the outcome of multiple parasitism.

We present here a model that includes such possibilities of differential survival within hosts infected by both a parasitoid and a pathogen in a detail-independent way, much as was done by Hogarth and Diamond (1984) for two parasitoid species interacting at the same time but with different degrees of competitive superiority. For the model developed below, the interference term ϕ reflects the dominance of one natural enemy over the other when co-occurring in the same host individual. It takes values between 0 and 1, with the parasitoid enjoying the advantage when ϕ approaches zero and the pathogen becoming the superior intrinsic competitor as ϕ tends to one. In the special case of $\phi = 0.5$, both competitors have an equal share of co-inhabited hosts. Hereafter, values of ϕ near 0.5 will be referred to as low-to-intermediate levels of (intrinsic) dominance within co-infected hosts, and values approaching either zero or one will be referred to as high levels of intrinsic dominance in favor of the parasitoid and the pathogen, respectively. This is elaborated in Appendix B.

The three-species system is

$$N_{t+1} = FN_t(1 - I)f(P_t), \tag{8a}$$

$$W_{t+1} = g[Q_t - (\Omega/\beta)\ln(1 - I)], \tag{8b}$$

$$Q_{t+1} = hW_{t+1}/g, \tag{8c}$$

$$P_{t+1} = cN_t\{(1 - \phi I)[1 - f(P_t)]\}, \tag{8d}$$

$$1 - I = \exp\{-[N_t(I\{f(P_t) + \phi[1 - f(P_t)]\}) + W_t/(\theta/\alpha - q)]/N_T\}, \tag{8e}$$

with the parasitoid functional response, $f(P_t)$, as described in equation (2). This set of equations merely combines the notions covered in both of the single-species models (i.e., eqs. 1, 2, 5), with the exception that now the natural enemies can interfere with one another. The interference interaction is discussed more thoroughly in Appendix B.

Invasion and Establishment of a Second Natural Enemy

Systems such as that of equations (8) need not be treated as static in species composition; populations can colonize new habitats and become extinct in established ones. The potential of a species to invade or to be excluded from a particular system can be determined by assuming that one of the natural enemies (the "invader") is at a very low density, whereas the other (the "resident") is at equilibrium with the host. This scenario is most applicable to relatively undisturbed systems in which invasion occurs by the chance introduction of one or a few colonists. It should be noted that the criteria for invasion (or persistence) set out below are exact only for cases in which the resident natural enemy regulates the host to constant densities in the absence of the competing (invading) natural enemy. If the interaction of the resident natural enemy and the host produces periodic or chaotic populations, then the conditions outlined below are only approximate and numerical simulations become necessary to delimit the criteria for invasion.

Let us imagine, then, that the host is at a constant equilibrium with the pathogen, and the parasitoid is present in very small numbers at the beginning of generation t. To invade this system (or to avoid extinction), the parasitoid population must grow between generations t and $t + 1$, which requires (Appendix C)

$$acN_T(1 - \tau) > (F - 1)/[F - \phi(F - 1)]\ln F. \tag{9a}$$

Thus, invasion requires that the parasitoid's relative dominance within the coinfected host $(1 - \phi)$ be high, and the product of its survival and searching efficiency must also be sufficiently high relative to pathogen survival and transmission efficiency (these processes are encapsulated in the left-hand side of inequality 9a). High values of F will aid parasitoid invasion only as the parasitoid becomes the dominant competitor within hosts that harbor both competitors. Notice that the distribution of parasitoid attacks given by equation (2) does not directly affect its ability to invade. Rather, the pathogen's (random) distribution of transmission, which is important, is reflected by $\ln F$.

An argument similar to that discussed above applies to the case in which the

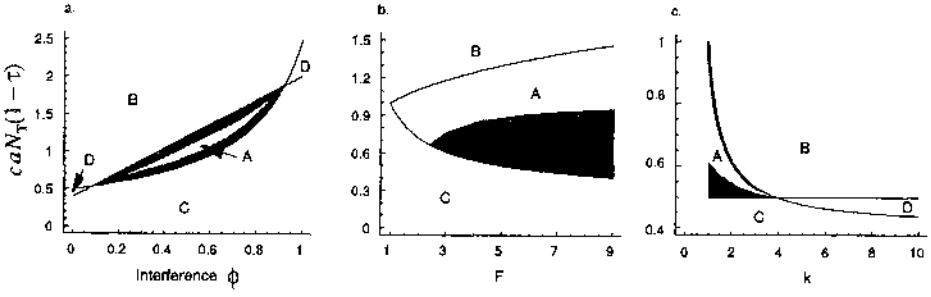

FIG. 4.—The outcomes of competition between a pathogen and a parasitoid. *a*, The effects of extrinsic competitive potential, $caN_T(1 - \tau)$, and intrinsic competitive potential, ϕ (with $k = 1000$). *b*, The effects of extrinsic competition and the host's finite rate of increase, F (with $k = 0.8$). *c*, The effects of extrinsic competition and the degree of the clumping of parasitoid attacks. Region A and shaded areas, Coexistence; B, the parasitoid wins; C, the pathogen wins; D, the parasitoid or pathogen wins, depending on initial population densities. Approximate parameter space in which the equilibrium point is unstable and perpetual cycles occur as indicated by the shaded regions. Other parameters general to the figures are the same as in figure 2 except $F = 5$, $g = 0.25$, $h = 0$, $c = 1$, $a = 1$, $k = 2$, $q = 0$, $\phi = 0$.

pathogen invades the established host-parasitoid system. Here, successful invasion requires (Appendix C)

$$1/acN_T(1 - \tau) > (F - 1)/k(F^{1/k} - 1)[1 + \phi(F - 1)]. \quad (9b)$$

Pathogen invasion is enhanced by its own relative dominance within co-infected hosts and when the product of its survival and transmission efficiency is sufficiently high relative to the parasitoid's survival and search capabilities. Once again, the distribution of attacks by the invader does not directly affect the invader's ability to invade the parasitoid equilibrium. However, any tendencies toward clumped attacks by the parasitoid may permit the invasion of the pathogen. Finally, high values of F always benefit the pathogen when $k < 1$; when $k > 1$, the pathogen benefits only if its intrinsic dominance is sufficiently high relative to that of the parasitoid. It is clear, therefore, that the importance of the distribution of attacks and levels of intrinsic dominance in inequalities (9a,b) is scaled by the host's finite rate of increase, F.

Figures 4 and 5 illustrate some representative cases of the effects of parameter values on the outcomes of invasion as set out in inequalities (9a,b). If both inequalities (9a) and (9b) hold, then either natural enemy can establish itself from rarity in the presence of its competitor (fig. 4, region A). This is equivalent to saying that neither competitor can be excluded from the system. The conditions under which both competitors can invade the system are (from inequalities 9a,b)

$$\frac{F - 1}{[F - \phi(F - 1)]\ln F} < caN_T(1 - \tau) < \frac{k(F^{1/k} - 1)[1 + \phi(F - 1)]}{F - 1}. \quad (9c)$$

The left-hand inequality reflects the ability of the parasitoid to invade the pathogen equilibrium, whereas the pathogen invades the parasitoid-regulated system when the right-hand side is satisfied. Note again that the mutual invasion condi-

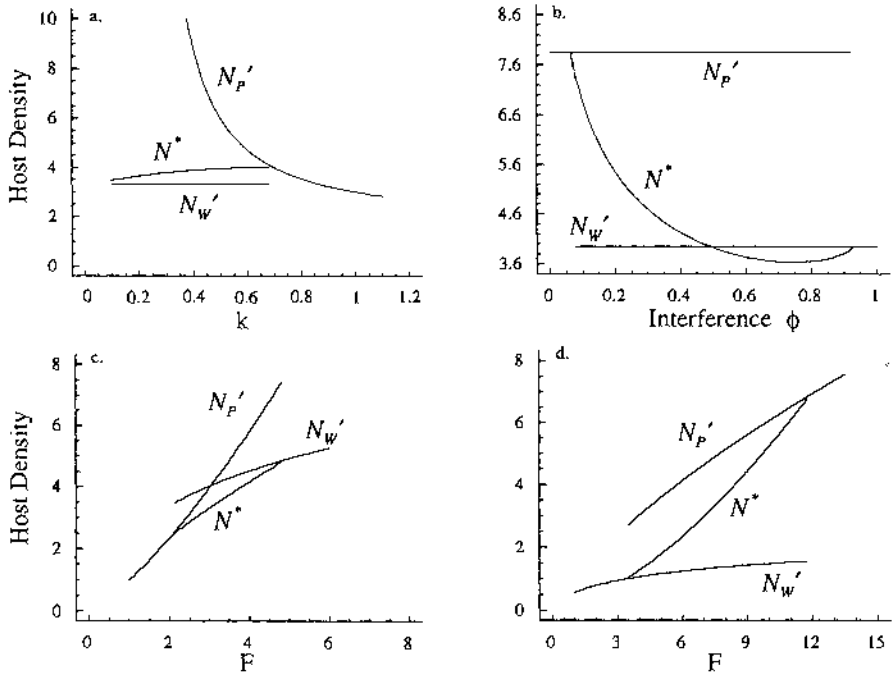

FIG. 5.—The effects of various parameter values on the outcome of competition between the pathogen and the parasitoid and on the equilibrium levels of the host. *a*, Effect of k, with $F = 3$, $g = 0.1$, $\mu = 0.1$, $\theta = 1$, $\phi = 0.25$; *b*, effect of ϕ, with $F = 5$, $k = 0.7$, $\theta = 2.5$; *c*, effect of F, with $k = 0.7$, $\theta = 2$, $\phi = 1$; *d*, effect of F, with $\mu = 0.1$, $k = 1.5$, $\theta = 5$, $\phi = 0$. N_P' is the equilibrium of the host when regulated by the parasitoid, N_W' when regulated by the pathogen, N^* when regulated by both natural enemies. Note that coexistence between the natural enemies occurs for parameter values where the levels of N^* are drawn. Where only N_W' is present, the pathogen eliminates the parasitoid. The parasitoid eliminates the pathogen for parameter values with only N_P' drawn. Parameters common to all four figures, except where otherwise noted: $g = 0.01$, $h = 0.9$, $\alpha = 1$, $\Omega = 0.05$, $\mu = 0.2$, $\beta = 0.05$, $c = 1$, $a = 1$, $q = 0$.

tions (9c) are exact only when either natural enemy regulates the host in the absence of its competing natural enemy.

Other outcomes may also arise. If the total competitive advantage of one natural enemy over the other is high enough, such that the inferior competitor cannot sustain itself on the available hosts, then one species will unilaterally exclude the other (i.e., one but not both of inequalities 9c is violated) (figs. 4*a–c*, regions B and C for the parasitoid winning and the pathogen winning, respectively). Finally, cases may also arise in which the winning species depends on the initial population densities (figs. 4*a*,*c*, region D). This "contingent" competition results when both of inequalities (9c) are violated. Note from figure 4 that in some cases small changes in parameter values may determine the difference between competitive outcomes (e.g., changes in exploitation parameters on the ordinate in figs. 4*a*,*c*), whereas in other instances very large parameter changes are required (e.g., changes in exploitation parameters for large F and very small k in figs. 4*b*,*c*).

Effects of Establishment on Host Abundance and Population Dynamics

When inequalities (9c) are satisfied and the enemies coexist, they need not both contribute to the depression of the host population (figs. 5*a*,*b*,*d*). For the special case of $k = \infty$ (Nicholson-Bailey parasitoid), one natural enemy always contributes to the depression of the host population, whereas the other always increases host densities. Which natural enemy plays which role is determined solely by the central term in inequalities (9c). If $ac < 1/N_T(1 - \tau)$, then the pathogen depresses host densities and the parasitoid increases them. Reversal of this inequality results in the reversal of the enemies' respective influences on host abundance. Nevertheless, both natural enemies may contribute to the depression of the host population (figs. 5*b*,*c*). This occurs as parasitoid search tends away from randomness and the competitive dominance within the host favors the pathogen. If the distribution of pathogen transmissions were aggregated, then mutual depression of the host population could be favored by intrinsic dominance of the parasitoid. In no case does the addition of both natural enemies contribute to increases in host abundance. (Note that this could occur if the attack distribution(s) of one or both natural enemies were under-dispersed.)

Competitive coexistence may be manifested by either constant or oscillatory dynamics (figs. 4, 6). Long-period cycles ensue as parasitoid search tends to become random (fig. 6*c*), whereas more-ragged, short-term cycles occur as the epidemic threshold becomes more pronounced, or as g tends to zero (a Kermack-McKendrick pathogen) (figs. 6*a*,*b*). In general, the instability of the equilibrium point is also promoted by large values of F (fig. 4*b*), extreme values of k (e.g., fig. 4*c*), and domination by the destabilizing natural enemy (i.e., the stabilizing competitor is close to unilateral exclusion; see the shaded regions of fig. 4).

In addition, the straightforward conditions that determine the population dynamics of the single-species models laid out in the preceding section do not necessarily hold when both natural enemies attack the host. For example, tendencies toward random search (which are otherwise destabilizing) by the parasitoid may actually damp the cycles produced by the pathogen, resulting in constant equilibrium dynamics (fig. 4*c*, region A; fig. 6*b*). This is due to high values of k that act to depress the host equilibrium away from the oscillatory region of the host-pathogen interaction (Hochberg, MS).

Finally, as noted in the preceding subsection, if the interaction between one natural enemy and the host is characterized by periodic or chaotic fluctuations, the competing natural enemy may, in principle, be able to persist even when its invasion criterion is violated. For example, cases may arise in which the pathogen is unilaterally excluded from the system (according to criterion 9b) but may aperiodically reinvade because of the oscillatory nature of the host-parasitoid interaction for $k > 1$ (fig. 6*d*). Similarly, the competitive exclusion of the parasitoid (as determined from inequality 9a) from an unstable host-pathogen system may give rise to aperiodic invasions and extinctions of the parasitoid. In either case, no three-species equilibrium exists, leading to the important conclusion that frequent colonizations and extinctions may themselves drive the dynamics of the system. Situations may even arise in which inequalities (9c) predict that

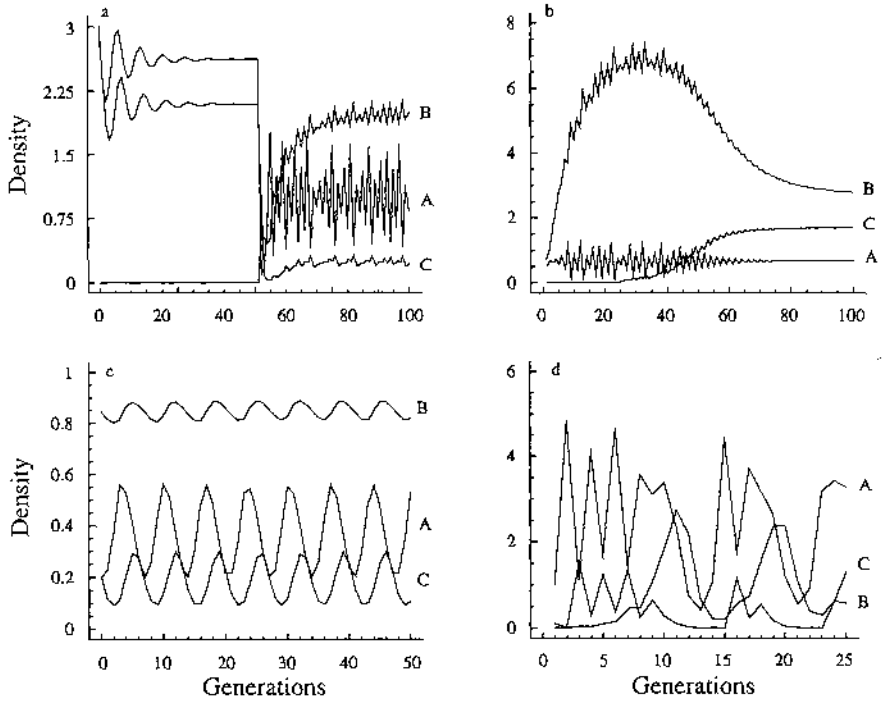

FIG. 6.—Population dynamics of the host and its two natural enemies. *a*, Short-period cycles (the pathogen invades at generation 51), with $F = 5$, $g = 0.001$, $\Omega = 0.01$, $\mu = 0.015$, $\beta = 0.05$, $a = 3$, $k = 0.7$, $\theta = 1$, $\phi = 0.55$; the density of the parasitoids is scaled by a factor of 0.2. *b*, Damped oscillations (the parasitoid invades at generation 25), with $g = 0.005$, $a = 3$, $\phi = 0.45$; the density of parasitoids is scaled by a factor of 5. *c*, Long-period cycles, with $a = 5$, $\phi = 0.27$; the density of pathogen stages is scaled by a factor of 0.25. *d*, Periodic reinvasion by the pathogen, with $h = 0.1$, $\mu = 0.1$, $a = 1$, $\theta = 1$, $\phi = 0.3$; the density of pathogen stages is scaled by a factor of 10. Curve A, Hosts; B, total pathogen stages; C, parasitoids. Parameters common to all four figures, except where otherwise noted: $F = 5$, $g = 0.1$, $h = 0.9$, $\alpha = 1$, $\Omega = 0.05$, $\mu = 0.05$, $\beta = 0.1$, $c = 1$, $k = \infty$, $q = 0$, $\theta = 3$.

the parasitoid excludes the pathogen, but because of an oscillatory host-parasitoid interaction, the pathogen invades and excludes the parasitoid.

DISCUSSION

Despite some fundamental differences between the biology of parasitoids and that of pathogens, there are clear analogies in the mechanisms by which each natural enemy can regulate its host population (May and Hassell 1988; Hassell and Anderson 1989). This is further evidenced by the host-parasitoid and host-pathogen models described in this paper. In addition, combining these pairwise interactions into a three-species system highlights the unexpected dynamics that can easily arise in simple multispecies systems (May and Hassell 1988).

In terms of biological control, one of the factors that can influence the establishment and effectiveness of an introduced natural enemy is the presence of another,

already established, natural enemy species. The introduction of the additional species may be competitively rebuffed or, if successful, result in stable or unstable coexistence, or even the exclusion of the resident natural enemy. These results are already known from a number of multispecies models (e.g., May and Hassell 1981; Kekehashi et al. 1984; Holt and Pickering 1985; Hochberg and Holt 1990), but no results are known to date from models involving pathogens and parasitoids acting together. Unfortunately, scant attention has been paid to field studies that compare the regulation of hosts by one and by several natural enemies. One notable example is that of the European corn borer, *Ostrinia nubilalis*, its microsporidian pathogen *Nosema* sp., and its parasitoids *Lydella thompsoni* and *Macrocentrus grandii*. It has been suggested (Lewis 1982; Siegel et al. 1986) that the observed breakdown in the regulatory abilities of *L. thompsoni* (e.g., Sandlan et al. 1983) and *M. grandii* (Andreadis 1980; Siegel et al. 1986) during the 1960s may have coincided with the introduction of *Nosema* sp. However, recent work based on the interaction of *L. thompsoni* and *Nosema* sp. within the host casts some doubt on the validity of this hypothesis (Cossentine and Lewis 1988). Our study suggests that population studies would have to be conducted to elucidate the possible role of the disease in the reduction or elimination of the parasitoid populations. Below, we briefly discuss those properties of pathogens and parasitoids that may make them amenable to biological control.

For pathogens that must kill their hosts (especially arthropods) in order to be transmitted, long-lived external stages appear to be an important means of ensuring the persistence of the pathogen population (Evans and Entwistle 1982; Hochberg 1989). When external stages are very short-lived, pathogen transmission is generally assumed to be directly proportional to infected density. Under these conditions, the pathogen must somehow reinvade the host population at the beginning of each generation. The result is invariably unstable dynamics (May 1985). Régnière (1984) considered the special case of pathogens that are able to span the intergenerational gap by vertical transmission and has shown that this promotes the persistence of the pathogen. In the absence of long-lived external stages, however, other mechanisms may be required for the pathogen to regulate the host to low and non-oscillatory densities, such as host recovery from infection (May 1985). Other forms of transmission, too, may be crucial to the system dynamics. Numerical simulations of variants of the host-pathogen model presented here and the results of a separate study (Hochberg 1989) suggest that pathogens can damp a host population if they (1) can amass a reservoir of long-lived stages and (2) can be sufficiently translocated from the protected reservoir to the habitat of the host at the beginning of the infectious season. If the rate of translocation (recycling) is very high, then oscillatory populations may again ensue (Hochberg 1989). Presumably, the amassing of such a reservoir would make parasitoid persistence less likely.

For the special case in which parasitoids search at random, coexistence of the two natural enemies simply requires balances in their respective capacities for attack, as characterized by both extrinsic factors (e.g., searching efficiency, transmission, survival rate) and intrinsic factors (e.g., competition within hosts, timing of host attack). This same result has been shown for competing parasitoids

(Zwölfer 1971; Miller 1977). Highly clumped search on the part of the parasitoids tends to blur this trade-off, and cases may arise in which both species coexist even when the parasitoid is both the better intrinsic and extrinsic competitor. Although our models did not include nonrandom distributions of parasitism on the part of the pathogen, heterogeneities in pathogen transmission should still influence the parasitoid's ability to establish in a way that is similar to the effect of the clumping of parasitoid attacks on pathogen invasion. The general property that heterogeneity enhances the persistence of competitors (de Jong 1979; Atkinson and Shorrocks 1981; Hanski 1981; Ives and May 1985) suggests that some degree of clumping of attacks in both parasitoid and pathogen is a desirable characteristic in biological-control programs.

Establishment of a second natural enemy leading to coexistence need not result in decreases in host density; increases are possible under some conditions. For example, after an epidemic of *Erwinia sphaerosperma* in the diamond-back moth, *Plutella xylostella*, native parasitoid populations were greatly reduced, and the host population subsequently equilibrated to levels higher than those before the epidemic (Ullyett 1947). Similar theory applies for systems of two parasitoids (May and Hassell 1981) and specialist and generalist natural enemies (Hassell and May 1986). Situations in which both natural enemies contribute to the depression of the host equilibrium are most likely to occur when both enemies are good extrinsic competitors (i.e., have high search or transmission rates and high survival rates) and when differences in their respective intrinsic abilities are low to moderate (timing of attacks and/or outcome of competition within the host is similar). This latter property is particularly important. For instance, as the clumping of parasitoid attacks increases, the value of ϕ for which the host population is maximally depressed shifts from near intrinsic equality ($\phi \approx 0.5$) to situations in which the pathogen is the better at this interference interaction (e.g., $\phi \approx 0.8$) but not necessarily completely dominant (i.e., $\phi \approx 1$). Although pathogens often have the upper hand over parasitoids in multiply infected hosts (e.g., Beegle and Oatman 1975; Levin et al. 1981; Powell et al. 1986), our model suggests that it is a misconception to conclude that parasitoids will be eliminated from the system or play a minor role in the regulation of the host. Furthermore, the commonly held notion that natural enemies must not interfere with each other may be misleading; some interference, in terms of overlap of attack times and/or the possibility of survival in a co-infected host, can be a desirable characteristic of systems with multiple natural enemies.

The levels of intrinsic competitive superiority do not themselves appear to contribute greatly to the stability of the three-species equilibrium. Even though oscillations are most likely to occur at the extremes of intrinsic dominance, the specific assumptions about the biologies of the three species (reflected by the values of parameters) determine the exact range of values of ϕ that permit constant or oscillatory dynamics at equilibrium. More generally, cycles are possible when one or both of the single-natural-enemy systems exhibits persistent oscillatory populations. This highlights the simple fact that the establishment of a natural enemy may itself render constant the oscillatory dynamics between the host and an already established natural enemy.

The models presented here suggest that biological control involving parasitoids and pathogens is most likely to be a sound strategy when (1) the pathogen produces external stages that may span the intergenerational gap; (2) parasitoid (and pathogen) attacks are moderately clumped; (3) both natural enemies have high rates of search or transmission; and (4) there is some degree of overlap in the timing of attacks of the enemies, and/or competition with the host is not invariably one-sided. What is now greatly needed are more long-term field studies comparing single and multiple introductions of natural enemies. Such investigations are certain to be valuable to future biological-control programs.

SUMMARY

The growing importance of pathogens in biological control warrants an investigation into the likely effects of disease on the widely successful class of biological-control agents, the parasitoid. Our models consider a parasitoid that can exhibit a range of attack behaviors ranging from clumped to random. The pathogen is assumed to be transmitted by external stages that can span periods when the host is rare or absent. When these two natural enemies occur simultaneously in a given host, the result can be the elimination of one enemy by the competitor in all co-infected hosts or it can be some intermediate outcome. We show through an analysis of invasion that both natural enemies may coexist with constant, cyclic, or chaotic populations. Other outcomes are also possible. One enemy may exclude its competitor either unilaterally or on the basis of the initial densities of the host and the competing natural enemies. The particular outcome depends on four main factors: (1) the finite rate of increase of the host, (2) the clumping of parasitoid (and pathogen) attacks, (3) the relative extrinsic potentials of the competitors, and (4) the relative intrinsic potentials of the competitors. We conclude that although situations may arise in which the host is best controlled by only one natural enemy, multiple introductions are likely to be a sound strategy for a wide variety of systems.

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APPENDIX A

The Derivation of the Host-Pathogen Model

This appendix gives some mathematical details of the derivation of the model for one host and one pathogen.

If the nontransmissible subpopulation (Q) is omitted from equations (4), then the various population densities within the infectious season are described by the following set of

differential equations:

$$dN/dt = \beta NW; \tag{A1}$$

$$dY/dt = \beta NW - \alpha Y; \tag{A2}$$

$$dW/dt = \theta Y - \mu W - q\beta NW. \tag{A3}$$

Here the parameters are as defined for equations (4a–c). In order to frame the system as a set of difference equations relating successive generations of the host population (which has discrete, nonoverlapping generations), it is necessary to integrate equations (A1)–(A3) over the time period of the infectious season. A problem immediately arises concerning the identification of the time at which infections no longer occur (and the epidemic ceases). If we choose an arbitrary end point to the epidemic, t_1, then we must numerically integrate equations (A1)–(A3) over a given time period (t_0, t_1). We can, however, maintain analytic simplicity by assuming that the season ends when the epidemic has run its course, or when Y and W tend to zero. These two assumptions preclude the possibility that some of the transmissible stages may survive between epidemics. The result is invariably chaotic dynamics (May 1985).

One way to allow for external stages of the pathogen that can span epidemics is to include, explicitly, a reservoir of protected stages. Equations (4) represent one way of including such a reservoir. Now, if we integrate (4a) over the time period t_0, t_1, we have

$$\int_{t_0}^{t_1} \frac{dN}{N} = -\beta \int_{t_0}^{t_1} W dt. \tag{A4}$$

This gives

$$\ln(N_1/N_0) = -\beta \overline{W}(t_1 - t_0), \tag{A5}$$

where $\overline{W}$ is the average value of $W(t)$ over the infectious season. For convenience, we assume that the infectious season lasts for one time unit, or $t_1 - t_0 = 1$. We now must find $\overline{W}$. This can be done by integrating equations (4b–d) with initial conditions $N(t_0) = N_0$, $Y(t_0) = 0$, $W(t_0) = W_0$, $Q(t_0) = Q_0$ and with final conditions $N(t_1) = N_1$, $Y(t_1) = 0$, $W(t_1) = 0$, $Q(t_1) = Q_1$. After substituting and rearranging terms, we have

$$\overline{W} = \frac{\theta/\alpha - q}{\mu + \Omega}\left(N_0 - N_1 + \frac{W_0}{\theta/\alpha - q}\right). \tag{A6}$$

Substituting $1 - I = N_1/N_0$ into equation (A5), rearranging terms, and then substituting for N_T (from eq. 5e) gives equation (5d). At the end of the infectious season, therefore, the population densities are

$$N_1 = N_0(1 - I), \tag{A7}$$

$$Y_1 = 0, \tag{A8}$$

$$W_1 = 0, \tag{A9}$$

$$Q_1 = Q_0 - (\Omega/\beta)\ln(1 - I). \tag{A10}$$

For the interval between infectious seasons, the following assumptions are made: (1) the host population grows at a finite rate of increase, F; (2) a fraction g of the pathogen stages in the reservoir have been translocated (e.g., by wind or rain) to the transmissible pathogen subpopulation; and (3) a fraction h of the stages remain in the reservoir. Thus, the difference equations relating generation t to generation $t + 1$ are given in equations (6). Although not covered here, the equilibrium point and its stability despite small perturbations are now found by routine methods (May 1975).

APPENDIX B

Interference in the Multispecies System

Priority in displacing a competitor from hosts that harbor both the pathogen and the parasitoid is accounted for by the parameter ϕ. As ϕ approaches zero, the parasitoid displaces the pathogen; when ϕ approaches one, the pathogen has an advantage over the parasitoid. At $\phi = 0.5$, both natural enemies have equal chances of surviving in multiply parasitized hosts and/or overlapping entirely in their times of action during the host life cycle. Multispecies models that do not account for intermediate outcomes in intrinsic dominance implicitly assume either that $\phi = 0$ (i.e., the parasitoid individual always wins in multiply infected hosts) or that $\phi = 1$ (i.e., the pathogen wins). The interference constant, ϕ, is determined a priori and thus is not affected by the details of the model.

During the generation t to $t + 1$, $If(P_t)$ of the hosts will be attacked only by the pathogen, and $\phi I[1 - f(P_t)]$ of the hosts attacked by both natural enemies will be won over by the pathogen. This gives a total fraction

$$I\{f(P_t) + \phi[1 - f(P_t)]\} \tag{B1}$$

of the hosts that produce pathogen progeny. In a like manner, the parasitoid is not contested in $[1 - f(P_t)](1 - I)$ of the hosts and displaces the pathogen in $(1 - \phi)I[1 - f(P_t)]$ of the hosts. Thus, a total fraction

$$(1 - \phi I)[1 - f(P_t)] \tag{B2}$$

of the hosts produce parasitoid progeny.

Terms (B1) and (B2) enter into equations (8) as exploitation terms (i.e., compound functional responses) to the initial host population, N_t. Term (B1) is the fraction of the initial host population that produces pathogen progeny after interference by the parasitoid is deducted (the phrase in oversized parentheses in eq. 8e). Term (B2), the fraction of N_t that produces parasitoid progeny after interference by the pathogen is considered, enters equation (8d) in the phrase enclosed in braces.

APPENDIX C

The Conditions for Invasion by Either Natural Enemy

The parasitoid can invade a system in which the host is regulated by the pathogen if and only if the parasitoid population tends to increase from generation t to $t + 1$ or, equivalently, if the absolute value of the eigenvalue (λ) of equation (8d) is greater than unity when the host and pathogen are at equilibrium (given by eqs. 6). This can be stated as the requirement that

$$|\lambda| = |\partial\{cN_t(1 - \phi I)[1 - f(P_t)]\}/\partial P| > 1. \tag{C1}$$

Explicit evaluation of this criterion for the system defined above leads routinely to inequality (9a), in the text.

The pathogen invades a system in which the host is regulated by the parasitoid if, and only if, the number of infected hosts tends to increase from one generation to the next, from very low densities. This requires that W and Q increase from generation to generation in equations (8b) and (8c) or that the absolute values of the eigenvalues of these equations are both greater than unity. Let $c_W(N_t, W_t, Q_t)$ represent the right-hand side of equation (8b), and $c_Q(N_t, W_t, Q_t)$ the right-hand side of equation (8c); the 2×2 invasion matrix then takes the form

$$\begin{bmatrix} \partial c_W(N_t, W_t, Q_t)/\partial W - \lambda & \partial c_W(N_t, W_t, Q_t)/\partial Q \\ \partial c_Q(N_t, W_t, Q_t)/\partial W & \partial c_Q(N_t, W_t, Q_t)/\partial Q - \lambda \end{bmatrix}. \tag{C2}$$

Matrix (C2) is solved by standard methods (May 1975) for the two eigenvalues. A straightforward calculation then gives inequality (9b) as the condition for the pathogen to invade.

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